The most important result of this scheme is that it is a financial success. A sum of money exceeding £700 is annually placed in this way to the credit of the patients, and great part of this is expended for them, and it is estimated by the asylum authorities—and let me suggest that the officials of Her Majesty's Treasury are not easily satisfied as to the propriety of expending the national funds—it is estimated that the sum thus expended is much more than recouped by the increased value of the labour that the patients are thus induced to perform.

CLINICAL NOTES AND CASES.

Cases of Hereditary Chorea (Huntington's Disease). By W. F. Menzies, M.D., B.Sc.Edin., M.R.C.P., Senior Assistant Medical Officer, Lancashire County Asylum, Rainhill. (*Illustrated.*)

(Concluded from p. 568 of Vol. xxxviii.)

A short account will next be given of the other cases I have had the opportunity of examining.

Case II.—Tickle (D. 16), sister to the previous patient, was admitted into Rainhill Asylum, 12th August, 1887, from the County Asylum, Lancaster, where she had been for four years. History: Ten years ago had a disappointment in love, and has ever since been of a sombre and depressed temperament. Six years ago the tremors commenced, and about the same time her mind became dull and her memory poor, while depression was more marked. The jerkings slowly increased, and fits of violence became common, so that she was dangerous to herself and others. On admission she was suffering from advanced phthisis, and was very thin and weak. The chorea was almost in abeyance. She was unable to stand, but could sit up. Pupils dilated and insensible to light; margins slightly irregular. Muscles of expression paralyzed, has right ptsosis. Thyroid gland enlarged. Patellar jerk increased, no ankle clonus, no superficial reflexes. Dementia is far advanced. She can barely tell her name, does not know her age, or where she is. Takes little notice of what goes on around her, says she feels weak and ill, but has no pain. Articulation most indistinct, lips and tongue tremulous. Heart weak, no bruit, urine normal. Thus the general weakness cloaked the usual signs, but Dr. Harbinson, of Lancaster Asylum, who himself twelve years ago published the first English recorded cases,

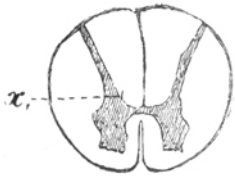

FIG. 1.

FIG. 2.

Fig. 1 is a diagram to show condition of Clarke's column between 11th and 12th dorsal.

Fig. 2 is copied from Obersteiner, and shows normal cord at 12th dorsal; x, Clarke's column.

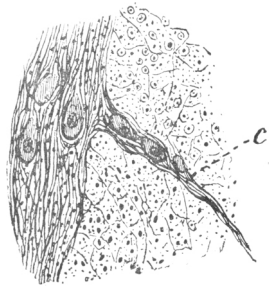

FIG. 3.

Fig. 3 shows Clarke's column as seen in a few sections just above 12th dorsal on the left side. The bulging "x" in Fig. 2 seems here to be represented by a process "c" from the posterior horn, containing a couple of ganglion cells. Though this process is indicated in Fig. 1, it was too small to be detected by the naked eye.

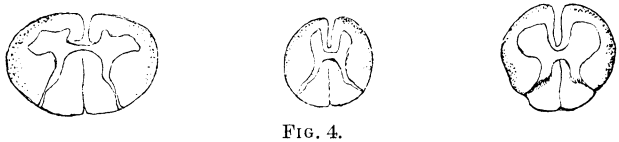

FIG. 4.

Fig. 4 shows areas of degeneration (dotted). It is not meant to indicate the relative amount in different parts accurately.

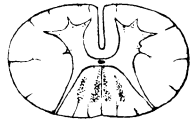

FIG. 5.

Fig. 5.—Cervical enlargement, showing increase of connective tissue in postero-median and postero-external columns.

TO ILLUSTRATE DR. MENZIES' CASE.

told me that while under his care the woman displayed the ordinary signs, but dementia was disproportionately advanced. She died fourteen days after admission. Post-mortem—besides the pulmonary tuberculosis, slight renal cirrhosis, and a small spleen, with fibrous capsule—there was nothing of note in the trunk cavities. Dura mater normal. Brain 1,061 grammes. Considerable excess of subdural fluid. Membranes thickened all over, general opacity of arachnoid. Brain, as a whole, unusually firm, membranes strip everywhere with abnormal ease. Right hemisphere 425 grammes; left 443 grammes, both stripped. Much general wasting, no local atrophy. Grey matter about $\frac{2}{3}$ normal thickness. Striation well marked. Cortex and white matter both rather pale, latter very firm, ventricles dilated, floor smooth. No noticeable change in basal ganglia or cerebellum. Pons and medulla firm. No microscopical examination was made. The brain was evidently overgrown with sclerosis, and the grey matter atrophied.

Case III.—Tickle (F. 5), a boy of 13. The only abnormality is an irregular jerking of the fore-arm and hand when he endeavours to grasp an object, as in eating; the thumb turns downwards and the object is often knocked over. When his attention is called to it he can control it, but it recurs as soon as he forgets about it. He is a decidedly intelligent lad. Knee-jerks normal. Whether this case will develop remains to be seen, but in any case the relationship here observed between the genesis of a habit spasm and an organic disease of the higher motorial regions is of more than passing interest.

Case IV.—Tickle (D. 48), male, an inmate of Prescot Union Infirmary, where, through the kindness of Dr. Hall, the medical officer, I was able to make an examination. He is totally confined to bed, but can sit up. The muscles are flabby, but not wasted. Subcutaneous fat abundant. The tremors marked in the thighs, trunk, upper extremities, and face, especially the two last, but in the legs have mostly given way to rigidity. He is continually throwing his arms about, turning and twisting his hands in and out, and going through every variety of movement. One or other side of the mouth may be drawn up, the head rotated or extended, the eyes blinked, or the corrugator supercilii thrown into strong contraction. The abdominal muscles, intercostals, diaphragm, and larynx all share, so that respiration is momentarily interrupted by a sharp snorting groan. Sensation is normal —no history of pain. He can describe accurately where he is touched, but cannot execute the movement of pointing out the place, the endeavour ending in more violent jerkings than usual. Cannot protrude his tongue at all. Knee-jerk and plantar reflex excessive, ankle and quadriceps clonus strongly marked, wrist and triceps jerk present, cremasteric, abdominal, and scapular reflexes absent. The left lower extremity is somewhat more affected than the right, and the same holds for the face.

Right pupil 3·5 m.m., left 3 m.m.; latter irregular in margin, both react freely. Vision, optic disc, and fundus normal. Hearing normal. No R.D. Heart perfectly normal and regular, action of bowels and digestion unimpaired. Urine normal. The cerebral condition is one of demented contentment. He smiles and nods his head in response to questions to which he cannot articulate an answer. Seems to understand most that is said to him, and knows where he is, but has a very imperfect idea of the flow of time. Attention poor. The emotional element becomes prominent when anyone gives him bad tobacco. He tosses it down, screams and ejaculates incoherently, and endeavours to strike the donor, while the movements become more tumultuous than ever. This is the most advanced case I have seen, and the cortical atrophy, with sclerosis in cord and brain, must be extensive.

Case V.—Dixon (C. 7), female, admitted into Rainhill Asylum, 10th September, 1887, aged 49. She has been married 30 years, and has six children. The jerks have prevailed for ten years. It does not certainly appear whether dulness and loss of memory were concomitant or sequent. For two years she has been more or less maniacal, and at last so violent that her relatives cannot keep her at home any longer. The excitement partakes almost entirely of the spasmodic emotional type. She has twice attempted suicide (drowning and hanging). The weakness and jerkings have progressed steadily. On admission she was too weak to stand alone. The movements were of the usual type. Sensation and muscular sense normal, plantar reflex absent, knee-jerk excessive, no ankle clonus, triceps jerk present. Heart, lungs, and abdominal organs normal, urine normal. She was weak-minded and happy, dull, uninterested in her surroundings. Memory very poor, attention small, judgment none. Probably the advanced dementia was the most prominent feature. During residence she had frequent attacks of emotional depression, and would cry aloud for hours. Opposition to her wishes was a common cause of these outbursts. On 9th February, 1888, she was seized with an apoplectiform attack, in no way differing from those seen in general paralysis; coma, stertor, and general flaccidity were present, all jerkings and reflexes abolished. Temperature reached 103° F. She pulled round in a few days, but remained permanently weaker and thinner, and the movements were less under control. Articulation was now unintelligible. This attack is of great interest, and is the only one I have been able to hear of in any case of the disease. The patient died 3rd March, 1889, of tuberculosis of lungs and intestines. The brain and cord were removed for examination, but there is no record of the result. I remember that there was general increase of neuroglia, as evidenced by unusual firmness. There was also a hæmorrhagic membrane on the cord, which may bear relationship to the apoplectiform attack.

Case VI.—Dixon (C. 14), male, 34, still living at home. He began to be affected at 26, and has for long been unable to work. The tremors are typical, but not very conspicuous. General muscular weakness is more prominent, and the gait is slow and unsteady. Sensation and muscular sense normal. He is a well-nourished man with no muscular atrophy. Plantar reflex absent, knee-jerk somewhat brisk, scarcely abnormally so. Tongue protruded in a jerky manner. Speech slow and dragging. He experiences a difficulty in starting his sentences, but these, when started, are fairly coherent. Pupils equal and normal in reaction, media clear, myopic shadow marked, disc and fundus normal, Vision $\frac{3}{3}$. Fields of vision normal. No R.D. Mentally he is fairly clear, but shows the same loss of apperception and attention as the other cases. He is contented and careless. His wife says his temper is most uncertain, but he has so far not been violent. He lately went to the Royal Infirmary, Liverpool, but after a few days grew restless and discontented, and refused to stay longer.

Case VII.—Dixon (C. 15), male, 32, younger brother to the last. This is an incipient case. At present his intelligence is good, and when I called to see him I found him engaged in solving quadratic equations. Contrasted with this excellent grasp of present events was his uncertain memory for the past. He was quite dubious as to how long he had been married, the age of his children, and the order in which they were born, and his wife remembered far more about his own family than he did himself. He presented a strange picture, aware that the fatal disease was commencing, yet most anxious to conceal it. He sat bolt upright, and did not attempt to rise when I entered, his knees were kept pressed together, hands clasped, and eyes fixed rigidly on the opposite wall, thus making every preparation to control and hide the slight jerks which were at intervals apparent in fingers, forearms, and legs. His tongue is steady, pupils normal, knee-jerks not exaggerated. During the conversation he frequently lapsed into dreamy inattention, from which he woke with a start when his wife spoke to him. I did not care to make a complete examination, dreading an outburst of temper, but shall endeavour to keep the case in sight.

Remarks.—Ætiology.—Huntington's chorea is probably one of the most hereditary of all diseases. In one of my families 25 per cent. of traced individuals were affected, and 50 per cent. of those over 12 years old; in the other, where the type was one of later appearance, nearly one-third of the adults suffered. Other observers have noted a higher percentage still. The age at which it appears, generally speaking, precesses generation by generation, but the exceptions are numerous,

and the rule far from strict. Those ascertained are here tabulated :—

Tickle.			Dixon.		
Case.	Age at onset.	Age at death.	Case.	Age at onset.	Age at death.
B. 1	—	70	B. 2	46	—
C. 2	40	44	B. 3	—	60
C. 4	30	—	B. 5	—	62
C. 5	24	38	C. 1	30	50
C. 6	—	43	C. 2	40	—
C. 7	—	36	C. 6	45	53
C. 9	16	—	C. 7	39	51
C. 11	—	53	C. 9	40	48
C. 12	30	50	C. 14	26	Alive (34)
D. 15	18	Alive (22)	C. 15	32	Alive (32)
D. 16	34	40			
D. 17	28	32			
D. 23	29	35			
D. 48	36	Alive (43)			
E. 8	26	40			
E. 9	31	Alive (33)			
E. 10	25	Alive (26)			
E. 11	20	Alive (23)			
Average age	27·6	43·7	Average age	37·2	54

For both Families.

Average age at onset ... ... 31·1 years.
" " death ... ... 47·3 "
" duration of case... ... ... 16·2 "

Sex.—The two families consist of 67 males and 71 females, as well as 36 of sex unascertained. There were affected 26 males and 16 females. This superiority in the males is not due to a preponderance of male patients, for those who had children were 18 males and 23 females, and of the affected members 14 males and seven females were parents, so that at

least twice as many men are affected as women. There is no tendency to alternation of the sexes in successive generations, either parent being liable to bequeath the disease to son or daughter.

Other points in the ætiology are the absence of any diathesis, especially a freedom from rheumatism.

Pathology.—The clinical signs of this disease are so identical with those of ordinary rheumatic chorea that we are driven to conclude that, whatever may be the nature of the lesion, the position in the nervous system is the same. Where may we reasonably seek for it? Hitherto the results of post-mortems have been most unsatisfactory, for early cases rarely die, and advanced ones show gross tissue changes quite sufficient to cloak the slight alterations presumably responsible for the symptoms. A minute microscopical examination of both hardened and fresh sections in an early case is still a desideratum.

We can at once exclude the cord, for the knee-jerk is never lost, and in early cases, sensation being normal, is not increased. Nuclear lesions of the medulla are always wanting, the elements of speech are always perfect, letters are never misplaced, syllables never omitted. The ataxia might suggest the cerebellum; but in one case of an acute lesion, which was for some days limited to one lobe, none of the cerebral signs here seen were noticed. A thalamic lesion has given rise to choreoid movements, but the association of mental defect with these limit our choice to the cortex.

Possibly the large motor cells of the third layer are defectively inhibited, either by disease of the higher cells or by interruption of connecting fibres. The latter is more probable, for the cerebral phenomena of an early case suggest no organic defect, but only a want of proper control, as evidenced by the rise of the emotional element. The simplest explanation thus assumes disease of the terminal fibres of the "cerebral segment," just as primary spastic paraplegia is the result of a similar lesion of the upper cerebro-spinal segment. Functional over-action may lead to the descending cord changes diagnosed in advanced cases, as happened to the hysterical girl mentioned by Charcot. Such a pathology reconciles the few facts of morbid anatomy hitherto collected, the degeneration of various cord tracts, with hypertrophy of the motor cells in the anterior horn, found by Cirincione and Mirto; the atrophy and sclerosis of the internal capsule with destruc-

tion of the cornual cells in the cervical cord, described by Harbinson; and the general sclerosis noted in my own cases and those of others. One condition always found is an overgrowth of the connective tissue element, giving abnormal firmness. Now it is possible that this growth of neuroglia may, by pressing upon the efferent, afferent, or commissural fibres, so affect either the sensations which regulate movement, the currents to the muscles themselves, or especially the direction, inhibition, and co-ordination of the large cells in the motor regions by those in the higher parts of the brain, wherever these may be situated, as to cause a partial dislocation of the muscle functions either by interruption or irritation. The implication of motion without sensation is just what we should expect in most cases, but in the more advanced ones some disorder might reasonably be looked for. It should be remembered that the special senses are not involved at all, and that for years there is no loss of the lower ideational centres, memory, judgment, reason; but only of the highest inhibitory functions. So with our present knowledge of brain function guesses at localization are futile.

The theory that certain embryonal connective tissue elements remain latent till late in life is one more easily formulated than either proved or disproved. The sclerosis is too slight and too wide-spread to render it probable, and the theory is less likely to hold water in hereditary chorea than in cancer, where it has been nearly proved to be false.

Evidence of a slowly acting micro-organism in the environments at home or at work is wanting, for although it be true that many of the unaffected leave the district while the sufferers remain, and although if the affection once cease it rarely reappears in a subsequent generation, yet there is no case in which it has developed in the wife of an affected husband, or *vice versâ*, even after the lapse of fifty years.*

* While this article was passing through the press, the woman Jane Tickle died of tubercular peritonitis. A post mortem examination was made by Dr. Wynne, Pathologist to Rainhill Asylum, and the notes are appended at the end of the paper. It will be seen that general sclerosis of the brain was present, but not to such a marked degree as in other cases. The atrophy (?) of the occipital lobes was most marked, but seems to have produced no symptoms. The myopia and nebulæ accounted for the defect in vision, and the fields and colour perception were normal. Probably the sclerosis of the antero-lateral ascending tracts deserves special attention, but in other respects the result of a minute microscopical search (over 100 sections) must be regarded as disappointing. The other cases I saw post mortem had no occipital atrophy. With regard to

Summary.—One of the first points which strike anyone reviewing the comparative literature is that different families appear to affect different clinical types. All writers agree that the disorder is essentially a chorea, that the jerkings are at first partly under control, that the knee-jerks are generally increased, and that cerebral defects are common. Yet all are not agreed whether or not the movements cease during sleep. According to Waters, signs of the ailment appear only after middle life, while Diller mentions a generation where ten cases all developed before twenty-five years of age. Huntington considered it more common in men, Sinkler in women. Caviglia thought it equally common in both. Therefore, with Dr. Reynolds, I would deprecate dogmatism until some more extensive series of cases have been collected, extending through more generations. My own cases explain that some observed differences arise from studying the disease at different stages of development, *e.g.*, the persistence of spasms during sleep. The essential points of the disease may be summarized thus: At first there are only the jerkings and the associated emotional state, leading to attacks of the so-called mania or melancholia, then descending changes cause increase of knee-jerks and general muscular weakness, while the cerebral sclerosis results in true dementia. Lastly the patient dies, not so much of the disorder itself as from some intercurrent affection, to which his paralyzed condition lays him open. The most common of these is tuberculosis, probably because he drifts into a workhouse hospital or asylum infirmary, where unfortunately, the bacillus is but too frequent.

To give in detail the results of other observers would swell this article much beyond its proper limits. These results can be obtained by reference to the original treatises; a relation of observed facts is of greater moment with our

Dr. Wynne's account of the post mortem changes, there was, in addition, a pronounced increase of connective tissue (almost a sclerosis) of the anterior ⅔ of the postero-median columns in the cervical enlargement, and to a less degree of the postero-external. The central canal was partially obliterated by connective tissue overgrowth in the cervical region, wholly in the dorsal and lumbar. That the processes of Clarke's column were less conspicuous than normal is not beyond the range of dispute. Other observers have met with sclerosis of the antero-lateral ascending tracts. Is it possible that interruption of the muscle sensations ascending to the cerebellum, causes erroneous reflex judgment, and so sets the muscles into clonic spasm, this spasm then causing a second upward sensation, which now reaches the cortical areæ of consciousness, and makes the patient aware of the contraction?

present knowledge of the disease than protracted discussion of theories more or less inadequate to account for the symptoms.

I close with a request to asylum medical officers to collect other cases. There cannot be many which do not, at some period or other, come under the notice of relieving officers of the various unions, through whose agency every affected family in England could without much expenditure of labour be traced, and thereby many questions regarding a morbid entity not wholly devoid of interest could be finally disposed of.

Summary of Post-Mortem Notes of Jane Tickle (Hereditary Chorea).

Post-Mortem made seventeen hours after death. Age 33. Body much wasted. No bed sores. No lividity.

Cranium.—The skull presented no abnormality either in shape or density. The sinuses were fairly full of partly clotted blood. There were no old thrombi. The main vessels were quite healthy.

Dura Mater.—A little above average thickness, but not adherent either to skull or pia-arachnoid.

Subdural Space contained a little excess of clear fluid.

Pia-arachnoid was absolutely free from any opacity or thickening and was nowhere adherent. There was a slight excess of fluid, chiefly in the sulci. There was no hyperæmia.

Cerebral Hemispheres.—The whole brain weighed 1,132 grms. The right hemisphere weighed 459 and the left 457 grms.

There was distinct, though slight atrophy of all parts of the cerebrum, as evidenced by the rounding off of the convolutions and widening of the sulci. In the temporo-sphenoidal lobes the atrophy was scarcely noticeable.

The occipital lobes presented a symmetrical diminution in size, which from the condition of the convolutions and sulci would seem to be at least in part developmental and not due to atrophy. The cuneus on the left side was very distinctly atrophied.

On the whole the atrophy was greater on the left than the right side of the brain, and more distinct in the paracentral region than in the frontal.

Cortex Cerebri.—The tint of the grey matter was quite normal. Its consistence was in all parts rather firmer than in health. In the temporo-sphenoidal lobes, it was, as is usual, less firm than elsewhere. There was slight narrowing in all, except the occipital lobes. The striation was rather more distinct than usual. Vascularity normal.

Lateral Ventricles were not dilated. The ependyma was slightly rough, but not distinctly granular.

Basal Ganglia presented nothing abnormal to the naked eye.

Cerebellum 139 grms. No abnormality.

Pons 15 grms. Rather softer than other parts of the brain. The grey matter seemed less pigmented than usual.

Medulla Oblongata weight 6 grms. Rather small. The grey matter of a paler tint than usual.

Spinal Cord.—Nothing beyond an unusual firmness detected in the fresh state.

THORAX.—The lungs were tubercular. Numerous small vomicæ in upper lobe of left, and small patches of consolidation scattered through right.

Heart.—A little hypertrophy of left ventricle.

ABDOMEN.—*Spleen.*—Capsule thickened. Increase of connective tissue.

Liver.—Fatty.

Kidneys.—Capsule thick, very adherent. Fibrotic kidney.

Intestines.—Matted together, and the whole peritoneum covered by tubercular deposits and lymph. The tubercles varied in size from the ordinary "miliary" to masses the size of marbles.

MICROSCOPIC EXAMINATION.—In considering the changes found in the brain it must be borne in mind that the patient was a fairly advanced phthisical subject, and for some time before death had tubercular peritonitis with diarrhœa.

Fresh sections from various parts of the cerebrum were examined, and all showed the same changes, differing only in degree according to the amount of atrophy present in the different regions. The changes noted were :—

1. A slight coarseness of the neuroglia in the first layer.
2. A slight thickening of the vessels.
3. Degeneration of the cells in all layers. The degenerate cells were characterized by loss of processes, a granular condition of the protoplasm, and very often vacuolation of the nucleus or cell-body. This vacuolation did not specially affect any particular layer of cells, but was more abundant in the deeper than the more superficial, or at least was more readily detected.
4. In hardened specimens spider cells were detected in very small numbers near the vessels in the white matter, but neither in fresh nor hardened specimens was there any appearance of miliary sclerosis.

Sections of the medulla showed a thickening of the ependyma, and an undue coarseness of the connective tissue beneath. Sections of the cornu ammonis and gyrus hippocampi showed vacuolation of the large pyramidal cells, and the presence of "colloid bodies" in the endothelial lining of the fimbria.

Spinal Cord.—In hardening, a one per cent. solution of bichromate of potash was used for twenty-four hours, followed by a two

per cent. solution frequently changed until the cord was ready for cutting. No alcohol was used until after the sections had been stained.

In staining, Weigert's, Pal's, and Marchi's methods were used, and some sections were stained with picrocarmine or with Ehrlich's hæmatoxylin alone. The results obtained by all the methods were the same.

White Matter.—In all parts of the cord Gower's tracts and the direct cerebellar tracts showed scattered groups of degenerated fibres. In the lumbar region a zone bounding the periphery of the cord from the anterior commissure to the exit of the posterior roots showed an almost entire absence of healthy fibres.

Grey Matter.—The cells were everywhere unusually pigmented and granular. Picrocarmine sections treated with osmic acid and those prepared by Marchi's method showed intensely black clumps of granules in nearly all the cells. This was most conspicuous in the cervical regions.

Clarke's column appears distinctly abnormal, especially in the lower dorsal region, where it should be most conspicuous. Sections from between the eleventh and twelfth dorsal nerves did not exhibit the characteristic swelling of the posterior horn produced by this group of cells. In some sections no cells at all could be seen; in others one or two highly pigmented oval cells with no processes. Sections at the junction of the dorsal and lumbar cord showed the cells of the column being displaced by growth of connective tissues, and their proper area encroached upon. Just below this the column was represented by an isolated cell, or not at all.

Throughout the upper and middle dorsal cord the column was ill-developed, and very often no cells were found. When cells were present they were always more numerous on the left side; that is, if there were two or three on the left side there would be one or none on the right.

Vessels.—Here and there distinctly thickened vessels were present in the anterior horns; elsewhere they were dilated and full of blood.

Medulla Oblongata.—Nothing abnormal was detected in sections prepared like the spinal cord. I was unable to make certain of any degenerated fibres.

From these observations it would seem that the degeneration of the cerebellar tracts and Clarke's columns was the characteristic lesion in this case.

There was undoubtedly a widespread though slight sclerosis, as evidenced by the unusual firmness of the brain and spinal cord. Fibrotic changes were also noted in the kidney and spleen. As regards the brain, the evidence of disease was abundant, but the changes met with were of a very common character, and presented as far as I could see nothing characteristic. The absence of

thickening, opacity, and adhesion of the soft membranes excludes, I think, any inflammatory origin of the cortical degenerations. What effect the bodily condition of the patient may have had on the cortical grey matter cannot be estimated, but I think a good deal of the cell degeneration may be set down to that cause.

I do not see any sufficient ground for assuming a cortical lesion for this disease. May it not more probably be a slight sclerosis sufficient to hamper but not abolish the control of movements? Such an obstruction might be situated in the cord.

At any rate, in future cases it would be well to search for the lesions here shown in the spinal cord. If these lesions are found to be constant it will be time enough to construct a theory of the pathology of hereditary chorea.

In view of the recent experimental work on the spinal cord, it is interesting to note that the patient in this case, though carefully examined, exhibited no alteration of sensation.—E. T. WYNNE, M.B.

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Case of Abnormal Development of the Scalp. By T. W. McDowall, M.D., County Asylum, Morpeth. (*With Plate.*)

The accompanying illustration represents what is believed to be a hitherto undescribed abnormality of the scalp. The condition was discovered accidentally. I observed an attendant cutting a lad's hair, and remarked that he was not doing it very well, as there appeared to be numerous scissor-marks. It was explained that the marks were due to the arrangement of the hair. This led to careful examination of the whole scalp. Its condition is very well represented in the illustration. On each side there are five deep furrows, passing from behind forwards. Those nearest the middle line are straight; the others slightly curved, and they are the more curved the further they are removed from the middle line, and at the same time they diminish in length. When the hair is of ordinary length the condition of the scalp would not be suspected; it is only when the hair is very short that the furrows become evident. For the purpose of preparing the accompanying illustration the hair was cut as short as possible, but the hair growing in the furrows was necessarily left somewhat longer than elsewhere, with the result of indicating with great clearness the course and arrangement of the depressions. The furrowing of the forehead is not abnormal, but is produced by the patient whenever he is annoyed, as he was when the photograph was taken.

The patient is an epileptic idiot, aged 22 years, and has been resident in the asylum nine years. He exhibits only slight traces of intelligence. He is above the average stature, and in good bodily health. The whole right side of the body is wasted, and the limbs contracted. He is stated to be the eldest of six children, and to have been epileptic since he was one year old. He is distinctly but not markedly microcephalic. Since this congenital deformity of the scalp was discovered I have examined the heads of all the male patients, with the result of discovering another example, but not so marked.