

Remarks on a case of syphilitic insanity / by J. Batty Tuke.

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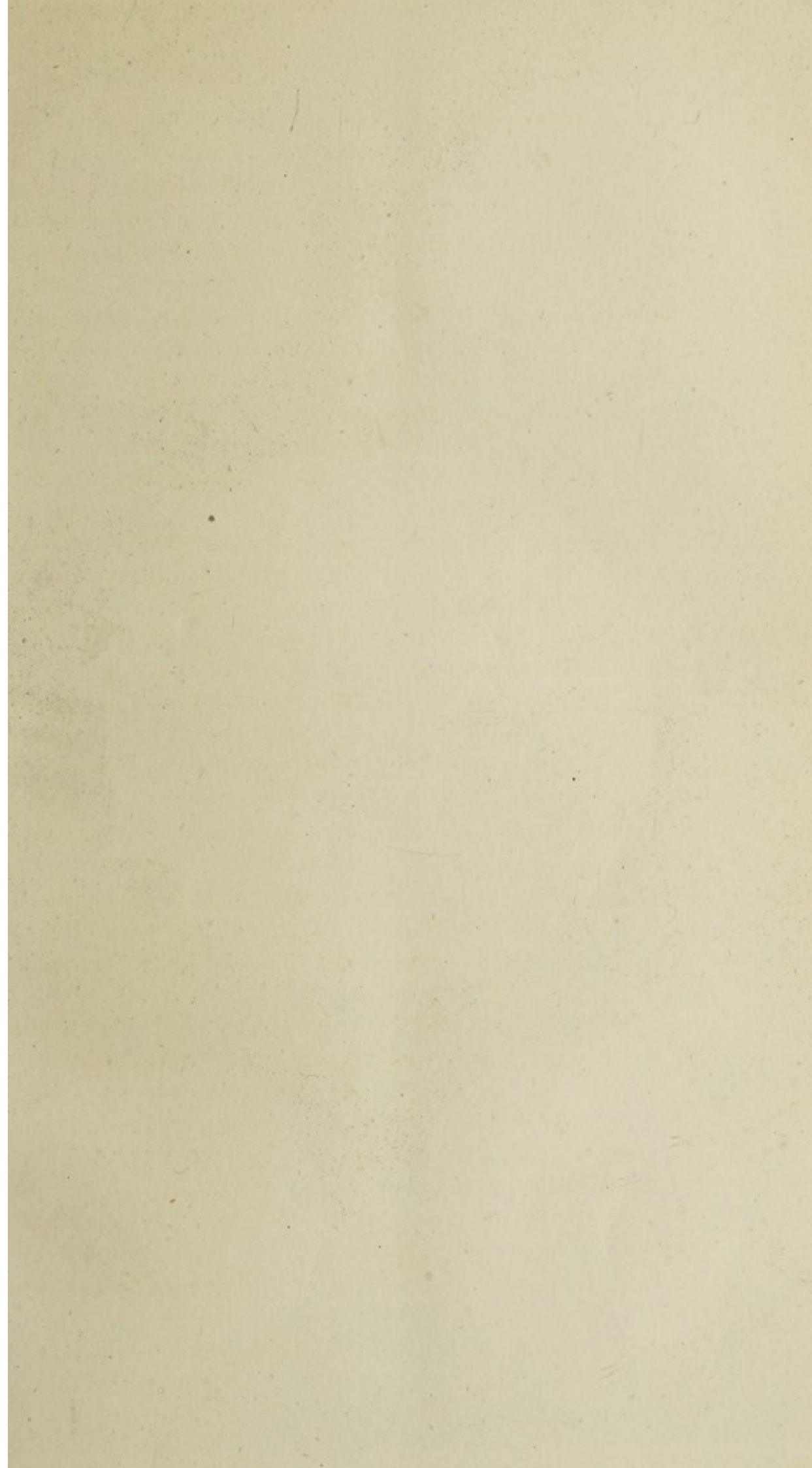
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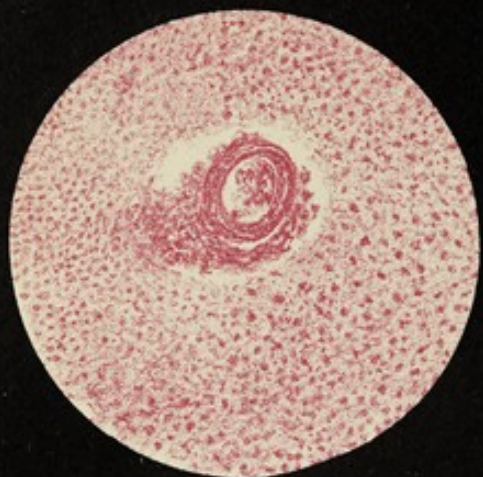
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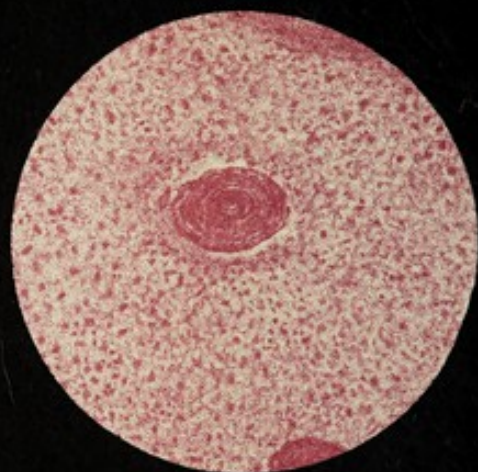


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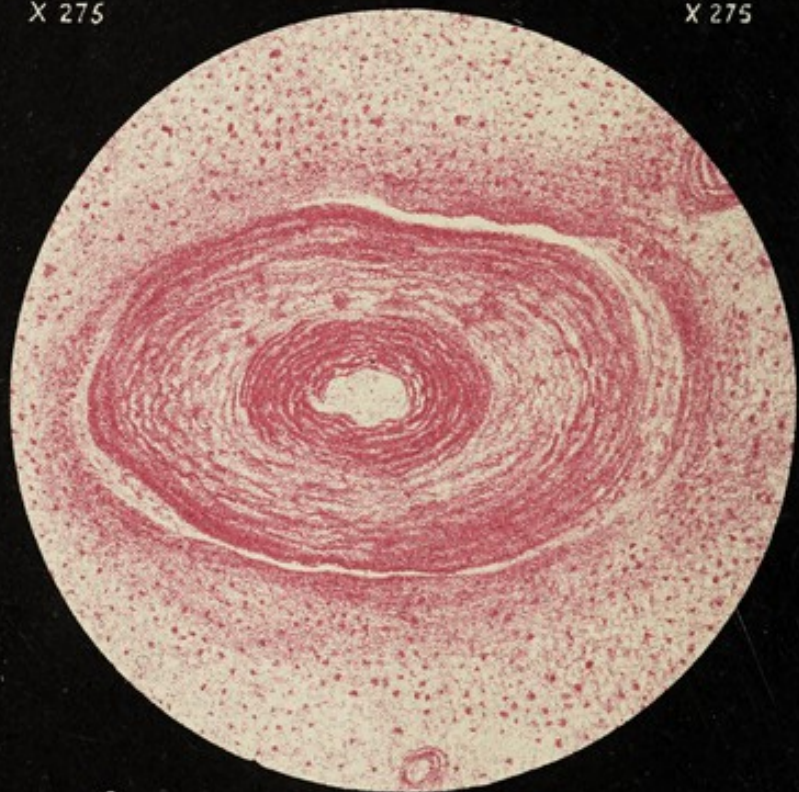


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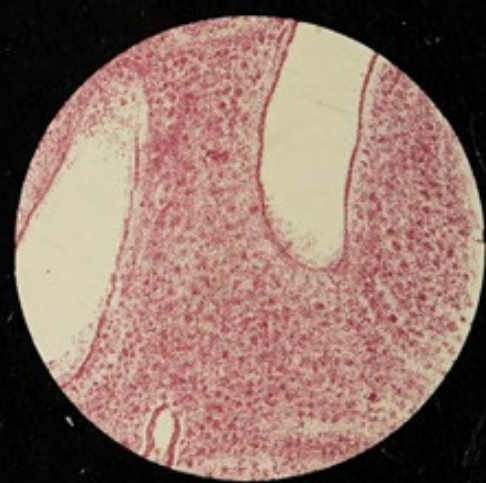
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REMARKS ON
A CASE OF SYPHILITIC INSANITY.

BY


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REMARKS ON A CASE OF SYPHILITIC INSANITY.

The details of the case I now propose to speak of have been published in the January and July numbers of "The Journal of Mental Science" of this year. In the January number the clinical history was reported as far as it went; shortly after its publication the patient died, and in the July issue the further progress and termination of the case were given, and the result of the post-mortem examinations detailed. I will not now detain you by reading the full report, but in order to carry you with me it will be necessary to recapitulate the main features of the case, supplementing them by a few particulars which have been evolved by further study of the pathological appearances presented in microscopic sections.

B. A. belonged to a very healthy family in which there was not the slightest history of predisposition to nervous instability in any form. His age, when he first came under observation, was 52. He had been an acute man of business, and although a somewhat free liver, by no means a drunkard. No undue anxieties had pressed upon him; in a word, there were no so-called moral causes to account for the incidence of insanity. Six years previous to his coming under my care (*i.e.*, at the age of 46), he contracted an infecting chancre, which was followed by the usual secondary symptoms, during the persistence of which he suffered at intervals from melancholy, and from what the Germans call "paralysis of energy." About eight months after partial recovery from this condition, he fell down in the street unconscious. The nature of this fit I have not been able to determine definitely, but the strong probability is that it was epileptic in character, for well marked epilepsy supervened in a short time, followed by maniacal excitement. An immediate sequence of the first fit was that he became amnesically and heterophasically aphasic. It is impossible to fix with absolute accuracy the period of incidence of the next important feature of the case, *viz*, progressive muscular atrophy:—all I

can say is that when he came under my observation early in 1873, this condition was very well marked on the right side of the body, and on the *right side of the body only*, accompanied by considerable loss of power. The muscles of the right hand were much wasted, the thenar and hypothenar eminences were obliterated, the interosseous muscles were reduced in bulk, as were those of the right thigh, and in a less degree the muscles of the right fore-arm and the right leg. In walking he "hoisted" the right leg. Common sensibility appeared to be unaffected, but difficulties were experienced, in consequence of the aphasia, in determining this point definitely. The hearing of the right ear was defective. The aphasia was amnesic and heterophasic in character. Articulation was defective. Memory on all points was confused; talking soon excited him; he was restless and irritable; when in good temper he displayed a good deal of *bien etre*. Both pupils were persistently much contracted. I much regret that no ophthalmoscopic observations were made. Whilst under my care B. A. had several epileptic fits, at long and irregular intervals. In one, which was most carefully observed by my friend and late assistant, Dr. Joseph Brown, the epilepsy was almost unilateral: after a shrill scream, convulsion commenced in the right side of the face, it next extended to the right arm, and right leg; at its height the left side of the body was but slightly affected.* I saw the patient shortly after the convulsion had ceased, and observed most definitely that the right side of the body was pale and blanched, the left being of a normally florid colour. (The case was reported up to this point in the January number of the Journal, and the diagnosis expressed that it was one of Syphilitic Insanity).

In February of this year my patient died of apoplexy. The full details of this attack are given in the July number of our Journal, and present many interesting points for the consideration of the pathologist and physiologist. But as my desire to-day is to restrict ourselves to the collation of the epilepsy, the progressive muscular atrophy, the aphasia, the insanity, with the pathological evidences of syphilitic changes found in the brain and spinal cord, I will not dwell longer on the conclusion of the case; suffice it to say that the symptoms were such as to suggest the diagnosis of an apoplectic clot in

* The patient's medical attendant, under whose care he was prior to his being committed to mine, informs me that all the epileptic fits which he observed were unilateral in character.

the neighbourhood of, although not involving, the right corpus striatum.

The autopsy was performed twenty-four hours after death (weather cold). The following were the more important naked eye appearances:—Dura mater adherent and arachnoid slightly opalescent; small local atrophies in the neighbourhood of the intra-parietal fissure. Apoplectic clot in the centre of the occipital lobe of the right hemisphere about the size of a walnut; below its level, and on a level with the corpus striatum of the same side, an apoplectic clot was found, measuring from five to six inches in length, and about one and a quarter inches in breadth at its widest part, extending from about an inch from the tip of the frontal lobe to about the same distance from that of the occipital lobe, bounded on the left by the motor tract, which was not implicated. The two clots were unconnected. In the left hemisphere a yellow softening, irregularly round in shape, and about the size of a large walnut, was found impinging on the corpus striatum, involving the extra-ventricular nucleus and claustrum, and extending to within a few lines of the grey matter of the convolutions. The external arteries were much thickened. On the basilar artery large deposits of a yellowish colour existed. The middle cerebral artery in the fissure of Sylvius on both sides were seen to be nodulated and rendered moniliform by this deposit.

Microscopic examination of recent specimens showed thickening of the vascular coats, on which irregular swellings were seen, consisting of molecular matter. In the grey matter cells undergoing fuscous degeneration were observed in large numbers. The adductor pollicis was examined, and no indications of disease were noticed, the striæ being markedly distinct. Various portions of the brain and spinal cord were prepared in chromic acid; sections were made from these specimens, certain of which now stand on the table. In the frontal lobes the muscular coats of the arteries were considerably thickened, and the capillary walls well defined. In the ascending parietal convolutions at the vertex transverse sections of the arteries showed that the muscular and outer fibrous coats were much thickened; surrounding the latter coat were concentric rings of a material in which were held corpora amylacea; in some instances empty spaces existed between this material and the brain substance, in others this interspace was filled with a colloid looking substance. In many of the smaller arteries perfect occlusion

had taken place. Fuscous degeneration of the cells of the fourth and sixth layers were observed in degree between simple and slight deposit and complete destruction. In the occipital lobes and cerebellum the same lesions were noted in a slighter degree. In the corpora striata and in the cerebral convolutions most contiguous to them the diseased condition of the vessels was more thoroughly marked than in any other region. In some instances the muscular coat was found at least four times thicker than normal, and the concentric rings of new material extended from the $\frac{1}{80}$ th to $\frac{1}{150}$ th of an inch around the larger arteries. The smaller vessels were very generally completely occluded. Immense deposits of hæmatoidin were found immediately below the ependyma ventriculorum and in the vascular canals. In the pons Varolii the vascular canals were in a state of extreme dilatation, resembling in a minor degree a preparation of Dr. Lockhart Clarke's, described by him in the *Medico-Chirurgical Transactions*, Vol. lvi., p. 106. In a section of the medulla oblongata immediately above the decussation of the pyramids, the cells in the course of the deep origin of the spinal accessory nerve were found reduced to fuscous masses. In the spinal cord the cells of the anterior vesicular column and those of the tractus intermedio-lateralis were seen bloated and swollen, their angles were obliterated, and their nuclei and nucleoli indistinct; they absorbed carmine but slightly; a few of those of the posterior column had undergone fuscous degeneration. In the posterior column large deposits of corpora amylacea existed, and the arteries on either side of the central canal were thickened. *In both cord and medulla the lesions were symmetrical.* The term bloated, as applied to the cells' appearance, is only applicable to their contour, for their greatest diameter was only the $\frac{1}{1000}$ th of an inch. In the posterior column large numbers of corpora amylacea $\frac{1}{2000}$ th of an inch in diameter existed. No evidence of syphilitic deposit was found in any other organ but the brain.

In this case we have presented a group of morbid nervous conditions, not only remarkable collectively, but which, when considered individually, are peculiar and anomalous: we have a man during the course of the secondary symptoms of syphilis undergoing intermittent changes in his psychical condition, and during the tertiary stages becoming the subject of epilepsy, amnesic aphasia, progressive muscular atrophy, impaired articulation, myosis, confirmed insanity with mental

symptoms not unlike those of general paresis, and, finally, apoplexy. This is the group of conditions, but these conditions are individually peculiar—thus we have unilateral epilepsy and unilateral progressive muscular atrophy, both in the same side of the body, and aphasia unconnected with any true paralysis. It is extremely difficult to know where and how to attack this case, but it appears to me that the best plan will be to consider the various indications of nervous degeneration in the order of their incidence, connecting them as far as possible with the morbid changes found after death, depending as little on hypothesis as possible, founding mainly on proved facts.

As has been already said, remittent melancholy and paralysis of energy were the first evidences of nervous degeneration; as they occurred during the secondary stage of syphilis it is probable that they were to some extent dependent on the anæmia, which is so generally present in that specific condition; but it is also probable that they were the result of commencing homologous changes in the tissues of the blood-vessels of the superior parts of the hemispherical ganglia. Microscopic dissection proved that the muscular and outer fibrous coats of the arteries of the various lobes were much hypertrophied, and that their calibre was thereby modified, a condition implying mechanical anæmia and loss of regulating power superadded to any actual modification in the character of the blood itself. That the psychical symptoms were remittent may be explained by improvement in the character of the blood due to medication and hygienic influences, and that the course of the cure showed a tendency to progressive deterioration of the nervous centres, by concomitant advance of the morbidity of the blood-vessels. We are taught by the highest authorities on syphilography that whereas homologous changes peculiarly characterise the secondary stage of syphilis, heterogenous morbidities are produced in the tertiary stage. Tracing this case through its clinical history we find at the period when the tertiary stage was approaching, or was approached, that an attack of an epileptic character occurred, followed closely by amnesic aphasia, deficient articulation, and unilateral progressive muscular atrophy. Microscopic dissection showed that the seat of the most thoroughly marked morbid changes was in the neighbourhood of the left corpus striatum, producing a softening, implicating its extra-ventricular nucleus and claustrum, and I hold it is a fair hypothesis that this locality was the one

which had been affected for the longest period, and, therefore, the spot to which we have a right to refer as the locus of lesion productive of the first epileptic attack. The specimens on the table show that heterogenous disease of the vessels is best marked in the immediate neighbourhood of diseased tracts, that the arteries are surrounded by extensive deposits, which, in the larger vessels, have modified the calibre, and, in the smaller ones, have produced complete occlusion. The fits occurred at long and irregular intervals, and their incidence may be ascribed to a gradual advance of the occluding disease, implicating by mechanical anæmia larger and larger portions of the motor tract. In support of this theory it may be fitting to remind you that the convulsions were unilateral. I found but little on the implication of the extra-ventricular nucleus and claustrum of the left corpus striatum in the production of amnesic aphasia; had the indication been ataxic aphasia, more might have been grounded on lesion of this part. I only regard the amnesia as indicative of a general degeneration of the grey matter of the convolutions, or, at the most, of some portion of it which was not definitely mapped out in this case.

But I would ask you to consider most carefully that, as far as the pathological investigations were prosecuted (and with all deference I may say they were not of a perfunctory character) the demonstration of lesions was shown to be bilateral, with the one exception of the softening of the left corpus striatum, its extra-ventricular nucleus and claustrum, and to collate with this the clinical fact that the two most prominent conditions, viz., epilepsy and progressive muscular atrophy, were unilateral. In the pons the cells and vessels were symmetrically affected. In the medulla the indications of disease were as distinct on one side as the other, and to the spinal cord the same observations extend.

As far as the unilateral convulsions are concerned, these pathological demonstrations do not present any points antagonistic to theories which have been advanced as to the causation of epilepsy; but the same cannot be said in regard to the progressive muscular atrophy. It may be in the power of some member to point to a fully recorded case of this condition in which the wasting was one-sided; such a one has not fallen under my observation, and although such a condition is cursorily alluded to by Hammond, it appears to be extremely rare. I need hardly remind you that a characteristic of this disease is, that atrophy of a set of muscles on one

side of the body is rapidly followed by wasting of the corresponding group on the other, and the inference drawn from this observation is that the affection of the trophic centres on one side of the cord extends after a short interval to those of the opposite side. But, in the case before us, we have a symmetrical condition of disease, as far as the cord is concerned, and an asymmetrical affection of the muscles. It may be objected that this was not a case of progressive muscular atrophy, inasmuch as that no lesion of the fibre could be detected on dissection. I would only refer any such objector to the clinical report published months before the patient's death, and challenge criticism of the diagnosis. I am not prepared to advance any theory as to the actual seat of trophic lesion, contenting myself by recording the fact that there was an old standing softening in the left motor tract, with atrophy of the muscles of the right side of the body, and that in the medulla and cord, although morbid appearances were well marked, that they were bilateral in every respect. In a case of atrophic hemiplegia, which I recorded in the "Journal of Anatomy and Physiology" (vol. vii.), atrophy of the left side of the body was shown to be co-existent with hypertrophy of the right cerebral hemisphere. It may be fair, from consideration of these two cases, to suggest that the trophic influence appears to be exercised by a nervous organ within the encephalon. It need not be necessarily inferred that this organ is in the neighbourhood of the motor tract, from the fact that in the present case a large lesion existed in that part of the brain, for, as is well known, circumscribed injuries affect the functions of tracts remote from them through the various systems of fibres which connect so intimately the various parts of the encephalon.

I will only refer to another of the difficulties presented by this case. It has been said that the cells in the course of the deep origin of the spinal accessory nerve were reduced to fuscous masses. This is not the first time that I have observed this lesion; quite lately I have found it in one case strongly resembling multiple cerebro-spinal sclerosis, and in another of old standing epilepsy; in none, however, has it been possible to connect it with any symptom observed during life.

This case, although it presents many other points of interest, might be dismissed at this point were it not that such a high authority as Dr. Samuel Wilks has lately, in the

“Journal of Mental Science” (April, 1874, p. 44), recorded his opinion that “if we refer to clinical experience and facts, the only cases of insanity connected with syphilis yet recorded are those where epileptiform symptoms, as well as temporary paralytic symptoms, proving the existence of an ordinary gummatous deposit, have, at the same time, been present. If this be true, we are not justified in admitting the existence of a disease which can, in correct pathological or clinical language, be styled syphilitic insanity; that is, there are not, as the term seems to convey to many persons, any morbid changes in the cerebral hemispheres attributable to syphilis. These cases also seem to show that the mania is quite independent of the epileptic attacks, and is not merely a phenomenon or precursor of them, unless indeed the mania might replace the epilepsy.” I am free to confess that I have not, after careful and repeated perusal of the paper from which this quotation is taken, been able to follow the course of reasoning which permeates it. The arguments of Dr. Wilks appear to be founded more on our existing ignorance of cerebral pathology, than on what we have a reason to hope will be elucidated by work and study. It may be of doubtful propriety to term a case one of syphilitic insanity which is, or appears to be, dependent on the presence of an ordinary gummatous tumour; on the other hand, the presence of such a tumour points to the probable existence of more diffused morbid conditions, especially to lesions of the blood vessels. It is even more open to criticism to apply the term to those cases in which melancholy and paralysis of energy may be dependent on the anæmia of the secondary stage; but even this more assailable position may be defended by the assumption, founded on analogy, that the cerebral vessels are undergoing the homologous changes peculiar to the condition. Knowing, as we do, that such morbid conditions do accompany the secondary stage in many organs of the body, the *onus probandi* that they do not take place in the brain almost falls on the objector. But when we meet with a case such as that before us, I fail to see cause why it should not be absolutely styled one of Syphilitic Insanity. In it there was no definite and circumscribed tumour, the gummatous deposits being confined to the vascular walls, not only occluding the vessels and rendering them unfit for the conduction of the fluid, on the regular supply of which the activity of the brain depends, but, if we accept the statements of Boll, His, Obersteiner, and others as to the existence of perivascular lymphatics, produc-

ing such changes as must have materially interfered with the return to the system of the products of waste and superabundant nutritive plasm. As a consequence, we have softenings, diseased cells, various morbid deposits and apoplexies, the result of the presence of which is affections of the various functions of the encephalon and spinal cord, motor, trophesial, and psychical. It may be said that the insanity was merely an accident in the case, and that the convulsive and trophesial abnormalities were the most important. But insanity was the first symptom of nerve degeneration on which these abnormalities supervened. There is under my care at present a case in which very similar psychical symptoms are present, following on the tertiary stage of syphilis, but in which there is no modification of motion or trophesis. Were this patient to die, and we were to find the same indications of syphilitic deposit on the vessels of the hemispherical ganglia and degeneration of their cells, would we not be entitled to designate it one of Syphilitic Insanity? There can be no objection to the periphrasis "insanity dependent on syphilis," in the same way as there could be none to "iritis dependent on syphilis," "rheumatism dependent on gonorrhœa," and scores of other diseases which are the results of special conditions of the system. The periphrasis implies, if it implies anything, that the insanity, iritis, or rheumatism would not have occurred if the patient had not contracted syphilis or gonorrhœa. But if to this assumption we can add the pathological fact that in a case of insanity dependent on syphilis a lesion of the vessels is present which has not been observed in any other etiological class of so called mental disease, we, I hold, have an absolute right to call the condition "Syphilitic Insanity." It is, perhaps, the only form of insanity in which a special lesion has been demonstrated, coinciding with those of other parts of the system under a similar condition. The descriptions and plates of *Edmansson* and *Fränkel* indicate a similar condition of the vessels of the syphilitic placenta, and reference need only be made to the thickened and occluded arteries of many other organs of the body in tertiary syphilis. From a merely clinical point of view this case is strongly illustrative of the acute remark of *Dr. Buzzard*, that the complexity and diversity of nervous symptoms in one individual are strong presumptive evidences of a syphilitic causation. With all deference to such an eminent pathologist as *Dr. Wilks*, I cannot refrain from reiterating the opinion that the clinical history and *post-*

mortem examination of this case warrants its being designated one of Syphilitic Insanity.

DESCRIPTION OF PLATE.

1. Section of large vessel, from white matter in the neighbourhood of the softening of the left corpus striatum, showing gummatous deposit and thickened coats. $\times 275$.
2. Section of right ascending parietal convolution at vertex, showing commencement of deposit. $\times 275$.
3. Ocluded small arteries in same position. $\times 275$.
4. Longitudinal section of vessel from same position. $\times 275$.
5. Section from Pons Varolii, showing dilated condition of Vascular canals.

