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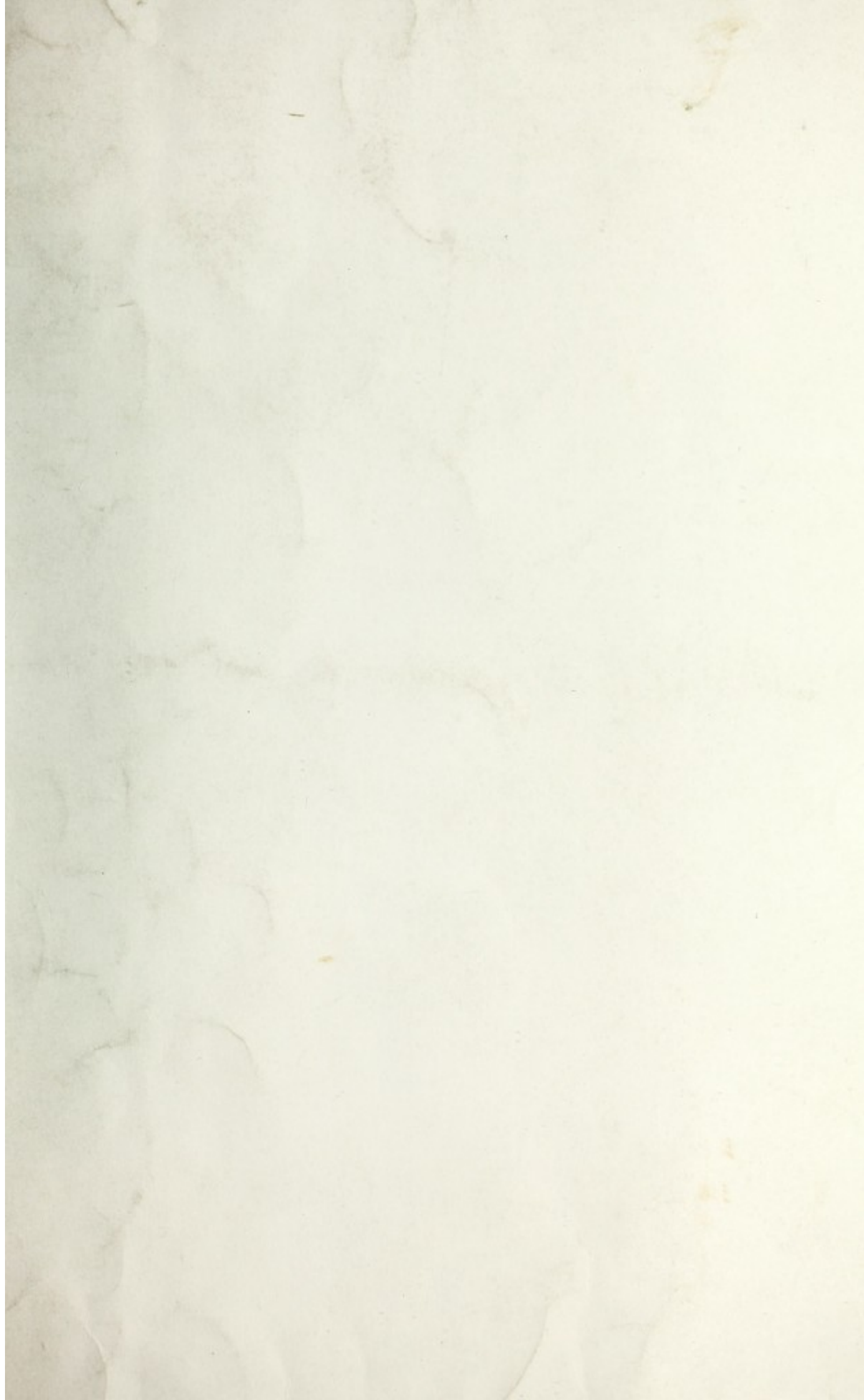
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MILIARY SCLEROSIS;

ITS

PATHOLOGICAL SIGNIFICANCE.

BY

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1874.

THE HISTORY OF THE

ANTHROPOLOGICAL INSTITUTE

OF GREAT BRITAIN

AND OF THE ASSOCIATION OF AMERICAN ANTHROPOLOGISTS

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1871

MILIARY SCLEROSIS;

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AN outline of the following remarks was embodied in a paper read before the Medical Microscopical Society, of London, on the 20th March. The object of that communication was to endeavour to draw the attention of histologists to a lesion which appears to be almost co-extensive with organic disease in the nervous centres, but which nevertheless has, until recently, been overlooked. This circumstance may excite surprise, when it is borne in mind how large a number of diligent and scientific pathologists have been for many years past engaged in the investigation of pathological histology.

The great diversity of diseased states in which miliary sclerosis is met with, in the brain and spinal cord, constitutes an important point in its pathological history, and possesses, in no less degree, an interest relatively to its clinical history, if indeed it can as yet be said to have any clinical history at all.

Miliary sclerosis was first described by Drs. Batty Tuke and Rutherford, in the pages of the 'Edinburgh Medical Journal,' 1868-69, and the lesion was so designated by them from the resemblance of the spots to millet-seeds in minuteness, compared with the more extensive forms of an analogous change which had been named by the French pathologists, general sclerosis, disseminated, annular sclerosis, *sclerose en plaques*, &c.

The name sclerosis is open, in some degree, to objection, inasmuch as hardness is not always its distinctive character. It would be more accurately expressed by the term "grey degeneration," of nerve tissue. Miliary sclerosis, or grey degeneration, is a change commencing in the neuroglia or its nuclei, the normal tissue having disappeared, is replaced by the minute millet-seed patches, either singly, or in clusters. These are very abundant in some instances. In one specimen that I examined from the brain of an idiot, they existed in the proportion of twenty-five thousand to a square inch of surface.

To attempt to describe in detail the characters of miliary sclerosis, would be merely to reproduce the exact and very precise description given by Drs. Tuke and Rutherford, as above mentioned; and still

more recently by Dr. Tuke again, in this Journal (July, 1873). In connection with this subject the reader may, with advantage, consult two papers on amyloid degeneration and brain sand, by Dr. Arlidge,¹ who has pointed out the tendency under morbid influence to the development in nerve tissue of albuminoid corpuscles, which in time undergo a calcareous transformation. The scope of the present remarks embraces mainly the *pathological relations* of the lesion under consideration. It is associated, as will be shown below, by widely different nosological conditions. It is associated also with other pathological changes, *e.g.* with amyloid and colloid bodies,—and differs essentially from what is known as the yellow granular degeneration, as well as from disintegration of nerve tissue, which may be either ante or post-mortem. The reactions of amyloid with codeine, in the brain and cord, are obscured by the presence of chromic acid employed in the previous hardening, but the spots of sclerosis may be distinguished from amyloid bodies by the use of the polariscope, which will give neither the concentric rings nor the black cross of amyloid bodies, while it renders evident the molecular character of the degeneration.

Colloid bodies may be distinguished by their clearly defined margins—miliary sclerosis presenting more or less irregular borders, from the broken ends of fibrils, vessels, &c., encroaching on their space—and from their clear homogeneous translucent contents, which do not take the carmine colouring. “In extreme cases the appearance of sections containing colloid bodies may best be compared to a slice of sago pudding, for they exist in such large numbers as almost completely to fill the field of the microscope, separated slightly from each other by a fine granular material.”² “Colloid bodies do not undergo the same gradations of development as miliary sclerosis, they do not push aside the fibres, and they never can be removed as a separate substance from the dried sections in which they exist.” These, Dr. Tuke points out, are the chief characteristic differences between colloid and miliary sclerosis. Having thus briefly alluded to the physical characters of miliary sclerosis, it becomes a point of great importance to determine, if possible, the pathological relations of these deposits,—their relation to previous diseases as observed during life. Here I think that at present we find ourselves at a loss. This lesion is found to co-exist with so many and varied morbid conditions, that we are puzzled in which direction to look for a clue to its proper place in neuro-pathology. In our present state of knowledge, we have no guide to the connection between this degeneration and the symptoms noted during life. In confirmation of this unsatisfactory view of the matter, I may state that over and above the examination of brains after death from insanity,

¹ ‘Brit. and For. Med.-Chir. Review,’ October, 1854, p. 470.

² Tuke ‘Brit. and For. Med.-Chir. Review,’ July, 1873, p. 208.

by Dr. Batty Tuke, already referred to, I have notes of my own observations of its presence in the brain or spinal cord, or both, in twenty different pathological conditions, as follow :—

1. *Acute meningitis* of the cord ; following upon fracture of the skull, with hernia cerebri—death after two months. This specimen was given to me by Dr. Moxon.

2. *Abscess of the brain.*

3. *Locomotor ataxy*,—in the posterior columns, principally.

4. *Idiocy*, in two cases. One of these cases an example of microcephalic brain of a boy, aged twenty years, whom I had known from the time of his birth. The second case also was microcephalic, twenty-one years of age, accompanied with mollities ossium ; idiotic from birth.

5. *Leukæmia*,—portion of spinal cord given to me by Dr. Moxon.

6. *Chorea.* (History wanting.)

7. *Tetanus*,—from several instances in which the course of symptoms had been rapidly fatal.

8. *Sub-acute myelitis*,—the spinal cord in this case was referred to Dr. Lockhart Clarke and myself, by the Pathological Society. (The Report thereon was presented to the Society in November last.)

9. *Pseudo-muscular hypertrophy.* The details of this case and its post-mortem examination were given by me in the 'Journal of Mental Science,' 1870. I should here observe that I at that time attributed to the presence of miliary sclerosis a more special pathological relationship to the case there related than a more extended observation has justified.

10. *Paralysis with Aphasia.* Case reported by Dr. Glover, in the 'Transactions of the Clinical Society,' 1873.

11. *Hydrorachis interna* ; two cases,—in the first, a portion of spinal cord exhibiting this condition was given to me by Dr. Moxon for examination. This case is one of pathological interest with reference to the importance of following up any indication of disease, however unlikely it may seem to promise useful results. Dr. Moxon, examining the body of a boy who had died of a disease not specially involving the nervous centres, observed a band of old lymph deposit, of about a hand's breath, encircling the inner surface of the costal pleura. This appearance led Dr. Moxon to examine the spinal cord, in the cervical and dorsal regions of which the central canal was found to have been dilated and distended with fluid. Both the white and grey matter of the spinal cord I found thickly studded with miliary sclerosis.

A second example of the same co-existence of miliary sclerosis and hydrorachis I found in a case of Progressive Muscular Atrophy, the particulars of which are reported by Dr. Headlam Greenhow in the fifth volume of the 'Transactions of the Clinical Society.'

12. *Apoplexy with hæmorrhagic softening*, in a portion of brain given me by Dr. Dowse, Superintendent of the Highgate Metropolitan Infirmary.

13. *Infantile convulsions*, in a child seven months of age, apparently healthy, but having a syphilitic history, suddenly attacked with fatal convulsions.

14. *Dementia*. A case of chronic mania that had been many years under my observation.

15. *General paresis*. Of the occurrence of this lesion in general paralysis, I have seen several examples.

16. *Malignant disease of the spinal cord*. For this pathological specimen I am also indebted to the kindness of Dr. Moxon. The preparation had been many years preserved in spirits of wine. It consisted of a small cauliflower growth, proceeding from the lining of the central canal.

17. *Glioma of the Pons varolii*. The opportunity of examining a portion of a tumour of this kind was afforded me by Dr. Hughlings Jackson. Besides the special structure of the glioma, this tumour presented some examples of the colloid bodies.

18. *Internal hæmorrhage*, the cause of sudden death. A woman aged twenty-five, eight months pregnant, giving way to a violent outbreak of passion, died suddenly. The peritoneal cavity was found to be filled with blood, but the source of hæmorrhage could not be found, nor could any change in the vessels be discovered. Miliary sclerosis was found in the brain.

19. *Puerperal mania*. This case which presented several points of pathological interest, has been already related to the Pathological Society of London. Briefly stated, the principal facts are as follow:—The patient, who had been under my own notice, had borne seven children. After the last four births, she had on each occasion an attack of puerperal mania of a very violent character. In the last she sank from exhaustion. Permission being given, we removed the brain and spinal cord, in both of which abundance of spots of miliary sclerosis were found. I may add, that a very remarkable condition was found in the cord, viz., a total absence of the cells of the posterior vesicular column, for about an inch in extent on the left side of the lower dorsal region. No symptoms existed during life by which attention was drawn to the spinal cord. We are ignorant of the exact functions of this column of cells, but they doubtless have some relation to sensation.

20. In the pages of this Journal, April, 1869, I described "a new lesion in the medulla oblongata." From the description then given, Dr. Tuke recognised "miliary sclerosis," and was good enough to forward to me a copy of the paper, above referred to, by Dr. Rutherford and himself, in the 'Edinburgh Medical Journal.' From this I learned the real nature of my "new lesion." I had no

history of the case whence it was taken; the specimen had been given me by Mr. Bryant simply as material for microscopical study.

To this catalogue I could have added several more, if I had carefully preserved notes of all my microscopical examinations. The list, however, is sufficiently extensive to arouse a desire to know if, among all these various morbid conditions, any one common feature can be traced. To myself the search has hitherto met with small success. There is little in common between acute meningitis and locomotor ataxy, or between tetanus and local softening in the brain. In acute disease of the nervous centres, such as meningitis or tetanus, we must assume that the lesion pre-existed, since we cannot suppose such wide-spread degeneration to have been produced in the course of a few days. There is, moreover, no other lesion that occurs to my mind which, like the present, is met with equally in acute and in chronic disease of the nervous structure. That it belongs to some chronic morbid condition of the nerve tissue, appears to be the inevitable conclusion. The relation of this condition to symptoms remains yet to be discovered.

It has been suggested to me by Dr. Dickenson, whose opinion, upon all matters pathological, commands respectful attention, that this supposed lesion is but a physical post-mortem change, resulting from the action of alcohol on the tissues. But to this it may be replied that the same thing is found in brain and cord that have been preserved in chromic acid only: moreover, it is not always found in brain or cord that has been preserved in spirits of wine.

That in miliary sclerosis we have a really morbid degeneration of nerve tissue there can, I believe, be found no good grounds to doubt. The multitude of its associated pathological states, its wide diffusion throughout both brain and cord, may present difficulties in the way of solution of the question of its relationship; but these circumstances afford no proof that it is not a veritable morbid change. What may be its starting-point is to be solved only by further investigations into the origin of the diseases with which it is associated.

So far as the present amount of evidence permits, this extends no further than to the inference that in a large number of diseases of the nervous centres, the significance of miliary sclerosis is a slow change or degeneration of the neuroglia, preceding or underlying the group of symptoms which have constituted the antecedent malady.

The accompanying illustrations present the characters of miliary sclerosis, as shown under low and high powers of the microscope, viz., from three and a half inch to one-eighth objectives.

EXPLANATION OF PLATE,

Illustrating Mr. W. B. Kesteven's paper on Miliary Sclerosis.

Fig. 1.—Section of spinal cord from a case of Tetanus, showing spots of miliary sclerosis. Seen under a three-inch object-glass. $\times 12$ diameters.

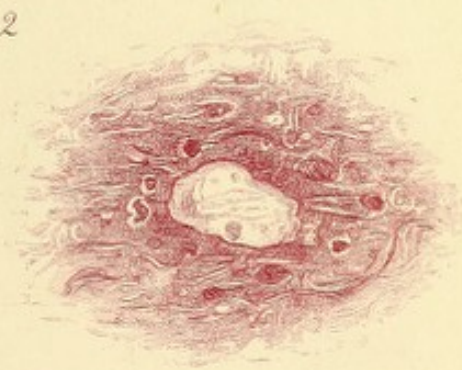
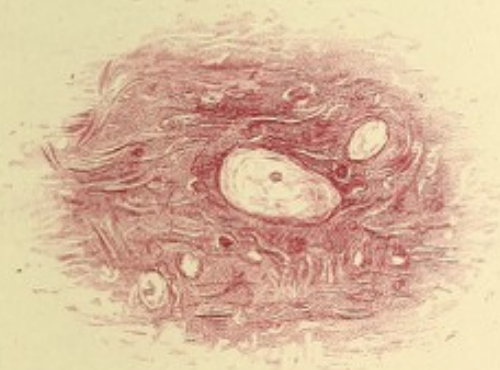
Fig. 2.—Spots of miliary sclerosis seen under a quarter-inch object-glass. $\times 195$ diameters.

Fig. 3.—The same under an eighth. $\times 420$ diameters.

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