which are descended from the epithelium of the testicle retain, even when living as parasites in foreign tissues, some of the specific secretory properties of their prototypes is not without interest to those of us who have been unable to accept the various theories which have been prevalent at times as to the causation of carcinoma by parasites from without and who have contended that it is only by research into the laws which regulate the development and growth of the normal tissues that we can hope to solve the all-important question of the method of causation of carcinoma.

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HUNTINGTON'S CHOREA AND DEMENTIA.

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THE interesting case of Huntington's chorea with dementia recorded in THE LANCET of Oct. 28th, p. 1252, and the annotation thereon (p. 1274) as to what extent the disease occurs in asylums for the insane prompt me to add another case of especial interest, insomuch as the clinical symptoms during life afforded the possibility of a diagnosis of chronic general paralysis with mental symptoms, a diagnosis which the results of the post-mortem examination, recorded by Dr. G. E. Peachell in his case, appeared also to suggest.

Having some considerable experience I venture to affirm that of all the gross pathological lesions found in cases of general paralysis "granulation" of the ependyma in the lateral ventricles, and particularly in the calamus of the fourth ventricle, is the most constant, and possibly also the most pathognomonic, of the naked-eye appearances—chronic and senile forms of insanity being of course excepted. The thickening of the membranes, the opaque gelatinous appearance of the pia arachnoid, the wasting of the frontal convolutions and the filling up of the inter-gyral space with fluid, as recorded by Dr. Peachell, further demonstrate the affinity which (some cases at any rate of) Huntington's chorea bears in its pathological findings to general paralysis.

The case here related is that of a married woman, aged 53 years. She had been in the asylum nearly seven years before death occurred. The disease was stated to have commenced shortly before admission, at the age of about 46 years, the mental condition being attributed by the friends to that vague but withal stormy crisis entitled the "change of life." Her previous history relates that she had been married 12 years, had borne two children who died young, that she had no miscarriages, and never had a seizure. Members of her own family stated that she was never "strong-minded" and that she had married "a man who always looked upon her as a poor thing, and gave her much worry." Upon admission she had a somewhat dull and heavy look, but she was restless and fidgety from large incoördinate choreiform non-rhythmic movements, which caused her to have some tendency to a "rolling gait." The movements particularly affected the head and face, but upon admission there appeared to be some facial paresis, more marked about the right side of the mouth. The reflexes were brisk, and the pupils were inactive to light (right pupil 3 millimetres, left 2½ millimetres) and reacting to accommodation (the right to 2½ millimetres and the left to 2 millimetres). The heart sounds were rapid and somewhat indistinct. Mentally, apart from delusions of a sexual character and that she imagined herself to be pregnant (accusing herself of infidelity), there was dementia—loss of memory being a prominent feature. For several years the weakmindedness increased and she gradually became more childish, her speech became more difficult, especially in pronouncing gutturals, but apparently there was little or no trouble with labials. The speech difficulty was greater when notice was taken of her. The last two years witnessed further general deterioration; the pupils at this time were unequal and inactive to light: right 4½ millimetres, left 4 millimetres; when accommodating: right 3 millimetres, left 2½ millimetres; and when accommodating strongly: right 2½ millimetres, left 2 millimetres. Later the choreic movements became very marked in the hands; she could not dress herself, and the left arm, owing to paresis, was weaker than the right. Both knee-jerks were increased but the left preponderated. At this stage both pupils measured one millimetre and were still inactive to light. She became very fidgety, especially when spoken to; her appetite became ravenous, her habits became defective, and she used to grind her teeth. She destroyed her clothing and required much attention. Gradually she became bedridden and very emaciated, and she died just under seven years after admission.

The family history is interesting. Her father, country born and a brick-burner by trade, was never very bright, being always fearful that he would have to go into the workhouse and he suffered from the same condition as the patient. Shortly after he reached the age of 50 years, having lost his wife, he went "a bit wrong in his mind" and had twitchings like his daughter had. He eventually became "quite silly" but was never in any asylum. He died from the "decay of nature" at the age of 58 years. The patient's mother had a congenital club-foot, being deformed from birth, but she was active and got about with the help of a special boot. She was described as being quite bright mentally and as being a great contrast to her husband. She died at the age of 54 years after a week's illness from what was described as an "apoplectic fit." Nothing is definitely known of the grandparents beyond the fact that they were respectable country villagers in Berkshire. The grandfather was a master brick-burner, for whom the patient's father himself worked. No history of any illness in this connexion nor of the cause of death is obtainable. As to the collaterals, there were seven members of the patient's family, three brothers and four sisters, and none of them are described as being particularly bright. One sister died in a fit in the village home and was "shaky" like the patient. Two brothers died from pulmonary tuberculosis and it is recorded that the mental state of one of them was characterised by impulsive and irritable outbursts.

The patient died from subdural hæmorrhage. The brain weighed only 1025 grammes (the average normal female brain weighs 1270 grammes). The right hemisphere weighed 415 grammes and the left 390 grammes. The dura mater was thickened on the left side and there was a great excess of red-stained fluid beneath it. There was a membrane over the vortex on both sides, thicker on the left than the right, and on this side also were about four drachms of recent blood clot which was probably only a few days old. The hæmorrhage extended over the whole of the base of the brain. It was membranous and probably of some duration in the anterior fossa where it existed in three films. It was also in films on the tentorium. The pia mater was opaque and thickened over the fronto-parietal region, stripping readily, and there was a great excess of subarachnoid fluid—a consequence of the loss of cerebral substance, probably through neuronic degeneration. The convolutions were extremely wasted throughout.[1] The actual nature of this wasting is imperfectly understood; the neuronic degeneration probably interferes with the nutritive and excretory systems of the cortex; an increase of glia tissue takes place which contracts and cerebro-spinal fluid or blood replaces the wasted cortex. The lateral ventricles were dilated and there were granulations of the ependyma, as also of the lateral sacs of the fourth ventricle. The vessels were apparently natural. The layers of the cortex in the pre-frontal region were carefully measured by Dr. J. S. Bolton (late assistant pathologist of the London Asylums and now at Rainhill) at the apex, side, bottom and flat part of the convolutions, and he found extreme narrowing of the pyramidal layer with considerable narrowing of the other layers, being identical in actual measurement with that of a full term fœtus and a six weeks' child.

The main interest of this case lies in the fact that it is not only a typical one of Huntington's chorea but it has also been most carefully worked out in Dr. F. W. Mott's laboratory and recorded by Dr. Bolton in his paper entitled "The Histological Basis of Amentia and Dementia," in Vol. II. of the *Archives of Neurology*—a paper which is itself evidence of colossal industry and is probably the first attempt through pathology to correlate mental states with the different areas of the cortical surface. I am of opinion that the classification of brain weights as to convolutional complexity and the comparative estimates of the cortical layers in actual measurements as worked out by Dr. Bolton and Dr. G. A. Watson are of the highest value, possibly more so than microscopical descriptions of the cells and their nuclei. I should like to invite

[1] See photograph, Archives of Neurology, vol. ii., plate iii., fig. 10, p. 618.

the special attention of those interested in asylum work to the excellent and elaborate thesis above referred to. It is, I think, an unusual and a valuable example of the combination of clinical and pathological investigations.

In the case under consideration the small weight and under-development of the brain are noteworthy and it would have been interesting to have had these referred to, as also the relative thickness of the cortical layers in the case previously described. The cortical measurements given by Dr. Bolton show a wasting of nearly 27 per cent., mostly in the pyramidal layer[2] which contains the highest association neurons and upon the integrity of which the most advanced needs of the individual depend—those needs based upon reason and intelligence, those which involve attention, comparison, and judgment, and which result from the interpretation, collection, and elaboration of incoming sensations arriving on the "platform" of the granular layer. The least wasting occurs in the polymorphic layer which Dr. Watson has shown to be related to the lower functions of the organic life, such as seeking for shelter, hunting for food, or those controlling the lower instincts, and it is only diminished in pronounced mental disease when the habits become degraded. The pyramidal layer has been shown by Dr. Bolton[3] to be directly related to the mental capacity and power of the individual. It is under-developed in amentia, is well represented in persons of high mental grade, and is, in the latter class, wasted proportionately to the degree of dementia caused by a degeneration of the neuronic elements in this layer.

As to Huntington's chorea, its pathology is hitherto not definitely ascertained. Some observers have ascribed the symptoms to vascular changes leading to hyperplasia of glia tissue, the neurons being secondarily affected. Others uphold a primary degeneration of the nerve elements, interstitial changes being secondary and due to the chronicity of the process. Its affinity with general paralysis may suggest that it is itself a pre-senile condition affecting the higher neurons, the pyramidal and Betz's cells, through a vicious organisation—some genetic deficiency of power to survive, a "hereditary malformation" becoming a *locus resistentiæ minoris*. We know that alcohol may select to make its ravages evident in some persons by cirrhotic changes, in some also by neuropathic changes, and in others by the psychopathies. In some persons decay is indicated by vascular changes, in others it is indicated primarily in the nervous system. We appreciate that in every tissue there is a certain inherent tendency to evolve towards a higher state of development or towards perfectibility in regard to the environment, but this perfectibility is accompanied—as a set off—by diminished durability. The more highly developed the neuron the more deficient it is in durability; the less highly developed the greater the durability. We are acquainted with cases of juvenile general paralysis in which slight stress causes little injury and they linger on for long periods, and we may contrast with these cases occurring in highly developed and capable persons where great and severe stress has led to rapid deterioration and degeneration when once this "breaking strain" has been reached. What determines the tendencies to dissolution we do not know, for hitherto we have but little knowledge of the recessive or dominant characteristics which determine hereditary tendencies in the different structures of the body. We may further instance the parellelism of general paralysis and Huntington's chorea; both are progressive and fatal and both are associated with some form of paralysis, and it might not be inappropriate to include the paralysis accompanying Huntington's chorea in that group of cases described as "pseudo-general paralysis"—a term applied by Fournier, and referred to by Dr. T. B. Hyslop,[4] as including ordinary general paralysis with remissions, also paralysis, more or less general, which may result from, or follow, alcohol, lead, or syphilis.

I am aware that the suggestion of pseudo-general paralysis is open to a retort from the logical mind that a thing is or is not, and that a disease is either general paralysis or it is not, but as clinicians we know that there are a number of atypical forms which for want of exact knowledge are most conveniently and expressively grouped as such. Whilst not personally subscribing to this somewhat comprehensive and inclusive classification of general paralysis, and whilst recognising the variety of mental affections accompanying true general paralysis, one's own experience can relate the association of mental deterioration with such cases as Friedreich's hereditary ataxia with choreiform movements, paramyoclonus multiplex, and some of the spinal system lesions (amyotrophic lateral sclerosis, bulbar paralysis, and chronic muscular atrophy) which at infrequent intervals are met with in the larger asylums for the insane.

It is only by such records as that furnished by Dr. Peachell that we shall succeed in further grouping the many varieties referred to as pseudo-general paralysis. In a recent report of all the nervous and mental cases that have come under treatment in one year in the Central Municipal Hospital for the city of Dresden, the proportion of cases of Huntington's chorea was, I believe, only three, but I speak from memory. In the statistics of St. Bartholomew's Hospital, London, for 1904 there are 64 cases of chorea, seven cases of insanity, but none indexed under the term Huntington's chorea. My own experience of over 10,000 male and female insane persons admitted into the London County Asylum at Claybury leads me to believe that this variety, excluding senile chorea, does not occur in asylums more often than about once in every 3000 cases—if as frequently.

Claybury.

[2] Archives of Neurology, p. 607, Case 19, Fig. 21.
[3] Ibid., p. 611.
[4] Transactions of the Medical Society, vol. xxvi

SOME REMARKS ON THE PREVENTION OF APPENDICITIS[1]

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IN bringing forward this subject before our society I think that some explanation is due to its members. In the first place, it has been discussed very fully of late; and in the second place, at first sight it may appear to have little to do with balneology. As my paper progresses I hope to give an answer to both statements. I think that there can be little doubt that appendicitis has much increased of late years. Although many years ago cases of appendicitis were almost invariably put down as peritonitis, yet these peritonitic cases were less numerous than we meet with to-day. The type of case I believe has also changed, as in many other diseases, for the so-called simple or medical cases we see less frequently than we used to. How this increase in number and alteration in type of cases have been brought about I hope to show later.

Before passing on to the clinical aspect of my paper it is well to say that there seems no doubt that the real starting point of the appendicular peritonitis is the presence of the bacillus coli communis in the appendix; yet this statement requires an explanatory note, for the above bacillus is always present in large numbers through the whole alimentary canal from the mouth to the anus; again, it varies greatly in its virulency. It is only when the bowel becomes the seat of any morbid change that the bacillus, so to speak, comes into play. Thus any change brought about in the bowel by constipation, diarrhœa, obstructed bowel, or congestion of the bowel becomes of immense importance. Thus it seems that for an attack of appendicitis to occur there must be some lesion of the appendix to allow the escape of the bacillus and the bacillus must be in a state of virulency. Ordinary or simple catarrh of the appendix is evidently of common occurrence. Post-mortem examinations show this. It generally leads to no symptoms and is not often clinically diagnosed. But there is a further stage of the trouble in which more or less inflammatory material is thrown out. This material may go on to the formation of pus or be entirely absorbed.

Coming now to the practical or clinical side of our subject, everyone must at once be struck with the change of mind which has taken place in respect to the treatment of appendicitis. A few years ago all cases of appendicitis were admitted into the medical wards and treated by the physicians. Now surgeons seem to claim these cases and all writers and speakers of to-day when discussing the subject of appendicitis refer to it almost entirely as requiring surgical treatment. This was particularly brought out at a meeting of the Royal Medical and Chirurgical

[1] A paper read before the British Balneological and Climatological Society on Dec. 7th, 1905.