

ART. IV.—THE PATHOLOGY AND MORBID HISTOLOGY OF CHRONIC INSANITY.

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The morbid histological changes occurring in insanity are, at the present day, undergoing microscopical investigation at the hands of many very skillful observers, both in our own country and in Europe, and these assume great importance when we reflect upon the fact that the pathological phenomena discovered in the brains of persons dying insane, all have for their basis, interference with the due nutrition, growth and renovation of the brain-cell, which, by interrupting the nutrition, stimulation, and repose of the brain, essential to mental health, results in the impress of a pathological state in the brain and disordered mental function. The investigation of both the normal and the morbid histology of the brain is a work requiring great labor, patience and perseverance, and also great judgment in the recording of observations, and, even with the most careful microscopists, mistakes may be made as to the nature and value of appearance met with in histological research. The naked-eye appearances which may be met with in the bodies of those dying insane are chiefly peculiarities in the form of the cranium, of which the most frequent is want of symmetry between the two sides; the shrunken and shriveled ear in chronic insanity, consequent upon hæmatoma auris; variations from the normal standard in the thickness or thinness of the cranium; changes in the membranes as to appearance and structure, and finally changes in the cerebral substance itself. In chronic insanity, the changes chiefly met with in the brain, have been, atrophy of the convolutions and brain itself; induration of both white

and grey matter; thickening and opacity of the membranes; chronic hydrocephalus; effusions into the sub-arachnoid space; pigmentation of the cortical substance, and extended and profound sclerosis of the brain. The pia mater is found to be thickened and adhesive to the brain, and its vessels tortuous and thickened in their walls. Atheromatous and fatty degeneration of the walls of the cerebral capillaries have also been noticed.

The pathology of epileptic insanity, which in cases under my care has generally been associated with chronic insanity or dementia, is a very interesting subject; one deserving of far more room and attention than is here devoted to it. I propose to merely briefly allude to it, reserving a more lengthy discussion of this very important subject for a future time. The pathology of the production of epilepsy occurring or associated with chronic insanity or dementia, consists, I think, primarily, in interference with the proper nutrition of the cerebral tissue of the fœtus, so that even during embryonic life, the brain of the infant undergoes pathological changes which induce deficient moral power, mental weakness and epilepsy, there being an ill-balanced and defective state of the whole central nervous system, predisposed to take on diseased action. The portion of the nervous system mainly affected by, or more strictly speaking, the portion which is the primary seat of epilepsy associated with chronic insanity or dementia, I believe to be the medulla oblongata, the corpus striatum, and the motor tract of the cervical region of the spinal cord and the cerebellum. From the fact that I have by means of the fluid extr. ergot, continued for some time, decreased the paroxysms of epileptic mania both in frequency and intensity, so that the maniacal excitement has entirely disappeared in many instances, the pulse and temperature becoming normal, I have inferred that the cause of the epilepsy in these cases was cerebral hyperæmia of the nervous centres, and that the disturbance was partly at least functional in character. On the other hand, in epilepsy, associated with chronic mania and dementia, I have never had anything but negative results from the use of ergot, and I have therefore inferred that the state of the nervous centres was one of organic degeneration, induration and anæmia. Examina-

tion of the urine in the former cases revealed an excessive elimination of the phosphates, while in the latter cases associated with chronic insanity the elimination of phosphates was below the average. The reaction of the urine in these latter cases was acid. In speaking of the epilepsy of chronic insanity as dependent upon anæmia, I of course recognize the fact that the clonic spasms of the second stage of the epileptic paroxysm, during which the alternate powerful muscular contractions occur, the pupils oscillating, the pulse being full, and palpitation of the heart occurring, are accompanied by notable cerebral congestion. I have only had the opportunity of making one post mortem examination in a case of chronic insanity associated with epilepsy. The case was one of chronic mania in a German, in the course of which epileptic paroxysms occurred. At times the maniacal excitement preceding the paroxysm would be such as to require mechanical restraint. The patient had fits very frequently and after a short time had them daily, and they increased both in frequency and intensity in spite of all medication. The patient finally died in a paroxysm after a succession of fits, lasting thirty-six hours. The post mortem examination revealed thickening of the membranes of the brain; the arachnoid opaque, and the pia mater much thickened. The brain was atrophied and indurated, and the lateral ventricles were filled with fluid. The cord was normal in appearance. The brain presented an anæmic appearance. Upon hardening the brain and medulla oblongata, and examining microscopically, there was a thickened and enlarged condition of the capillary vessels of the medulla, and some vascularity in the fourth ventricle extending through the medulla. In the larger nerve cells of the deeper layers of the convolutions of the brain, which I consider analogous in their functional activities to the ganglion cells of the anterior cornua of the cord, was seen pigmentation, atrophy, and degeneration. On examination of the cells of the grey matter of the corpora striata, there was found degeneration and atrophy.

Having devoted considerable time to the investigation of both the normal and morbid histology of the brain, I desire to call attention to an appearance which I have

noticed in the brains of those dying insane, and to which my attention has been drawn from the interest it assumes when viewed in the light of a possible ultimate cause of the nutritive defect which results in chronic insanity. We know, that for the proper nutrition and healthy functional activity of the brain-cell, the proper nutrient supply is required, and that we cannot have healthy mental function without a due supply of healthy blood to normal and healthy brain substance. We also know, that if any agent operates in the influencing of the circulation unfavorably, so that a morbid condition of the cerebral capillaries be induced, we shall inevitably have resulting morbid changes, set up and maintained in the cerebral cells.

In my writings on insanity, I have called attention to the fact, that a microscopical examination of blood from insane patients, as compared with an examination of blood from the same number of healthy individuals, revealed in the blood of the insane a marked increase in the number of white corpuscles. In making microscopical examinations of brain tissue from chronic insanity, I have noticed repeatedly in different cases lymphoid cells, or white blood corpuscles, and also red corpuscles in small numbers, in the substance of the brain tissue, evidently having emigrated from the blood-vessels. From what I have observed, I think that under conditions of inflammatory irritation of the brain, an emigration of lymphoid cells takes place on a large scale, the cells, or corpuscles, by virtue of their vital contractility passing through the walls of the vessels and penetrating into the brain tissue. It will be remembered that both Dr. Bastian and Dr. Blandford have noticed a plugging up of the blood vessels by small embolic masses composed of aggregations of white corpuscles in insanity. Ecker found that the vessels of the gray matter were generally dilated in insanity, and Raman also noticed the same thing in the vessels of the pia mater, while Dr. Major has described a dilatation of the arteries in "brain wasting," a condition which appertains to chronic insanity.

We have here two factors, which operate, I think, in the production of the appearance in the brain of the lymphoid cells, and in some cases of the red corpuscles. First, the undue

predominance and accumulation in the blood vessels of the white corpuscles, which obstruct the capillaries, as they move so much more slowly than the red corpuscles, giving us as a result an impeded circulation and an increased pressure on the coats of the vessels; and second, the dilatation of the vessels before alluded to. These two conditions are favorable to the rapid emigration of the white, and also the red corpuscles through the walls of the vessels, and also, perhaps, the same condition may be produced at times by the obstruction in the capillary vessels becoming great enough to rupture them, permitting in this way the escape of a few blood globules into the brain tissue. (Such a condition would be analogous to what, I think, often occurs in the lungs, as I endeavored to explain in a paper on consumption, published in the *New York Medical Record* of September 18, 1875.) Such lymphoid cells would act probably as foreign bodies, and a slow course of inflammation would be likely to be set up to get rid of the intruders. Such an inflammatory process would naturally be of slight intensity and long duration, and these collections of lymphoid cells would tend to become developed into a fibroid structure, resulting in induration of the brain, such as we find in chronic insanity. I am also forcibly impressed with the idea that, if I am correct in my conclusions, we have here the solution of the problem as to the relation which exists between tuberculosis and insanity. Dr. Clouston, in the *Journal of Mental Science*, for April, 1863, showed that of 282 patients who died with tubercular disease at the Royal Edinburgh Asylum, 153 passed rapidly into the state of chronic insanity, the acute stage being of very short duration, the patients all manifesting a decided tendency towards chronicity. He also noticed that the prognosis relating to mental recovery was eminently unfavorable, and that apparent recoveries proved to be only remissions. In these cases, where the development of the two diseases seemed to be nearly contemporaneous, there was probably, owing to a want of proper hygienic and sanitary surroundings; want of sunlight, fresh air and exercise, this undue development of the white blood corpuscles and the tuberculosis was very likely induced by the escape of some of these lymphoid cells into the meshes of the connective tissue

of the lungs where they became the nucleus of tubercular deposit. Again, these cases may have been the result of too little blood being sent to the lungs as a result of want of proper exercise, and close confinement at business or trades; the upper part or apices of the lungs being allowed gradually to collapse and tubercles appearing as the circulation ceased to affect them. I think this occurs more often than we are aware of, especially in persons who inherit the predisposing, neurotic element. That there exists such an hereditary, neurotic or morbid element or force, present in both insanity and phthisis, I most firmly believe, and I also believe that there is a correlation of morbid force which renders these diseases mutually convertible. I have repeatedly seen this borne out by undeniable facts, children of one family being affected with both insanity and phthisis in many different instances. I have no doubt other observers have also witnessed the same thing frequently. I have also come to believe that skin disease, in many of its forms, may be included in this same class of mutually convertible diseases, as I have had occasion to notice several times, psoriasis, herpes and eczema alternating with attacks of insanity. To return more immediately to our subject,—respecting the dilatation of the vessels which I before alluded to, it appears to be probable that the general obstruction in the capillaries of the brain, causes primarily a compensatory hyperæmia, and as this gradually becomes permanent, the small arteries would naturally become enlarged, as they have been found to be by Ecker and Dr. Major, and their walls would become thickened, as we find them to be, *post mortem*, in chronic insanity. Such long continued mechanical hyperæmia causes an impairment of vitality and function, and this we find exemplified by the retrogressive changes which occur in the substance of the brain in chronic insanity, viz.: atrophy, induration and degeneration of the nervous elements of the brain, with a resulting dynamical state of loss of psychological activity and profound physical and mental weakness, with the exception of cases of apoplexy in which large clots have been discovered, *post mortem*, I am not aware that any observer has described any such lymphoid deposit in the brain, which may, or may not, have undergone fibroid metamorphosis or

degeneration. I think, therefore, that from both a physiological and pathological standpoint, these observations (if confirmed by further researches) become of the highest clinical significance.

I desire not to be misapprehended as regarding the presence of the lymphoid deposits in the brain as the ultimate cause of insanity. I *do* however, think, that by their presence we are enabled to explain many of the changes incident upon chronic insanity and think their presence must affect, very materially, the ultimate molecular changes in the brain, upon which, functional activity depends, and regard it as a very strong probability that such foreign deposits in the brain, may, by interfering with the molecular changes just alluded to, destroy both functional excitability and activity. It would appear very probable the prominent alterations taking place in chronic insanity; viz., atrophy of the convolutions and of the brain itself and induration of the two substances, with degeneration and atrophy of nerve cells may be considered, fairly, to depend upon this abnormal state in the mutual relationship between the blood and the tissues, which becomes the ultimate cause of the nutritive defect, which results in chronic insanity.*

Clinical Cases illustrating the Pathology and morbid Histology of Chronic Insanity. Case I.—*Dementia and Paresis.* Death resulting from pulmonary hemorrhage. T. A., male, aged 22, single; occupation, wagon maker. Admitted to asylum June 24th, 1874. Upon admission was demented with symptoms of paresis; laughed vacantly when addressed and stared unmeaningly about him. No appreciation of condition or surroundings. His gait was staggering, and lips and tongue were affected with muscular tremors. He never spoke but once and that was upon the occasion of a visit from his brother. His speech at that time was hesitating and trembling. He had an attack of sub-acute meningitis in October, and died in January from an exhausting hemorrhage

*The best results in microscopical examinations of the brain tissue, can be obtained by immersing the brain immediately after removal in the following:

R.—Bichromate Ammonia,	160 grs.
Methyl Alcohol,	10 oz.
Distilled Water,	30 oz.

M. To remain until hardened.

from the lungs. *Post-mortem*: Upon removing the calvarium the membranes were adherent to the skull; subarachnoid effusion existed; also large effusion between the pia mater and the brain; the pia mater was thickened in patches. There was effusion at the base of the brain, fluid in the spinal canal and the spinal cord was atrophied. There was miliary tuberculosis throughout the brain. Upon making an examination of the chest, the left lung was found to be partially destroyed by the breaking down of the caseous products of pneumonia as a result of which, large cavities were formed. The heart gave evidence of recent endocarditis. The surface of the heart and endocardium were covered with miliary tubercles. The walls of the heart were atrophied and exhibited traces of fatty degeneration. The kidney, spleen and liver normal. Upon hardening the spinal cord, making sections, and employing carmine-staining, there was found to be, upon microscopical examination, atrophy and degeneration of the nerve elements of the posterior column, with increase of connective-tissue. Sections of hardened brain-tissue being made, there was observable in the cerebral cells of the frontal convolutions a diffused granular degeneration. No change could be discovered in the cells of the cervical sympathetic, which was carefully examined. Lymphoid cells and white corpuscles were found in aggregated masses in the brain having become developed into fibroid structure causing shrinking and induration.

Case II.—M. A. R., female, aged 29, single, occupation, servant. Admitted to asylum Dec. 29th, 1873. Form of insanity, dementia ending in paresis. The speech was slurring and hesitating; the gait was staggering and the mental faculties very much enfeebled; would become very angry at trifling incidents and then would relapse into silence, which lasted sometimes for weeks. She suffered from gradually progressing paralysis which involved the sphincters of the rectum and bladder. The cutaneous and muscular sensibility was impaired and there was likewise loss of electro-muscular contractility, so that disease of the antero-lateral and posterior columns of the spinal cord was diagnosticated before death. The paresis was attributed to spinal injury when quite young. She died from exhaustion, March 24th, 1874. *Post-mortem*: The dura

mater was firmly adherent to the cranium; the pia mater was thickened and infiltrated and the arachnoid thickened and opaque. The convolutions of the brain were atrophied and the brain-substance indurated, and throughout the brain were collections of altered white corpuscles. There was fluid in the spinal canal, and the cord was atrophied and softened in patches; the heart was small and flabby; spleen atrophied; stomach, liver and kidneys, normal; the uterus was in a rudimentary condition, apparently never having been developed properly. The spinal cord after being hardened and sections being made, revealed, upon microscopical examination, loss of neuroglia and connective tissue and degeneration of posterior columns and loss of nerve tubules of white substance; the ganglion cells of both anterior and posterior cornua were disintegrated and atrophied, and granular and fatty matter occupied their place.

Case III.—M. P., a female, aged 23 years, was admitted to the asylum Sept. 30th, 1873. Upon admission was depressed and melancholy and in a delicate state of health. She became gradually demented and paretic. Physical exploration of the chest revealed pulmonary tuberculosis, with cavities at the apices of both lungs. Patient died from exhaustion from paresis and tuberculosis July 18th, 1875. *Post mortem*: Brain anæmic, atrophied and indurated. The spinal cord was about of a normal size, its membranes were thickened and the pia mater thickened and opaque. The lung presented extensive disease; the heart was small and flabby and the kidneys atrophied and anæmic. Upon hardening the cord, the posterior columns together with the posterior section of the lateral column were found to be affected. The posterior columns presented atrophy and disintegration of nerve elements and plates of connective tissue in different places. In the posterior lateral columns were granular and fatty corpuscles and new connective tissue.

Case IV.—J. W., male, aged 27; occupation, student; admitted to asylum, April 30th, 1873 with acute mania. Upon admission was violent, requiring the restraint of a camisole. As soon as the mania subsided, dementia supervened, and he became gradually paralyzed. His mental faculties seemed entirely lost. He did not speak; required to be dressed and un-

dressed, and put to bed like a child, and led to the table for his meals, which he took from a spoon which had to be put in his mouth by an attendant. On the morning of March 27, 1875, he became suddenly comatose and died in a short time. *Post mortem*: The dura mater adherent to skull; arachnoid opaque and thickened; pia mater thickened and infiltrated and the blood-vessels enlarged and varicose. A varicose vessel had ruptured, giving rise to extensive hemorrhage which pressed upon both hemispheres causing death. The brain was anæmic, atrophied and indurated. There was effusion at the of the brain and in the lateral ventricle. Upon examination the lungs were found diseased, a large cavity being found at the apex of the left lung; the stomach, liver, heart and spleen were normal. The kidneys were hypertrophied and undergoing fatty degeneration. Many more cases might be cited but at the expense, I am afraid, of your patience, and those which have been inserted are typical of chronic insanity.

ART. V.—CASES OF INJURY OF THE BRAIN, INVOLVING SPEECH.

By P. R. Hoy, M. D., Racine, Wis.

IN October, 1842, I was called to see James Lawson, aged 18, a resident of New Haven, Ohio.

I found him comatose. There was a fracture of the skull, occupying the anterior superior angle of the left parietal bone, caused by the kick of a horse. He was insensible during the operation of trephining and removing fragments of bone, which left an opening as large as a half dollar. Soon after the operation he fully recovered his senses.

On the morning of the third day I found the patient comatose. I cut the stitches and opened the wound, when there escaped a clot of blood and a small quantity of bloody serum.