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Report on the Examination of One Hundred Brains of Feeble-Minded Children.*

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THE paper which I present to-day gives the results of *post-mortem* examinations, including microscopic study, of one hundred cases, including children of all grades under our care, thirty-four of which belonged to the school department, or were employed as aids in the asylum department, and sixty-six were children belonging properly to the asylum department of the institution. Some of these cases have been fully described in previous papers and will receive only passing mention in this.

The central nervous system consists practically of ingoing fibers from the various organs of sense and nerve cells for receiving and retaining impressions received through these fibers, and by some mysterious power of co-ordination combining these impressions and evolving new combinations, which are manifested to other individuals by impulses sent through a set of outgoing fibers to the various organs of motion. It is also well known that a constant and large blood supply is necessary to maintain the normal activity of the nervous system, and the circulatory apparatus of the brain is especially adapted to this end. It is evident that disease of any part of this delicate mechanism, or any condition which inteferes with its nutrition by interfering with its blood supply, is liable to disturb its perfect working. Should this occur before the seventh year, during the period of its active growth, the mischief is liable to be irreparable and life-long disturbance of mental growth be the result.

* Read before the Association of Medical Officers of American Institutions for Idiotic and Feeble-Minded Persons, Faribault, Minn., July, 1890.

In the following paper I design giving illustrative cases of the different morbid conditions found in the series, rather than to give them all in detail.

Probably the most frequent morbid condition met with is sclerosis in its various forms. The most destructive of these to the functional activity of the part involved is the so-called sclerose tuberense. It is usually seen on removing the membrane, as one or more areas of a white color and considerably harder in consistence than the surrounding tissue, slightly elevated above the level of the neighboring convolutions. It may be single but is usually multiple. It seems to be formed by a finely granular exudate, probably albuminous in character. If it occurs in the motor regions of the brain, the resulting paralysis is very complete, and if a large portion of the cortex is implicated, profound idiocy is liable to be the result.

In one case only have we been able to watch a case of this disease through its entire course and verify the diagnosis by an autopsy.*

71–1527.—A boy, aged 16, apathetic idiot. No family history of nervous disease except that the paternal grandfather died with one leg paralyzed. The boy was seized with frequent spasms of short duration, invariably beginning in the right leg and affecting the right side only. Little or no fever. Spasms not followed by stupor, recovery being almost immediate. All the usual remedies were applied and pushed to the limit of safety without effect. After continuing several days they became less frequent and finally ceased, but the right leg was left completely paralyzed, and contraction of the limb rapidly followed. After his death, which occurred a few months later, a block of this form of sclerosis was found in the paracentral lobule, very firm to the touch and of the characteristic whitish color. Besides this lesion, defective development of both frontal lobes was found.

Another case is 58–1296.—Male; born in 1877. The

* A second case came under observation since the presentation of this paper, in which the symptoms were nearly identical, the area of tissue involved was more extensive and death occurred in a few days, due to pressure from effusion.

maternal grandmother died of paralysis. Her sisters were insane. The mother was epileptic after marriage up to within three months of this boy's birth. Was a bright-looking little fellow; mute; no paralysis; had some idea of form, music and locality; learned a few of the simplest kindergarten exercises, but seemed to soon reach the limit of his capacity for learning, and did not improve to any extent during the last half of his three years' stay with us, though some especial effort was made by his teacher in his case. Died of membranous croup. The skull and brain were large and well formed. The sclerosis was found quite extensively through the cortex, though it had not attained the degree of hardness found in some cases. In the "motor" region only the posterior third of both third frontal gyri were attacked. The diseased areas were not large, though numerous.

66–312.—Adult female; age unknown. No history of nervous disease in the family. A prolonged labor, terminated by the use of some medicine (probably ergot), the child being cyanosed when born. Was taken to Philadelphia when six weeks old, contracting a "severe cold" on the journey, with obstinate constipation and convulsions. The convulsions continued as long as she lived. Profound idiot; mute; right hemiplegia; spent most of her time standing or sitting in a favorite corner mildly waving a dumb-bell in her hand, and occasionally getting up in the night to practice this form of calisthenics. The brain, removed during my absence, in hot weather, unfortunately softened in the lower part. The upper part showed this form of degeneration in places; a block in the left ascending frontal gyrus explaining the right hemiplegia from which she suffered.

[illegible] presents an extreme instance of this disease. Born in 1869. There seemed to have been no hereditary influence, except that the father is hardly up to the average mentally, though able to pursue his business as a butcher. Her mind seems an almost perfect blank. Mute; deaf; unable to find her way about, however often she may have traveled the same way before; has to be even led to her own bed. On her admission, food had to be placed in her hand before she would eat. Partially paralyzed in her right leg and left hand. So much of her brain was destroyed by this lesion that a detailed

description would be needlessly long. The "motor regions" were not implicated, with the exception of a small nodule on each side, but the rest of the cortical substance of the hemispheres was so thoroughly diseased that at least two-thirds of their surface was attacked.

This case, with 98–1412, to be described later, demonstrates how life may be preserved, and the animal functions carried on so long as the lower brain is intact.

Another form of sclerosis, in direct contrast with that just described, is marked by shrunken tissue, the fibrous character of its structure, is more liable to be found in localized mass than in disseminated nodules, and is apparently less destructive to the function of the parts implicated unless it has reached an advanced stage.

5-806 has already been described in a previous paper (ALIENIST AND NEUROLOGIST). A case where right hemiplegia with aphasia resulted from two blocks of sclerosis in the left ascending frontal gyrus.

47–1476.—Born 1877. Weighed only three and one-half pounds at birth. Spasms began at twenty months old. Was free from them from 1881 to 1883, when they again began and continued with intervals of about two weeks until her death. Was very slow of comprehension and her memory very defective. Vision defective, having as far as we could ascertain, hemianopsia. A large block of sclerosis was found in the right hemisphere, involving the angular gyrus and encroaching on the second occipital, supermarginal and posterior portions of the first and second temporal gyri. The right hemisphere weighed five ounces less than the left, notwithstanding the fact that she was left-handed.

82–1336.—Was born in 1874. Instrumental labor. Father emotional and silly to the verge of imbecility. Paternal aunt had puerperal mania. The child had whooping cough followed by "brain-fever" at two years of age. Left hemiplegia with epilepsy; has also violent explosions of temper. Learned to recognize simple colors and the alphabet, and could count a little. His ability to learn seemed to stop here. Sight and hearing good. Died of cerebral meningitis in 1889. The shape of the

skull is peculiar; high at the vertex and low in front, with a peculiar flatness of the right fronto-temporal region, as if the head had been subjected to pressure against a level surface. The membranes were strongly adherent over the right hemisphere, and the hemisphere itself shrunken and sclerosed in front of a line drawn parallel with, and about one and one-half inches posterior to the fissure of Rolando, including the tip of the temporal lobe. The left hemisphere weighed eighteen and one-half ounces; the right, only twelve and one-half ounces.

The same lesion was found in 75–1261, who was born in 1872. We have nothing of her early history. Had left hemiplegia; speech slow and labored, but distinct; could understand language and answer simple questions very intelligently; learned to do simple manual work but *could not* learn in school. In this case the entire right hemisphere was shrunken and atrophied.

98–1412 illustrates an extreme case of this disease. Born in 1870. Hemiplegia, with epilepsy, followed a severe burn at one year old. Spasms ceased for some years, to begin again. The father was a periodical drinker. The girl is not capable of receiving either manual or school training. Articulation fair; answers simple questions about herself rather intelligently; is liable to extreme outbreaks of temper on slight provocation; died in 1889. The skull has a depressed appearance on the right side, over the parietal lobe, with a corresponding fullness on the opposite side, as if the skull had been pushed over. The left hemisphere was large and well-formed with the exception of the convolutions forming the medial surface of the tip of the frontal lobe, which were slightly atrophied. The right hemisphere was almost entirely destroyed, a few of the gyri forming the medial surface of the occipital lobe alone retaining any resemblance to normal brain tissue. The remainder of the hemisphere seemed to consist of connective tissue containing cyst-like cavities filled with fluid.

29–1317 is an idiot, whose brain suffers from this lesion in conjunction with a peculiar lack of development of the commissural system. Born in 1876; is a mute idiot. There is no heredity acknowledged by the parents

except that his maternal grandmother died paralyzed. His infirmity is said to date from an attack of scarlet fever when six weeks old (?), accompanied by convulsions and right hemiplegia, which persisted until his death. The commissural system is very deficient. The corpus callosum is only about two-thirds its usual width, ending just in front of the pineal gland. Middle and posterior commissure wanting. There was no optic commissure, a slight protuberance only, existing on the inner side of each optic tract in the usual position of the commissure. Two blocks of sclerosis were found in the left hemisphere, one in the posterior portion, one in the motor region.

56–1146.—Age unknown, about 18 years; there was considerable improvement on entering the institution; became fairly expert in school work, but soon reached a point where further advancement was found impossible. During her later years acquired the habit of eating leaves, sticks and other rubbish unless closely watched. At the autopsy, in the right hemisphere, a small mass of sclerosed tissue included a part of the fusiform and anterior portion of the third occipital gyri. In the left hemisphere, all the convolutions posterior to the fissure of Rolando, except the extremities of the ascending parietal, a small portion of the supramarginal and the superior parietal, and those gyri lying posterior to the parieto-occipital fissure, were shrunken and destroyed. The motor area was curiously spared in the destructive process.

A condition probably closely allied to the first form of sclerosis described, if not identical with it, was found in 103–908. Born in 1867. Had chorea during early childhood; epileptic spasms began in 1876; family history unknown. When he first came to the institution he worked among the school grade of children, but insane periods became so frequent and he was so unruly, that little could be done with him. His deterioration was slow but constant. He gradually lost his power of speech, and died in 1890, of cerebral effusion with convulsion. Both hemispheres were somewhat hardened in the middle portions, notably in the Island of Reil on both sides, which were distinctly sclerosed. In the tip of the right frontal lobe, a number of cavities could be seen, pin-head in size and cylindrical in form. In other places, small

spots are seen under the microscope, in the brain substance, consisting of loose connective tissue and a few small round cells; in other places again, these spots can be resolved into a finely granular material effused into the normal brain substance, having usually a small vessel for a center. Miliary aneurism of the arterioles were occasionally found. Those small foci of degenerative changes were most plentiful in the frontal regions, especially in the vicinity of the Island of Reil on both sides.

99–845.—Came to us without history. Profound mute and knows little except to scream and eat. Is not paralyzed or epileptic, but moves very slowly. The brain, which weighed thirty-five ounces, was seemingly perfect in structure, but unusually firm in consistence. Microscopic examination shows, in the frontal lobe, effusion of small round cells beneath the pia-mater, abundance of the same small cell-elements in the first or non-cellular layers of the cortex, and throughout the cortical substance of this region, a great number of the same elements, with few, if any true ganglionic cells. As we pass backward, the cellular elements approach more nearly the normal type, but everywhere the connective tissue cells are more plentiful than in the normal brain.

I have dwelt at considerable length on these various forms of sclerotic changes, because sclerosis is a comparatively rare disease, to show to what extent it may attack the brain without destroying life, and the improbability of great or permanent improvement, where any form of this disease exists.

It is interesting to note, that in this lesion, as in localized atrophy, the frontal and occipital regions suffer more frequently than other parts of the brain. This may be due to the blood supply being less direct, or that these portions of the cerebrum are more recently developed than the middle portions; and hence a greater liability to degenerative changes exists.

General cerebral atrophy was found in only two cases, both demi-microcephalic. The symptoms were general decadence of mental and physical powers until death seemed to come from general inanition. The brain in both cases was shrunken, considerably smaller than the

skull; the gyri smaller than usual and the sulci wider and more open.

97–1663 (colored), age unknown, probably about 14 years. At 3 years old had several spasms. Sight and hearing defective. Nutrition so low that it is difficult to keep his fingers and toes from ulcerating from even moderate exposure to the cold. Inproved in school, though but slowly. Over the first and second left frontal convolutions, the vessels of the membranes are greatly increased, both in number and size, while the cortex below is shrunken and atrophied. The same condition to a considerably less extent was found over the right angular gyrus. A tumor (glioma) occupies nearly the whole of the left occipital lobe, over which the membranes are strongly adherent, both to the surface of the brain and to the skull.

Localized atrophy is usually associated with sclerosis, and requires no lengthy description. In my second paper is an account of a case when atrophy of the frontal lobe resulted from pressure of plates of bone in the dura-mater. In two microcephalic brains it was found at the extremities of both hemispheres, resulting perhaps from pressure of the small skull. When not associated with sclerosis it is as soft, or nearly as soft in consistence as the normal brain tissue, the gyri are not so large as the surrounding convolutions, but do not exhibit the multiplied foldings seen in undeveloped brain.

Non-development is found in several forms. A portion of the cortical substance may be thin, and instead of following the typical arrangement of the fully developed brain, form a number of irregular folds, which may be so small and numerous as to resemble a mass of angle worms. Again, the gyri may be of a normal size and appearance, but show a diminution in number, or absence of ganglionic cells in certain layers. Or, again, the convolutions may begin of normal size and development, but soon diminish and sink beneath the surface of the hemisphere, or perhaps entirely disappear, before acquiring near their normal length.

59–1070, brought us no history. Was probably about 20 years old. Nearly mute. Partially deaf. Was always very expert in using his hands, but it was very difficult for him to learn anything in school aside from manual exercises. Anything relating to figures especially, being entirely beyond his comprehension. He was excitable and insane for some years after puberty. Speech indistinct and vocabulary limited. Brain very large and well formed, showing no deficiency except Broca's convolution, which was very small. In the first temporal gyrus of the left side some degenerative change was found in the white matter. On examining sections from the tip of the right frontal lobe, a light band was seen running through the middle of the gray matter in the stained section. By microscopic examination, the middle layers were found to be almost destitute of true ganglionic cells; such cells as were present being attenuated and poorly formed, while the neuroglia shows a preponderance of connective tissue elements. Vessels normal.

96–1381.—Microcephalic, brain weighing but twenty-eight ounces. Mute. Had but little intelligence. Probably defective hearing. No nervous disease acknowledged in family history. Some spasms during dentition. The arrangement of the convolutions are of the simplest type, and some of them very imperfect in development. The first temporal begins of normal size, but instead of running along the upper border of the temporal lobe, sinks almost immediately into the fissure of Sylvius and disappears. The third frontal on each side is also poorly developed in its posterior half.

88–892.—Born in 1866. Was very ill at eight months old. Profound mute; nearly deaf; but little improvement under training; learned with difficulty plain sewing and sweeping. Skull well formed except small in the frontal region and distinctly hypertrophied. On section, some portions of the brain were found lighter in color than normal, notably the first left temporal gyrus, which is of a pale pink instead of the gray-red of other parts of the cortical substance. Microscopical examination shows an almost complete absence of true cortical cells, with abundance of small granule cells in this region.

In 34–1050, who was completely paralyzed in both legs, partially paralyzed in both arms, profound mute and almost devoid of intelligence; was found an exaggerated

form of this condition. The whole of the anterior and middle portions of the cerebrum exhibited this appearance, the posterior portions appearing to the eye nearly or quite normal. Previous injection with zinc chloride made a microscopic examination impossible.

Of the variety of non-development, where considerable areas fail to attain their normal growth, but form an atypical group of small imperfect convolutions, good examples have been given in former papers.

Hydrocephalus.—The clinical history and pathological features of ordinary hydrocephalus are too well known to require detailed description here, with the exception of one case which presents somewhat unusual features.

53–997 was a dwarf, less than four feet tall; supposed to be about twenty years old. Had excited considerable attention by his memory and ability to recite passages from standard authors and short Latin quotations. His actual knowledge, nevertheless, was very limited. A large quantity of fluid distended the ventricles and lay between the skull and brain. In the ventricular cavity was found a large cyst containing approximately six ounces of fluid. The walls of this cyst were evidently formed by the parts originally forming the roof of the third ventricle, all these parts being wanting except the corpus callosum, which was stretched over the top of the cyst. It had no connection with the lateral ventricles.

Internal hydrocephalus, with its destructive effects, have been so exhaustively described by the late Dr. H. M. Knight, in a paper before this Association, in 1879, that I can add nothing to what he has written. We found but one case where this condition existed, without compensatory distension of the skull, with most destructive effect on the entire cerebral function.

Microcephalic brains show the result of two entirely different conditions; when premature closure of the sutures checks the active growth of the contained brain, and when the skull seems to stop its growth simply because the brain, from some cause, has ceased its growth, and the skull no longer receives the stimulus of the growing

organ within. This same effect is seen to a limited extent in cases of hemiplegia, where a portion of the brain has been destroyed by infantile disease, the portion of the skull over the affected tissue showing a marked flattening.

The first variety is well shown in 35–1121, a mute idiot, paralyzed in both lower extremities; born in 1880. There is a strong tendency to phthisis in the mother's family, where of twelve first cousins, ten have died of this disease. There is no history of nervous disease except that the maternal grandmother was "very nervous" at times. On opening the skull (which was very thin) the brain was found so firmly compressed in the skull case that the fissures were seen only as narrow lines, the convolutions being pressed together so firmly as to obliterate the usual depression marking the sulci.

Illustrating the effect of extensive hemorrhage in infancy, which failed to cause death, we have 26–576. When seven months old was dropped by her nurse, striking on her head. The fontanelle bulged immediately. Convulsions followed and never altogether ceased until her death several years after. She was an excitable idiot, mute, and walked on the ball of her foot; was fond of lying on her side with her knees drawn up, with her hands slowly swinging past her eyes; spasms of hystero-epileptoid character; very sensitive to changes of disorder or change of place, and exceedingly irritable. At the autopsy was found much thickening of the frontal bones; strong adhesion of the membranes to the skull and to the brain; thickening of the membranes over the right parietal lobe. The principal lesion was in the frontal lobes, more particularly in the left, where the cortical substance was shrunken and discolored. Microscopic examination demonstrated this cortical substance replaced by firm connective tissue, with blood crystals thickly scattered through the mass. The hemorrhage from the old injury had completely destroyed the normal structure and then by its absorption, left this cicatricial mass.

94–915 is one of a class, not less interesting, but much less satisfactory to the pathologist than those previously described. Born in 1867. Was very ill for three weeks after birth. Father and mother both of nervous temper-

ament. Considerable insanity and some epilepsy among father's relations. Was vaccinated at six months old, spasms beginning four days after that event. From that time until his death, in 1889, his course has been steadily downward. Speech very imperfect; sight, hearing and general sensation defective; walk and prehension faulty; in fact, a general decline of all powers, physical and mental. The brain was of good size, well formed, and no change, macroscopic or microscopic, was found. The only explanation seemed to be that the gradual destruction of functional activity was due to trophic changes, induced perhaps by disordered circulation, the result of frequent severe spasm.

Five cases of "Mongolian idiocy" are included. In all these the brains are of good size for imbecile brains, the pons and medulla alone being very small, weighing in each case about one-half ounce, whereas the usual weight is nearly twice as much. The cerebral vessels are inclined to be much thinner than in healthy brains. In some places, evidences of old arteritis were discovered. In one instance the vessels showed a tendency to form angiomatous groups, four or five different vessels in a bunch, having somewhat the appearance of erectile tissue. The defective nutrition and circulation of these children lead one to suspect that the defective condition of the vessels may be a general condition. From the small size of the pons and medulla in every instance, there seems to be a strong probability that the low nutrition, and possibly the other anatomical peculiarities of this group, may be due to the imperfect development or absence of certain cell-groups in this region.

There are also included three cases of pseudo hypertrophic muscular paralysis, described at length by Drs. Kerlin and Mills in the "Transactions of the American Medical Association" of 1880. A detailed description of one case is given in our "Proceedings" of 1886, where degenerative changes were found not only in the cord, but in the brain itself. In the other two brothers, who have died since, the examination of the cord and brain

gave negative results, though the cords were examined carefully throughout their length. The cause of the atrophic change seems inherent in the muscle, and the lesions found in the first case examined, were probably from some intercurrent affection. In all these cases the bones were found unusually dense and hard, and in two, hypertrophy of the skull existed. The change in the muscle seemed to be a swelling of the fibers, followed by atrophy, the fibers retaining, for the most part, their transverse striation until a very advanced stage of atrophy was reached, the place of the fibers being taken, first, by fat cells, afterward, by connective tissue.

In seventy five cases, or in all where injection of zinc chloride, or extensive destruction of the brain substance made weighing valueless, the brain was carefully weighed. The average weight was 38 3 ounces. In fourteen cases, the weight was below 30 ounces. Thickening of the skull to an extent to constitute hypertrophy was found in eight instances, while in eight, the skull was usually thin, not including cases where there was distention from hydrocephalus.

The 100 cases may be summarized as follows: Sclerosis with atrophy, 12; sclerose tuberense, 6; diffuse sclerotic change, 7; degenerative changes in vessels, ganglionic cells or medullary substance, not constituting true sclerosis, 15; hydrocephalic, 5; general cerebral atrophy, 2; non-development in various forms, 16; infantile hemorrhage, 1; extensive adhesion of membranes from old meningitis, 3; angiomatous condition of cerebral vessels (with degenerative changes), 1; glioma (with sclerosis), 1; porencephalous (with non-development), 1; of thirty-one cases where actual disease or imperfect development of the brain proper was not demonstrated, there was hypertrophy of the skull, 6; acute softening (recent), 2; demi-microcephalic, 2; when the brain was above usual weight, but the convolutions large and very simple in their arrangement, 2.

In closing, I would briefly call attention to the com-

paratively large number of cases of actual cerebral disease, in contrast with the relatively small number where imperfect development seems the causative agent of the mental defect. With this fact, we see a corresponding improbability of a large majority of these cases ever attaining a full mental development. That a large majority of them can be greatly improved, we have daily evidence in our schools. Children inheriting inactive brain from parents below the usual average of intelligence, without history of infantile disease or epilepsy, seem the most promising of general and permanent improvement. The brain diseases of infancy, on the other hand, are peculiarly destructive, and not only leave a permanent injury to the brain, preventing its growth and development, but are liable, from the irritation they leave, to kindle the epileptic habit with its destructive effects. Realizing these facts, the physician should be more cautious in giving the customary diagnosis of "Arrested development that special training will remove," thereby arousing hopes in the minds of the parents that can only result in disappointment.

In closing, I desire to acknowledge my indebtedness to Dr. I. N. Kerlin, not only for the clinical and school histories of the cases here described, but also for many valuable hints in conducting this work.