

there is any failure in the general health or recurrence of the neurasthenia.

In one of the cases which I have reported the rumination ceased when the patient's general health had improved and did not return as long as he was under observation, although the attacks of migraine, for which he had originally come for treatment, persisted. There are practically no therapeutic measures of use in these cases, with the exception possibly of nerve sedatives and tonics. If there is gastric indigestion this must be corrected. Lavage is an important procedure in the management of cases of rumination dependent upon indigestion. The diet should be carefully regulated, and what is of great importance, the amount of liquid taken with a meal should be reduced to a minimum. Thorough and complete mastication of the food is also an essential matter in this disease. The patient should be trained to control the inclination to regurgitation of the food. Hypnotic suggestion will probably prove useful in the management of such cases, although I have seen no record of any treatment by this measure. Hammond stated that the patient whose case he reported before the American Neurological Association was mentally defective, and with a view of relieving the psychical condition he trephined the skull, first in one parietal region and then in the other. The merycism ceased after the first operation and had not returned at the time that the case was reported. In this instance, no doubt, the checking of rumination was due to suggestion.

#### DISCUSSION.

Dr. JAMES HENDRIE LLOYD—This would seem to be a neurotic state allied to the condition of hysteric vomiting. I do not think in hysteric vomiting it is common to have true rumination, and there is constant attempt at retching—probably more a condition of esophagismus, a tendency to regurgitate through the esophagus. In an idiotic child that came under my observation there was regurgitation from the mouth, the food being disgorged, reintroduced into the mouth and eaten over again. The procedure resorted to in the case reported by Dr. Hammond seems hardly worthy of being followed. The treatment is by suggestion, and we can get ideas into people's skulls without making holes to put them in!

Dr. CHARLES H. HUGHES—I think the better treatment is by digestion rather than suggestion. I have been accustomed to associate the majority of these cases with conditions of dyspepsia in persons of neuropathic diathesis. I have seen these cases in neurasthenia. When I first read Trousseau on "Apepsia Nervosa," and subsequently gained a great deal of experience with nervous dyspeptics, I found that there were many of these conditions in which regurgitation took place and in some instances the food was rechewed.

Dr. SINKLER—I think the point is that rumination is not a condition of indigestion; the food when returned is sweet and pleasant. As soon as it becomes fit for intestinal absorption it passes out of the stomach and ceases to be returned to the mouth. Dr. Lloyd's remarks remind me that several cases of rumination have been reported in idiots. There is one suggestion that can be made in regard to this habit, that it is a form of digestive perversion like sexual perversion. I think a lack of thorough mastication is probably one of the primary causes.

### A CLINICAL AND PATHOLOGIC REPORT OF A CASE OF PROGRESSIVE DEMENTIA.<sup>1</sup>

Presented in the Section on Neurology and Medical Jurisprudence, at the Forty-eighth Annual Meeting of the American Medical Association held at Philadelphia, June 1-4, 1897.

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The case under consideration presents the following history:

Patient aged 64 years; married; has had five chil-

dren, three of whom are living and in good health. She was always of a nervous, somewhat irritable, temperament, but mentally bright and clever, with linguistic and other accomplishments. After the birth of her first child she had an attack of mania; when about 23 years of age she had an attack of chorea which lasted several weeks. At 35 years of age, apparently as the result of unusual worry owing to sickness, she became more irritable and her temper was afterward capricious. For about ten years previous to her death she was subject to spells of excitement which almost amounted to transient derangement, but she had no tangible delusions, although she had a tendency to persecutory ideas, frequently believing without cause that she was abused and ill-treated by others. During the same period she began to show a decided amnesia for names, this gradually but surely increasing, so that for several years before her death it was almost impossible for her to recall the names of acquaintances, and occasionally of objects with which she was familiar. She had, however, no motor aphasia and could converse and write well, having an unusual facility for letter writing until within three years of her death.

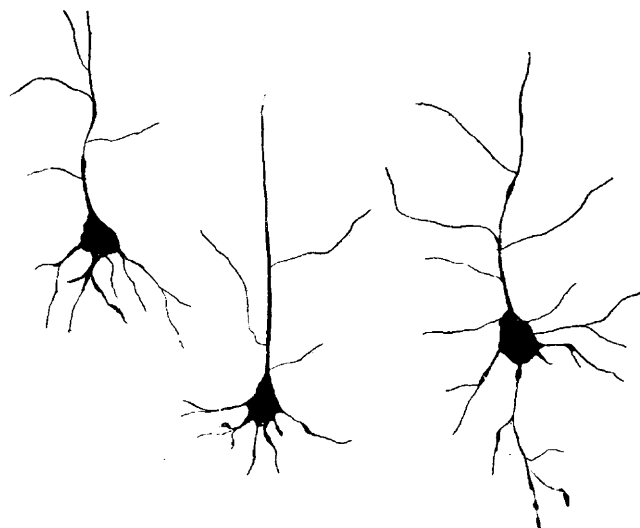


FIG. 1.—Long pyramidal cells, showing moniliform swellings of basilar and apical dendrites. Region of second frontal convolution.

Up to this time she had been less vigorous, but always attended to her business affairs, kept house and performed what other duties she had to do. During this third year previous to death she became so unreasonable that it was impossible to live peaceably with her, she having at times outbursts of uncontrollable passion. General failure of memory was first noticed about two years before death, during which time she became half bedridden.

In January, 1895, she had what appeared to be an attack of grippe; there was rise of temperature and she complained of intense pain in the right side of the head, also of pain in the back, extending down the legs in the course of the sciatic nerve. This attack lasted about a week and she apparently regained her usual health. In March of the same year she had a second attack, which was also accompanied by intense backache and pain in the head, the latter pain being persistent and always referred to the right parietal region. The patient did not improve as she

<sup>1</sup> An abstract of a portion of this paper, with other illustrations, has been published as a "Preliminary Report," in the Proceedings of the American Medico-Psychological Association for 1897.

had done after the previous attack, except that the lumbar pain diminished. There was marked insomnia.

After two or three weeks she began to have marked delusions, which related to her surroundings and also to her children. She believed that she had been taken away from home and was living in a strange house. She spoke of having visits from her father and mother, both of whom had been dead for many years. Sleep was only obtained by the use of hypnotics.

In April, 1895, a consultation was held by Drs. Sinkler and Mills. At this time the patient had marked delusions, talked volubly and at random; was unable to walk without assistance and if she attempted to do so staggered, always pitching to one side.

During the last eighteen months of her life she was confined to bed continuously. Her chief symptoms during this time were vertiginous attacks; difficulty in orientating; marked amnesia, not only for names but for recent events. She gradually became feebler mentally and a few months before her death was in a state of decided dementia, with occasional periods of excitement. Her attempts at conversation were childish and incoherent; she had numerous unsystematized delusions; she failed to recognize her children and attendants and lost memory of everything.



FIG. 2.—Long pyramidal cell from the second frontal convolution, showing roughening and swelling of the apical dendrite; deformity and loss of basilar dendrites; absence of gemmule.

An ophthalmoscopic examination made in April, 1895, and again in the early part of 1896, revealed no change in the fundus. One pupil was dilated. There were no bed sores, and no paralysis at any time.

November 5, 1896, she became suddenly comatose and died the next day. The case was one in which the entire fabric of the mind seemed gradually to break up, and step by step with failure of physical powers mental failure progressed.

The postmortem examination made by Drs. Burr and Kelly, November 7, revealed the following pathologic conditions: The dura was somewhat thickened; the pia-arachnoid opaque. The internal carotid, posterior communicans and basilar arteries were atheromatous; the left anterior communicans showed aneurysmal dilatation. Miliary aneurysms of the pial

vessels were present on the ventral surface of the pons. Portions from six regions of the cortex were hardened in alcohol, the remainder of the brain was placed in Müller's fluid.

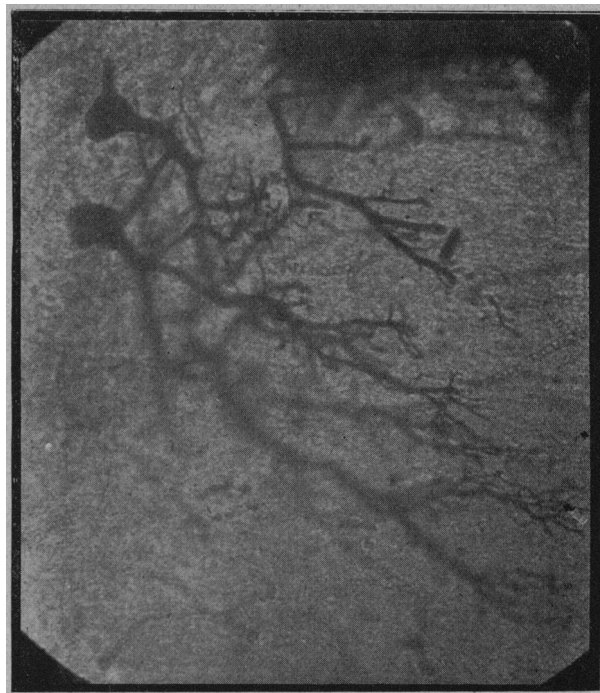


FIG. 3.—Purkinje cells of cerebellum, showing atrophy of the dendrites; stumpy branches in place of feathery dendrites.

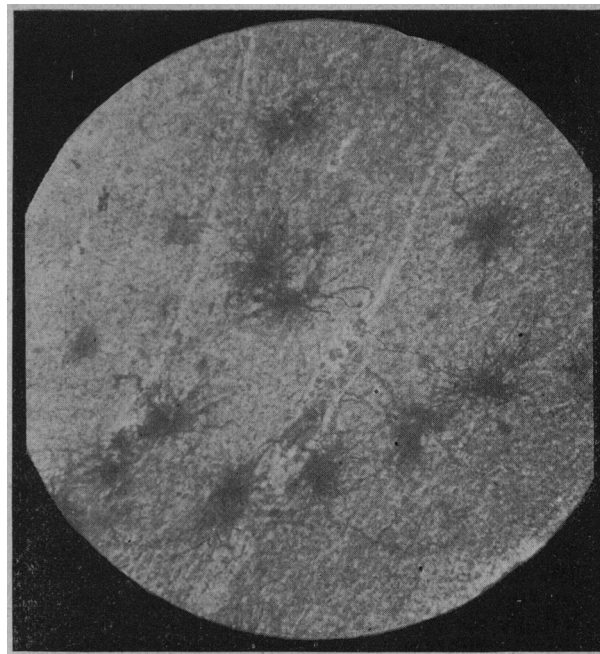


FIG. 4.—Protoplasmic glia cells from middle occipital region; some presenting a botryoidal appearance, others showing stages of disintegration.

The methods employed in the microscopic examination of the brain were the following: The silver phospho-molybdate method of Berkley; Nissl's methylene blue; thionin according to Lenhossek; Weigert Pal; eosin and hematoxylin; also hematoxylin, picric acid and fuchsin. (Van Gieson.)

Microscopic examination reveals the following pathologic conditions:

The neuron shows internal changes which consist in deeper staining of the cell body; the chromophilic particles are irregularly arranged; absent from some areas and aggregated in others, giving the protoplasm a vacuolated appearance. In most cases the chromophilic particles are arranged as a layer of fine dust around the nucleus, next there is a surrounding clear space and beyond this a layer, either continuous or interrupted, of chromophilic particles along the cell wall. The chromophilic particles of the cell processes are either wanting or sparsely scattered throughout. The nucleus is regular in contour; the larger chromophilic particles are absent, while the finer dust-like particles and normally clear karyoplasm stain irregularly. The nucleolus is somewhat increased in size and shows a markedly increased receptivity to stain.

External changes in the neuron are shown in roughening deformity and in some cases excavation of the cell-corpus. The basilar dendrites show moniliform swellings along their course, or are shortened, with

are areas of atheromatous degeneration. The elastic lamina is intact. Both vessels contain thrombi.

The left posterior communicans is twice the diameter of the right. In both, the intima, media and adventitia are equally thickened, and thrombi are present.

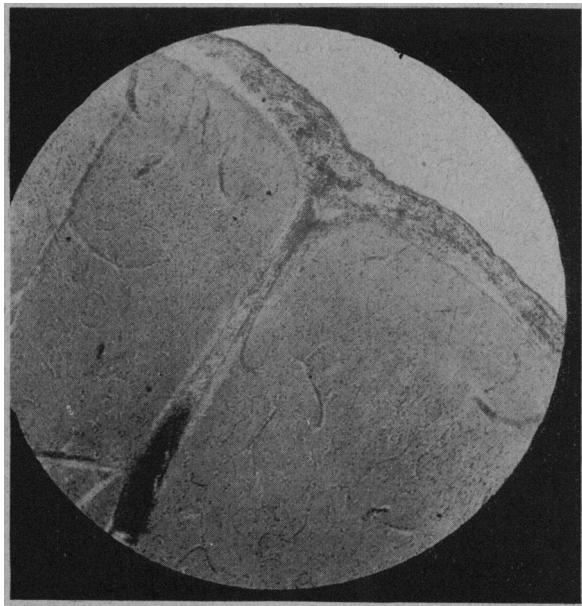


FIG. 5.—Section in region of second temporal convolution, showing thickening of the pia-arachnoid, stasis and over-distension of cortical vessels.

clubbed extremities, or some of the dendrites are absent. The apical dendrite is roughened and irregular in contour. The fine collaterals and terminals are wrinkled and irregular in contour. Gemmulæ are absent in the regions of moniliform swellings of the dendrites and at other points. The long pyramidal cells seem to be most affected, although similar changes do occur in the fusiform and polymorphic cells they are not so marked. These changes of the neuron are not limited to any special region of the cortex. The Purkinje cells of the cerebellum show thickened stems with short stumpy branches in place of the feathery dendrites of the normal cell.

The basilar and internal carotid arteries show an increase in the number of endothelial cells and a growth of new connective tissue derived from the endothelium. This growth consists of branching cells, proliferated nuclei and basement substance, the latter being very dense. Scattered through this new growth



FIG. 6.—Section of the right optic nerve, showing similar changes to those found in the cortical vessels.

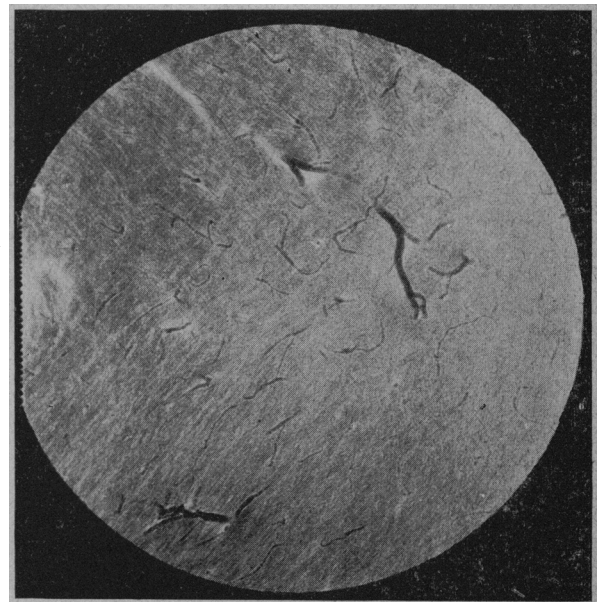


FIG. 7.—Section of optic chiasm; similar vascular changes.

The anterior communicans exhibits aneurysmal dilation; the endothelium is absent; there is proliferation of adventitial elements with increase in basement substance, the latter being so marked as to give the whole vessel wall a hyaline appearance. There are areas of atheromatous degeneration in this increased basement substance; the elastic lamina is absent. The vessel wall opposite to this thickened area shows

absence of the media and thinning of the adventitia. The vessel contains recent and partially organized thrombi.

The pia is increased in thickness shows nuclear proliferation and evidences of extravasated blood in the form of groups of corpuscles and hematoidin crystals. The pial vessels show thickening of their walls and stasis; they are markedly tortuous and present aneurysmal dilatations in their course. Miliary aneurysms found on the ventral surface of the pons have the appearance of grape-like clusters. On section the walls of these vessels show nuclear proliferation of the protoplasmic cells of the adventitia, also extravasation of blood corpuscles and pigment into the spaces between individual vessels. Stasis and over-distension is marked.

The cortical vessels of all sizes are over-distended with blood corpuscles; they are exceedingly irregular and tortuous in their course (many being twisted several times upon themselves); they appear much more numerous than normal. The perivascular lymph-

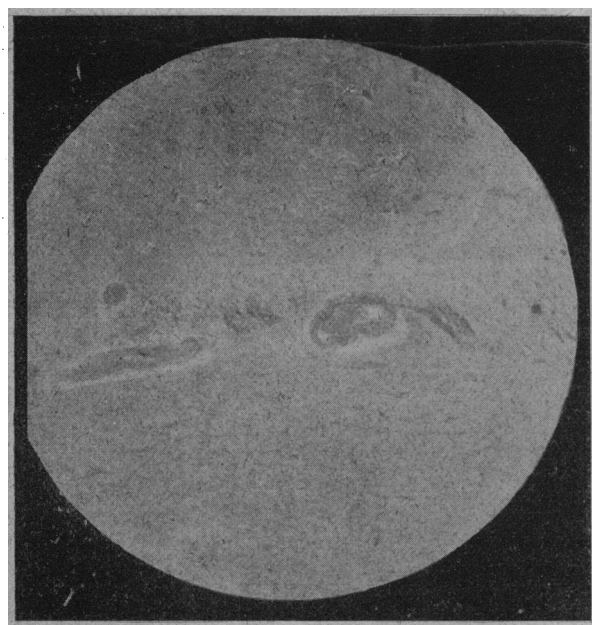


FIG. 8.—Transverse section of vessels of anterior perforated space, showing stasis, thickening of their walls and distension of the perivascular lymphatic space.

phatics are distended, showing wide spaces between the vessel sheath and the brain substance. The pericellular lymph spaces are also enlarged.

The protoplasmic glia cells present a series of transitional changes; their fine mossy granulation appearance is lost; the pseudopodia show varicose swellings in their course, thus far they retain their vascular attachment. Many cells show an irregular botryoidal appearance and loss of vascular attachment. Finally, evidences of disintegration of cells is to be observed. Deiter's cells are numerous both in cerebrum and cerebellum.

Areas of softening occur in right ascending parietal region, they consist of a reticulated stroma surrounding a central cavity which contains portions of blood vessels; groups of blood corpuscles, hematoidin crystals and fragments of nerve and neuroglia tissue. Areas of coagulation necrosis are present in the left ascending parietal region.

Medullated fibers show different stages of myelin

degeneration; these appearances are demonstrable in the ascending parietal region, the optic chiasm and in irregularly scattered areas of the pons and medulla.

The pathologic conditions occurring in this case of progressive dementia may be summarized as follows: 1, internal and external changes of the neuron; 2, changes involving the cortical and pial vessels, also the vessels of the base of the brain; 3, changes in the protoplasmic glia cells; 4, multiple areas of softening in the ascending parietal region; 5, myelin degeneration.

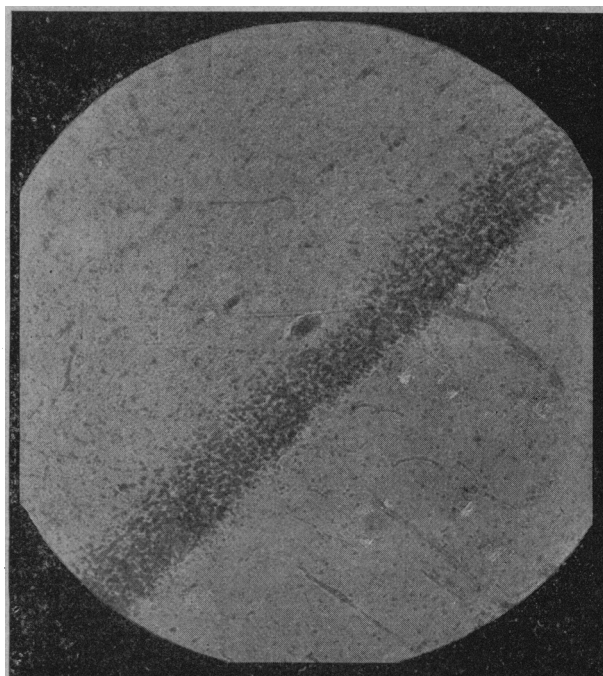


FIG. 9.—Section of cerebellum, showing over-distension and multiplication of vessels.

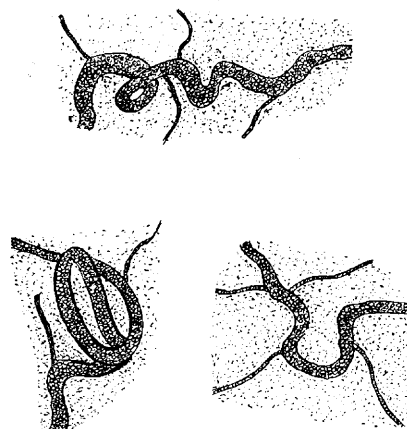


FIG. 10.—Tortuous vessels from the ascending parietal region.

The above described changes in the neuron correspond to those found by Berkley in experimentation upon alcohol poisoning, and in examination of cases of alcoholic dementia. They also correspond to the changes described by Andriezen as occurring in alcoholic dementia and other insanities. A survey of the views of these authors upon the pathology of insanity is therefore in order.

Berkley considers that conduction of the nerve stimuli to the cell corpus takes place principally through the medium of the gemmulæ, and that these are the



first division of the neuron to atrophy. If the gemulae lose their vitality or become diseased conduction of impulses is impossible. Inco-ordination of thought and motion would then result from loss of direction as well as from want of originative impulses. Permanent dementia would be the result of the degenerative process involving a large number of cortical neurons.

flow of ideas followed by fatigue and drowsiness, and this in turn by a sub-conscious condition in which there is a continuous and distressing whirl of nerve currents through the brain." The repetition of such attacks causes permanent damage to the nerve cell. Delusions of suspicion and maniacal outbursts are explained by him as the result of destruction of the neuro-protoplasmic plexus of the molecular and ambiguous layers.

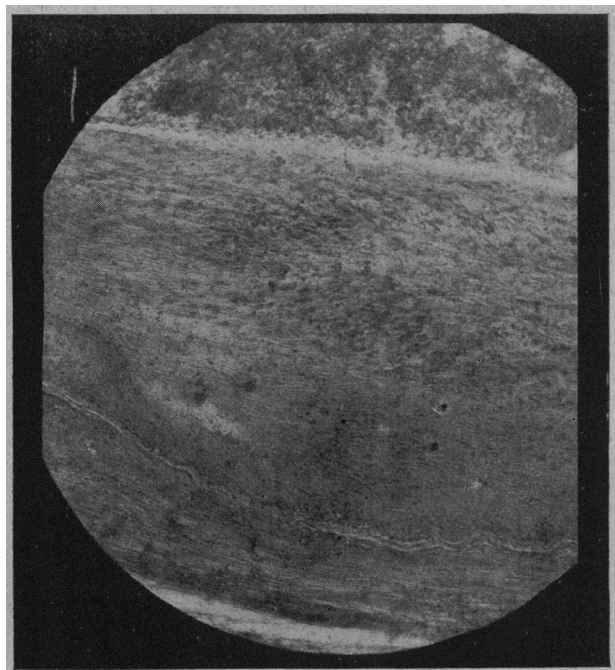


FIG. 11.—Section of atheromatous portion of the basilar artery.

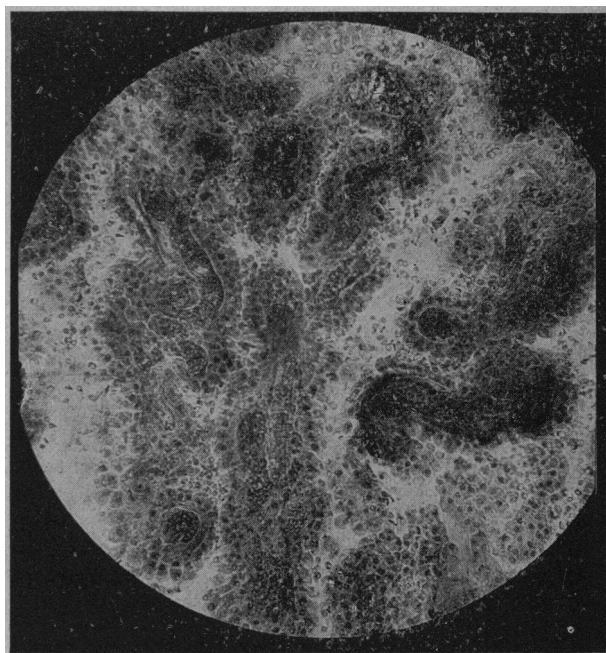


FIG. 13.—Higher magnification of the preceding.

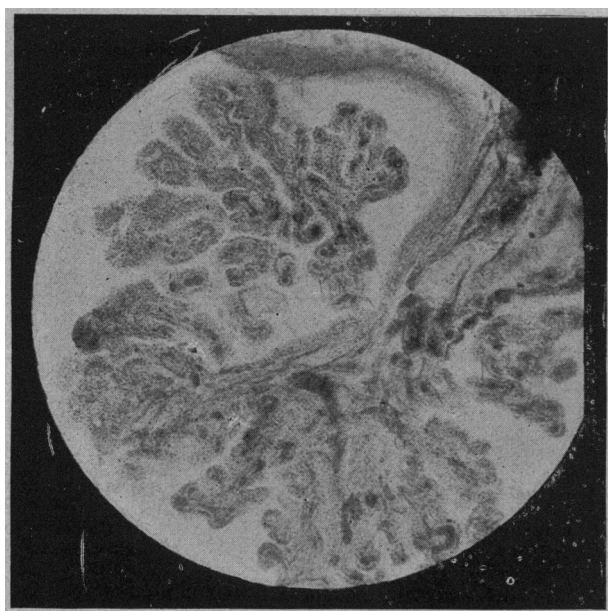


FIG. 12.—Miliary aneurysms, pial vessels ventral surface of the pons.

According to Andriezen the series of early changes in dementia illustrated by insomnia, amnesia, incapacity for attention and mental exertion and fatigue can be explained pathologically by progressive nutritive and dynamic changes. The nerve cell of the dement is stimulated beyond its average limits and at a greater pace than it would be in health. As he describes it, "The result is quickness, rapidity and over-

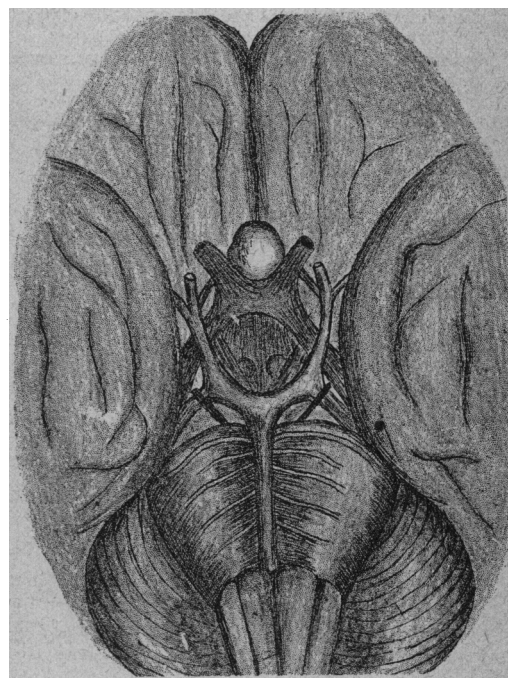


FIG. 14.—Aneurysm of anterior communicating artery.

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#### DISCUSSION.

Dr. CHARLES K. MILLS—The lady was a patient in private

practice. There was no possibility of either alcoholism, syphilis, or any of the common exciting or predisposing causes of insanity of this kind being present, so it furnishes us with a non-specific and non-alcoholic case. This case is interesting in connection with the entire question of neurasthenia, and also in relation to the pathology of melancholia and non-specific dementia. The paper bears out the views advanced by Dr. Dercum, which we endeavored to place upon a firm basis of the pathology of neurasthenia, and bears out the views as perhaps best elaborated in the remarkable series of papers which were eventually put into a monograph by Dr. Cowles of Boston, on "Neurasthenia and the Development of Insanity." Doubtless this patient had some inherent tendency to mental disorder, but that does not alter the question as to the bearing of these investigations upon the pathology of the subject.

Dr. F. X. DERCUM—My own conception of the pathology of insanity, presented some years ago, is about as follows: It embraced the changes more especially in the neurons, but especially did I lay stress on the possible toxic causes of insanity. There are a great many facts to prove that we have toxins circulating in the blood; certainly the toxicity of the urine is very pronounced in some forms of insanity, while it is diminished in others. It is diminished, for instance, in melancholia, while the toxicity of the blood is increased in mania.

It seems that these changes in the nerves and blood vessels are only referable to some irritant, something that necroses the collaterals, the dendrites, and that destroys the chemic constitution of the protoplasm in the nerve cell.

Here we have changes taking place in a non-specific and non-alcoholic case, and yet, to my mind, there must have been some substance in the blood which acted in this destructive manner upon these delicate structures in the brain and in the nerve centers. When we have a case that appears to be auto-toxic the general autopsy is of the utmost importance. In this case the most elaborate studies would have been repaid, including the study of the viscera as a whole, and of the bone marrow. Even studies of the blood in the dead subject might have been repaid. We see here terminal changes, resulting changes; the primary cause is still to be sought after. In the various laboratories of the State hospitals for the insane a large amount of work should be done, not only upon the dead brain, but upon the living individuals, and it should be largely experimental in character. We are still far behind in our knowledge of the chemistry of the urine, and we have to adopt coarse physiologic methods to determine the presence of toxic bodies there. Especially should there be experiments as to the changes which arise in the nerve cells of guinea pigs to see whether they are similar to the changes which occur in the human. In the same way we ought to experiment with the serum of the blood of the insane.

### ALCOHOL AS A CAUSATIVE FACTOR IN DISEASES OF THE CENTRAL NERVOUS SYSTEM.

Presented to the Section on Neurology and Medical Jurisprudence, at the Forty-eighth Annual Meeting of the American Medical Association held at Philadelphia, Pa., June 1-4, 1897.

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Next to syphilis, alcohol is the most frequent cause of diseases of the brain. In many instances it is more virulent, rapid and pronounced in its effects and is now fully recognized as a poison of the narcotic class.

Its general action on the system is seen in irritation, deranged functional activities, diminished force and narcosis. Some instances show marked narcosis from the beginning. The first effect of alcohol is to produce functional disturbance, and such poisons are called narcotic and neurotic, because their most conspicuous action is on the nervous system. Apparently this effect is transient, and the injury is supposed to be indicated by the repetition of the functional disturbances. A fatal dose acts like other tissue poisons, producing narcosis and necrosis. Alcohol while being carried to all parts of the body acts, like other poisons, on those parts which it reaches with the least amount of dilution, particularly the stomach and liver. Unlike

other poisons, it seems to have a special affinity for the brain and nervous system. The vascular disturbance and consequent nutritional derangement are most prominent. The effects of alcohol in what are called chronic states, may vary widely in persons who are termed steady drinkers, and others who are not supposed to be injured by alcohol, up to those who are notoriously degenerated from its use, may be grouped as follows:

1. General temporary exaltation of the brain and emotional activities, followed by depression and feebleness, loss of memory and change of character and conduct.

2. General diminished power of attention and volition, particularly in the sensorial and kinesthetic centers and their connections.

3. Diminished energy and adaptability of conduct to the conditions of life.

4. Diminished muscular power and demand for help or stimuli to do the ordinary work of the system.

5. From the first a marked blunting and deepening obscurity of the moral and ethical sense.

6. Insomnia, diminished power of sleep and recuperation, with a nutritive break down of the stomach and brain.

7. The relations of the *ego* to the external world become disturbed followed by melancholy, suspicion, delusions, hallucinations and changed emotional states all merging into various chronic insanities.

These groups of symptoms are seen in all cases, not in a continuous order, but varying widely. Sometimes one is much more prominent than the other, but in all cases the psychical disturbances are present. The diminished sensory functions and the lowering and obscuring of the higher ethical sense of duty and the relation to the surroundings indicate a profound nutritive and dynamic failure of the nerve elements of the brain. While these are the well-marked symptoms of all cases who use alcohol excessively they are by no means absent in those who are supposed to use it in moderation. There are no dividing lines on one side of which the poisonous action of alcohol can be seen, while on the other it is absent. The fact is that alcohol, like other poisons, varies in its effects on different persons, producing in certain organs more prominent effects than in others; in certain cases concealed for a long time, then bursting out, revealing a degree of degeneration unsuspected; concentrating on the liver, stomach, kidneys or heart, or suddenly developing into serious lesions of the higher brain centers.

To understand more clearly the influence of alcohol in the causation of disease of the central nervous system, a study of some central facts of the dynamics of brain energy and force will be useful. All nerve force and brain activity is physical energy in motion. This energy is gathered and released and is technically only a variety of motion the same as is electricity. It is transmitted motion, but not electricity, although the latter is transmitted in the same way. It comes from nutrition, from chemic and molecular changes and is stored up and released by certain activities. It may be quickened, retarded, changed, increased or withdrawn.

It is the transmission of this energy along the nerve tracts to all parts of the body that constitutes life, and with all its attractions and repulsions and the continual readjustments of the delicate equilibrium necessary to sustain the work of life this is carried on with exact and absolute precision. Nothing in the