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Preliminary Report, Clinical and Pathological, of a Case of Progressive Dementia.

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PRELIMINARY REPORT, CLINICAL AND PATHOLOGICAL, OF A CASE OF PROGRESSIVE DEMENTIA.*

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The case presented the following history: Patient—aged sixty-four years; married; 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 twenty-three years of age she had chorea which lasted for several weeks. When thirty-five years of age, apparently as the result of unusual worriment, she became more irritable and her temper was afterwards capricious. For a period of ten years preceding 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. About the same time, she began to show a decided amnesia for names; this gradually, but surely, increased. She had, however, no motor aphasia but could converse and write well until within three years of her death. During the third year previous to her death, she became so unreasonable that it was almost impossible to live peaceably with her, she having at times outbursts of uncontrollable passion.

During the second year previous to death she had two attacks of what appeared to resemble la grippe; during which time she complained of intense pain in the head and back; from the first of these attacks she recovered, but from the second she did not, the pain in the head being persistent and always referred to the right parietal region; insomnia was marked. General failure of memory was observed about this time and she became half bedridden. During the last eighteen months of her life she was

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confined to bed continuously. Her chief symptoms during this period were vertiginous attacks; difficulty in orientating herself, and marked amnesia not only for names but for recent events. She gradually became feebler mentally and during the few months preceding death was in a state of decided dementia with occasional spells of excitement. Her attempts at conversation were childish and she had numerous transient, unsystematized delusions. An ophthalmoscopic examination made one year previous to death was negative. There were no bed sores and no paralysis at any time. The case was one in which the entire fabric of the mind seemed gradually to break up and step by step failure of the mental and physical powers progressed. On November 5, 1896, she suddenly became comatose and died the next day.

The post-mortem examination was made on November 7th by Dr. Burr and Dr. Kelly. The following pathological conditions were present: The dura was somewhat thickened; the pia-arachnoid was opaque. The vessels of the base of the brain were atheromatous; the anterior communicating artery showed aneurismal dilatation. The pial vessels on the ventral surface of the pons showed miliary aneurisms. Portions from six regions of the cortex were removed and hardened in alcohol; the remainder of the brain was placed in Müller's fluid.

Microscopical examination reveals the following pathological changes: Sections stained with thionin (according to the method of Lenhossek) and by methylene-blue (Nissl's method) show internal changes in the neuron. These changes consist in irregular arrangement of the chromophilic particles of the cell-body—disappearance of these from some areas and aggregations in others—giving the cell-body a vacuolated appearance. The chromophilic particles are absent from or sparsely scattered through the cell processes. The nuclear changes consist in absence of large chromophilic particles, while the finer dustlike particles and the normally clear karyoplasm stain irregularly; the nucleolus stains deeply.

External cell changes (as demonstrated by the silver phosphomolybdate method of Berkley) consist in roughening and deformity, extending in some cases to excavation of the cell corpus. The basilar dendrites show moniliform swellings along their course or present clubbed extremities; in some cells there is loss

FIG. I.—Long pyramidal cell from the middle occipital convolution, showing roughening of the cell corpus and apical dendrite; moniliform swellings on the basilar dendrites; also loss of gemmulæ.

FIG. II.—Long pyramidal and fusiform cells from the second frontal convolution, showing irregularity in contour; also excavation of the cell corpus; deformity of basilar dendrites; also moniliform swellings of the apical dendrites.

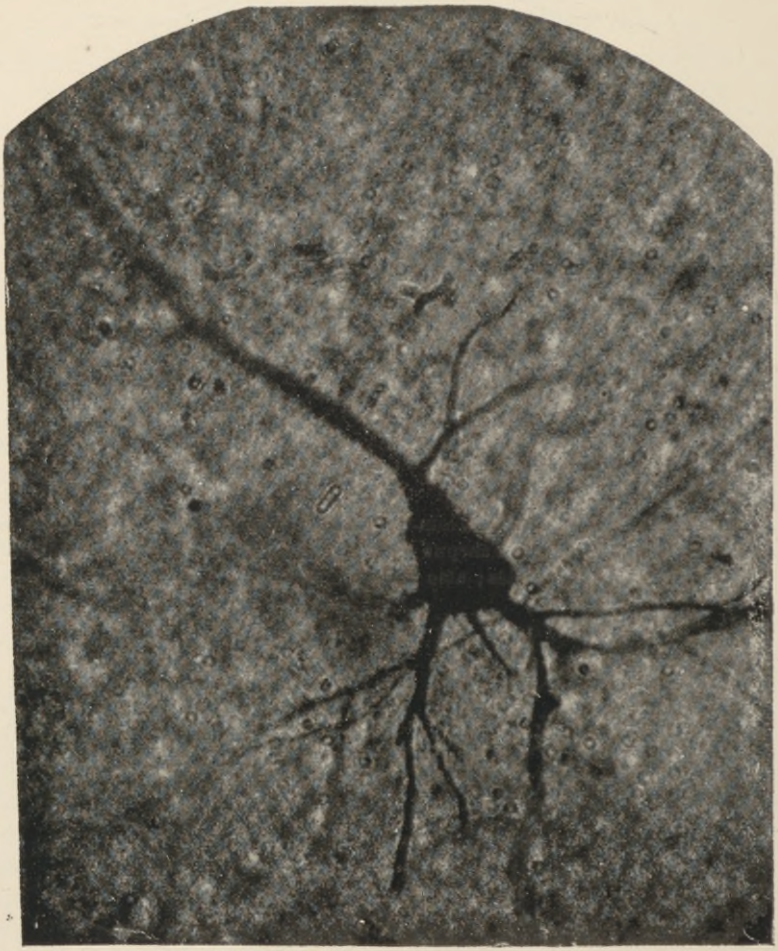

FIG. III.—Photo-micrograph of a long pyramidal cell from the ascending frontal convolution, showing roughening of the cell corpus and of the apical dendrite; also moniliform swellings of some of the basal dendrites.

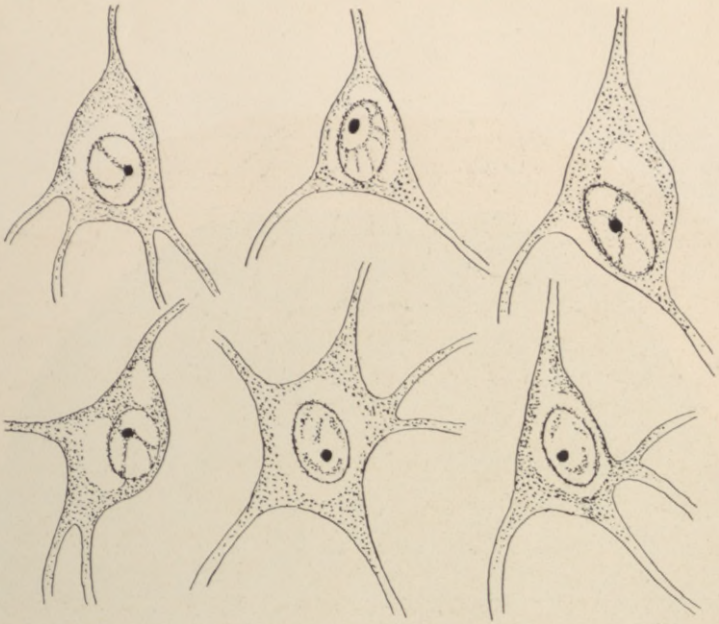

FIG. IV.—Long pyramidal cells from the second frontal convolution, showing vacuolated appearance of the cell body; diminution of chromophilic particles in cell processes; and nuclear changes.

FIG. V.—Protoplasmic glia cell, showing loss of fine mossy granulation, and the presence of varicosities on the pseudopodia.

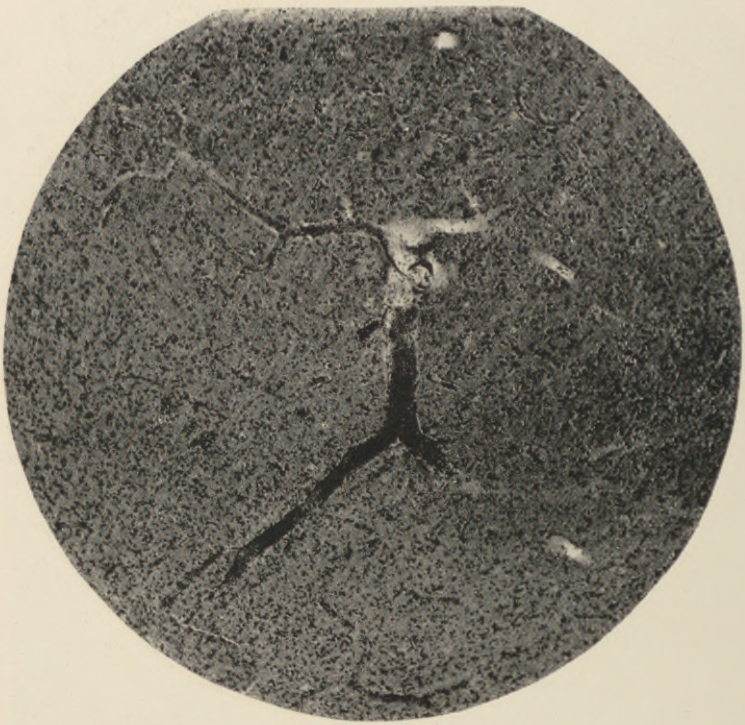

FIG. VI.—Section from the middle frontal convolution, showing stasis, over-distension of the vessel walls, enlargement of perivascular lymphatic space; also area of softening.

FIG. VII.—Section from the ascending parietal convolution, showing stasis; over-distension of the vessel walls; tortuosity and multiplication of vessels.

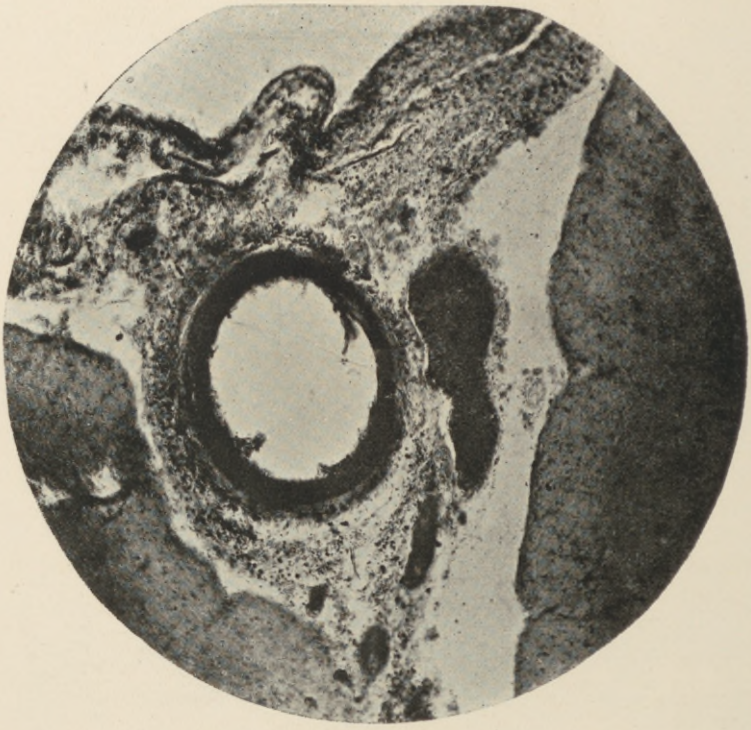

FIG. VIII.—Section in the second temporal region, showing changes in the meninges; thickening of the pia-arachnoid; evidences of extravasated blood; thickening of vessel walls.

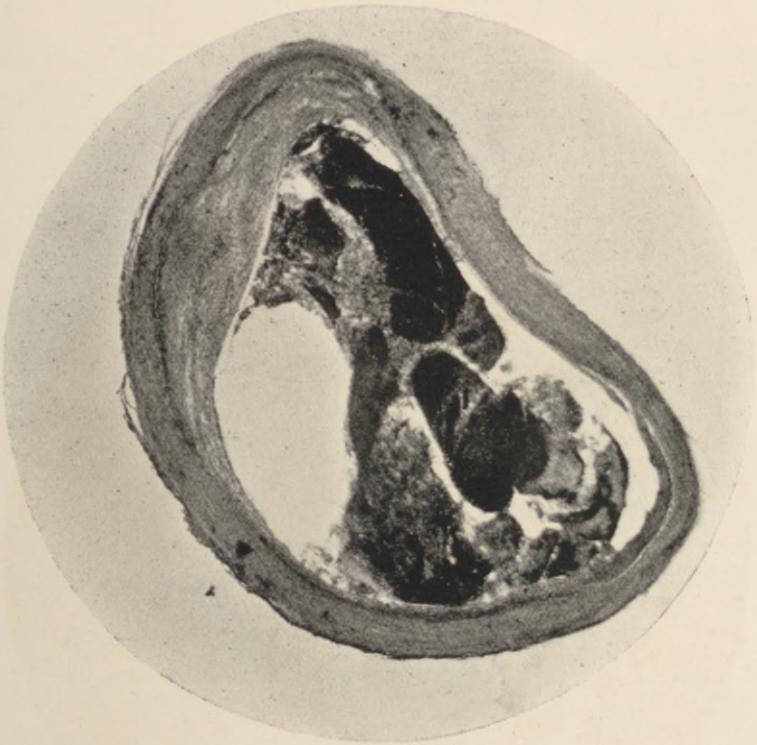

FIG. IX.—Section of basilar artery, showing atheromatous condition of walls; also thrombus.

FIG. X.—Area of softening from the ascending parietal region, containing portions of blood-vessels, blood corpuscles and hæmatoidin crystals.

of several dendrites. The apical dendrite is roughened and deformed in contour. The axis cylinder remains intact. The long pyramidal cells seem to be most affected by these changes. The Purkinje cells show thickened stems with short, stumpy branches in place of the complex feathery dendrites of the normal cell.

The protoplasmic glia cells present a series of transitional changes; the fine mossy granulation appearance of the pseudopodia is lost and varicose swellings appear in their course. Other cells take on an irregular, botryoidal appearance and finally evidences of disintegration are to be observed. Deiter's cells are numerous both in cerebrum and cerebellum.

The basilar and internal carotid arteries show increase in the number of true endothelial cells and a growth of new connective tissue derived from the endothelium. This growth consists of branching cells, proliferated nuclei and basement substance with areas of atheromatous degeneration. In the posterior communicating arteries the intima, media and adventitia are about equally thickened. The anterior communicating shows aneurismal dilatation. Thrombi are present in all these vessels.

The pial vessels show stasis, aneurismal dilatations, tortuosity and thickening of their walls, with nuclear proliferation. In all regions there are evidences of extravasated blood in the form of groups of corpuscles and hæmatoidin crystals. The region of miliary aneurisms shows similar changes.

All cortical vessels are over-distended with blood corpuscles; they are exceedingly irregular and tortuous in their course and appear to be greatly increased in number. The perivascular lymphatics are distended.

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

Medullated fibres of the ascending parietal region, optic chiasm, and of scattered areas in the pons and medulla show different stages of myelin degeneration.

In addition to the methods above referred to, sections were stained with hæmatoxylin and eosin; hæmatoxylin, picric acid and fuchsin, and according to the Weigert-Pal method.

In conclusion the pathological features of the case may be summarized as follows: First, Internal and external changes in the neuron; second, changes in the protoplasmic glia cells; third, changes involving the cortical and pial vessels, also the vessels at the base of the brain; fourth, multiple areas of softening in the ascending parietal region; fifth, myelin degeneration.