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CATALOGUE
OF A
SERIES OF SPECIMENS ILLUSTRATIVE
OF THE
MORBID ANATOMY
OF THE
BRAIN AND SPINAL CORD.

Exhibited at Ottawa Meeting of Canada Medical Association, Sept. 1st and
2nd, 1880, by William Osler, M.D., M.R.C.P., Lond.

BRAIN.

No. 1. Section of brain (made with Dalton's section-cutter)
showing large apoplectic clot *in situ*.

Woman, aged 40, cirrhotic kidneys, hypertrophied heart, sudden
hemiplegia with coma, and death in two hours.

No. 2. Hæmorrhagic softening, probably from embolus.

The lesion in this case involved chiefly the band of white matter
(int. capsule) between the caudate and lenticular nuclei of the corpus
striatum, and it illustrates the truth of Charcot's view that the motor
path is in the anterior part of the internal capsule.

No. 3. Cortical softening from hæmorrhage of traumatic
origin.

This specimen is exhibited to illustrate how beautifully superficial
lesions are shown in brains preserved by Giacomini's method (see
N. Y. Med. Record, April, 1880).

No. 4. Apoplexy of pons.

No. 5. Cicatrix of apoplectic clot in cerebellum.

Both of these specimens are from the same case, a woman aged
40, dissipated. The lesion in cerebellum dates from a year before
patient's death, and is a good example of a healed injury to brain
substance. She remained dull and stupid after the attack, and there

was inability to use the legs freely, though they were not paralyzed; sensation was impaired. Death followed the hæmorrhage into the pons. The cerebral vessels were extensively diseased.

No. 6. Abscess in left temporo-sphenoidal lobe.

From a case of mastoid disease. There was no paralysis; headache, dullness and occasional inability to express himself freely were the chief brain symptoms.

No. 7. Embolism of left middle cerebral artery.

The embolon is seen *in situ*. Case of young girl with mitral stenosis and numerous vegetations on the valves. Sudden right-sided hemiplegia with aphasia. Red softening of third left frontal convolution and neighboring parts supplied by middle cerebral.

No. 8. Aneurism of left middle cerebral artery.

Aneurisms on branches of the cerebral arteries are more common than is supposed. I have met with six cases in four years, two on the basilar, three on branches of the left middle cerebral and one on the anterior communicating. In four of these death was caused by bursting of the sac.

No. 9. Miliary aneurisms on small cerebral arteries.

The structures, described by Charcot & Bouchard, are supposed to play an important rôle in the causation of cerebral hæmorrhage, particularly in old persons. They result from a periarteritis which weakens the wall, leads to a local dilatation and final rupture. The statement of these authors, that they are to be found in every case of apoplexy in old persons, is not, in my experience, correct.

No. 10. Coarse tubercle of brain.

Irregular masses about the vessels in the sylvian fissures.

No. 11. Section of coarse tubercle of brain.

The part shown is just at the border of a mass the size of a pea; There are numerous small tubercle cells embedded in a granular matrix, two giant cells are also seen. Internally there is a granular degeneration of the cells (caseation) and an obliterated vessel can be seen.

No. 12. Miliary tubercles on small arteries.

From case of acute hydrocephalus. The tubercle cells are seen in outer coat (adventitia) of the small arteries. The increase of these causes a bulging which can be, in small arteries, seen with the naked eye, and the calibre of the vessel may be greatly deduced or even obliterated.

In the case from which this specimen was taken, there was no *basilar meningitis*, i. e., no exudation of lymph about the base,

but on carefully removing the pia mater and examining the vessels, particularly those of the perforated spaces, the little tubercles were seen on the small arteries.

No. 13. Syphilitic arteritis.

From a man aged 36; syphilis 18 months before death, which followed rupture of an aneurismal dilatation of the basilar caused by the arteritis. In this specimen, the alteration consists in a very great thickening of the *intima*, which in places is of greater diameter than the other coats together; the cellular elements are few in number, the chief part of the new growth consisting in a low form of fibrillated tissue.

No. 14. Glioma of corpus striatum.

Tumor consists of small round cells, like those of a small-celled sarcoma, embedded in the meshes of a reticulum of fine fibres.

No. 15. Pachymeningitis.

A localized spot upon the frontal lobe in the case of a young man, the subject of severe epilepsy.

There is thickening of the dura mater and adhesion to the arachnoid and pia. There has been extravasation in the thickened membranes as evidenced by numerous hæmatoidin grains.

No. 16. Insular sclerosis.

Localized areas of fibroid transformation, usually in the white matter, the result, it is supposed, of a chronic inflammatory process. The substitution of the white substance by a fibrillar growth is well seen in this specimen. Many of the fibres are in connection with elongated corpuscles. The development of these patches in the brain and spinal cord causes a well-recognized form of disease, characterized by a remarkable tremor, &c.

No. 17. Medullary neuroma.

New growth (heterotopia) of grey matter on thalamus opticus, with extension into third ventricle; chronic hydrocephalus from pressure on venæ Galeni. Intellectual faculties retained. Girl aged 16.

The section shows the finely granular grey matter, a ganglion cell and numerous smaller (nerve) corpuscles.

No. 18. Pigmentary degeneration of cerebral vessels.

In case of apoplexy of the pons, cerebral vessels were much diseased, whereas the general arteries of the body were but slightly involved. Many of the smaller arteries present the peculiar pigmentary change in the adventitia, seen in the specimen, the deposition being chiefly in spider-like connective tissue cells.

CORD.

No. 19. Locomotor ataxia, posterior spinal sclerosis.

Thickening of the neuroglia with compression and atrophy of the nerve cylinders constitutes the essence of the disease termed sclerosis or grey degeneration.

No. 20. Descending degeneration of crossed pyramidal column.

This specimen illustrates the secondary degeneration which takes place in the cord after a destructive lesion in the brain, which has involved the motor path. In this case there is no degeneration as there often is in the situation of the direct pyramidal fasciculus, *i. e.*, in the part of the anterior column next the median fissure on the same side as the lesion.

No. 21. Antero-lateral sclerosis.

Degeneration of anterior horns of grey matter with sclerosis of antero-lateral columns. It is characterized clinically by atrophy of the muscles with contractures, and must be distinguished from progressive muscular atrophy, in which the anterior grey matter is alone diseased and there are no contractures.

I am indebted to my friend Dr. Gowers for this beautiful specimen.

No. 22. Ascending degeneration of posterior median columns.

When there is a focus of chronic disease at any point in the cord which impairs its function, these columns above the seat of disease become atrophic. No symptom is known to be connected with this process.

No. 24. Annular myelitis.

Sclerosis chiefly cortical, deepest in posterior parts of lateral columns. Section from level of 6th dorsal nerve, in case of spastic spinal paralysis.

No. 24. Lateral sclerosis.

In case from which preceding specimen was obtained. In the lower dorsal the sclerosis was chiefly, as seen in this specimen, in the hinder part of lateral columns and near the posterior nerve roots. The special symptom connected with disease of these columns is rigidity of the muscles.

No. 25. Tumour, probably syphilitic, of the cord.

Specimen from Dr. Gowers.