THE MANNER OF INVASION AND DESTRUCTION OF BRAIN TISSUE BY SPONGIOBLASTOMA.

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In July 1918 Strauss and Globus,¹ and shortly thereafter Ribbert,² reported a type of cerebral neoplasm which grew very rapidly, which was made up of undifferentiated glia cells, and which was termed by them 'spongioblastoma.'

In August 1925, the former writers³ reported a new group of sixteen cases showing the same embryonal undifferentiated glia pictures, which they now called 'spongioblastoma multiforme' in distinction to the 'medulloblastomas' of Bailey and Cushing.⁴

The present case also began acutely, progressed very rapidly to a fatal termination (within fifty-three days) and at autopsy revealed a spongioblastoma, the size of a hen's egg. Histopathologically, this single case shows all the pictures previously described, i.e., giant cells, rosette-formation, glandular appearance with radial arrangement of cells and circular cell arrangement round blood vessels.

The body of the tumour is highly cellular, very vascular and in the main of ectodermal character. The cells are either pyriform or round and show little cytoplasm; the nuclei are well stained and contain numerous coarse granules. Elongated cells are also present, as well as numerous giant cells containing many nuclei peripherally placed. We wish to lay stress on the manner of invasion of normal tissue and the formation of rosettes.

The tumour advances in a pseudopodium-like manner (Fig. 1), engulfing and then completely surrounding areas of normal brain tissue as well as blood vessels. In advance of the invading tumour there are numerous areas of colloid degeneration (Figs. 1 and 2). From the sides of the pseudopodia offshoots are seen. The cells in the sides of the invading pseudopodia are arranged in parallel fashion (Fig. 3). When viewed closely, there are areas where the most advanced part of a pseudopodium has joined a neighbouring similar part and has formed an alveolar gland-like structure, which still retains the parallel cell arrangement. It is in this way that a rosette is formed. Another manner of rosette-formation is easily understood by imagining a

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pseudopodial offshoot cut in a plane horizontal to that seen in Fig. 1. Since the cells are polarized towards the normal tissue and also towards the core of the offshoot, rosette-formation must occur on cutting it across near its apex. Later, glia fibres are seen to arise and converge towards the centre, which then breaks down and finally gives rise to an area of degeneration (Fig. 4).

The cells in the pseudopodium-like advancing tumour are always polarized in the direction of growth, i.e., their longest diameter points in this direction (Fig. 3). This polarization recalls that seen in embryonic connective tissue. The forces producing it are dependent, we believe, upon a distinct physico-chemical difference between the normal tissue invaded and the tumour tissue itself. This polarization and resulting parallel arrangement of the cells, combined with the pseudopodium-like advance, engulfing normal tissue, are, as suggested in less detail above, the origin of rosette-formation. Neurobiotactic processes are therefore responsible for the typical pictures of spongioblastomata (Figs. 5 and 6).

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Fig. 1.—Showing the pseudopodium-like invasion of normal tissue by the tumour, and the colloid degeneration within the normal tissue in advance of the tumour. (See also Fig. 2.) As these pseudopodia close in on each other rosettes are formed. Hematoxylin-eosin. Leitz obj. 1, oc. 1. A, pseudopodia; B, colloid degeneration; C, rosette formation; N, normal brain tissue; T, tumour.
Fig. 2.—Colloid degeneration in normal tissue in advance of the invading tumour. Hæmatoxylin-eosin. Zeiss obj. 16 mm. ap., oc. 10 x comp.

Fig. 3.—Showing parallel arrangement of the cells of the advancing tumour in the periphery of the pseudopodia. Hæmatoxylin-eosin. Zeiss obj. 4 mm. ap., oc. 10 x comp.
Fig. 4.—Rosette with glia fibres converging towards the centre of the core. 1st modification. Zeiss obj. 16 mm. ap., oc. 15 x comp. Rio-Hortega stain.

Fig. 5.—General appearance of tumour. O.R., old rosette formation; G. C., giant cells (See also Fig. 6). Haematoxylin-eosin. Zeiss obj. 16 mm. ap., oc. 7 x comp.
CASE REPORT.

U. D., male, age 43, iron-worker, admitted to Lebanon Hospital, July 3, 1924, and died July 23, 1924. Chief complaints: (1) frontal headaches; (2) noise in head and ears; (3) weakness in both legs for the past three weeks; (4) stiffness of neck. The patient enjoyed good health until four weeks before admission, when he began to suffer from attacks of dizziness and faintness, accompanied by a severe burning sensation in the region of the left triceps. These attacks came on every morning and continued for two weeks. Headaches at this time were accompanied by a loud roaring sound, followed by vomiting. Stiffness of the neck and progressive weakness of both legs compelled him to take to bed.

Neurological Examination: July 9, 1924.

Cranial Nerves: (I) bilateral anosmia. (II) a beginning bilateral papilledema. (III), (IV), (VI) pupils widely dilated (atropin). Weakness of left levator palpebræ; left palpebral fissure wider than right. Slight weakness of left abduceens. Nystagmoid movements on right lateral fixation. (VII) a central left facial paresis. (VIII) hearing diminished to watch and tuning fork bilaterally. Bone conduction affected on left. (XII) tongue slightly deviated to left.

Movement and Co-ordination: Motor power diminished in left upper extremity, including grasp. Left lower extremity, motor power affected. Slight paresis, right upper and lower extremities. Station and gait: no Romberg, no staggering, but head tilted to left. Slight ataxia in finger-nose test on left. No spontaneous past-pointing.

Sensation: Repeated sensory examinations were of little value, because of non-cooperation on the part of the patient, but it was thought there was some hypesthesia and hypalgesia over the trunk and both lower extremities.

Reflexes: Deep reflexes moderately exaggerated in left upper and lower extremities, but distinct diminution of left triceps jerk. Abdominals and cremasterics absent on left. An inconstant Oppenheim reflex present bilaterally.

Summary: A definite left pyramidal tract involvement, some implication of right side, and a beginning bilateral choked disc.
Course: Repeated neurological examinations showed changing phenomena. On July 15, 1924, a definite bilateral papilloedema of three to four diopters was present. Left facial paresis cleared up entirely. Knee jerks and ankle jerks present and normal in response. A right Oppenheim reflex was now elicited, but no Babinski response or ankle clonus. Abdominals and cremasterics present. A constant absence of the left triceps jerk was an outstanding feature. Sensory examination, performed repeatedly, also varied, but a persistent belt of hyperaesthesia from the third to the sixth cervical segments was present. Rigidity of the neck and a bilateral Kernig sign were found, as well as a left Babinski response and right Oppenheim reflex. The definite belt of hyperaesthesia from C3 to C6 persisted.

An X-ray of this region showed slight haziness of the spinous processes. A laminectomy from the second to the seventh cervical segment revealed an edematous dura with spinal fluid under increased pressure. A probe passed up and down encountered no obstruction. (Dr. L. Miller Kahn of Lebanon Hospital.)

Caloric examination was difficult as the patient did not cooperate well. However, it was shown that responses in the horizontal positions were normal. On July 23, he became very drowsy and feeble. A subtemporal decompression was done, but he died a few hours after the operation was terminated.

Necropsy: Limited to brain only. There was some increase in the size of the ventricles, and hemorrhage at the sight of decompression. Incision revealed a subcortical tumour, brownish tinged in colour, the size of a hen’s egg, situated in the right temporoparietal region. It was firmer in consistency than the surrounding brain tissue, and apparently encapsulated.

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