

(right): broncho-pneumonia and abscess of the lung (left): chronic congestion of the liver: duodenal ulcer.

The brain, after removal of the dura, showed flattening of the convolutions in the third frontal, ascending frontal and parietal regions of the left hemisphere, also a slight reddening of the cortex over the third left frontal region. The pia was smooth and glistening throughout. The first temporal convolution is compressed by the bulging of the upper boundary of the Sylvian fissure.

On section after hardening the brain, a tumour was found extending from near the anterior end of the Sylvian fissure in the third frontal convolution backwards almost to the posterior end of the sulcus. The growth measured two inches from without in, and two and a quarter inches from before back. The edge is irregular and no definite capsule was evident except at the upper and anterior end of the tumour in the ascending frontal convolution, where it was immediately subcortical. Behind this level it merged with the grey matter of the cortex and came very close to the surface, especially in the tissues forming the roof of the Sylvian fissure. The inner border of the growth was ill-defined and extended in for two inches from its outer edge.

Several small dark brown hæmorrhagic areas were scattered through the growth. Microscopically, the growth proved to be a spindle-celled sarcoma.

The diagnosis on admission seemed to lie between hæmorrhage and syphilitic softening, the former being regarded as more probable. With the progress of the case neither of these hypotheses seemed quite satisfactory, but it seemed possible that a progressive specific arteritis with extending thrombosis of the vessels might account for the increasing paralysis and deepening torpor and lethargy.

The usual symptoms of cerebral tumour were absent. Headache, although present for a short time, was never severe or persistent; vomiting only occurred on two occasions, and optic neuritis was absent a month before death. Had more weight been laid on the gradually increasing stupor, on the slow development of paralysis and spasm in the leg and on the two convulsions, a more correct opinion might have been reached; the sudden onset, however, was so strongly in favour of a vascular origin that these symptoms did not secure sufficient consideration.

The localisation of the lesion offered less difficulty than its pathological character. A cortical condition was improbable owing to the absence of early Jacksonian attacks, whilst the fact that the leg escaped paralysis in the earlier stages suggested the subcortical region rather than the internal capsule as the most probable site.