rupture of some small vessel. No miliary aneurisms were found in the neighborhood. The nutritive disturbances in the vicinity were not so much in the thalamus, immediately below the extravasation, but in the upper part of internal capsule and the outer section of lenticular nucleus—*i.e.*, in the regions supplied by the vessels from which the extravasation took place.

Second, *the absence of sensory paralysis in the first case and its quick disappearance in the second.* The localization of the lesions may be considered to afford a satisfactory explanation. The band of white matter known as the internal capsule, and which occupies a position between the caudate and lenticular nuclei on the one hand, and between the latter and the thalamus opticus on the other, is a prolongation of the fibres of the crus cerebri. Now it has been proved experimentally that destruction of the anterior part of the internal capsule —*i.e.*, that portion between caudate and lenticular nuclei—produces motor hemiplegia only of the opposite side of the body, no loss of sensation; and the same evidence is afforded by pathology. This is the territory supplied by the lenticulo-striated arteries. If the posterior part of the internal capsule be injured—*i.e.*, that part between the lenticular nucleus and the optic thalamus—the result is anæsthesia of the opposite side without motor paralysis. This is the region supplied by the lenticulo-optic arteries. Case I affords an illustration of the truth of this view, viz., that the course of the motor impulses from the hemispheres to the cord is in the anterior part of internal capsule.

Thirdly, In case II, a point of considerable interest is the early onset of the rigidity or contraction of the muscles of the paralyzed side. This condition may be quite transitory, coming on with the attack; more commonly it is a late symptom supervening after weeks or months. In this case it came on on the third day, and persisted. It is to be attributed, most probably, as suggested by Todd, to the irritative effects of the lesion on the motor nerve tract, but it is difficult to see why it should come on so early in one case and not in another. Ferrier states that the irritable lesions inducing spasm are almost always