

us first deal with the sensory affections, and later with the motor. In this way we shall be better able to appreciate the etiology of the condition.

The most fertile source for the production of organic hemianæsthesia is hæmorrhage into the internal capsule. In hysteria, the patient is not conscious of the anæsthesia present, while in organic trouble he readily appreciates the loss. In the first instance the disease affects the cell bodies themselves, while, in the organic, the cell bodies are not involved, but the axones are, and at some distance from it, with the result that, the cell bodies being uninjured, they are aware of not receiving the usual stimuli. As a comparison, one might mention the difference between a general and local anæsthetic. If local anæsthesia was produced in my leg, I should at once have the impression that my leg was dead; while, if put under a general anæsthetic, I would, of course, not be aware of the condition present.

In the hysterical subject hemianæsthesia, pure and simple, is a very common symptom. Hemianæsthesia alone, in organic disease seldom, if ever occurs, because when a lesion takes place in the internal capsule where the sensory fibres are so closely associated with the motor fibres anteriorly, and the fibres of the eye behind, a pure sensory paralysis is impossible. The presence of motor involvement, homonymous hemianopsia and hemianæsthesia are a frequent occurrence in organic diseases for the above reasons. In fact, either a motor enfeeblement plus anæsthesia, or homonymous hemianopsia and anæsthesia are sure to be evident in those diseases.

Another differential sensory disturbance in hysteria is the characteristic islets of anæsthesia throughout the body. Their position at once shows that the anæsthesia is not due to peripheral involvement, as it is not in the course of any nerve supply, nor due to involvement of the cord. The explanation is that the sensory fibres as they leave the internal capsule on their way upwards, subdivide, and end separately in the cortex. And, therefore, small cortical areas if functionally depressed would give rise to limited areas of sensory deficiency in the skin.

Another form is the glove and stocking anæsthesiæ in the extremities. This also cannot be due to peripheral disease from its distribution. In a case reported by Pitres, where the patient was suffering from anæsthesia of the arm, he exposed the ulnar nerve, and upon pricking it with a sharp instrument, no sensation was present, yet the muscles connected with the nerve contracted, showing that conductivity must have been present, as the motor impulses were carried. This case again shows that the sensory anæsthesiæ must be of cortical origin.