

Rockwell (A. D.)



INTERMITTENT HEMIPLEGIA,

WITH

RESULTS OF A POST-MORTEM EXAMINATION.¹

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THE following case was, in its symptoms and progress, unique, interesting, and instructive; and as a *post mortem* was fortunately obtained, although with difficulty, I think it worthy of brief mention.

C. H. B., a stair-builder, aged forty-nine, and in the seeming enjoyment of a fair degree of health, was suddenly seized, one afternoon in the latter part of July, 1876, and while at work in his shop, with symptoms of dizziness. His power of speech was lost, and the left side became completely paralyzed. In about twenty minutes these symptoms entirely disappeared, leaving him quite well. On the following day he remained quietly in his house during the morning, but in the afternoon resumed work, and at about the same time (4 P. M.) the attack recurred, but with less severity. For some three weeks

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thereafter paroxysms occurred every other day, and invariably in the latter part of the afternoon, rarely exceeding in duration, however, from ten to fifteen minutes. About the middle of August he was prostrated by a similar but much more severe attack, this time, however, 11 A. M. being the hour of onset; and thereafter, until the 3d of September, an attack occurred every day at 11 A. M., lasting some fifteen minutes; and between that hour and 4 P. M. the paroxysms would recur from three to four times.

On the 5th, 6th, and 7th of September, the symptoms increased in severity, attacks occurring early every morning, and repeating themselves at intervals of two or three hours during the day. From the day of the first attack until the latter part of August the patient's health remained pretty good; appetite fair; bowels regular; sleep in excess of normal. In the more severe attacks he was utterly unable to walk or speak; in those of less severity he could move with difficulty and speak indistinctly; but in all attacks the sensory symptoms were profoundly marked.

On the 8th of September, when he came under my observation, he was beginning to show the effects of these repeated strokes, in a condition of pallor and general muscular weakness.

As to the sequel of treatment, I give it with some hesitancy, since, in the light of subsequently-ascertained pathological changes, it seems difficult to believe that the method employed, or indeed any form of treatment, could have availed much. I submitted the patient to a mild *séance* of general faradization.

On the following day he returned, stating that no attack had occurred. He received similar treatment, and, in addition, directions to take the small dose of two grains of quinine three times daily. Two days subsequently, when he presented himself, he had had but one slight premonition of his difficulty.

On the 25th of September he was discharged as approximately well, and so remained until December 4th, when I was called to see him.

I found him in his shop in Twenty-eighth Street, with his left side completely paralyzed, articulation imperfect,

inability to swallow, but with intelligence unimpaired. He was taken in a sleigh to his home in Harlem, where he died the following night. At a *post mortem* held December 6th, by Dr. Henry T. Pierce—present, Drs. Brockway and Forbes, and the late Dr. H. H. Gregory—the following conditions were revealed: Venous congestion of the surface of the brain; pia mater covered with a thin film of organized lymph from old inflammation; texture of brain softer than normal; choroid plexus enlarged and cystic; basilar artery and part of the circle of Willis enlarged and atheromatous, with a considerable amount of serous effusion at the base of the brain. The mitral valve, as well as the liver, was in a condition of fatty degeneration.

No artery was ruptured, neither was we able to detect, in the course of a thorough and careful examination, any evidence of embolism or thrombosis. As the pathological changes above recounted seem hardly sufficient to account for the unusual course of the symptoms, or for the suddenness of death, it becomes of especial interest to consider what was the probable cause. The kidneys were not examined, although it is probable that they were the seat of fatty or other degenerative changes; but, from the fact that the patient was entirely conscious up to within a few moments of death, it is safe to exclude uræmia as a factor in the production of the final result. In cases where death occurs from the brain, and examination reveals no blood-effusion and no obstruction of vessels, it has been customary to refer to the case as one of simple apoplexy—a nomenclature as unsatisfactory as it is unscientific. Sudden and violent congestion of the brain, causing what Trousseau aptly calls “cerebral surprise,” may, it is believed, be in itself sufficient to produce death; but the whole history of our patient readily excludes the possibility of this as a cause.

There remains but one other condition in which we may reasonably hope to find an explanation of the above phenomena, viz., a spasm of vessels, which may be supposed to be associated with, or actually caused by, molecular changes in the brain-tissue, rendering it unfit to discharge its proper function. The regularly-intermittent character of these attacks of hemiplegia justified the inference that there was a

malarial influence in the case, and renders, perhaps, the temporary results of treatment less unaccountable. The brain was not galvanized, the faradic current alone being used; but it does not seem difficult to believe that a current of sufficient tension, applied to the cilio-spinal centre so as to affect the sympathetic and its cervical ganglia, might, either reflexly or directly, exert a beneficial influence upon spasm of the smaller vessels of the brain.

