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BY

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ASSISTANT PHYSICIAN IN THE STATE HOSPITAL FOR THE INSANE, AT DANVILLE, PA.

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The following case is interesting viewed from several standpoints, and I think it is quite worthy of being noted. The history which accompanied the patient, though meagre and unsatisfactory, is still of value in the study of the case.

Mrs. D——, widow, admitted into the Danville Hospital, June 4, 1887; married 22 years ago; had one still-born child seventeen years ago; supposed cause of insanity, domestic troubles and money matters; prominent symptoms consist of incessant talk of a rambling sort. She is said to have had attacks of epilepsy or apoplexy several years ago. When irritated she has threatened to jump out of the window. For more than a year there has been alternate mental depression and excitement. Various physicians have been employed and different remedies have been

tried, but with no good result. When admitted her general health was impaired, for which a tonic was ordered. She had various erratic ideas concerning brain clots, her ideas being often disjointed and mixed.

On June 15, 1888—about a year after her admission to the hospital—her powers of locomotion were slowly becoming impaired. On her arrival here she was able to play some old familiar airs on the piano, but now she was unable to strike simple chords. Her language and power of expression had altered quite materially. She was very fond of creature comforts.

By Nov. 1, 1888, the patient had a peculiar gait, taking short steps, with her feet dragging on the floor. She had many symptoms which appeared to be of an hysterical character; for example, she asked if she were not fatally ill, and whether she would live until morning. She asked for a piece of brown bread, or a soft-boiled egg, with as much earnestness as though it were a matter of momentous importance. She spoke quickly, and in a jerky manner. She often stopped abruptly in the midst of a sentence and with a look of earnest appeal on her face would say: "What is it? What is it?" If the word was supplied, her face would at once light up, and she would be profuse in her thanks. She referred all her trouble to her brain, often saying: "There is a weakness" (indicating her head). Since June, 1887, nineteen convulsive seizures had

been noted as follows: In 1887, June, one; July, none; August, two; September, none; November, none; December, two. In 1888, January, none; February, none; March, three; April, none; May, two; June, five; July, none; August, two; September, two.

Immediately after the occurrence of some, if not all, of the seizures, there was greater or less mental lethargy or confusion. The aphonia on one or two occasions, was complete. The ataxia usually increased while the general motor power decreased. The symptoms gradually lessened in intensity up to the date of the next attack; so that the patient had periods of great physical and nervous disturbance, alternating with periods in which the symptoms were much less pronounced. Her convulsions were not minutely noted, but they coincided with the description of none which follow a well-defined course. In one which I had a chance to observe she called out that she was dying, then, with her eyes rolled toward the ceiling, she was seized with a violent general convulsion.

It was the opinion of the writer at this time that she had many symptoms of hysteria—superadded to those resulting from some obscure, ill-defined organic trouble, probably a gross brain lesion.

On November 18, 1888, at five o'clock in the morning, the night nurse discovered that the patient was breathing very heavily

and was unconscious, and at once notified me. The pulse was slow, full and strong (about 65); breathing stertorous; cheeks puffing out with each expiration, but regular in rhythm. The head and mouth were slightly drawn to the left side; pupils small, equal, and both responsive to stimulus of light. Tickling the soles of the feet and palms of the hands failed to produce any reflex action. Four drops of croton oil, with a little whiskey, were placed upon the tongue. An ice-cap was applied to the head, which was elevated. The oil failed to operate. The patient died at 8.50 A.M., the same day.

Autopsy.—26 hours after death. Rigor mortis well marked.

Brain-Envelopes.—On removing the calvarium, the dura mater which was presented to view was of a dark blue tinge in the lower or posterior half, the color being most marked along the longitudinal sinus. Upon cutting through the dura, this coloration was found to be due to a large amount of extravasated blood, partly clotted, between the brain proper and the membranes; but the pia mater and arachnoid were perforated at one point, so that the blood was directly beneath the dura. The convolutions of the brain were well marked, the sulci unusually deep, but the layer of gray matter rather thin. The vessels of the pia mater were somewhat engorged.

Cerebellum, left side.—Upon section, a

cavity somewhat larger than a walnut was discovered, occupying the centre of the lobe. The walls of the cavity were quite ragged and at many points there were adherent to them small clots. These were mostly grayish or yellowish-white in color, and seemed to be well organized. At three or four points small fibrous bands (old blood-vessels) were joined to the clots from the cavity wall. The principal portion of the cavity, however, was occupied by a single, large, dark-red clot, apparently of recent origin. There was direct communication between the blood in this cavity and that upon the outside, already referred to, by means of a small ragged laceration in the gray matter of the cerebellum, which formed the posterior wall of the cavity.

Cerebrum, right side.—In the bottom of the sulcus between the first and second convolution, and midway between the anterior and posterior extremities of the frontal lobe, was a well organized, reddish-brown clot, of the size of a pea. It principally occupied the gray matter. In the centre of the Island of Reil, just beneath the gray matter, and occupying the greater portion of the island, was a large reddish-brown, or rusty colored, clot. Midway between the anterior and posterior boundaries of the parietal lobe, near the great longitudinal fissure, was a clot the size of a pea. It was situated partly in the gray and partly in the white matter, and was grayish-white in color.

Cerebrum, left side.—In the lower extremity of the ascending frontal convolution, a small reddish brown clot was found of the size of a pea In the ascending frontal convolution, near the longitudinal fissure, was another small clot, well organized. In the occipital lobe, near the junction of the parieto-occipital and longitudinal fissures, on the outer aspect of the brain, a rusty-brown, moderately firm clot about size of a shellbark was found.

Base of brain.—The vessels were atheromatous in numerous places, this condition being especially marked in the middle cerebrals. It was thought that some of the immense cerebellar hemorrhage found its way into the fourth ventricle, although no means of communication between the two cavities could be demonstrated.

The principal points of interest in this case are as follows:

1. The immense cavity in the cerebellum, with the contained clot.

2. The large number of old clots on the cerebrum.

3. The obscurity and complexity of the symptoms which the case presented.

4. The fact that such extensive destruction of brain matter was compatible with life.

5. The possible confirmation of a theory, to be mentioned hereafter.

Almost all the white matter and the included "*arbor vitæ*" of the left cerebellum

was disintegrated. Evidently some morbid process had been active here for a long time prior to the death of the patient. Most likely hemorrhages had occurred here from time to time. Clots were formed. These broke down into pus and were re-absorbed; but the process of disintegration had also involved brain substance. Hence the large extent of the cavity. This process of retrograde metamorphosis also involved the blood-vessels in this region. The large clot which was found in the cavity was evidently of recent origin and due to the rupture of a vessel of considerable size. This hemorrhage, with the pressure which it caused in this region—and most likely too in the floor of the fourth ventricle—was doubtless the immediate cause of death.

Some of the "attacks" which occurred at irregular intervals since 1886, were doubtless symptoms resulting from cerebral hemorrhage, while others were "motor explosions" or "pressure symptoms." It is not unlikely that several of them were principally or wholly of an hysterical character. It is interesting to note that the almost entire destruction of the function of one side of the cerebellum produced no train of characteristic symptoms, or symptoms by means of which the condition could have been diagnosticated.

That such extensive destruction of white and gray matter should produce as few disturbances as it did seems to me to be an

argument in favor of the theory which Brown-Séquard recently advanced, that there are, for many, and perhaps all brain centres, supplemental centres. If the main or primary centre be destroyed the secondary or auxiliary centre is capable of taking upon itself a good portion of the work of the primary centre, and the extra work thrown upon the secondary centre tends to develop its capacity and power: just as we see unusual or compensatory development of a leg or arm when its fellow has been removed. At least this seems to me a very plausible theory by which to account for the gradual disappearing and recurrence of the aphasia from time to time in the case just described, when at the autopsy an almost entire destruction of the speech centre (lower part of left ascending frontal convolution) was noted.

The gradual lessening of the ataxia, the temporary increase in the muscular power, and the decrease and subsequent increase of the power of volition, would all seem to point to a confirmation of the theory.