Oculomotor paralysis accompanied by facial palsy, neuroparalytic keratitis and hemiplegia / by Burton Chance.

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OCULOMOTOR PARALYSIS ACCOMPANIED BY FACIAL PALSY, NEUROPARALYTIC KERATITIS AND HEMIPLEGIA.[1]

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Through the kindness of Dr. Schwenk I am allowed to report the following case, interesting on account of the multiplicity of important symptoms, the vagaries in the modes of their production, and the marked regain of power by the patient.

On August 24, 1905, a tall negro woman came to the hospital complaining of the sudden closure of her left eye. The eye had been inflamed for six weeks past. Two weeks before coming to the hospital the sight was totally obscured by the falling of the lid over it. The woman was aged thirty years, a housekeeper, and had been married for eight years. She had been pregnant only once, six years ago. Two days before the end of term she fell down stairs and gave birth to a dead child. She denied having specific disease. She had been in good health until March, 1905, when she was seized with violent pains in her head, and was treated for rheumatism. In July her left eye became inflamed, and she had applied for treatment at another hospital; this inflammation was not very painful.

On examination the left brow and cheek were seen to be discolored, and near the outer angle of the orbit there was a large circular maculation, which had resulted from long blistering. The eye was completely covered by the upper lid. There was facial palsy on the left side and the mouth was drawn toward the right. When the lid was raised the globe was seen to be rotated outward. It was deeply injected, and the epithelium of the cornea was macerated and steamy over nearly the entire surface. The summit was œdematous and infiltrated; and it stained deeply when fluorescin solution was instilled. No view of the fundus could be obtained, nor could the iris be seen. Atropine suspended in oil was prescribed, and the patient was instructed to take a saturated solution of potassium iodide, beginning with fifteen drops and rapidly increasing the dose.

The periphery of the cornea was less œdematous in two days,

[1] Read at a meeting of the Wills Hospital Ophthalmic Society, Philadelphia, March 2, 1909.

and soon became sparkling. The ulcerous area rapidly became sharply outlined. In a few days, on forced effort, there was slight ability to raise the upper lid, but no effort could overcome the paresis of the internal rectus, though there was a faint reaction of the superior oblique muscle. By September 21 the ball remained divergent and fixed at the outer extremity. Facial paralysis was complete on the left side, and in the distribution over the superior and inferior maxillary branches of the fifth nerve there was anesthesia. The cornea continued hazy and anesthetic. About this time the patient's left arm and leg became numb and powerless. She was seized suddenly in the night in the midst of a severe headache; in the morning she could not lift her arm nor raise her leg in walking.

On September 28 the patient was sent to Dr. Wm. G. Spiller, at the Polyclinic hospital, for an examination of her general state. His letter, dated October 12, reported that the patient had cerebral syphilis. On October 24 the solution of atropine was discontinued, and by November 11 the "mixed solution" which had been ordered three weeks before was withdrawn, and mercurial ointment substituted. By November 28 the patient showed signs of ptyalism, and all medicines were withheld. Toward the end of December, when efforts were forcibly made the globe could be brought to the vertical line, but not within the meridian, and when the exertions were made it was noticed that the palpebral fissure widened so that the globe was entirely uncovered by the lid. At this time marked increase in the general muscular power began; the facial palsy was reduced, and the leg and arm regained much of their lost functions.

On January 5, 1906, the cornea again became denuded. To allay the irritation the lids were sealed with plaster and a firm bandage was kept on for two days; on the removal of this the exposed area was seen to be closed, but the surface was shaggy, as though it were studded with epithelial shreds like the remains of ruptured herpes. These shreds were examined by Dr. Goldberg and found to consist of epithelial cells only. Toward the end of the month the epithelium became restored. From this time on the return of power was marked. The ptosis disappeared almost entirely. The eye could be rotated to the primary position, but not within the vertical nor above or below the horizontal meridian. The woman could walk with greater comfort. A certain childishness manifested in the past gave place to a decidely acute intelligence. Before the first of February, 1906, the patient ceased her visits, and did not return again until January, 1909, in response to a letter from me. By this time she had regained all her lost weight and much more besides, for she weighed 215 pounds. In the past three years she had been able to keep house and to work as a laundress. She has had a constant dull ache over her left brow, and, when overtired, she has attacks of headache, and the arm and leg feel like "pins and needles." She is very much annoyed by diplopia when she attempts

to look up or down, yet even in the primary position there are two images, the left being the false one and the higher. The sight is good, but with the left eye objects appear far away and small. There is now partial ptosis when at rest, but by wrinkling her forehead she can draw the lid up to the limbus. There is paralysis of the superior and inferior recti, the inferior oblique, and the superior oblique muscles; the ocular excursions are limited to the extent of free adduction and abduction, while supraduction is abolished and infraduction is only faintly perceptible. The diplopia is upward. There is no accommodation power, for while the woman can read types 0.50 D. with the right eye, she has great difficulty in seeing type 2 D. with her left. The vision, however, in each eye equals 5/10. The pupil of the left eye is not circular and is apparently fixed. The fundus is healthy. The cornea is perfectly smooth, and, even with the stereoscopic loupe, nothing is found marring the clearness of the membrane. It is, however, anesthetic, and so is the conjunctiva. There is a free flow of tears. The adjustment of spherical lenses + 1.25 D. with which she sees 5/5, has relieved the woman in the past month of this annoying epiphora. The facial movements have been regained; she can wrinkle her brows and protrude her tongue straight. The face is anesthetic to pin pricks, including the nose, tongue, all of the upper part of the head except that portion comprised in a line from the lobe of the ear and ramus of the jaw forward to about three-fourths of an inch from the meridian line of the chin up beyond the angle of the mouth, thence obliquely to the ear.

In order to know the patient's general condition, I sent her to Dr. T. H. Weisenberg for an examination. He fortunately remembers having seen the woman in Dr. Spiller's clinic at the Polyclinic hospital four years ago. As Dr. Weisenberg points out, the separate occurrence of left hemiplegic with left oculomotor palsy is most unusual. It is probable that the hemiplegia was caused by vascular disturbance in the right side of the brain, while the oculomotor palsy arose through a disturbance at the base on the left side, in advance of the nerve fibers supplying the extremities. The hemiplegia has almost entirely disappeared; it is rather strange, therefore, that the oculomotor disturbance remains. This palsy has been most extensive, affecting both the intrinsic and the extrinsic mechanisms. The widening of the palpebral fissure during the middle period of the ptosis was rather startling in appearance, and was the first instance of that phenomenon in my experinece. The facial palsy has all but disappeared, the anesthesia being the most annoying subjective symptom in this connection. It is most fortunate that the early keratitis did not extend to the true corneal tissue.