PARALYTIC ALTERNATING STRABISMUS OF
? CONGENITAL ORIGIN

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THE case to which these notes and photographs apply, I venture to call a Paralytic Alternating Strabismus of Congenital Origin. Though "alternating" is usually applied to concomitant squints, this action here is a striking feature in the picture. A fairly diligent search through ophthalmic records has failed to yield a similar case.

History.—E. H., female, aged 5½ years, was seen by me on July 7, 1931. She was a healthy looking, well nourished and intelligent child. Her mother said she had been born naturally and was always strong and well. When a few months old, and after a slight accident caused by the fall of a blind on her perambulator, the mother noticed her eyes peculiar. She was taken to a doctor who has been good enough to send me the notes he made at the time, viz., "She fixed with the left eye which showed some lid ptosis, and there was a marked upward and inward deviation of the right eye." Atropine was ordered and the fixing eye to be covered during part of the day.

There was no further medical attendance until, two years later, when she was taken to an eye surgeon in another town. The treatment was similar, but soon after her return home she became very ill: this illness was attributed to atropine poisoning. The drug was stopped and partial covering of the left eye advised. The child was

brought again in a year when no change was ordered but advice was given to return in a couple of years for operation. She was then three years old. After persisting, as directed, until early in 1931, her mother left off the shade entirely as she said it was injuring the good eye which had "gone small."

Her tonsils had been removed in 1929.

Early in the treatment a Wassermann test had proved negative.

On examination, I found each eye could fix equally well, though at a subsequent visit I thought the right fixed the better. When the right eye fixed, the left eye looked in and the left lid drooped. On the left eye fixing, the right eye turned up, and in—the eye being then widely open.

There was also a facial paralysis of the left side extending to tongue and lips; a weakness of the levator of the left lid and a slight deafness on the left side.

R.V. = 6/6. L.V. = 6/6. No diplopia. Under atropine, each eye gave 6/6 and a normal refraction. Pupils evenly dilated. Reaction to light and convergence had not been noted. Occasionally, by stimulus or strong effort, a slight outward deviation was obtained in each eye. Knee jerks active and the Babinski reflex definite but not marked. As syphilis seemed negatived, a congenital nuclear deficiency or an encephalitis was considered. I could find no reason for the latter.

In seeking help to localize the lesion I found abundance of information in the recent work, chiefly that of Leslie Paton and Gordon Holmes.

Obviously the external rectus of each side appears defective, therefore the sixth nerve or its nucleus. For the facial paresis the proximity of the seventh nerve nucleus with its fibres sweeping around the sixth nucleus gives a clue. To account for the levator palpebrae superioris weakness is more difficult, as the nucleus of the third nerve is comparatively distant, but the diagram of the course of the fibres from the sixth nucleus in Parsons's¹ "Diseases of the Eye" shows the connection with the third nerve nucleus. He shows the anterior part of the great large-celled lateral nucleus of the third nerve is the probable source of the levator palpebrae superioris nerve supply.

Whitnall² says that though this was the source given by Bernheim, other workers have considered the posterior end of the nucleus as the origin of the fibres to the levator.

The up, and in, deviation of the right eye when the left fixes, indicates a probable involvement of the supply to the superior oblique muscle. The very close proximity of the fourth nerve nucleus to the third establishes touch here.

In trying to account for the defective hearing on the left side, I found guidance in the fine diagrammatic set of coloured plates in Leslie Paton's article "Vestibulo-ocular Reflex Paths."³ It shows the nucleus triangularis of the eighth nerve, in the floor of the

fourth ventricle giving "some direct fibres to the nucleus of the sixth nerve," which lies closely mesial to it at its proximal end. He also here traces the fibres coming from the nucleus of the descending root and passing into the posterior longitudinal bundles. "In all probability this nucleus forms an important relay on the reflex path, linking up labyrinthine receipts with movements of the eyes and of the limb muscles."⁴

Duke-Elder⁵ quoting Kappers (1920) emphasises the "position of the oculo-motor nuclei, with their close anatomical relationship to the posterior longitudinal bundles and vestibular system." The slight outward movements, noted occasionally on effort, might be due to reflex action, a paralysis of movement rather than of muscle; as Gordon Holmes suggests "the lesion responsible for it must involve certain paths converging on the oculo-motor nuclei rather than these nuclei or the nerves that arise from them,"⁶ also that such movements may be elicited by labyrinthine stimulation or other means.

One is forced to recognise the outstanding importance of the posterior longitudinal bundles in all ocular palsies. Their value as central relay stations is emphasised by the authorities I have mentioned. In the present state of my knowledge I suggest the sixth nerve nucleus on the right side as the chief seat of the defect in the patient. Unfortunately for a more complete history, she left here for a distant town and, so far, attempts to trace her have failed.

REFERENCES

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 4. *Ibid.*, p. 262.
 5. Duke-Elder.—Recent Advances in Ophthalmology, p. 330.
 6. Gordon Holmes.—Spasm of Fixation. *Trans. Ophthalm. Soc. of U.K.*, Vol. L, p. 254.
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A CONJUNCTIVAL BAND SIMULATING A PERSISTENT NICTITATING MEMBRANE

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UNDER the heading conjunctival bridges and pouches Herbert describes folds of the conjunctiva in the region of the superior fornix with or without adhesion to the tarsal conjunctiva.