PARESIS OF THE ABDUCENS NERVE FOLLOWING SPINAL ANESTHESIA *

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PALSY of the abducens nerve following spinal anesthesia undoubtedly is more frequent than reports indicate, and many cases of slight paresis are certain to be overlooked when patients complain of blurred vision which clears in a few days. Reber (1), in 1910, discussed the literature and summarized 36 cases, including 5 cases which he reported from the service of W. W. Babcock. Blatt (2) reviewed 97 cases in 1929, 6 of which occurred after spinal puncture, with no anesthesia; there were 6 cases of palsy of the oculomotor nerve, 4 of the trochlear nerve and the remainder of the abducens nerve. In 1950, Di Marzi (3) reported 7 cases. Fairclough (4) summarized 10 cases in 1945. Since then, other reports have appeared. In most cases the paresis follows spinal anesthesia, but Dattner and Thomas (5) described one case in which it followed simple lumbar puncture. In addition, Woltman (6), in 1936, reviewed 2 cases of palsy subsequent to ether anesthesia. The reported incidence of abducens palsy following spinal anesthesia varies from 10 in 2021 cases (4) to 0 in 2500 cases (7). Involvement of almost every cranial nerve has been reported, but in over 90 per cent the abducens nerve has been affected.

Hayman and Wood (8) advanced three theories as to the etiology: mechanical, toxic and inflammatory. (1) *Mechanical:* the long course of the nerve exposes it to injury. Fairclough (4), however, stated that the trochlear nerve has the longest intracranial course. If this theory is reliable, it is probably owing to the fact that the abducens nerve is relatively fixed in the cranium, is tight in Dorello's canal and, therefore, more vulnerable to stretching. (2) *Toxic:* direct action upon nerves or nuclei, or associated with reflex involvement. Koster and Weintrob (9) mentioned the animal experiments of Van Lier, who demonstrated swelling of ganglion cell nuclei after spinal anesthesia; they suggested this as a possible cause. Spielmeyer (10), in discussing stovaine spinal anesthesia in 1908, said that although many cells may be affected by the anesthetic agent, the outcome is probably more evident in abducens nuclei than in other nuclei since there are fewer cells in the nuclei of the eye muscles, particularly of the abducens, and damage is more apparent

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clinically. It must be remembered, also, that the abducens nerve has a superficial relation to the fourth ventricle, which communicates with the subarachnoid space into which the anesthetic agent is injected. Abducens paresis has occurred after anesthetization with almost every known spinal agent, and continues to occur despite the lower toxicity of present day agents. (3) *Inflammatory:* usually attributed to low grade meningitis. Babcock (11), in 1928, referring to 5 of his cases reported by Reber (1) in 1910, stated that these instances of paresis were caused by contaminated solutions of imported stovaine and tropococaine.

In 1945, Fairelough (4) proposed a new theory, indicating the loss of binocular single vision in esophorics as the basic entity. Nine of his patients examined after recovery were found to have esophoria. The 2 cases to be reported were exophoric. Although this theory may explain certain cases of transient blurred vision following anesthesia, it does not give the reason for the usual latent period or for the pronounced weakness of only one lateral rectus muscle. The mechanical theory explains neither the latent period between spinal anesthesia and the onset of paresis nor the predominance of unilateral lesions. Those cases which are produced after spinal puncture alone disclaim the toxic theory. Evidence against the supposition of an inflammatory cause lies in the fact that specimens of spinal fluids examined during the paresis have been found to be normal.

Abducens paresis is often said to be preceded by headache, nausea, dizziness, photophobia and stiff neck. In the patients reported there were no complaints of photophobia or stiff neck. Should the nausea and dizziness be directly related to the diplopia, the only relatively constant prodromal symptom is headache. The majority of cases occur from the third to the fifteenth postoperative day, and 90 per cent of the patients recover in eight weeks or less. The only treatment is the covering of one eye to eliminate diplopia and prevent nausea, and this covering should be discontinued as soon as possible. It is generally believed that surgical intervention should be deferred for a period of two years since a few patients have experienced spontaneous recovery twelve to eighteen months after the paresis occurred.

Of a total of 1581 spinal anesthesias at the Cleveland Clinic during 1950, 2 cases of abducens paresis occurred subsequently.

REPORT OF CASES

Case 1. A white woman aged 56 years was admitted to the hospital on February 27, 1950, with the diagnosis of cystocele, chronic cervicitis and old, third degree laceration of perineum. She had had diabetes mellitus for ten years. On March 2, 1950, an anterior colporrhaphy was performed under spinal pentothal anesthesia which consisted of pontocaine, 10 mg., and procaine, 100 mg. Ninteen cubic centimeters of 2.5 per cent sodium pentothal was also given during the course of the operation. Nitrous oxide and oxygen were administered by mask throughout the procedure. The blood pressure reading was 120 mm. systolic and 80 mm. diastolic when anesthesia was begun and fell to 70 mm.

systolic and 50 mm. diastolic thirty-five minutes later. Neosynephrine was administered and the blood pressure promptly rose to 120 mm. systolic and 80 mm. diastolic. The remainder of the anesthetic course was uneventful. The duration of administration was seventy-five minutes.

Postoperative Course.—On the first postoperative day, the patient complained of some blurring of vision on looking to the left. The blurring was not evaluated. She did not sit up until the eighth postoperative day, when she complained of severe, right-sided occipital and parietal headache, relieved upon lying down. She was discharged on the sixteenth postoperative day, but headache persisted, and she was unable to maintain the erect position with comfort until the twenty-fifth day after operation. On the twenty-first postoperative day, the patient's son noticed that her eyes seemed crossed. Seven days later she was observed in the Department of Ophthalmology. She stated that blurred vision had remained unchanged since the operation, although deviation had been noticed only one week previously. At the time of examination homonymous diplopia and paralysis of the left lateral rectus muscle were present. She had been seen previously in the Department of Ophthalmology on January 21, 1949, at which time eye movements appeared normal. On May 1, 1950, two months after operation the patient could rotate the left eye outward 15 degrees, but blurred vision was still present. This symptom disappeared in July 1950, approximately four months after its onset.

The patient was readmitted to the hospital on September 18, 1950, and posterior colporrhaphy was done under gas-oxygen-ether anesthesia on September 19, 1950. Both the operation and the postoperative period were uneventful and she had no eye complaints following this operation. On November 13, 1950, there was normal muscle balance with full lateral excursions. Exophoria of 7 prism diopters at 33 cm. and 3 prism diopters at 6 M was found.

Case 2. A white man aged 36 years was admitted to the hospital September 19, 1950, with a diagnosis of recurrent disk protrusion at the fifth lumbar segment. He had undergone a laminectomy for the same complaint in March 1948. The previous operation had been performed under spinal anesthesia at Johns Hopkins Hospital, and he had experienced no eye symptoms subsequently. A spinal puncture was done on the day of admission to the Clinic Hospital; the findings were normal. Exploration of the fourth and fifth lumbar and first and second sacral segments was performed on September 20, 1950, under spinal anesthesia, which consisted of 10 mg. of pontocaine and 59 cc. of 2.5 per cent pentothal. Nitrous oxide and oxygen was given by mask throughout the procedure. The anesthetic course was uneventful, and the blood pressure remained stable.

Postoperative Course.—The patient did not void spontaneously until twenty-four hours after operation, and he experienced mild difficulty in voiding for seventy-two hours. A moderately severe prefrontal headache was present for three days while the patient was still lying flat in bed; he was not allowed to be ambulatory until the tenth postoperative day. He did not experience an occipital headache or a typical postspinal puncture headache at any time. The patient's head was elevated in bed on the tenth postoperative day, and at that time he became aware of blurring of distant objects. On the following day, while sitting in a wheel chair, he noticed definite double vision in the distance, and experienced nausea. Examination revealed homonymous diplopia at distance, and on looking to the left at 33 cm. There was a pronounced esophoria at near vision in the

primary position, but no tropia. The left lateral rectus muscle was definitely weak. On the following day diplopia at near vision was demonstrated. The paresis remained stationary until the patient was discharged on the fourteenth postoperative day; it did not progress to complete paralysis. The patient was obliged to cover the left eye to prevent nausea. He used the eye shield for two and a half weeks, and then was conscious of diplopia only on reading or on looking to the left. Twenty-one to twenty-five days after the onset he noticed only occasional diplopia on looking to the left, and after this experienced no difficulty. On the thirtieth day after the onset of paresis, muscle balance was normal and no residual weakness could be demonstrated. At this time, esophoria of 1 prism diopter at 6 M, and exophoria of 3 prism diopters at 33 cm. were present.

SUMMARY

It is interesting that the first patient was subsequently anesthetized with ether, with no return of abducens paresis. The second patient had had previous spinal analgesia with no abducens involvement. It is unusual that a particular patient should experience abducens paresis following one episode of spinal anesthesia and not another. This lends support to the theory of a low grade infection caused by some flaw in the technic of administration although it is conceivable that the abducens nerve may have been damaged during the first spinal anesthesia to the extent that the second spinal anesthesia produced clinical symptoms.

The literature has been discussed, and 2 cases of paresis of the abducens nerve following spinal anesthesia have been presented. Symptomatic treatment only was used and recovery was complete in both cases.

Muscle surgery should not be performed early in these cases since recovery is usually spontaneous.

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