BILATERAL PARALYSIS OF THE ABDUCENS AND HYPOGLOSSAL NERVES FOLLOWING SPINAL ANESTHESIA *

CASE REPORT

CAPTAIN JACK L. MILOWSKY AND
MAJOR RAUL R. BETANCOURT

Medical Corps, Army of the United States

THE occurrence of cranial nerve paralyses following spinal anesthesia has been reported in the literature. The abducens nerve was involved in over 90 per cent of these reports. This complication is not limited solely to patients in whom anesthetic solution has been placed in the subarachnoid space. Less frequently, it has been reported following spinal puncture made for diagnostic and therapeutic reasons (1). Woltman reported two cases of paralysis of the abducens nerve following inhalation anesthesia (2).

The theories relating to the pathogenesis of this complication are legion. Hayman and Wood (3), in a review of etiologic factors, mentioned three possibilities: mechanical, toxic and inflammatory. Proponents of the mechanical theory attach prime importance to the suction and pressure effects produced by alterations in blood and spinal fluid pressure. The high incidence of the abducens nerve paralyses is explained by these mechanical factors acting on a nerve which is particularly susceptible because of its anatomical situation. "The long course of this nerve, its close relationship to a bony prominence and the extreme narrowness of the foramen through which it has to pass make it extremely vulnerable to environmental changes" (3). It is difficult, however, to postulate that the toxic and inflammatory causes are the sole etiologic agents, because paralyses have been reported following inhalation anesthesia and because spinal fluid findings following paralyses are negative in a majority of cases.

A definite syndrome has been described in which the onset of paralysis is preceded by the prodromal symptoms of headache, stiff neck, visual disturbances, nausea and vomiting. Very often, findings of meningeal irritation may be elicited. The time interval between operation and the manifestation of these symptoms varies from one to twenty-one days. Spontaneous gradual remission has been reported in the majority of patients. A guarded prognosis should be entert

* From the anesthesia section and the urological section of Schick General Hospital, Clinton, Iowa.

tained, however, as there are reports of permanent paralyses. Therapy should be directed along symptomatic lines. Correction of ocular deformities by resection or recession of extra-ocular muscles should not be considered until two years following the onset of symptoms.

Case Report:

An unmarried 21 year old Pfc. was admitted to the urologic service because of signs and symptoms of urinary calculi in the left kidney and ureter of one year's duration. The past history was essentially negative, except for occasional dizzy spells, lasting from five to ten minutes. On physical examination, no abnormalities were found.

The patient was brought to surgery for a nephrolithotomy. Preoperative medication consisted of morphine sulfate 0.016 Gm. and scopolamine 0.0004 Gm., given one and a half hours before operation. In the operating room, the blood pressure was 140 mm. systolic and 80 mm. diastolic, pulse 80 and respirations 18. Two hundred milligrams of procaine diluted in 4.5 cc. of spinal fluid was injected between the second and third lumbar vertebrae. Anesthesia extended up to the fifth dorsal segment. One-half hour after the spinal anesthetic was given, the blood pressure was 78 mm. systolic and 60 mm. diastolic, pulse 60 and respirations 14. Fifty milligrams of ephedrine sulfate was given intramuscularly. After fifteen minutes, the systolic pressure had risen to 120 mm. of mercury and the diastolic to 80 mm. of mercury. The pulse increased to 88. The remainder of the course of anesthesia and operation was uneventful.

Fifteen days postoperatively, the patient noticed marked double vision and difficulty in moving his tongue. At that time he had an occipital headache of the throbbing type, with pains referred up over the top of his head in the frontal area. Questioning revealed the presence of malaise and slight headache for two days prior to the onset of the diplopia. On physical examination, the pupils were dilated, equal and regular. Reaction to light was present and minimal. The patient used his left eye for vision and was able to fix it on objects. When doing so, the right eye was turned inward and downward. When the left eye was closed, the patient was able to use his right eye for vision. The extra-ocular muscles were normal except for bilateral paralysis of the external recti muscles. Examination of the fifth cranial nerve revealed no sensory abnormalities. Corneal reflexes were present and equal. The seventh and eighth nerves were within normal limits. The twelfth cranial nerve was paralyzed, as evidenced by inability to protrude or move the tongue to either side. No abnormalities of the deep reflexes of the upper and lower extremities could be elicited. No specific muscle weakness or incoordination could be demonstrated. Positional, vibratory, pain and temperature sensations were intact. The remainder of the physical examination was essentially negative.

Spinal fluid, taken the day of the onset of the diplopia, showed normal pressure. The specimen was clear. On microscopic examination 2 lymphocytes per cubic centimeter were found. No bacteria were obtained on smear or culture. Sugar, protein and chlorides were within normal limits. Serologic tests and colloidal gold curve were also negative.

Two weeks after onset of symptoms, the patient was able to protrude and move his tongue slightly to either side. Speech, which had been slurred, improved markedly. No change was found in the eye signs. One month after onset, there was almost complete restoration of lateral rotation of each eye separately, but conjunctive diversion still was poor. Two months later the patient was discharged to a convalescent center. At this time it was noted that there was complete restoration of lateral motion of each eye separately and the ability for conjunctive diversion had improved markedly. Movements of the tongue seemed normal.

Conclusion:

A case of bilateral paralysis of the abducens and hypoglossal nerves following spinal anesthesia has been reported.

Possible etiologic factors in the production of this syndrome have been discussed.

REFERENCES

1. Barrcux, A., and Bordes, L. A.: A Case of Transitory Paralysis of the Sixth Nerves Secondary to Spinal Puncture, Revue d'Oto-Neuro-Ophthalmologie, Paris 15–58 (Jan.) 1937.
2. Davis, L.; Haven, H.; Givens, J. H., and Emmett, John: Effects of Spinal Anesthetics on the Spinal Cord and its Membranes, J. A. M. A. 97: 1781–1785, 1931.
3. Hayman, Irving R., and Wood, Paul M.: Abducens Nerve (VI) Paralysis Following Spinal Anesthesia, Ann. Surg. 115: 864–868 (May) 1942.
4. Hingson, Robert A.: Ferguson, Charles H., and Palmer, Louis: Advances in Spinal Anesthesia, Ann. Surg. 118: 971–981 (Dec.) 1943.
5. Hyslop, G. H.: Spinal Anesthesia, Nervous System Sequelae, Surg., Gynec. & Obst. 1: 799–802, 1933.
6. Fawcett, K. R.: Extra-Ocular Muscle Paralysis Following Spinal Anesthesia, Minnesota Med. 14: 648–649, 1931.
7. Lesser, L. H.: Peripheral Neuritis as a Sequelae of Spinal Anesthesia, J. A. M. A. 101 31–32 (July) 1933.
8. Light, G.; Sweet, W. H.; Livingstone, H., and R. Engel: Neurological Changes Following Spinal Anesthesia, Surgery 7: 138–156 (Jan.) 1940.
9. Lundy, John S.; Essex, Hiram E., and Kernohan, James W.: Lesions Produced in the Spinal Cord of Dogs by a Dose of Procaine Hydro-Chloride Sufficient to Cause Permanent and Fatal Paralysis, J. A. M. A. 101: 1546–1549, 1933.
10. Thompson, K. W.: Spinal Anesthesia—Experimental Study, Surg., Gynec. & Obst. 58: 852–866 (May) 1934.
11. Waner, W. L.: Spinal Anesthesia, Illinois M. J. 67: 45–50, 1935.
12. Woltman, H. N.: Neurologic Complications, Post-Operative, Wisconsin M. J. 35: 427–436, 1936.