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Ipsilateral Mydriasis Following Carotid-artery Puncture during Attempted Cannulation of the Internal Jugular Vein

JOHN E. FORESTNER, M.D.

Attempted cannulation of the internal jugular vein may be complicated by injury to structures near the vein in the neck and superior mediastinum. With the various techniques reported for internal jugular venipuncture, a small incidence of pneumothorax, hematoma, and transient Horner's syndrome has been reported.[1,2] Recently, a right carotid artery was punctured during an attempted internal jugular vein cannulation, and subsequently it was found that the patient's right pupil was dilated and unresponsive to light. Evaluation of the possible mechanisms involved would suggest that stimulation of sympathetic pupillodilator pathways in the neck had occurred.

REPORT OF A CASE

A 59-year-old black woman was scheduled for an exploratory laparotomy because of an apparent mass in the gastric antrum. She was slightly obese, weighing 90 kg. Her only medications were chlorthalidone, for control of mild chronic hypertension, oral potassium supplements, and an unknown oral hypoglycemic agent, which the patient had been taking for two years for adult-onset diabetes mellitus. She reported occasional paresthesias in both feet, but the results of neurologic examination were entirely normal. A preoperative brain scan, chest x-ray, and electrocardiogram were also normal. Fasting blood glucose was 156 mg/dl, and a serologic screen for syphilis was negative.

An hour after premedication with diazepam, 7.5 mg, and scopolamine, 0.2 mg, im, anesthesia was induced with thiopental, 400 mg, iv, and continued with inhalation of 3 per cent enflurane in nitrous oxide and oxygen, 2:1. The trachea was intubated after paralysis with 5 mg pancuronium. A pad was placed under the patient's right shoulder, the jaw rotated slightly to the left, and the operating table was placed in a 15-degree head-down tilt. With a high anterior approach proceeding lateral to the carotid pulse, a 6-inch 16-gauge Massa needle was directed toward the internal jugular vein.[3] After return of bright red blood under high pressure was observed, the catheter was immediately withdrawn and firm pressure applied to the right side of the neck. The left external jugular vein was subsequently cannulated, and access to the central circulation obtained with a catheter passed over a J-wire.[4] During the laparotomy a duplication of the gastric antrum was resected and pyloroplasty performed.

An hour after incision, it was noticed that the right pupil was dilated and did not react to light, while the left pupil was miotic. Vital signs remained stable and no other neurologic abnormality was observed. The patient was mechanically ventilated for a short time in the recovery room, and on emergence from anesthesia was intact neurologically, except for a widely dilated right pupil that reacted poorly to light and did not react consensually with the left pupil, or accommodate to near vision. After four days the pupil could constrict to 3–4 mm in diameter and reacted briskly to light. Ten days postoperatively, pupil size and reactivity had completely returned to normal, with equal pupils fully reactive to light, both directly and consensually, and with good accommodation.

DISCUSSION

Mydriasis can be due to parasympathetic inhibition or sympathetic stimulation. Because parasympathetic innervation to the pupil is entirely intracranial, the sustained pupillary dilatation in this case was probably caused by stimulation of cervical sympathetic pathways. Pupillodilator fibers ascend in the neck in the sympathetic chain and ansa subclavia, to synapse in the superior cervical ganglion. They leave the chain to branch around the carotid artery within the temporal bone and proceed into the orbit. While considerable anatomic variation is reported, pupillodilator fibers have not been detected in the common carotid artery or its sheath.[5] It would therefore seem most likely that cervical sympathetics near the carotid artery were transiently irritated, from direct trauma or from pressure resulting from hematoma, after the carotid artery was inadvertently punctured.

While Horner's syndrome is the expected result of injury to the cervical sympathetics, mydriasis secondary to sympathetic irritation is occasionally seen prior to development of fullblown miosis and ptosis. This has been reported to occur with Pancoast tumors or tuberculosis of the apex, during early compression of the sympathetic chain, and following thyroid surgery.[6] Acute unilateral mydriasis has also been seen with therapeutic pneumothorax during tuberculosis treatment. Mydriasis in these cases can resolve, as seen in this patient, or progress to complete cervical sympathoplegia with a typical Horner's syndrome.

* Assistant Professor, Department of Anesthesiology, University of Mississippi Medical Center, Jackson, Mississippi. Present address: The Department of Anesthesiology, Emory University School of Medicine, Atlanta, Georgia.

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Address reprint requests to Dr. Forestner: Department of Anesthesiology, Henrietta Egleston Hospital for Children, 1405 Clifton Road, N.E., Atlanta, Georgia 30322.

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Hematoma, trauma, or extravasation of fluid or drugs may damage vulnerable neurologic structures in the neck and superior mediastinum. Significant neurologic deficits resulting from attempted percutaneous puncture of the internal jugular vein have been reported. In one patient both carotid arteries were punctured during attempts to locate the jugular veins.[7] The resulting hematomas caused recurrent laryngeal nerve paresis and firm apposition of the vocal cords. A tracheostomy was necessary until normal cord function returned in several weeks. In another case, extravasation around an indwelling internal jugular catheter damaged the sympathetic chain, cervical plexus, and the last four cranial nerves on the left side.[8] The neurologic deficits resulted in chronic pulmonary aspiration and were major factors in the patient's eventual death.

Direct ocular trauma, topical mydriatics, and concomitant neurologic disease were considered as other possible causes of unilateral mydriasis in this patient. The most plausible explanation for the clinical findings, however, would be sympathetic pupillodilator stimulation, due to direct trauma or pressure from hematoma around the carotid puncture. While the finding of unilateral pupillary dilatation seemed somewhat ominous when first discovered during anesthesia, it resolved, with no permanent neurologic deficit.

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Dextrose Affects Gravitational Spread of Epidural Anesthesia

Young R. Park, M.D.,* and Douglas W. Eastwood, M.D.†

Gravitational influence on the spread of local anesthetics in the epidural space has been identified by several investigators.[1–4] However, as a result of other factors, lumbar epidural anesthesia tends to spread to the thoracic area to a greater extent than to the caudal area. This is disadvantageous for the patient who needs perineal anesthesia for the second stage of labor. Many factors may influence the extent and direction of spread of anesthesia in the epidural space. This study was designed to determine whether administering the local anesthetic in a dextrose, 5 per cent, solution would decrease the thoracic spread of lumbar epidural anesthesia in pregnant women at term.

* Instructor in Anesthesiology.
† Professor of Anesthesiology.
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Address reprint requests to Dr. Eastwood.

Materials and Methods

In a double-blind design, 60 patients for whom epidural anesthesia had been selected for vaginal delivery or for cesarean section were randomly assigned to control or study groups. The Human Research Review Committee approved the study protocol, and informed consent was obtained from each patient. Obese patients (weighing more than 100 kg) and patients whose progress in labor was so rapid that determination of the anesthetic level was not feasible were excluded.

Chloroprocaine, 0.5 per cent; was used to provide analgesia for labor, and 2.7 per cent was used for delivery or cesarean section. Patients in the study group received anesthetic solutions containing dextrose, 5 per cent, while solutions for the control groups were diluted with physiologic saline solution. Solutions were prepared and numbered according to a random table by the hospital pharmacist. Anesthesiologists selected the desired anesthetic con-

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