

started, if it was not already being given, or increased if it was. During that final preoperative day the patient should be monitored closely to ensure adequate blood pressure control with the hydralazine. This should avoid any hypertensive crisis, but if it does not, SNP may be used temporarily to regain control until effective hydralazine therapy is established.

Enflurane would be the current anesthetic of choice, since catecholamine-induced cardiac arrhythmias are less likely to occur with this agent than with others. Postoperatively, oral medications may be resumed fully as soon as the patient can manage to swallow and retain them. Obviously, those who have had gastrointestinal operations and are receiving gastric suction therapy will need maintenance therapy with parenteral drugs. Of the three vasodilators, hydralazine, prazosin and clonidine, only hydralazine is available for injection.

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Anesthesiology
51:92-94, 1979

Post-spinal Headache or Intracranial Tumor after Obstetric Anesthesia

DAVID D. ALFERY, M.D.,* M. LOU MARSH, M.D.,† HARVEY M. SHAPIRO, M.D.‡

Headache and sixth-cranial-nerve palsy are well-known complications of spinal anesthesia.¹⁻⁶ Subarachnoid blocks have also reportedly exacerbated symptoms of pre-existing neurologic disease.⁷⁻¹⁰ We are unaware of any report of a case of an intracerebral lesion that initially manifests as headache and abducens-nerve palsy following spinal anesthesia. We recently encountered such a case, in which symptoms

developed following a saddle block for a normal vaginal delivery.

REPORT OF A CASE

An 18-year-old Mexican-American primigravida was admitted to another hospital in labor after an uncomplicated term pregnancy. A low forceps vaginal delivery was facilitated by the use of a subarachnoid block administered by the obstetrician. A 25-gauge needle was introduced at the L₄₋₅ interspace atraumatically, and 40 mg (5 per cent sol.) lidocaine were injected. Paresthesias were not elicited. The patient was discharged from the hospital the following day.

Two weeks later she consulted a physician in Tijuana, Mexico, complaining of the recent onset of a retro-orbital headache, partially relieved by recumbency. There was no previous history of headache occurring during the pregnancy. She was unsuccessfully treated with ergotamine tartrate-caffeine tablets (Cafergot) and aspirin. Five weeks post partum she returned to her obstetrician with persistent headache, which he ascribed to cerebrospinal fluid (CSF) leakage. Bed rest, increased oral hydration, but no

* Senior Resident in Anesthesiology.

† Assistant Professor of Anesthesiology.

‡ Associate Professor of Anesthesiology and Neurosurgery.

Received from the University of California, San Diego, and Veterans Administration Hospital, La Jolla, California. Accepted for publication December 7, 1978.

Address reprint requests to Dr. Marsh: Department of Anesthesiology, University of California, San Diego, University Hospital, 225 West Dickinson Street, A.I.D. 7700, San Diego, California 92103.

medication was prescribed. Ten days later the patient returned to her obstetrician, complaining of diplopia and sustained headache. She was then referred to the Neurology Department at the University of California, San Diego for evaluation of these symptoms. On examination, a right sixth-cranial-nerve palsy and early papilledema were found. Extensive neurologic evaluation for suspected intracranial mass was then undertaken.

Roentgenograms of the skull, visual fields and analysis of CSF disclosed no abnormality. However, CSF pressures were not recorded. A computerized axial tomographic (CAT) scan revealed obliteration of the third ventricle and suprasellar cistern with moderate symmetrical dilatation of the lateral ventricles. Pneumoencephalogram showed a hypothalamic mass obstructing the right foramen of Monro and occupying the floor of the third ventricle. A diagnosis of hypothalamic tumor was made.

Seven days after admission the headache and papilledema were markedly increased. The patient underwent bilateral ventriculoperitoneal shunting, which resolved her symptoms.

DISCUSSION

Headache is a well-recognized complication following subarachnoid block. Dripps and Vandam described a 14 per cent incidence after 10,098 spinal blocks.¹¹ Moore and Bridenbaugh reported headaches in only 1.4 per cent of 11,574 patients who received subarachnoid block.¹² The pain is thought to be due to CSF leakage with resultant traction on pain-sensitive structures, primarily vascular and meningeal. Typically, the pain begins several minutes to a few days after the lumbar puncture. It is generalized over the calvarium, throbbing, and aggravated by the upright position.¹³ Most cases resolve within two weeks following conservative management, including supine bed rest, adequate hydration, and oral analgesics.

Cranial nerve palsies are also recognized sequelae of spinal anesthesia.¹⁻⁶ Ninety per cent involve the sixth cranial nerve. Dripps and Vandam found eight cases in 10,098 (.08 per cent),¹¹ Moore and Bridenbaugh, only one in 11,574 (.009 per cent),¹² and Phillips *et al.* reported eight in a series of 10,440 subarachnoid blocks (.08 per cent).¹⁴ Lateral rectus muscle weakness, a result of traction on the abducens nerve, almost always follows headache by several days. Nausea and photophobia may be concurrent. Typically, the palsy occurs within three weeks of the subarachnoid block, and it is most often unilateral. Ninety per cent of the cases resolve spontaneously within eight weeks of onset.

Although these findings occasionally complicate the post-spinal-anesthetic course, the need to investigate other causes for such neurologic symptoms cannot be overemphasized. Several reports have detailed exacerbations of pre-existing peripheral neurologic disease following spinal anesthesia.⁷⁻¹⁰ Dripps

and Vandam reported 12 patients in 10,098 who had exacerbation of neurologic deficits following subarachnoid blocks.⁹ The postoperative sequelae involved primarily peripheral phenomena: reappearance of backache, exacerbation of sciatica, leg weakness and sensory changes, urinary incontinence. An intracranial problem was documented in only one patient, a 12-year-old child in whom meningitis developed. Two patients manifested spinal cord compression syndromes caused by tumor; no patient had a pre-existing undiagnosed intracranial tumor. General anesthesia, however, has reportedly unmasked previously undiagnosed intracranial abnormalities. Edwards and Bras described repeatedly abnormal neurologic findings in a child awakening from anesthesia on two separate occasions.¹⁵ Subsequent operation revealed a tumor of the cerebellar pontine angle as the basis of the unilateral hypertonic emergence pattern. Thus, both regional and general anesthetics have served to reveal previously undiagnosed neurologic disease.

This case report is unique in that the patient's intracranial process first manifested as two recognized complications of the spinal anesthesia. It is impossible to determine whether the headache was due to the subarachnoid block or the intracranial mass. It is not unreasonable to consider that both processes may have contributed to it. The mass lesion and resultant increased intracranial pressure may have exaggerated a CSF leak caused by dural puncture. The abducens-nerve palsy could likewise have been dismissed as an anesthetic sequela, though its onset, at six weeks, was well beyond normal limits. Papilledema, however, could not be related to the subarachnoid block without invoking an infectious process, such as meningitis, in a patient who was otherwise asymptomatic.

In summary, a patient who underwent spinal anesthesia subsequently had a persisting headache followed by diplopia. Initially, the headache was felt to be a complication of anesthesia, but when diplopia occurred a thorough neurologic evaluation was undertaken. The primary cause was found to be a hypothalamic tumor, and the patient underwent a palliative procedure. Patients who have neurologic complaints following spinal anesthesia, especially those with unusual features or time courses, deserve proper evaluation, including a simple fundoscopic examination, to rule out the possibility of serious underlying neurologic disease.

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