15. Benedetti TJ, Kates R, Williams V: Hemodynamic observations in severe preeclampsia complicated by pulmonary edema. Am J Obstet Gynecol 152:330–334, 1985

16. Hatjis CG, Swain M: Systemic tocolysis for premature labor is associated with an increased incidence of pulmonary edema in the presence of maternal infection. Am J Obstet Gynecol 159: 723–728, 1988

17. Dantzker DR, Brook CH, DeHart P, Lynch JP, Weg JG: Gas exchange in adult respiratory distress syndrome and the effect of positive end-expiratory pressure. Am Rev Respir Dis 120:1039–1052, 1979

18. Cugell DW, Frank NR, Gaensler EA, Badger TL: Pulmonary function in pregnancy. I. Serial observations in normal women. Am Rev Tuberc 67:568–597, 1953

19. Cheek TG, Gutsche BB: Maternal physiologic alterations during pregnancy, Anesthesia for Obstetrics. Edited by Shnider SM, Levinson C. Baltimore, Williams & Wilkins, 1987, pp 3–13

20. Knuttgen HG, Emerson K: Physiological response to pregnancy at rest and during exercise. J Appl Physiol 36:549–553, 1974

21. Hagerdal M, Morgan CW, Sumner AE, Gutsche BB: Minute ventilation and oxygen consumption during labor with epidural analgesia. ANESTHESIOLOGY 59:425–427, 1983

22. Ueland K, Hansen JM: Maternal cardiovascular dynamics. III. Labor and delivery under local and caudal analgesia. Am J Obstet Gynecol 103:8–18, 1969

23. Shoemaker WC: Physiologic monitoring of the critically ill patient, Textbook of Critical Care Medicine. Edited by Shoemaker WC, Ayres S, Grenvik A, Holbrook PR, Thompson WL. Philadelphia, Saunders, 1989, pp 145–159

Anesthesiology
72:386–388, 1990

Intraoperative Diagnosis of Acute Subarachnoid Hemorrhage Using Continuous Pressure Monitoring *via* a Lumbar Subarachnoid Catheter

ALBERT A. KALUSTIAN, D.O.,* RANDALL S. GLIDDEN, M.D.†

Lumbar cerebrospinal fluid (CSF) drainage is often helpful in reducing intracranial volume during the surgical management of intracranial aneurysms, thereby decreasing the need for brain retraction and improving surgical exposure.[1,2] At our institution the surgeon often requests that a catheter be inserted in the lumbar subarachnoid space to withdraw CSF intraoperatively. It is our routine practice to continuously monitor the lumbar CSF pressure (LCSFP), usually a reflection of intracranial pressure (ICP).[3]

We report a case of a patient about to undergo clipping of a middle cerebral artery aneurysm, in whom aneurysmal rupture occurred shortly after induction of general anesthesia. The information provided by the lumbar catheter led to a major change in the operative plan and subsequent patient management.

* Resident in Anesthesiology, University Hospital.

† Assistant Professor of Anesthesiology, Boston University School of Medicine.

Received from the Department of Anesthesiology, University Hospital, and Boston University School of Medicine, Boston, Massachusetts. Accepted for publication September 18, 1989.

Address reprint requests to Dr. Glidden: Department of Anesthesiology, University Hospital, 88 East Newton Street, Boston, Massachusetts 02118.

Key words: Anesthesia, neurosurgical: intracranial aneurysm. Monitoring: cerebrospinal fluid pressure.

CASE REPORT

A 38-yr-old white woman was admitted to the hospital following a subarachnoid hemorrhage (SAH). Ten days after admission she was brought to the operating room for clipping of a middle cerebral artery aneurysm, at which time her Hunt clinical grade was III. Arterial and intravenous (iv) catheters were inserted and general anesthesia was induced with sufentanil, thiopental, esmolol, and lidocaine. Blood pressure and heart rate remained essentially unchanged during induction. General anesthesia and paralysis were maintained with sufentanil, oxygen, 60% nitrous oxide, and pancuronium. The patient was placed in the lateral decubitus position and a lumbar subarachnoid catheter was inserted at the L4–5 interspace, using an epidural anesthesia set. The CSF was clear and flowed freely; however, care was taken to avoid loss of more than 1–2 ml of CSF. The catheter was attached to a pressure transducer, and the LCSFP was continuously displayed by analog waveform and digital display. A waveform that fluctuated with respirations was observed, and an initial pressure of 11 mmHg was measured. The patient was positioned supine and placed in a Mayfield fixation apparatus without significant change in monitored parameters. During preparation and prior to skin incision, a sudden increase in the LCSFP from 10 to 150 mmHg was noted (fig. 1). This was accompanied by a sudden increase in the arterial blood pressure from 108/36 to 200/108 mmHg, and by an increase in heart rate from 52 to 130 beats/min. The patient was immediately treated with iv thiopental and esmolol, and 1% inspired isoflurane was added. The LSCFP, blood pressure, and heart rate gradually decreased and returned to baseline values within 30 min, at which time isoflurane was discontinued. Arterial blood gases, serum electrolytes, and complete blood count values were measured as normal following this event. Small aspirates of CSF from the lumbar catheter were initially clear but became blood-tinged within 5 min: cell count, 40,000 red blood cells (RBC) and 7 white blood cells (WBC) per millimeter.[3] Based upon this

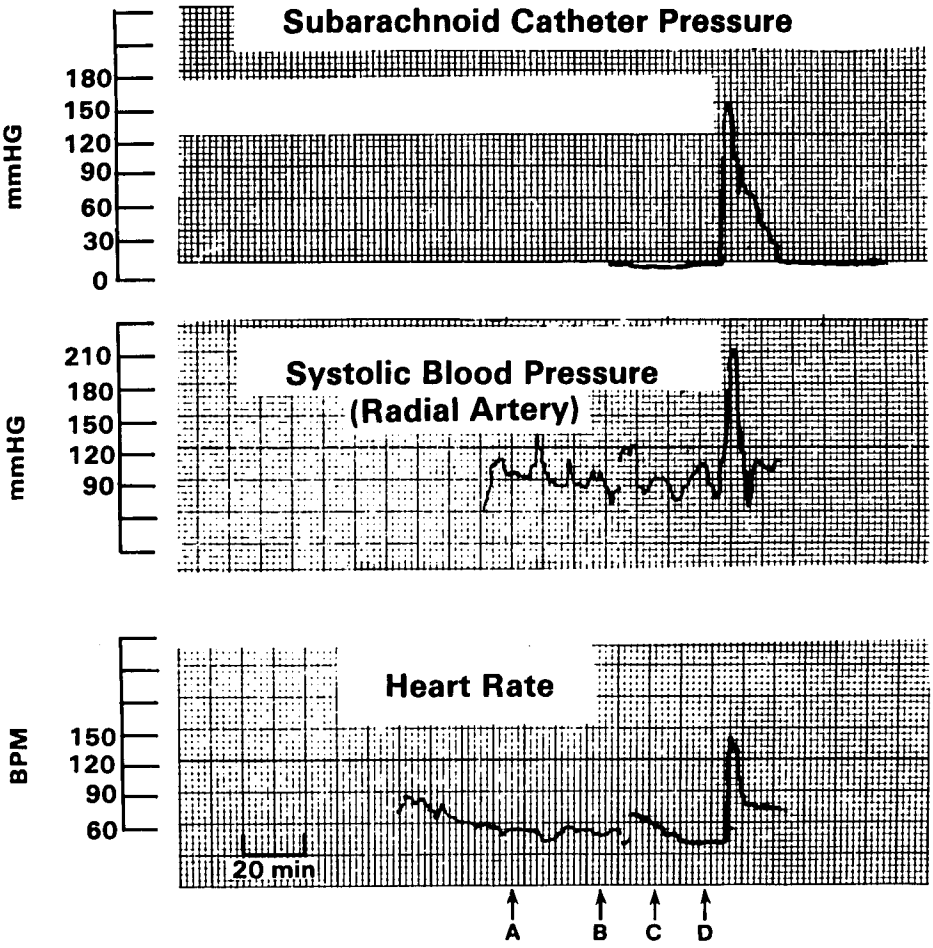

FIG. 1. Recordings of lumbar subarachnoid pressure, systolic blood pressure, and heart rate superimposed to reflect simultaneous measurements. Each large box equals 10 min. Arrows correspond to the following events: A = induction of anesthesia; B = lumbar subarachnoid catheter placement; C = application of Mayfield pins; D = surgical skin preparation.

information, the patient was presumed to have suffered a recurrent SAH. The planned operation was cancelled and a subarachnoid ICP bolt was inserted. The subarachnoid bolt pressure was found to be 7 mmHg, which was identical to the LCSFP measured at that time. The patient was transferred to the postanesthesia care unit for further management. Subsequent head computed tomographic (CT) scan later that day confirmed the diagnosis of a new SAH.

DISCUSSION

Our case demonstrates the usefulness of intraoperative LCSFP monitoring in a patient with an intracranial aneurysm. It has been suggested that monitoring the LCSFP during craniotomy may be useful in providing reassurance that aneurysm rupture has not occurred.[4] In this case, however, the sudden increase in LCSFP accompanied by hypertension and tachycardia aided in the diagnosis of acute SAH. Our suspicion that an SAH was responsible for the sudden increase in LCSFP was supported by the appearance of blood in the catheter aspirate and later confirmed by head CT scan. The diagnosis of an acute SAH prior to actual skin incision allowed for a change in the operative plan (*i.e.*, cancelling the craniotomy), which may not have occurred without this information.

After reviewing the literature, we could find no previously reported case in which LCSFP was monitored at the time of a SAH. The degree of increase of LCSFP and the associated arterial hypertension seen in our patient are similar to the findings in patients who suffered SAH while being monitored by continuous intraventricular catheters.[5] Hypertension is thought to represent a reflex response that may help maintain cerebral perfusion pressure in the presence of increased ICP.‡ The tachycardia accompanying our patient's SAH, rather than the bradycardia associated with the so-called Cushing's triad, has previously been reported in response to elevated ICP.[6]

‡ Brian JE, Eleff S, McPherson RW: Immediate hemodynamic management following subarachnoid hemorrhage during embolization of cerebral vascular abnormalities. J Neurosurg Anesth 1:63–67, 1989.

Because of the potentially useful information obtainable and the low additional risk incurred, we would recommend that LCSFP be continuously monitored in those patients in whom lumbar subarachnoid drainage is utilized.

References

1. Ojemann RG, Heros RC, Crowell RM: Intracranial aneurysms: General aspects of surgical treatment, Surgical Management of Cerebrovascular Disease. Edited by Ojemann RG, Heros RC, Crowell RM. Baltimore, William & Wilkins, 1988, pp 163–177
2. Yasargil MG: Anesthesia for microsurgical procedures in neurosurgery, Microneurosurgery. Edited by Smith RD, Young PH, Teddy PJ. New York, Thieme-Stratton, 1984, pp 272–278
3. Adams RW, Gronert GA, Sundt TM, Michenfelder JD: Halothane, hypocapnea, and cerebrospinal fluid pressure in neurosurgery. ANESTHESIOLOGY 37:510–517, 1972
4. Colley PS: Cerebral aneurysm management, Anesthesia and Neurosurgery. Edited by Cotrell JE, Turndorf H. St. Louis, CV Mosby, 1986, pp 407–443
5. Voldby B, Enevoldsen EM: Intracranial pressure changes following aneurysm rupture. III: Recurrent hemorrhage. J Neurosurg 56:784–789, 1982
6. Fitch W, McDowall G, Keaney NP, Pickerodt VWA: Systemic vascular responses to increased intracranial pressure. J Neurol Neurosurg Psychiatry 40:843–852, 1977