ists, there will be no effect and he may proceed as planned. However, in the presence of significant abnormality of pseudocholinesterase, a brief period of apnea and diminished twitch tension will ensue. The anesthesiologist may then be forewarned and avoid a normal dose of succinylcholine.

**CASE REPORT**

**Meningitis as a Complication of Anesthesia in a Patient with a Basal Skull Fracture**

**Luke M. Kitahata, M.D., Ph.D.,** and **William F. Collins, M.D.**

Although inhalation anesthesia by mask in a patient with a communication between the nasal cavity and the subarachnoid space might be expected to predispose to infection in the subarachnoid space, failure to consider this possibility and our inability to locate a report of this complication in the literature prompted this report. Our patient did not have overt cerebrospinal fluid rhinorrhea but did have other clinical evidence of post-traumatic cerebrospinal fluid fistula to the nasal sinuses. The severity of the complication and the ease with which it could probably have been averted makes its consideration mandatory in treating patients with trauma to the base of the skull and in elective cases of nontraumatic cerebrospinal fluid fistulae. A pressure gradient between the contaminated nasal cavity and the subarachnoid space with communication during mask ventilation appears to be necessary to produce the complication seen in this patient. Use of techniques other than positive-pressure mask anesthesia would avoid the complication.

**CASE REPORT**

A 23-year-old man was admitted to the Yale-New Haven Medical Center following an automobile accident. He was unconscious, responding only to painful stimuli with semipurposeful movements. Initial diagnoses were: cerebral concussion, basal skull fracture, multiple facial injuries, comminuted fracture of the left tibia, and soft tissue edema at the left elbow. X-rays revealed clouding of the right maxillary antrum and fluid levels in the ethmoid and sphenoid sinuses. During the initial evaluation and while x-rays were being taken, the patient became more alert but remained confused and combative. The tibia was aligned and immobilized in plaster, and as his neurologic status improved, he was taken to the operating room where the elbow wound was debrided under axillary block, using 20 ml of 1.5 per cent lidocaine with 1:200,000 epinephrine. Postoperatively, he continued to show improvement and was placed on ampicillin, 1 g q.i.d., as prophylaxis against infection from the basal skull injury. By the fifth hospital day, he was coopera-

---

* Assistant Professor of Anesthesiology.
† Professor of Neurosurgery.
Received from the Yale University School of Medicine, New Haven, Connecticut.
tive, oriented, showed no evidence of focal neurologic deficit, and was afebrile.

On the sixth day, closure of the left elbow wound, reanimation of the left tibia, and fixation of the fracture were performed with the patient under general anesthesia administered as follows: following intravenous administration of 200 mg of 2.5 per cent thiopental, nitrous oxide (4 l/min) and oxygen (2 l/min) were given by face mask. Respiration were briefly controlled by positive pressure (20 cm H₂O) ventilation following intravenous administration of 100 mg of succinylcholine. Endotracheal intubation with a #38 tube was accomplished without difficulty. Anesthesia was maintained with 60 per cent nitrous oxide and 40 per cent oxygen supplemented with intravenous d-tubocurarine. Respiration were controlled throughout by positive-pressure (up to 15 cm H₂O) ventilation. Duration of anesthesia was one hour and 45 minutes. The patient was awake and talking within a few minutes after the end of the procedure.

Approximately eight hours postoperatively, the patient's temperature rose to 104 F. He was placed on respiratory exercises and given aspirin, following which his temperature dropped to 102 F. During the next day he continued to have temperature elevation to 102.5 F and became confused and somnolent. On examination, he had a stiff neck with positive Kernig's and Brudzinski's signs. Lumbar puncture revealed an opening pressure of 170 mm H₂O. The fluid was turbid, slightly xanthochromic with 300 erythrocytes and 67,750 leukocytes/cu mm (100 per cent polymorphonuclear leukocytes). Gram-negative rods and gram-positive cocci were seen on smear. Medication was changed from ampicillin to gentamicin 75 mg q.s. 3h. im and oxacillin 2 g q.4h. iv.

The culture of the cerebrospinal fluid grew Klebsiella, sensitive to gentamicin. Nasopharyngeal culture taken at the time of the evening elevation of temperature also revealed the same organism.

During the next four days, he continued to have fever without improvement in meningeal signs or general level of consciousness. Two more lumbar punctures were attempted, but these showed pressures so low it was difficult to be certain that the needle was in the subarachnoid space. It was felt this signified either a block in the subarachnoid space secondary to infection or a decrease in pressure secondary to a cerebrospinal fluid fistula. Because the patient showed no improvement, the dose of gentamicin was doubled, and cisternal puncture was done with installation of 4 mg of gentamicin in the subarachnoid space. The cisternal puncture yielded a yellow, turbid fluid with approximately the same characteristics as the initial lumbar tap. Four mg of gentamicin were also introduced into the lumbar subarachnoid space on each of the next two days. Following this, the lumbar cerebrospinal fluid showed evidence of clearing, the fever began to subside, and the patient became more alert. He continued to improve, the cerebrospinal fluid cultures remained negative after the initial cisternal puncture, and, on the thirteenth postoperative day, the lumbar spinal fluid was clear with only a few lymphocytes and the pressure was normal. Oxacillin and gentamicin were continued for 19 and 21 days, respectively. The patient remained afebrile, alert, and was discharged on the twenty-fourth day after his second operation.

**Discussion**

There is a reasonable basis to support a direct relationship between the meningitis and the induction of anesthesia in this case. The main evidence is temporal. The patient was afebrile for four days before operation and then developed high fever eight hours after induction and florid meningitis about 24 hours after positive-pressure ventilation using a face mask. Nasopharyngeal culture showed the same organism as the spinal fluid culture. It is reasonable to assume that the basal cisternal fluid communicating with the paranasal sinuses was near atmospheric pressure at the time of the second operation. Use of positive pressure (20 cm H₂O) during controlled respiration with a face mask, prior to endotracheal intubation, would allow this pressure gradient to drive fluid from the nasal sinuses into the subarachnoid space.

Although this patient did not show obvious signs of cerebrospinal fluid rhinorrhea, the presence of fluid levels in the paranasal sinuses was an indication of a communication with the subarachnoid space. The subsequent clinical course emphasized to us that inhalation mask anesthesia with positive-pressure ventilation should have been avoided. Regional anesthesia, when feasible, is probably the best anesthetic technique in these patients. If general anesthesia is required, a direct tracheal airway should be established prior to the induction of anesthesia. This may be done either by an awake endotracheal intubation or by tracheostomy. Since our experience with this case, a patient with spinal fluid communication to facial wounds has been managed by awake intubation and endotracheal anesthesia, with a resultant smooth postoperative course.

**Reference**