A 4-year-old sexually intact female French Bulldog (weight, 7.3 kg [16.1 lb]) was evaluated at the MJR-VHUP because of lethargy, anorexia, and chronic rhinitis-sinusitis. The dog had an 18-month history of nasal discharge and had been treated with antimicrobials, prednisolone, and antihistamines. A rhinotomy was performed 3 months prior to the initial evaluation.

Clinical Findings—On initial evaluation, intraventricular pneumocephalus and sinusitis were diagnosed; CSF analysis revealed high total protein concentration and mononuclear pleocytosis. The dog’s condition improved with treatment. Two weeks after discharge, it was treated by a local veterinarian because of upper airway obstruction; 3 days later, the dog was referred because of seizures. Computed tomography revealed a large fluid-filled, left lateral ventricle and a soft tissue mass protruding through a cribiform plate defect. The mass was histologically consistent with brain tissue. Findings of cytologic examination of a CSF sample were indicative of septic, suppurative inflammation, and bacteriologic culture of CSF yielded Escherichia coli.

Treatment and Outcome—Amputation of the herniated olfactory bulb and antimicrobial treatment resolved the septic meningoencephalitis, but neurologic deficits recurred 6 weeks later. Definitive correction of the cribiform plate defect with bone and fascial grafts was attempted. Postoperative rotation of the bone graft resulted in cerebral laceration and hemorrhage, and the dog was euthanized.

Clinical Relevance—Findings suggest that following dorsal rhinotomy and nasal polyectomy surgery, the dog developed herniation of the left olfactory bulb, intraventricular pneumocephalus, and septic meningoencephalitis because of a cribiform plate defect. Care must be taken to prevent rotation of bone grafts used in cribiform plate defect repair. (J Am Vet Med Assoc 2006;229:240–245)
The next morning, the dog was anesthetized for CT and rhinoscopic examinations. The CT scan revealed an absence of the nasal turbinates, nasal septum, and portions of the nasal and frontal bones (consistent with previous rhinotomies). A focal mass of soft tissue density partially obstructed the rostral aspect of the nasopharynx. A defect with sharp borders was detected in the left ventral aspect of the cribriform plate; the defect was associated with a 7 × 5 × 5-mm pocket of gas in the left frontal lobe. Further caudally, a large amount of gas was present in the left lateral ventricle (ie, intraventricular pneumocephalus) and a lesser amount of gas distended the third ventricle. There was a moderate midline shift of the falx cerebri to the right that was associated with the large left lateral ventricle (Figure 1). Rhinoscopy revealed inflamed, erythematous nasal mucosa with serous discharge. Via an endotracheal wash procedure, samples were collected to rule out an infectious cause of the bronchointerstitial pattern that had been detected radiographically. Cytologic examination of wash specimens revealed low numbers of neutrophils with occasional ciliated respiratory epithelial cells and mucus, which were consistent with mild, acute inflammation; however, subsequent aerobic bacterial culture of wash samples yielded no growth. Because of the cribriform defect, history of infectious rhinitis, and presence of the pneumocephalus, a cisternal CSF aspiration was done. Cytologic examination and fluid analysis of the CSF sample revealed high total protein concentration (54 mg/dL; reference range, < 25 mg/dL) and mononuclear pleocytosis (7 nucleated cells/µL; reference range, < 5 nucleated cells/µL). The nucleated cells consisted of small lymphocytes (78%) and macrophages (22%). Bacterial culture of CSF did not reveal growth of aerobic or anaerobic bacteria.

The dog recovered from anesthesia without complications and was eating and drinking well the following day. The dog was discharged, and the owner was instructed to administer trimethoprim sulfamethoxazole (15 mg/kg [6.8 mg/lb], PO q 12 h) for 2 weeks and continue oral administration of prednisone and azithromycin. The dog did well at home for approximately 2 weeks, at which time it developed stridor and lethargy. An obstruction of the upper portion of the respiratory tract and an elongated soft palate were diagnosed by the local veterinarian, who performed a soft palate resection. The dog improved over the following 3 days but then developed another obstruction of the upper portion of the respiratory tract. A tracheostomy was performed, and the dog’s respiratory status improved; however, the following day, the dog had 2 seizure events (at an interval of approx 2.5 hours) and responded to treatment with diazepam (0.5 mg/kg [0.23 mg/lb], IV) on both occasions. The dog became stuporous and was referred to the MJR-VHUP for further evaluation.

On arrival at the MJR-VHUP Emergency Service, the dog was laterally recumbent and minimally responsive; menace response was absent bilaterally. General proprioception was decreased in all 4 limbs and worse on the right side. Prosencephalic disease, in which the left side of the brain was more affected than the right, was suspected. At this time, the dog weighed 5.9 kg (13 lb). A CBC revealed mild mature neutrophilia (16,180 cells/mL); mild normocytic, normochromic, nonregenerative anemia (Hct, 36.4%; reference range, 40.3% to 60.3%); and moderate thrombocytosis (562,000 platelets/mL). Results of serum biochemical analyses were unremarkable. Thoracic radiography revealed a mild, diffuse bronchointerstitial pattern throughout all lung lobes.

Because of the rapid neurologic deterioration, the dog was anesthetized for an emergency CT scan, which revealed a large fluid-filled left lateral ventricle and associated midline shift of the falx cerebri to the right (Figure 2). Following IV administration of iohexol (2.2 mL/kg [1.0 mL/lb]), the choroid plexus of the left lateral ventricle appeared more hyperdense on the CT image than the choroid plexus of the right lateral ventricle, suggestive of local inflammation of the former. A non–contrast-enhanced soft tissue mass that was contiguous with the left olfactory bulb was detected in the nasal cavity; this mass was presumed to be either herniation of the left olfactory bulb through the defect in the cribriform plate or extension of inflammatory debris from the nasal cavity into the cranial vault.

After the CT procedure, a cisternal CSF sample was collected for analysis; cytologic examination revealed evidence of severe suppurative inflammation with bacterial infection. The CSF total protein concentration was 441 mg/dL, and the nucleated cell count was 19,895 cells/µL; the nucleated cells consisted of variably degenerated neutrophils (98%), macrophages (2%), and scant RBCs. Rarely, neutrophils contained intracellular coccobacilli or short rods. Escherichia coli that was resistant to ampicillin, amoxicillin trihydrate-clavulanate potassium, cefazolin, cefoxitin, celtazidine, ceftriaxone, enrofloxacin, marbofloxacin,
and ciprofloxacin was grown on bacterial culture of CSF. The organism was susceptible to amikacin, cefotaxime, imipenem, and tobramycin. A large soft tissue mass in the nasal cavity was confirmed rhinoscopically. Results of histologic evaluation of biopsy samples of the mass were consistent with brain tissue with associated hemorrhage and neutrophilic inflammation.

The dog recovered from anesthesia and was moved to the intensive care unit at the MJR-VHUP. Initial postoperative treatment included administration of cefotaxime (25 mg/kg [11.4 mg/lb], IV, q 4 h) and clindamycin (10 mg/kg [4.5 mg/lb], IV, q 8 h) pending results of bacterial culture of the CSF sample. During the next 5 days, the dog improved and was able to ambulate without assistance; the dog had no additional seizures. Because the dog had been treated with prednisolone for several months, administration of prednisolone was continued at a tapering physiologic dosage (initially, 0.25 mg/kg [0.11 mg/lb], PO, q 12 h; tapered to 0.1 mg/kg [0.045 mg/lb], PO, q 48 h over 3 weeks; and then discontinued).

Six days after the second evaluation at the MJR-VHUP, the dog was anesthetized and underwent debridement of the necrotic brain tissue; also, an attempt was made to repair the defect in the cribriform plate. A large amount of green mucoid material was present in the frontal sinuses and the nasal cavity; a sample was collected for microbial culture before the material was removed via gentle lavage with physiologic saline (0.9% NaCl) solution and suction. There was no growth of aerobic or anaerobic bacteria or fungi on bacterial cultures of the mucoid material. The defect in the cribriform plate was identified, as were additional defects in the left frontal bone (dorsal to the cribriform plate defect). The defects were debrided; during surgery, it was noticed that the bone was softer than normal. A mass of necrotic tissue was identified in the nasal cavity after lavage. Following resection, a portion of the mass was submitted for histologic assessment; findings were consistent with subacute inflammation and associated granulation tissue, and there was dense fibrous connective tissue (which may have been dura mater) that was bordered by clusters of lymphocytes and plasma cells. Because of the severe necrosis, it was not possible to definitively determine histologically whether this tissue was brain or nasal mucosa. Sections of tissue were Gram stained or stained specifically to identify fungal organisms, but results were negative. A fat graft was harvested from the left paralumbar region and was placed over the approximately 3- to 4-cm-diameter cribriform defect as well as over defects in the frontal bone. A temporal fascial graft was then harvested and sutured in place over the fat graft in the cribriform plate defect with 5-0 polydioxanone suture in a simple interrupted pattern. The dog recovered from anesthesia without complications and continued to improve neurologically over the subsequent 6 days.

Attempts to remove the temporary tracheostomy tube were unsuccessful because of regurgitation and continued obstruction of the upper portion of the respiratory tract. Six days after surgical repair of the cribriform plate and frontal bone defects, the dog underwent surgery to create a permanent stoma at the tracheostomy site. At that time, CSF aspiration was repeated and cytologic examination of the fluid revealed mild mixed-cell pleocytosis (2 nucleated cells/µL consisting of large mononuclear cells [84%], small lymphocytes [14%], and well-preserved neutrophils [2%]); total protein concentration was 35 mg/dL. The dog recovered from anesthesia and was discharged from the hospital 5 days later. At this time, the dog's weight had increased to 7.6 kg (16.7 lb). The owner was instructed to administer cepodoxime (3.0 mg/kg [2.3 mg/lb], PO, q 12 h) and phenobarbital (1.0 mg/kg [0.45 mg/lb], PO, q 12 h). At

Figure 2—Axial CT images of the skull of the dog in Figure 1 following rapid neurologic deterioration approximately 2 to 3 weeks after the initial evaluation. A—Image obtained before IV administration of contrast medium. Notice the dilated, fluid-filled left lateral ventricle (white arrowheads). B—Image obtained after IV administration of contrast medium. A mass with soft tissue density extends through the defect in the cribriform plate (white arrow) into the nasal cavity and is contiguous with the left olfactory bulb. See Figure 1 for key.
suture removal 10 days after discharge, the dog had moderate pelvic limb ataxia but findings of a neurologic examination were otherwise unremarkable. A mild mucopurulent nasal discharge was evident. Administration of cefpodoxime was discontinued, and treatment with chloramphenicol (35 mg/kg [15.9 mg/lb], PO, q 8 h) was started. Because the dog had no seizure activity and moderate pelvic limb ataxia, the dosage of phenobarbital was decreased to 1 mg/kg, PO, every 24 hours.

Eight weeks later, the dog was reexamined. It had continued to gain weight (9.75 kg [21.45 lb]). The dog had several episodes of mucopurulent nasal discharge as well as mucopurulent discharge from the tracheostomy site, for which the referring veterinarian had prescribed enrofloxacin and amoxicillin-clavulanic acid. Also, the dog had recently developed a cough after a new dog was introduced to the household. Results of a neurologic examination were within normal limits. On physical examination, mild erythema of the tracheostomy site and mild bilateral mucopurulent nasal discharge were evident. An endotracheal wash specimen was obtained for bacterial culture, and *Bordetella bronchiseptica* was identified; the organism was susceptible to chloramphenicol. No abnormalities were detected via a CBC and serum biochemical analyses. The dog was anesthetized, and MR images of the head were obtained by use of a 1.5-T magnet. The MR images were anesthetized, and MR images of the head were obtained by use of a 1.5-T magnet. The MR images revealed that the cribriform plate defect repair was intact. On the T1-weighted image, there was evidence of bilateral frontal sinusitis as well as a small defect in the left frontal bone adjacent to the frontal sinus; there was a small amount of epidural enhancement in the region of the defect after IV administration of gadolinium contrast medium (0.2 mL/kg [0.09 mL/lb]). Compared with previous CT findings, the degree of lateral ventricular hydrocephalus was decreased. Pending results of bacterial culture of the endotracheal wash specimen, administration of chloramphenicol was continued.

Approximately 6 weeks after the MR imaging procedure, the dog developed behavioral changes at home, including difficulty climbing stairs, polyphagia, and episodes of abnormal mentation that were consistent with brief absence seizures. Because of concerns regarding the abnormalities detected on the initial MR images and the change in neurologic status, an otolaryngologist (AGC) from the Hospital of the University of Pennsylvania was consulted about potential surgical intervention. In discussions, a concern was raised that the fat graft, which had been placed during the defect repair surgery in an area of potentially active infection, could serve as a continued nidus of infection. A decision was made to attempt a definitive repair of the cribriform plate defect. Eight weeks after the last evaluation, the dog was returned to the MJR-VHUP for surgery. At that time, results of clinicopathologic analyses were unremarkable; mild general proprioceptive deficits in the right thoracic and right pelvic limbs were detected, which were consistent with left prosencephalic disease. The dog was treated in the right thoracic and right pelvic limbs were detected, which were consistent with left prosencephalic disease. The dog was treated in the hospital for 5 days with cefotaxime (25 mg/kg, IV, q 6 h) before undergoing surgery. A cisternal CSF aspiration procedure was performed on the day of surgery; the CSF sample contained < 1 nucleated cell/µL (cell types included neutrophils [43%], large mononuclear cells [43%], and small mononuclear cells [4%]), and the total protein concentration was 34 mg/dL, all of which was suggestive of mild, acute inflammation.

During surgery, a large amount of yellow, gelatinous fluid was located in the frontal sinuses; samples were collected for microbial culture before the fluid was removed via suction. Aerobic and anaerobic bacterial and fungal cultures of the fluid yielded no growth. Scar tissue (with no evidence of necrosis) was firmly adhered to the previous defect of the left frontal bone; the brain was not adhered to the scar. A 1×1.5-cm piece of bone was removed from the left wing of the ilium and shaved down to a thickness of 2 mm. It was placed within the bony defect between the cribiform plate and the brain. A 2×2-cm fascial graft from the left temporal muscle was then obtained and positioned over the defect within the nasal cavity. A microfibrillar collagen hemostatic material (2 g reconstituted with sterile physiologic saline solution) was then packed into the nasal cavity against the defect.

During the evening after surgery, the dog had 1 seizure event and was given phenobarbital (4.0 mg/kg [1.8 mg/lb], IV, q 4 h [2 doses]) as well as mannitol (0.25 g/kg [0.11 g/lb], IV, q 12 h [1 dose]). The dog recovered well and was willing to eat on the following day. No additional seizures occurred until 3 days later, at which time the dog also became more lethargic. The next morning, the dog developed status epilepticus and was treated with constant rate IV infusions of diazepam (1 mg/kg/h) and propofol (0.1 mg/kg/h). Computed tomography revealed rotation of the bone graft into the brain parenchyma, severe intracranial hemorrhage, and intraventricular pneumocephalus of the left lateral ventricle (Figure 3). Because of the severe neurologic deterioration and poor prognosis, the owner elected...
euthanasia. Necropsy revealed bone graft displacement with severe, acute encephalomalacia of the olfactory peduncle and left frontal lobe and hemorrhage. Histopathologic findings included linear chronic encephalomalacia extending from the frontal gyrus into the mesencephalon; mild distension of the left lateral ventricle with chronic ependymitis, ependymal loss, ependymal proliferation, and subependymal gliosis; and moderate, multifocal, subacute lymphoplasmacytic, histiocytic, and neutrophilic meningoencephalitis.

Discussion
Pneumocephalus is a rare disorder in which gas accumulates within the cranium. Potential sites of gas accumulation include epidural, subdural, subarachnoid, cerebral, or intraventricular locations. If the gas accumulates under pressure, thereby causing localized or diffuse neurologic signs, it is termed tension pneumocephalus; most commonly, this develops intraventricularly. In humans, clinical signs are generally vague and include apathy and mild disorientation. Although rare, pneumocephalus has been reported as a complication of nasal polyectomy in people, most commonly secondary to damage to the cribriform plate or other parts of the ethmoid bone. To the authors’ knowledge, there are no descriptions of pneumocephalus or septic meningoencephalitis secondary to rhinotomy in the veterinary medical literature. In the dog of this report, we propose that the pneumocephalus resulted from damage to the cribriform plate at the time of rhinotomy and polypectomy or from a congenital nasoencephalocele that was present concurrently with the nasal disease, and that the olfactory bulb may have been inadvertently damaged at the time of surgery. Unfortunately, because CT or MR images were not obtained prior to the first rhinotomy, the latter proposal cannot be confirmed or refuted. The most common complications of rhinotomy reported in cats and dogs are subcutaneous emphysema, intraoperative bleeding from the conchae, oronasal fistulae, and persistent nasal discharge. In 2 case reports of pneumocephalus in dogs, both had tension intraventricular pneumocephalus involving the lateral ventricles that developed as a complication of transfrontal cranietomy. One of those dogs had severe neurologic signs including seizures and an absent menace response, whereas the other dog was more mildly affected and had signs of mental depression and mild postural reaction deficits. Both dogs had serous nasal discharge, considered by the authors of those reports to be CSF rhinorrhea, and both recovered well after surgical correction of the dural defect.

Intraventricular pneumocephalus suggests the presence of a defect in the dura (allowing entrance of air into the cranium) as well as a fistula in the brain parenchyma to the ventricular system. Two main mechanisms have been proposed to explain the entry of air into the cranium: the ball valve (or positive-pressure) mechanism and the CSF leak (or negative-pressure) mechanism. When a defect in the dura mater and a fistula in the brain parenchyma are present, coughing or sneezing results in increased extracranial pressure in the region of the defect and air is forced through the dural defect. At the cessation of the cough or sneeze, the intracranial pressure is then higher than pressure on the other side of the dural defect and the brain parenchyma seals the defect, trapping air within. The air can then pass through the fistula into the ventricle. In addition, in the presence of a CSF leak and a parenchymal fistula, pressure decreases within the ventricular system because of the loss of CSF, which can result in an influx of air. Persistent serous nasal discharge was present in the dog of this report after both rhinotomies, and although it was postulated at the time that the nasal discharge was attributable to inflammation, it is possible that the discharge was in fact CSF rhinorrhea. Cerebral spinal fluid rhinorrhea can be diagnosed by measurement of glucose concentration in the serous nasal discharge; concentrations of glucose > 10 mg/dL in the nasal discharge in the absence of blood contamination are suggestive of CSF rhinorrhea. However, rhinoscopy was performed immediately after CT in the dog of this report, resulting in persistent blood contamination of the nasal discharge, making it impossible to definitively diagnose CSF rhinorrhea on the basis of the glucose concentration. Other options for diagnosis of CSF rhinorrhea in the presence of blood contamination include high-resolution CT, CT cisternography, radionuclide cisternography, and intrathecal administration of fluorescein into the lumbar portion of the vertebral canal during surgical exploration of the nasal cavity. Given the CT evidence of a nasoencephalocele, it is possible that CSF rhinorrhea was present in the dog of this report, but a definitive diagnosis was not obtained.

Current recommendations in the human medical literature for the management of CSF rhinorrhea following surgery depend on the clinical course. In patients with an onset of CSF rhinorrhea within 48 hours of surgery and no evidence of pneumocephalus, conservative medical management with administration of antimicrobials, laxatives (to reduce straining during defection), and cough suppressants and restricted activity is preferred. For patients with concurrent pneumocephalus, delayed onset of CSF rhinorrhea, or neurologic signs, surgical or endoscopic correction of the defect is recommended because of the high risk of meningoencephalitis.

The association between pneumocephalus and the development of septic meningoencephalitis has not been well documented, but there is some evidence that the presence of pneumocephalus is a risk factor for the latter, independent of the presence of CSF rhinorrhea. A recent study of humans with mild to moderate head trauma and pneumocephalus revealed that approximately 20% of patients developed septic meningoencephalitis. In addition, CSF rhinorrhea and intracranial hemorrhage were identified as independent risk factors.

To the authors’ knowledge, this is the first case report of intraventricular pneumocephalus and septic meningoencephalitis that likely developed secondary to dorsal rhinotomy and nasal polypectomy in a dog. The development of septic meningoencephalitis in the dog of this report 2 weeks after diagnosis of the intraventricular pneumocephalus suggests that pneumocephalus may be
a risk factor for the development of septic meningoencephalitis in dogs. However, there are other potential mechanisms for entrance of bacteria into the ventricular and CSF spaces in dogs, including CSF drainage pathways into the lymphatics of the nasal mucosa along the olfactory nerve and around the cribriform plate; such pathways may have contributed to the development of septic meningoencephalitis in the dog of this report. In addition, immune suppression as a result of the administration of corticosteroids may have contributed to the development of septic meningoencephalitis.

The decision to attempt a final, definitive repair of the bone defect in the dog of this report was made on the basis of the development of neurologic deficits and the potential for the fat graft (placed at the time of the first repair surgery) to serve as a nidus of infection. Although samples of the mass of necrotic tissue obtained at the time that the graft was placed yielded negative results via microbial culture, samples were collected 6 days after diagnosis of severe septic meningoencephalitis for which the dog had been treated IV with antimicrobials, thereby increasing the likelihood of false-negative culture results. After the debridement was performed during the first surgery, the size of the dog’s skull defect was approximately 3 to 4 cm. In human medicine, it is considered standard of care to repair skull-base defects > 3 cm with bone grafts as an underlay against the dura to provide structural support and to place fascial grafts over the defect to add more layers and provide a tight seal to reduce the risk of infection. Fascial grafts alone act as scaffolds for scar tissue formation, but across large defects, they commonly tent and fail to provide a complete seal. It is possible that the development of neurologic signs in the dog of this report 6 weeks after the recheck MR imaging was the result of failure of the fascial graft. It is likely that rotation of the bone graft placed during the final surgical intervention occurred because of the previous amputation of the left olfactory bulb, which resulted in a focal intracranial void in the area of the graft. We suspect that securing the graft to the bone at the time of surgery or the use of multiple layers of fascia lata and temporalis fascia rather than a bone graft may have avoided this complication.

Given the presence of pneumocephalus and the development of serous nasal discharge 4 weeks after the initial rhinotomy, this case provides some support for the concept that current recommendations from the human medical literature for management of pneumocephalus and CSF rhinorrhea may also apply in veterinary medicine. However, it was not possible to definitively diagnose CSF rhinorrhea in the dog of this report, and this conclusion should be interpreted cautiously. The current recommendation for treatment of pneumocephalus and CSF rhinorrhea in humans suggests that although clinical signs may be mild, early surgical intervention may be warranted to prevent the development of septic meningoencephalitis.

References