# Transorbital craniectomy for treatment of frontal lobe and olfactory bulb neoplasms in two dogs

**Kathryn L. Duncan** DVM
**Charles A. Kuntz** DVM, MS
**James O. Simcock** BVSc

From Southpaws Specialty Surgery for Animals, Moorabbin, VIC 3189, Australia.

Address correspondence to Dr. Duncan (kathryn.l.duncan@gmail.com).

**CASE DESCRIPTION**
An 8-year-old spayed female Shih Tzu crossbreed dog (dog 1) and a 13-year-old neutered male Miniature Fox Terrier (dog 2) were evaluated for removal of neoplasms involving both the frontal lobe and olfactory bulb.

**CLINICAL FINDINGS**
Physical examination revealed decreased menace response and behavioral changes in both dogs. For dog 1, neuroanatomic localization of the lesion was the left forebrain region; for dog 2, neuroanatomic localization of the lesion was the right forebrain region. Both dogs underwent CT, and dog 1 also underwent MRI. Results of diagnostic imaging were consistent with frontal lobe and olfactory bulb neoplasia in both cases. Dog 1 had lysis of the frontal bone adjacent to the neoplasm.

**TREATMENT AND OUTCOME**
Both dogs underwent a transorbital craniectomy to permit surgical tumor removal. Dog 1 was discharged from the hospital 48 hours after surgery, at which time its mentation and cranial nerve examination findings were considered normal. Dog 2 developed neurologic deterioration after surgery but was ultimately discharged from the hospital after 72 hours, at which time its mentation appeared normal.

**CLINICAL RELEVANCE**
The transorbital approach to the cranium provided excellent access to facilitate removal of frontal lobe and olfactory bulb neoplasms in these 2 dogs. (*J Am Vet Med Assoc* 2021;258:1236–1242)

An 8-year-old 7.7-kg (16.9-lb) spayed female Shih Tzu crossbreed dog (dog 1) was referred for investigation and treatment of cervical hyperesthesia. Fourteen days earlier, the dog had been examined by the primary care veterinarian because of lethargy and aggression. The primary care veterinarian had sedated the dog and performed survey radiography of the cervical, thoracic, and lumbar portions of the vertebral column; no abnormalities had been identified. Results of a CBC performed by the primary care veterinarian indicated that all assessed variables were within reference limits. Serum biochemical analysis revealed low amylase activity (429 U/L; reference range, 500 to 1,500 U/L) as the only abnormality. The dog had been treated by the primary care veterinarian with robenacoxib (10 mg, PO) every 24 hours, gabapentin (100 mg, PO) every 8 hours, and tramadol (20 mg, PO) every 8 hours for 4 days with minimal improvement.

Clinical examination of dog 1 at our referral hospital confirmed cervical hyperesthesia. No neurologic deficits were identified. The dog was anesthetized and underwent CT[a] of the cervical, thoracic, and lumbar portions of the vertebral column. Lumbar subarachnoid puncture was performed to collect a sample of CSF, and CT myelography was also performed. No structural abnormalities were evident on the CT images. Cerebrospinal fluid analysis identified cytoalbuminologic dissociation with a total nucleated cell count of 2 cells/μL (reference range, < 5 cells/μL) and high total protein concentration (2.50 g/L; reference range, < 0.45 g/L). Serum C-reactive protein concentration (1.9 mg/L) was within reference limits (reference range, 0.0 to 10.0 mg/L).

On recovery from anesthesia, repeated neurologic examination of the dog revealed mild generalized proprioceptive ataxia with absent paw placement and delayed hopping on the right pelvic limb. Cranial nerve examination identified an absent left menace response and intermittent menace response on the right side. All other cranial nerve responses were intact. These findings were consistent with multifocal neurolocalization of the lesion.

Five days later, dog 1 was anesthetized and MRI[b] of the brain (before and after contrast medium administration) was performed, including acquisition of sagittal plane T2-weighted sequences; transverse plane T2-weighted FLAIR, T2*gradient recalled echo, and diffusion-weighted imaging sequences; and dorsal plane T1-weighted, Fourier-acquired, steady-state spoiled gradient recalled sequences. Magnetic resonance imaging revealed a large extra-axial mass at the level of the left frontal lobe and olfactory bulb, which was predominantly hypointense relative to gray matter on T2- and T1-weighted images **(Figure 1)**. A small ovoid region caudal to the mass was noted to be hyperintense on T2-weighted images and hypointense on T1-weighted images. The mass had heterogeneous gadolinium contrast enhancement with meningeal enhancement extending along the falx cerebri at the

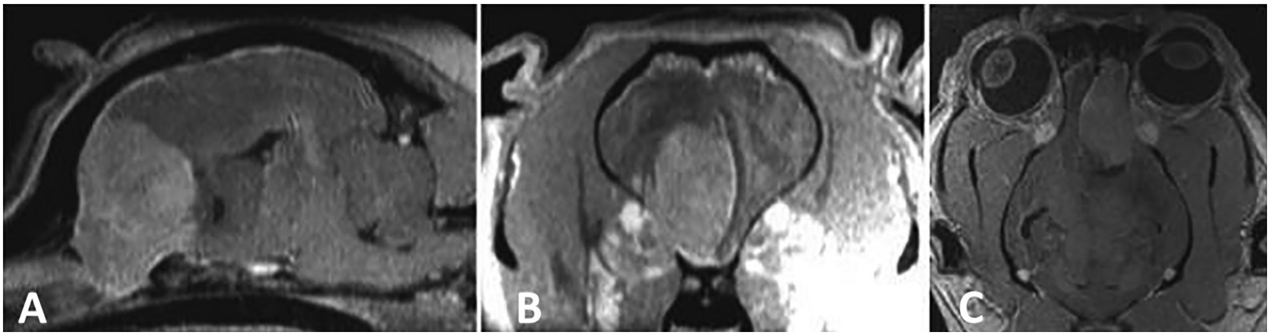

**Figure 1**—Sagittal (A), transverse (B), and dorsal (C) T1-weighted MRI images of the cranium of an 8-year-old Shih Tzu crossbreed dog (dog 1). The images were obtained after IV administration of gadolinium contrast medium. Notice the large mass within the left frontal lobe and olfactory bulb.

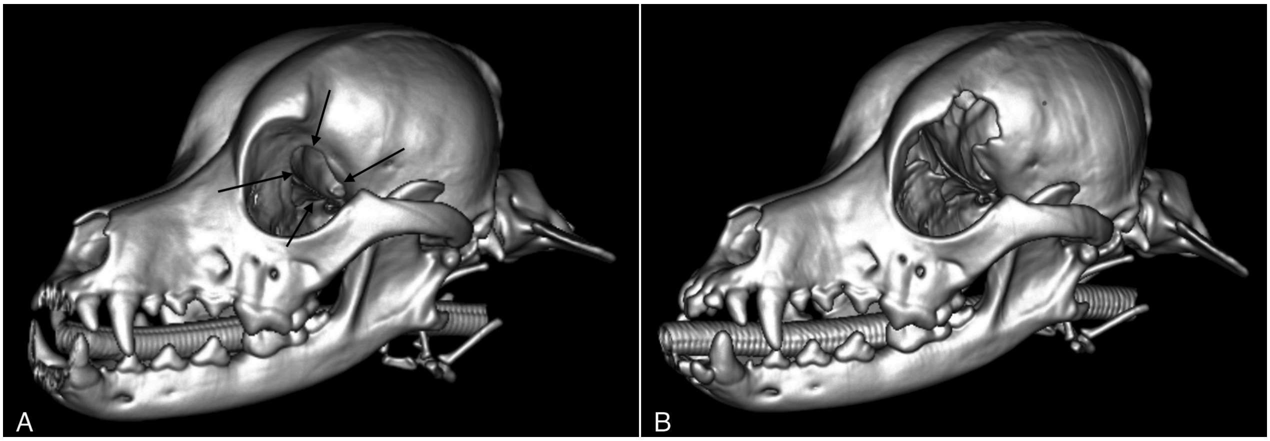

**Figure 2**—Computed-tomographic 3D volume rendering of the head of the dog in Figure 1. A—Preoperative image. A region of bone lysis in the suborbital region of the left frontal bone (arrows) is present. B—Postoperative image. Notice the extent of frontal bone removal following the transorbital craniectomy.

level of and caudal to the mass. There was a marked mass effect with rightward displacement of the falx cerebri, compression and displacement of the left lateral ventricle, and mild displacement of the third ventricle. Syringomyelia of the cervical portion of the spinal cord was also evident. The primary differential diagnosis was neoplasia with adjacent vasogenic edema. A cystic meningioma was considered most likely.

After discussion of all treatment options for the dog, including palliative medical care, resection, and radiation therapy, the owner elected to proceed with surgical intervention to remove the intracranial mass. To aid in surgical planning, CT scanning of the dog's cranium following IV administration of contrast medium was performed **(Figure 2)**. The mass involving the left frontal lobe and olfactory bulb was adjacent to a region of lytic frontal bone within the orbit. Because of the lysis of the adjacent frontal bone, a novel surgical approach was elected, which involved craniotomy via a transorbital approach.

The dog was anesthetized and positioned in sternal recumbency. The dorsal and left lateral aspects of the head and neck were clipped to remove the hair, and the skin was aseptically prepared for surgery. The head was fixed in position by placement of a custom metal brace under the mandibles and application of adhesive tape, ensuring the jugular veins were not compressed.

Cefazolin (22 mg/kg [10 mg/lb]) was administered IV to the dog at the time of anesthetic induction, and repeated injections were given every 90 minutes until the end of the surgical procedure. Pantoprazole (1.0 mg/kg [0.45 mg/lb], IV), mannitol (1.0 g/kg, IV), and methylprednisolone sodium succinate (15 mg/kg [6.8 mg/lb], IV) were administered. A continuous rate infusion of fentanyl (4 to 8 µg/kg/h [1.8 to 3.6 µg/lb/h], IV) was administered for intraoperative analgesia in combination with a continuous rate infusion of propofol (6 to 24 mg/kg/h [2.7 to 10.9 mg/lb/h], IV). The dog was intubated with a reinforced endotracheal tube for oxygen delivery.

A left supraorbital skin incision was made, which extended in a curvilinear fashion from slightly dorsomedial to the left medial canthus and ended caudodorsal to the lateral canthus **(Figure 3)**. The incision was continued through the subcutaneous tissues. The frontalis and temporalis muscles and periosteum were reflected laterally from the frontal

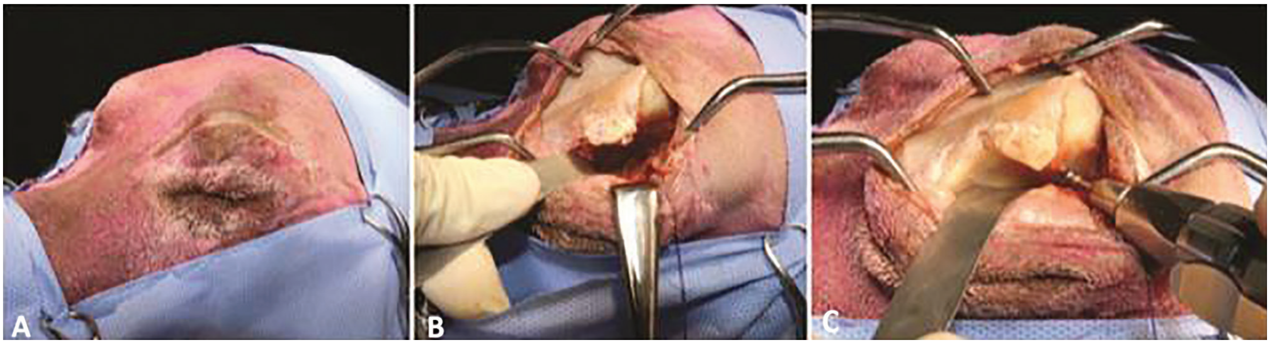

**Figure 3**—Photographs of a cadaveric reproduction of the surgical approach for transorbital craniectomy in dogs 1 and 2. A—Initial supraorbital skin incision. B—Ventrolateral retraction of the globe with a malleable ribbon retractor. C—Surgical exposure to facilitate craniectomy with a high-speed pneumatic drill.

bone. The orbital ligament was identified and transected. By use of Gelpi self-retaining retractors and a malleable ribbon retractor, the left globe was retracted ventrolaterally to facilitate exposure of the frontal bone on the medial aspect of the orbit. Gentle probing of the frontal bone confirmed the location of the bone defect overlying the mass, and after the dura was incised, grossly abnormal tissue was directly visualized through the bone defect. This defect was expanded with Kerrison rongeurs to improve exposure of the abnormal tissue. Caudal to the mass, brain tissue appeared grossly normal with macroscopic anatomic characteristics of gyri. The tumor tissue was gently excised by a combination of directly applied suction and blunt dissection with a nerve probe and Love-Gruenwald pituitary rongeur forceps. During dissection, the cribriform plate was inadvertently breached. Minimal intraoperative hemorrhage was controlled by gelatin sponge application and bipolar electrocautery. Retraction of the left globe was discontinued. The orbital ligament was not reconstructed. The temporalis and frontalis muscles were closed with 3-0 polydioxanone suture in a simple continuous pattern. Subcutaneous tissue was closed with 3-0 polydioxanone suture, and skin closure was achieved with 3-0 polydioxanone intradermal sutures, resulting in a normal appearance of the head. Computed tomographic examination was repeated immediately after surgery to assess the extent of the craniectomy (Figure 2).

Immediately after the surgical procedure, while the dog remained anesthetized, serum biochemical analysis revealed that it had hyperlactatemia (6.3 mmol/L; reference range, < 2.5 mmol/L]). There was no evidence of hypoperfusion at this time, and the dog recovered from anesthesia without complications. Serial assessment of serum lactate concentration was performed every 2 to 4 hours. Two hours after surgery, serum lactate concentration was 7.8 mmol/L; at 5 hours after surgery, hyperlactatemia had worsened (13.4 mmol/L). Over the subsequent 38 hours, serum lactate concentration consistently decreased without specific treatment but failed to return to the reference range (4.6 mmol/L).

The dog was maintained on a continuous rate infusion of fentanyl (2 to 8 µg/kg/h, [0.9 to 3.6 µg/lb/h], IV), and a fentanyl transdermal patch (25 µg/h) was applied. Pantoprazole (1 mg/kg) administration was continued IV once daily, and prednisolone (0.5 mg/kg [0.23 mg/lb]) was administered orally every 24 hours. In addition, ondansetron (0.5 mg/kg) was given orally to the dog every 12 hours.

Twelve hours after surgery, dog 1 was alert and ambulating well and had an excellent appetite. The dog was discharged from the hospital 2 days after surgery with apparently normal mentation and cranial nerve examination findings; it continued to have an excellent appetite. At that time, the dog's rectal temperature, heart rate, respiratory rate, and blood pressure measurements were within reference ranges.

The excised mass underwent histologic examination, the findings of which were consistent with a malignant anaplastic neoplasm. Marked cellular atypia and features of malignancy were present, and the cellular origin of the mass was not determined by routine staining. Differential diagnoses included histiocytic sarcoma and malignant (grade III) meningioma. Immunohistochemical analysis to further characterize the tumor was recommended but was declined by the owner, as was treatment of the dog with adjunctive radiation therapy.

Repeated examination of dog 1 at 2 weeks after surgery revealed no neurologic deficits and resolution of all hyperesthesia and aggression that was present prior to surgery. Metronomic chemotherapy was initiated to delay local tumor recurrence. The metronomic chemotherapy protocol included chlorambucil (4 mg/m$^2$) administered orally every 24 hours and lomustine (60 mg/m$^2$) administered orally once monthly for 5 doses.[c] Given the inconclusive nature of the histologic diagnosis, a tumor-specific chemotherapy protocol was not available. We elected to use the aforementioned protocol because of the ability of lomustine to penetrate the blood-brain barrier and preliminary evidence that metronomic chlorambucil may penetrate the blood-brain–tumor barrier in dogs.[c]

Dog 1 was reevaluated 101 days after surgery owing to the development of generalized tonic-clonic seizure activity. Repeated CT imaging of the dog's brain confirmed local tumor recurrence, and the owner elected euthanasia at this time. No postmortem examination was performed.

A 13-year-old 6.3-kg (13.9-lb) neutered male Miniature Fox Terrier (dog 2) was presented for investigation of suspected forebrain abnormalities. Four days earlier, the dog had been examined by the primary care veterinarian because of altered mentation with aggressive tendencies, generalized tonic-clonic seizure activity, and apparent blindness.

On physical examination, the dog displayed compulsive circling to the right and had obtunded mentation and reduced menace response bilaterally, all of which were signs consistent with neuroanatomic lesion localization to the right forebrain region. The remainder of the examination findings were unremarkable. A CBC was performed, and all variables were within reference ranges except for mean corpuscular hemoglobin (26.0 pg; reference range, 21.2 to 25.9 pg). Serum biochemical analysis was performed and revealed no abnormalities.

The dog underwent CT of the brain, and images were obtained after the dog received an IV injection of iodinated contrast medium. Computed tomography revealed a large, extra-axial, contrast-enhancing right frontal lobe and olfactory bulb mass with a marked midline shift **(Figure 4)**. The primary differential diagnosis was meningioma.

All medical and surgical options were discussed with the owner, and while options were being considered, treatment of the dog was commenced. The dog was administered prednisolone (0.5 mg/kg, PO, q 12 h), phenobarbital (4 mg/kg, IV, q 6 h as a loading dose), and levetiracetam (20 mg/kg [9.1 mg/lb], IV, q 8 h). The dog's neurologic status deteriorated over the following 12 hours. The dog's compulsive circling changed to circling in a counterclockwise direction, and it had reduced ability to ambulate. The owner elected that the dog should undergo resection of the mass the following day.

Dog 2 was anesthetized, positioned, and prepared for surgery in the same manner as dog 1; however, the dorsal and right lateral aspects of the head and neck were clipped to remove the hair. The anesthetic protocol used for dog 2 was identical to that used for dog 1, with the exception of a reduced dose (0.5 g/kg, IV) of mannitol administered at the time of anesthetic induction.

A right supraorbital skin incision was made, which was extended in a curvilinear fashion that started from a position slightly dorsomedial to the right medial canthus and ended caudodorsal to the lateral canthus. The initial surgical approach was identical to that previously described for dog 1, although a right-sided approach was used for dog 2. Following exposure of the frontal bone medial to the orbit, a 5-mm high-speed pneumatic drill was used to penetrate through the frontal bone, and Kerrison rongeurs were used to expand the bone defect. After the dura was incised, tumor tissue was directly visualized and an obvious gross demarcation between the neoplastic tissue, and adjacent unaffected brain parenchyma was evident. The tumor tissue was gently excised, as previously described for dog 1. The cribriform plate was breached during dissection, but caudally located nasal tissue was not encountered. Minimal intraoperative hemorrhage was treated with gelatin sponge application and bipolar electrocautery. Retraction of the right globe was discontinued. The orbital ligament was not reconstructed. Closure of the surgical site was achieved as described for dog 1, and the cosmetic outcome was considered excellent.

The dog recovered from anesthesia but developed persistent bradycardia with marked variation in arterial blood pressure measurements and a tendency for hypoventilation. Ongoing supportive measures were instituted including IV administration of mannitol (1.0 g/kg) and fluids. Administration of pantoprazole (1.0 mg/kg, IV) was continued once daily. Despite commencement of treatment with phenobarbital and levetiracetam only 1 day earlier, those medi-

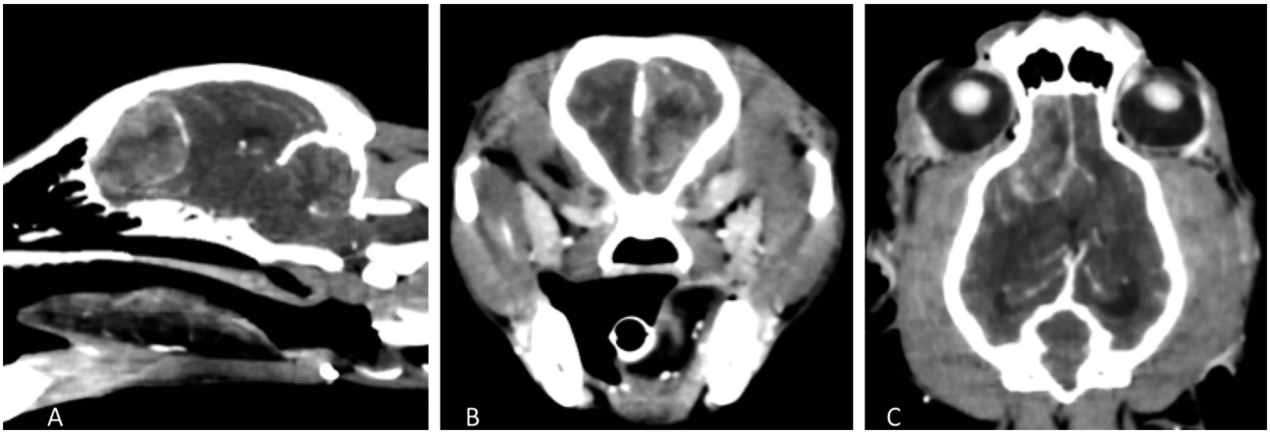

**Figure 4**—Sagittal (A), transverse (B), and dorsal (C) CT images of the cranium of a 13-year-old Miniature Fox Terrier (dog 2). The images were obtained after IV administration of iodinated contrast medium. Notice the large mass within the right frontal lobe and olfactory bulb.

cations were discontinued in the early postoperative period in case they were contributing to the hemodynamic abnormalities (bradycardia and blood pressure changes). In the immediate postoperative period, the dog was hyperlactatemic (3.3 mmol/L) despite having had a preoperative serum lactate concentration that was within the reference range. Hyperlactatemia (2.6 to 4.9 mmol/L) persisted during hospitalization, although the dog was assessed as having adequate perfusion. Twenty-four hours following surgery, the dog had persistent altered mentation with dysphoria and ongoing bradycardia, both thought to be secondary to increased intracranial pressure. A constant rate infusion of propofol (6 mg/kg/h, IV) was recommenced to control the dysphoria, and the constant rate infusion of fentanyl was continued. Forty-eight hours after surgery, there were no longer episodes of breakthrough dysphoria and the propofol infusion was discontinued. The dog was quiet, alert, and responsive and had an improved neurologic status with the ability to stand unassisted. The bradycardia had resolved, and blood pressure measurements were within reference ranges. The dog had an excellent appetite and displayed no further aggressive behaviors. There was rapid improvement in the dog's clinical signs over the subsequent 12 hours, with progression to unassisted ambulation and apparently normal cranial nerve examination findings. Three days after surgery, the dog was discharged from the hospital with only mild residual postural deficits on the left side.

Histologic examination of the excised tissue revealed evidence of spindle cell neoplasia suggestive of a meningioma (papillary type). Adjuvant radiation therapy was recommended for prolongation of survival time but declined by the owner.

Reexamination of the dog 1 week and 8 weeks after surgery revealed no neurologic deficits and resolution of all aggressive episodes and seizure activity. Vision was considered normal. At 294 days after surgery, dog 2 was alive but had recurrence of neurologic signs with intermittent generalized tonic-clonic seizure activity of approximately 3 months' duration, suspected to be secondary to recurrence of the neoplasm. The owners declined investigation of the dog's deteriorating neurologic status and elected not to commence treatment with anti-epileptic medication.

## Discussion

The cases described in the present report highlighted successful removal of intracranial frontal lobe and olfactory bulb neoplasms via a transorbital approach to the skull in 2 dogs. To the authors' knowledge, this approach has not been reported previously in the veterinary medical literature. A supraorbital approach to the cranium has been described in the human medical literature for treatment of various anterior and middle cranial fossa lesions,[1] including aneurysms and neoplasms, and there are some similarities between that supraorbital approach and the transorbital approach used in the 2 dogs. Craniotomy and craniectomy techniques commonly undertaken for removal of intracranial masses in dogs and cats include a transfrontal approach, modified transfrontal approach, temporoparietal approach, and suboccipital approach.[2–6] The decision regarding choice of surgical approach to the cranium ultimately relies on accurate localization of the lesion to be treated, which is typically determined from results of cross-sectional imaging.

Magnetic resonance imaging is an excellent technique for diagnosis of intracranial neoplasia and has been reported to be the gold standard for this purpose.[7] Compared with MRI, CT provides reduced parenchymal detail, but despite its comparatively poorer accuracy for localization of intracranial lesions, CT is more readily available and can also provide important information regarding surrounding bone structures, which can aid in surgical planning via 3-D volume rendering. Of the dogs in the present report, dog 1 underwent MRI to obtain a diagnosis and subsequent CT to enhance surgical planning and dog 2 underwent CT alone for purposes of diagnosis and surgical planning.

In dog 1, CT enabled identification of lysis of the frontal bone adjacent to the neoplasm in the left frontal lobe. Proposed causes for this lytic region of bone adjacent to the neoplasm included pressure necrosis from the mass as well as direct neoplastic invasion of the frontal bone. The presence of this bone defect adjacent to the neoplasm provided the impetus to consider a novel approach through this region.

For both dogs, a transorbital frontal craniectomy was considered to be superior to the more commonly used transfrontal approach for several reasons. With regard to dog 1, an approach to the neoplasm through the lytic bone region would permit resection of the abnormal region of cranium, which might then reduce the tumor burden if the bone lysis was secondary to direct invasion of the tumor tissue. In addition, the results of advanced imaging for dogs 1 and 2 indicated the tumors were directly adjacent to the suborbital region of the frontal bone without interposed parenchyma. Thus, the risk of iatrogenic injury to unaffected brain parenchyma was greatly decreased, compared with that associated with a standard transfrontal craniotomy.

Subcutaneous emphysema as a complication of intracranial surgery has previously been reported.[8] Breaching the frontal sinus or nasal cavity during intracranial surgery can result in leakage of air into the subcutaneous tissue. Although typically self-limiting, subcutaneous emphysema can take a prolonged time to resolve and poses a cosmetic concern for owners. An approach to the cranium by use of the transorbital technique negated the potential for breaching the frontal sinus and was therefore considered to reduce the risk for subcutaneous emphysema development. Although the nasal cavity was inadvertently entered via the cribriform plate in both dogs of the present report, the transorbital approach provided more overlying soft tissue structures for reconstruction, compared with the transfrontal approach, which likely improved the tissue apposition, reduced the dead space present, and prevented development of subcutaneous emphysema. A cranioplasty was not performed in these 2 dogs; however, such a procedure could be implemented, if required, in other cases.

Pneumocephalus is the accumulation of air within the cranium. Postoperative nonpathological (simple) pneumocephalus has been reported in the human medical literature and develops in up to 100% of human patients undergoing intracranial surgery.[9] Individuals with simple pneumocephalus are asymptomatic, and no specific treatment is indicated.[10] By comparison, postoperative pathological tension pneumocephalus in human patients results in symptoms such as headaches, seizures, and depressed neurologic status and requires emergency treatment.[11] Pathological tension pneumocephalus has been reported for only 3 dogs.[9,12,13] The development of this condition in humans is suspected to result from communication between the intracranial space and the atmosphere with a valve-like mechanism, allowing air to enter the cranium but preventing it from escaping, thereby causing development of a pressure gradient.[11,14–17] A surgical approach to the cranium that involves breaching the frontal sinus or cribriform plate may increase the risk of development of a such communication. Among the 3 dogs with pathological tension pneumocephalus,[9,12,13] the condition developed following a transfrontal approach to the cranium in 2 dogs and following rhinotomy with disruption of the cribriform plate in 1 dog. Unlike the transfrontal approach, the transorbital approach does not involve penetration of the frontal sinus; the risk of tension pneumocephalus may therefore be considered less likely, although the use of autologous tissues or synthetic materials for dural replacement in intracranial surgeries mitigates this risk. In both dogs of the present report, dural closure was not undertaken.

Early postoperative neurologic deterioration has previously been reported to occur in as many as 22 of 49 (45%) dogs undergoing intracranial surgery for a variety of underlying conditions.[8] Dog 2 of the present report developed postoperative clinical signs consistent with an increase in intracranial pressure. In addition, this dog had postoperative neurologic deterioration characterized by persistently altered mentation and dysphoria. The dog was treated with a continuous infusion of propofol while strategies were undertaken to normalize the intracranial pressure. The neurologic deterioration improved and ultimately resolved within 48 hours after surgery. Thereafter, the dog showed signs of steady improvement in mentation.

Both dogs of the present report had hyperlactatemia in the postoperative period despite perfusion variables that were within reference ranges. Hyperlactatemia in dogs with meningioma that are undergoing general anesthesia has previously been reported.[18] The high serum lactate concentration is thought be a result of local hypoxia of the tumor site and adjacent brain parenchyma. It has also been theorized that alteration in carbohydrate metabolism may be an underlying cause. Administration of corticosteroids has been associated with hyperlactatemia in healthy dogs,[19] and dogs 1 and 2 received corticosteroids within 12 hours after surgery. For either dog, serum lactate concentration did not normalize during hospitalization. The clinical importance of this postoperative finding is unknown.

Histopathologic findings for dogs 1 and 2 differed. Dog 1 had a malignant anaplastic neoplasm, whereas dog 2 had a papillary type meningioma. The prognosis for dog 1 was difficult to determine because the owner declined immunohistochemical analysis to further characterize the neoplasm. In a systematic review[20] of studies of brain tumor treatment in dogs, dogs with intracranial neoplasms had a median survival time of 312 days (range, 27 to 2,104 days) following surgical treatment alone. However, median survival time varies greatly on the basis of histologic classification of the neoplasm. In a previous study,[21] dogs with rostrotentorial meningiomas that were treated with resection alone had a median survival time of 422 days (range, 10 to 2,735 days), whereas dogs with rostrotentorial gliomas that were treated with resection alone had a median survival time of 66 days (range, 10 to 730 days). There is evidence to suggest that the addition of radiotherapy to the postoperative treatment regimen may improve the outcomes for dogs with intracranial meningiomas.[4,22] In both dogs of the present report, adjuvant radiation therapy was recommended but declined by the owners.

For the 2 dogs of the present report, transorbital craniectomy for removal of frontal lobe and olfactory bulb neoplasms allowed adequate visualization and preservation of perilesional brain parenchyma. Removal of the neoplasms ultimately resulted in resolution of clinical signs in both cases. One dog had postoperative deterioration of its neurologic status (suspected to have been secondary to increased intracranial pressure), which resolved with medical treatment. A transorbital approach to the cranium should be considered for dogs with lateralized frontal lobe and olfactory bulb neoplasms that are closely associated with the suborbital region of the frontal bone.

## Footnotes

a. 16-slice Aquilion Lightning CT scanner, Canon, Otawara, Japan.
b. 1.5-T Signa HD MRI machine, GE Healthcare, Milwaukee, Wis.
c. Bentley RT, Cohen-Gadol A, Jones D, et al. Metronomic chlorambucil chemotherapy for canine gliomas: a phase I/II clinical trial (abstr), in *Proceedings*. Am Coll Vet Intern Med Forum 2015;903.

## References

1. van Lindert E, Perneczky A, Fries G, et al. The supraorbital keyhole approach to supratentorial aneurysms: concept and technique. *Surg Neurol* 1998;49:481–490.
2. Parker AJ, Cunningham JG. Transfrontal craniotomy in the dog. *Vet Rec* 1972;90:622–624.
3. Kostolich M, Dulisch ML. A surgical approach to the ca-

nine olfactory bulb for meningioma removal. *Vet Surg* 1987;16:273–277.

4. Uriarte A, Moissonnier P, Thibaud J, et al. Surgical treatment and radiation therapy of frontal lobe meningiomas in 7 dogs. *Can Vet J* 2011;52:748–752.
5. Glass EN, Kapatkin A, Vite C, et al. A modified bilateral transfrontal sinus approach to the canine frontal lobe and olfactory bulb: surgical technique and five cases. *J Am Anim Hosp Assoc* 2000;36:43–50.
6. Oliver JE. Surgical approaches to the canine brain. *Am J Vet Res* 1968;29:353–378.
7. Edelman RR, Warach S. Magnetic resonance imaging (1). *N Engl J Med* 1993;328:708–716.
8. Forward AK, Volk HA, De Decker S. Postoperative survival and early complications after intracranial surgery in dogs. *Vet Surg* 2018;47:549–554.
9. Cavanaugh RP, Aiken SW, Schatzberg SJ. Intraventricular tension pneumocephalus and cervical subarachnoid pneumorrhachis in a bull mastiff dog after craniotomy. *J Small Anim Pract* 2008;49:244–248.
10. Ihab Z. Pneumocephalus after surgical evacuation of chronic subdural haematoma: is it a serious complication? *Asian J Neurosurg* 2012;7:66–74.
11. Schirmer CM, Heilman CB, Bhardwaj A. Pneumocephalus: case illustrations and review. *Neurocrit Care* 2010;13:152–158.
12. Garosi LS, Penderis J, Brearley MJ, et al. Intraventricular tension pneumocephalus as a complication of transfrontal craniectomy: a case report. *Vet Surg* 2002;31:226–231.
13. Fletcher DJ, Snyder JM, Messinger JS, et al. Ventricular pneumocephalus and septic meningoencephalitis secondary to dorsal rhinotomy and nasal polypectomy in a dog. *J Am Vet Med Assoc* 2006;229:240–245.
14. Wohlgemuth MV. Tension pneumocephalus. *J Neurosurg Nurs* 1985;17:146–154.
15. Andrews JC, Canalis RF. Otogenic pneumocephalus. *Laryngoscope* 1986;96:521–528.
16. Gozur JA. Postoperative tension pneumocephalus. *J Neurosci Nurs* 1987;19:30–35.
17. Arbit E, Shah J, Bedford R, et al. Tension pneumocephalus: treatment with controlled decompression via a closed water-seal drainage system. Case report. *J Neurosurg* 1991;74:139–142.
18. Sullivan LA, Campbell VL, Klopp LS, et al. Blood lactate concentrations in anaesthetized dogs with intracranial disease. *J Vet Intern Med* 2009;23:488–492.
19. Boysen SR, Bozzetti M, Rose L, et al. Effects of prednisone on blood lactate concentrations in healthy dogs. *J Vet Intern Med* 2009;23:1123–1125.
20. Hu H, Barker A, Harcourt-Brown T, et al. Systematic review of brain tumour treatment in dogs. *J Vet Intern Med* 2015;29:1456–1463.
21. Suñol A, Mascort J, Font C, et al. Long-term follow-up of surgical resection alone for primary intracranial rostrotentorial tumors in dogs: 29 cases (2002–2013). *Open Vet J* 2017;7:375–383.
22. Axlund TW, McGlasson ML, Smith AN. Surgery alone or in combination with radiation therapy for treatment of intracranial meningiomas in dogs: 31 cases (1989–2002). *J Am Vet Med Assoc* 2002;221:1597–1600.

**From this month's *AJVR***

## Effects of perineural administration of ropivacaine combined with perineural or intravenous administration of dexmedetomidine for sciatic and saphenous nerve blocks in dogs

Vincent Marolf et al

**OBJECTIVE**
To evaluate the effects of using ropivacaine combined with dexmedetomidine for sciatic and saphenous nerve blocks in dogs.

**ANIMALS**
7 healthy adult Beagles.

**PROCEDURES**
In phase 1, dogs received each of the following 3 treatments in random order: perineural sciatic and saphenous nerve injections of 0.5% ropivacaine (0.4 mL/kg) mixed with saline solution (0.04 mL/kg; DEX0PN), 0.5% ropivacaine mixed with dexmedetomidine (1 µg/kg; DEX1PN), and 0.5% ropivacaine mixed with dexmedetomidine (2 µg/kg; DEX2PN). In phase 2, dogs received perineural sciatic and saphenous nerve injections of 0.5% ropivacaine and an IV injection of diluted dexmedetomidine (1 µg/kg; DEX1IV). For perineural injections, the dose was divided equally between the 2 sites. Duration of sensory blockade was evaluated, and plasma dexmedetomidine concentrations were measured.

**RESULTS**
Duration of sensory blockade was significantly longer with DEX1PN and DEX2PN, compared with DEX0PN; DEX1IV did not prolong duration of sensory blockade, compared with DEX0PN. Peak plasma dexmedetomidine concentrations were reached after 30 minutes with DEX1PN (mean ± SD, 338 ± 190 pg/mL) and DEX2PN (786 ± 549 pg/mL), and bioavailability was 54 ± 40% and 73 ± 43%, respectively. The highest plasma dexmedetomidine concentration was measured with DEX1IV (1,032 ± 415 pg/mL) 5 minutes after injection.

**CONCLUSIONS AND CLINICAL RELEVANCE**
Results suggested that perineural injection of 0.5% ropivacaine in combination with dexmedetomidine (1 µg/kg) for locoregional anesthesia in dogs seemed to balance the benefit of prolonging sensory nerve blockade while minimizing adverse effects. (*Am J Vet Res* 2021;82:449–458)

June 2021

See the midmonth issues of *JAVMA* for the expanded table of contents for the *AJVR* or log on to avmajournals.avma.org for access to all the abstracts.