Dogs develop intracranial neoplasms more commonly than other large domesticated animal species and humans, and brain tumors are a common etiology of neurologic dysfunction referable to the brain in middle-aged to geriatric dogs.1–3 In necropsy-based studies,1–3 intracranial neoplasms account for 1% to 3% of all canine deaths, with secondary intracranial tumors being more prevalent than primary tumors. Several studies1–3 have identified meningiomas and gliomas (as well as other primary brain tumors) as the most common brain tumors diagnosed in clinical practice, there is currently little comparative information available regarding the influence of various treatment modalities, including purely palliative treatment, on the survival time of dogs with brain neoplasms. Although corticosteroids and anticonvulsants have been shown to transiently improve clinical signs and tumor-associated secondary complications (ie, peritumoral edema) in dogs with spontaneous and experimentally induced brain tumors, palliative treatments are universally associated with poor survival times in existing reports.1,2,6,7 With median survival times ranging from approximately 30 to 90 days in dogs with primary brain tumors.

It has been established that irradiation or gross, complete macroscopic resection of intracranial tumors, either alone or in combination, can provide a significant survival time benefit to dogs with a variety of intracranial neoplasms, including meningiomas, pituitary tumors, and gliomas.2,5,7,17 However, interpreta-

## Objective
To analyze survival time and identify prognostic factors associated with outcome following discharge in dogs with primary brain tumors treated palliatively.

## Design
Prospective case series.

## Animals
51 dogs with 5 histopathologic types of brain tumors.

## Procedures
Owners with dogs examined from 2004 to 2008 were invited to participate if dogs had CT or MRI evidence of a brain mass that was histopathologically confirmed as a neoplasm upon death, dogs survived for ≥ 48 hours after hospital discharge, and treatments following discharge were limited to administration of prednisone or phenobarbital. Prognostic factors, including malignancy, clinical signs (including duration), tumor type, tumor location, degree of peritumoral edema, lesion burden, and prescribed treatment, were evaluated. Survival time was estimated and animal- and tumor-specific variables evaluated as potential prognostic factors.

## Results
The median survival time in all dogs was 69 days (95% confidence interval [CI], 18 to 201 days). Multivariate analyses identified neuroanatomic location as the only significant prognostic variable, with the survival time of dogs with infratentorial tumors (n = 18) being significantly shorter (median, 28 days; 95% CI, 19 to 68 days) than survival time of dogs with supratentorial (33) tumors (median, 178 days; 95% CI, 119 to 270 days). Seizures were the most common clinical sign associated with supratentorial tumors (24/33 [73%]) and central vestibular dysfunction with infratentorial tumors (12/18).

## Conclusions and Clinical Relevance
Dogs with palliatively treated primary brain tumors, particularly those with tumors in the cerebellum, pons, or medulla, had a poor prognosis. However, dogs with supratentorial tumors had survival times > 3 months. (J Am Vet Med Assoc 2013;242:193–198)
H&E-stained sections of each neoplasm were histomitted for examination. For the purposes of this study, but in select cases, only the brain was sub-
date of death or euthanasia. For the purposes of this study, but in select cases, only the brain was sub-

tion of survival time data in some studies, particularly those that specifically examine radiotherapeutic treatments, is complicated by the fact that histopathologic diagnoses were not stringent inclusion criteria, preventing analysis of tumor type on survival time or leaving the possibility that some treated lesions were nonneoplastic conditions.

Few canine studies have investigated prognostic factors in dogs with histopathologically confirmed tu-

tumors, and results of those studies provide ambiguous information regarding the influence of tumor-associated variables such as lesion burden or histopathologic type and grade. However, the severity of neurologic dysfunction and multifocal or infratentorial neuroana-
tomic tumor locations have been previously and nega-
tively associated with survival time in existing canine studies. The purposes of the study reported here were to analyze overall survival time and identify prognostic factors in a cohort of dogs with histologically confirmed primary intracranial neoplasms that received palliative treatments.

Materials and Methods

Case selection criteria—Owners with dogs ex-

amined from August 2004 to August 2008 at the Vet-

erinary Teaching Hospital, Virginia-Maryland Regional College of Veterinary Medicine, Virginia Polytechnic Institute and State University, Blacksburg, Va, were in-
vited to participate in the study if the following inclu-
sion criteria were satisfied: clinical or historical signs of intracranial disease, CT or MRI evidence of an intracranial mass interpreted as consistent with intracranial neoplasia by board-certified radiologists, subsequent discharge from the hospital, and survival for at least 48 hours following the initial examination or intracranial imaging study. Dogs were included in the final analysis if antemortem treatments administered following hospi-
tal discharge for the identified brain mass were limited to administration of prednisone or phenobarbital, and survival for at least 48 hours following the initial examination or intracranial imaging study. Dogs were included in the final analysis if antemortem treatments administered following hospi-
tal discharge for the identified brain mass were limited to administration of prednisone or phenobarbital, and brain lesions identified on diagnostic imaging examina-
tions were histopathologically confirmed as intracranial neoplasms upon death. All study procedures were ap-
proved by the Institutional Care and Use Committee, Virginia-Maryland Regional College of Veterinary Medi-
cine, Veterinary Teaching Hospital Review Board.

Procedures—The endpoint of this study was death or euthanasia resulting from tumor-related factors. Po-
tential prognostic factors examined included the signal-
ment, clinical signs of brain disease, duration of clin-
cal signs prior to diagnostic imaging, histopathologic tumor type, circumferential location of the neoplasm in relation to the brain parenchyma, neuroanatomic loca-
tion of the brain lesion, degree of peritumoral edema, lesion burden, and prescribed treatment. Survival time was measured in days, from the day of diagnostic imaging documentation of an intracranial mass lesion to the date of death or euthanasia.

Necropsies were performed in all dogs enrolled in the study; but in select cases, only the brain was sub-
mitted for examination. For the purposes of this study, H&E-stained sections of each neoplasm were histo-
pathologically classified and graded according to stan-
dard World Health Organization criteria via consens-
sus between the pathologist attending the necropsy and by a pathologist (JLR or KLZ) blinded to the original diagnosis. If diagnostic discordance between the necropsy report and blinded pathologist was encountered, a third blinded pathologist reviewed the slides and a consensus diagnosis, representing the majority opinion of the 3 pathology reports, was reached, because tissue samples from some of the dogs included in this study were used in multiple investigations.

The clinical severity of neurologic dysfunction at the time of enrollment was graded on a scale of 0 to 3: 0 (normal) = historical evidence of brain dysfunction only (ie, seizures or behavior change) with otherwise normal findings on neurologic examination; 1 (mild) = neurologic deficits detected on examination only with animal alert and ambulatory; 2 (moderate) = overt clinical neurologic dysfunction present in ambulatory ani-
mal (ie, circling, paresis, vestibular or cerebellar ataxia, head turn, or blindness) with or without depressed level of consciousness; and 3 (severe) = nonambulatory or stuporous to comatose.

Diagnostic imaging reports were reviewed for the following information: neuroanatomic location of the tu-

mor, circumferential location of the tumor relative to the brain (intra- or extra-axial), subjective severity of any peritumoral edema (none, mild, moderate, or severe), and list of differential neuroradiologic diagnoses (me-

ningioma or choroid-plexus tumor, glioma, or other). When this information was not available in the radiolo-
gy report, a board-certified veterinary radiologist (JC) blinded to the tissue diagnosis was asked to review the case and provide the missing data. Differential diagnoses were made on the basis of imaging characteristica of histopathologically confirmed brain tumors. On the basis of the results of antemortem brain imaging, neoplasms were defined as occupying one of the follow-

ing neuroanatomic regions of the brain: supratentorial region (cerebrum, basal nuclei, and diencephalon), in-
fratentorial region (mesencephalon, cerebellum, and pontomedullary area), or ≥ 2 of these regions (multifo-

cal) with or without evidence of metastatic disease.

Estimates of lesion burdens were obtained from MRI scans with commercial, open-source image ana-

lysis software by an observer unaware of the histopath-
ologic diagnoses via a described method, which ex-

presses the lesion burden as a percentage of the brain volume. Because MRI was performed with multiple scanners (0.2, 1.0, and 1.5 T) and imaging sequences were not standardized, lesion burdens were estimated with hand-drawn regions of interest around the tumor and any associated edema on transverse T2-weighted images as well as the entire brain on every slice of the transverse T2-weighted series because these were avail-
able in multiple planes from all dogs with MRI scans and edema volumes estimated from fluid-attenuated inversion recovery or proton density–weighted im-
ages. Thus, the total lesion burden was determined by dividing the total number of lesion voxels by the total number of brain voxels. In dogs in which CT scans were performed, lesion volumetric burdens were es-
timated with a commercial CT software workstationb from the mean of 3 hand-drawn tumor regions of inter-
tomic tumor location variables were strongly associated with use of the clinical signs and neuroanatomic structures) were prescribed prednisone (0.5 mg/kg [0.23 mg/lb], PO, q 12 h for 14 days; then 0.5 mg/kg, PO, q 24 h for 14 days, followed by 0.5 mg/kg, PO, q 48 h indefinitely). Dogs with seizures and peritumoral edema, obstructive hydrocephalus, or mass effect were treated with phenobarbital. Dogs with moderate or severe peritumoral edema, obstructive hydrocephalus, or mass effect also received 1 g/kg (0.45 g/lb) of 20% mannitol IV as a 20-minute constant rate infusion followed by a 0.75 mg/kg (0.34 mg/lb) IV bolus of furosemide during anesthetic recovery from their intracranial imaging procedure.

Statistical analysis—Survival times were defined from the date of the intracranial imaging procedure to the date of death or euthanasia. In survival analyses, only cases in which death or euthanasia was directly attributable to the tumor were considered deaths, with other causes of death being censored on the date of death.

Normal probability plots showed that age was normally distributed and that duration of illness was skewed. The prognostic factors examined included age (1 to 5 years, 6 to 10 years, or 11 to 15 years), breed, body weight (≤15 or >15 kg [33 lb]), sex, clinical signs, duration of illness (≤30 or >30 days), histopathologic tumor type (choroid plexus tumor, meningioma, glioma, or neuronal tumor), neuroanatomic tumor location (supratentorial or infratentorial), circumferential tumor location (extra- vs intra-axial), lesion burden (<15%, 15% to 25%, or >25%), peritumoral edema severity (none, mild, moderate, or severe), severity of neurologic dysfunction (none, mild, moderate, or severe), and type of treatment administered (prednisone, phenobarbital, or both). To account for the potential effects of these variables on survival time and account for censoring, the Kaplan-Meier product limit method and a log-rank test were used. Prognostic factors that in the univariate analysis had P < 0.1 were evaluated for an effect on survival time by use of a Cox proportional hazards model. In the initial modeling procedure, results obtained with use of the clinical signs and neuroanatomic tumor location variables were strongly associated, so clinical signs were removed and the neuroanatomic tumor location was included in the remainder of the modeling. The overall model adequacy was assessed via Cox-Snell residuals. Values of P < 0.05 were considered significant in the final model. Data were analyzed by means of commercial statistical software.

Results

In the 4-year study period, 189 dogs with clinical and diagnostic imaging evidence of brain masses consistent with neoplasms were eligible for inclusion in the study, of which 51 of 189 (27%) met the final inclusion criteria. The reasons for exclusion of the 138 eligible dogs were failure to obtain histopathologic confirmation of the diagnosis upon death (78/138 [57%]), death or euthanasia occurring during hospitalization (35/138 [25%]), brain mass identified on diagnostic imaging confirmed to be a secondary neoplasm (10/138 [7%]) or nonneoplastic at necropsy (4/138 [3%]), client deviation from prescribed palliative treatment course to pursue surgery or radiotherapy (5/138 [4%]), poor client compliance with study protocol (4/138 [3%]), and severe postmortem autolysis that prevented accurate histopathologic diagnosis of the neoplasm (2/138 [1%]).

The mean age of the 51 dogs was 8.9 ± 2.7 years, and 18 breeds were represented in the population. The most common breeds were mixed (19/51 [37%]), Boxer (8/51 [16%]), Golden Retriever (6/51 [12%]), and Boston Terrier (4/51 [8%]), with 14 other purebred dogs represented by a single individual. There were no significant (P = 0.29) associations between breed and survival time. The histopathologic types of 51 tumors included in the study were as follows: 25 (49%) meningiomas, 14 (27%) gliomas (8 oligodendrogliomas and 6 astrocytomas), 8 (16%) choroid plexus tumors, and 4 (8%) primitive neuroectodermal (neuronal) tumors. Brain masses were diagnosed via CT in 10 dogs and MRI in 41 dogs.

The median duration of clinical signs of brain disease prior to referral was 30.5 days (95% CI, 4 to 78 days; range, 0 to 240 days). Empirical and palliative treatments were administered to 36 of 51 dogs prior to referral, including anticonvulsants (n = 21; phenobarbital [14] and diazepam [7]), various antimicrobials (15), NSAIDs (7), topical otic medications (3), prednisone (5), dexamethasone (4), and selegiline hydrochloride (2).

The clinical signs associated with brain tumors in this study were focal and consistent with the neuroanatomic location of the tumor in 40 of 51 (78%) cases. However, 11 of 51 (22%) dogs had ≥2 clinical signs referable to neurologic dysfunction in multiple parts of the brain. Seizures, behavioral changes, and central vestibular dysfunction were the most common clinical signs observed. Seizures occurred in 30 of 51 dogs, including 24 of 33 (73%) dogs with supratentorial tumors and 6 of 18 dogs with infratentorial tumors. In dogs with infratentorial tumors and seizures, 5 of 6 had imaging evidence of obstructive hydrocephalus, and all 6 temporally developed seizures after the vestibular dysfunction was apparent. Behavioral changes were observed or reported in 13 of 33 (39%) dogs with supratentorial tumors. Central vestibular dysfunction, in-
excluding the vestibulocerebellum, was the predominant clinical manifestation of infratentorial tumors, occurring in 12 of 18 cases, but was also observed in 2 of 33 (6%) dogs with supratentorial tumors associated with secondary complications of transsientorial or foraminal brain herniations. Dysphagia attributable to cranial nerve IX, X, or XII dysfunction was the second most common sign of infratentorial tumors, being noted in 3 of 18 cases. In this study, all 18 dogs with infratentorial tumors received prednisone treatment, and 6 of 18 received phenobarbital in addition to prednisone.

The median survival time of the entire cohort of dogs was 69 days (95% CI, 18 to 201 days; range, 2 to 761 days). There were no associations between age, sex, body weight, histopathologic tumor type, peritumoral edema severity, tumor burden, type of treatment, duration of clinical signs before neuroimaging diagnosis, or severity of neurologic dysfunction with survival time (Table 1). The prognostic variables identified as significant ($P < 0.10$) in the univariate analysis that were incorporated into the multivariate model included circumferential tumor location (intra-axial versus extra-axial [hazard ratio, 0.6; 95% CI, –1.3 to 5.2]) and neuroanatomic tumor location. Of these confounders, only the neuroanatomic tumor location was found to be significant in the final multivariate model. Compared with that of supratentorial tumors, the presence of an infratentorial tumor was significantly ($P = 0.02$) and negatively associated with survival time (hazard ratio, 4.8; 95% CI, 2.4 to 11.6). The median survival time in dogs (n = 18) with infratentorial tumors (median, 28 days; 95% CI, 19 to 68 days; Figure 1), was significant.

**Table 1**—Association between potential prognostic factors and survival time in dogs (n = 51) with palliatively treated primary brain tumors.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Category</th>
<th>No. (%)</th>
<th>HR</th>
<th>95% CI</th>
<th>$P$ value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex</td>
<td>Spayed female</td>
<td>22 (43)</td>
<td>0.69</td>
<td>0.34–1.89</td>
<td>0.59</td>
</tr>
<tr>
<td></td>
<td>Male</td>
<td>28 (57)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Sexually intact male</td>
<td>3 (10)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Neutered male</td>
<td>26 (90)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age (y)</td>
<td>1–5</td>
<td>12 (24)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>6–10</td>
<td>25 (49)</td>
<td>2.06</td>
<td>0.39–5.12</td>
<td>0.49</td>
</tr>
<tr>
<td></td>
<td>11–15</td>
<td>14 (27)</td>
<td>1.27</td>
<td>0.41–2.91</td>
<td>0.36</td>
</tr>
<tr>
<td>Body weight (kg)</td>
<td>≤15</td>
<td>24 (47)</td>
<td>0.89</td>
<td>0.57–1.36</td>
<td>0.26</td>
</tr>
<tr>
<td></td>
<td>&gt;15</td>
<td>27 (53)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tumor type</td>
<td>Meningioma</td>
<td>25 (49)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Supratentorial</td>
<td>20 (80)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Infratentorial</td>
<td>5 (20)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Choroid plexus tumor</td>
<td>8 (16)</td>
<td>1.95</td>
<td>0.63–4.24</td>
<td>0.38</td>
</tr>
<tr>
<td></td>
<td>Supratentorial</td>
<td>4 (50)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Infratentorial</td>
<td>4 (50)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Glioma</td>
<td>14 (27)</td>
<td>2.94</td>
<td>0.97–5.78</td>
<td>0.12</td>
</tr>
<tr>
<td></td>
<td>Supratentorial</td>
<td>9 (64)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Infratentorial</td>
<td>5 (30)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Neuronal tumor</td>
<td>4 (8)</td>
<td>2.29</td>
<td>0.86–5.28</td>
<td>0.29</td>
</tr>
<tr>
<td></td>
<td>Supratentorial</td>
<td>2 (50)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Infratentorial</td>
<td>2 (50)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Circumferential tumor</td>
<td>Extra-axial</td>
<td>33 (65)</td>
<td>3.15</td>
<td>1.67–6.74</td>
<td>0.01*</td>
</tr>
<tr>
<td>location</td>
<td>Supratentorial</td>
<td>24 (73)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Infratentorial</td>
<td>9 (27)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Intra-axial</td>
<td>18 (55)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Supratentorial</td>
<td>11 (61)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Infratentorial</td>
<td>7 (39)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tumor burden (% total brain</td>
<td>&lt;15</td>
<td>30 (59)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>volume)</td>
<td>15–25</td>
<td>13 (25)</td>
<td>0.56</td>
<td>0.04–3.43</td>
<td>0.24</td>
</tr>
<tr>
<td></td>
<td>&gt;25</td>
<td>8 (16)</td>
<td>2.35</td>
<td>0.64–7.33</td>
<td>0.51</td>
</tr>
<tr>
<td>Neuroanatomic tumor</td>
<td>Supratentorial</td>
<td>33 (65)</td>
<td>5.22</td>
<td>2.34–18.1</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>location</td>
<td>Telencephalon</td>
<td>22 (67)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Diencephalon</td>
<td>9 (27)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Multifocal</td>
<td>2 (6)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Infratentorial</td>
<td>18 (35)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Cerebellum</td>
<td>7 (39)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Pons or medulla</td>
<td>7 (39)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Peritumoral edema</td>
<td>None</td>
<td>4 (18)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Mild</td>
<td>17 (33)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Moderate</td>
<td>11 (22)</td>
<td>0.94</td>
<td>0.38–1.98</td>
<td>0.48</td>
</tr>
<tr>
<td></td>
<td>Severe</td>
<td>4 (8)</td>
<td>2.21</td>
<td>0.48–5.01</td>
<td>0.23</td>
</tr>
<tr>
<td>Treatment</td>
<td>Prednisone</td>
<td>21 (42)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Referent</td>
<td>11 (22)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Phenobarbital</td>
<td>15 (29)</td>
<td>4.11</td>
<td>0.37–6.22</td>
<td>0.35</td>
</tr>
<tr>
<td></td>
<td>Prednisone and phenobarbital</td>
<td>15 (29)</td>
<td>2.09</td>
<td>0.97–5.76</td>
<td>0.42</td>
</tr>
<tr>
<td>Duration of clinical signs</td>
<td>≤30</td>
<td>29 (57)</td>
<td>1.74</td>
<td>0.43–9.13</td>
<td>0.51</td>
</tr>
<tr>
<td>to imaging (d)</td>
<td>&gt;30</td>
<td>22 (43)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Severity of neurologic</td>
<td>None</td>
<td>14 (27)</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>dysfunction</td>
<td>Mild</td>
<td>20 (39)</td>
<td>1.05</td>
<td>0.43–3.26</td>
<td>0.38</td>
</tr>
<tr>
<td></td>
<td>Moderate</td>
<td>12 (24)</td>
<td>1.46</td>
<td>0.71–2.57</td>
<td>0.27</td>
</tr>
<tr>
<td></td>
<td>Severe</td>
<td>5 (10)</td>
<td>3.84</td>
<td>0.19–8.82</td>
<td>0.54</td>
</tr>
</tbody>
</table>

*Value was significant ($P < 0.10$).

HR = Hazard ratio. NA = Not applicable.
Reports\textsuperscript{1,2} that dogs with primary brain tumors treated
infratentorial tumors (9/30).

dialogue that prompted euthanasia, whereas progressive
dysfunction was the most common manifestation of tumor progres-
and 8 (16%) died, with 47 (92%) deaths being tumor-
related. In 30 of the 43 dogs that were euthanized,
information in the medical record was available regarding
the tumor-related clinical factor contributing to eutha-
nasia. None of the deaths in this study were attributed
to adverse effects associated with the prescribed pallia-
tive treatments, although owner-perceived polyuria and
polydipsia was common, being noted in the postdiag-
nosis medical history or client communications at least
once for 44 of 51 dogs and was noted in dogs receiv-
ing all of the prescribed treatments. Owners of 21 of
30 dogs on phenobarbital also reported that their dogs
developed or had exacerbations of existing sedation
(n = 15) or ataxia (6) after starting the treatment. In
dogs with supratentorial tumors, the development of
epilepsy refractory to phenobarbital treatment (n = 30)
was the most common manifestation of tumor progres-
sion that prompted euthanasia, whereas progressive
gait dysfunction resulting in an inability to walk was
the principal reason cited for euthanasia of dogs with
infratentorial tumors (9/30).

Discussion

The results of the present study corroborate earlier
reports\textsuperscript{1,2} that dogs with primary brain tumors treated
palliatively have a poor prognosis, as evident from the
overall median survival time of 69 days. Although our
data demonstrated that the median survival time of the
entire cohort of dogs was similar to those reported, we
demonstrated that there were specific and recognizable
clinical features of dogs with primary intracranial tu-
mors that influenced survival time.

The results indicated that the presence of an in-
fratentorial tumor had a significant and negative ef-
fect on survival time in this population of palliatively
in dogs (33) with supratentorial tumors (median, 178
days; 95% CI, 119 to 270 days).

In this study, 43 of 51 (84%) dogs were euthanized,
and 8 (16%) died, with 47 (92%) deaths being tumor-
related. In 30 of the 43 dogs that were euthanized,
information in the medical record was available regarding
the tumor-related clinical factor contributing to eutha-
nasia. None of the deaths in this study were attributed
to adverse effects associated with the prescribed pallia-
tive treatments, although owner-perceived polyuria and
polydipsia was common, being noted in the postdiag-
nosis medical history or client communications at least
once for 44 of 51 dogs and was noted in dogs receiv-
ing all of the prescribed treatments. Owners of 21 of
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developed or had exacerbations of existing sedation
(n = 15) or ataxia (6) after starting the treatment. In
dogs with supratentorial tumors, the development of
epilepsy refractory to phenobarbital treatment (10/30)
was the most common manifestation of tumor progres-
sion that prompted euthanasia, whereas progressive
gait dysfunction resulting in an inability to walk was
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The results indicated that the presence of an in-
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Multiple deaths in this study were attributed
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Discussion

The results of the present study corroborate earlier
reports\textsuperscript{1,2} that dogs with primary brain tumors treated
palliatively have a poor prognosis, as evident from the
overall median survival time of 69 days. Although our
data demonstrated that the median survival time of the
entire cohort of dogs was similar to those reported, we
demonstrated that there were specific and recognizable
clinical features of dogs with primary intracranial tu-
mors that influenced survival time.

The results indicated that the presence of an in-
fratentorial tumor had a significant and negative ef-
fect on survival time in this population of palliatively
in dogs (33) with supratentorial tumors (median, 178
days; 95% CI, 119 to 270 days).

In this study, 43 of 51 (84%) dogs were euthanized,
and 8 (16%) died, with 47 (92%) deaths being tumor-
related. In 30 of the 43 dogs that were euthanized,
information in the medical record was available regarding
the tumor-related clinical factor contributing to eutha-
nasia. None of the deaths in this study were attributed
to adverse effects associated with the prescribed pallia-
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The palliative treatments in this study were eco-
nomical and clinically well tolerated, with no dog being
withdrawn from the study or dying because of adverse
drug effects. Not surprisingly, given that all dogs were
treated with phenobarbital or prednisone, the most com-
mon adverse effects noted were polyuria and polydipsia.
In addition, sedation or ataxia was observed in 70% of
dogs treated with phenobarbital. These drug-related ef-
fects should be anticipated in dogs and therefore includ-
ed during education of clients who elect to palliatively
treat dogs with brain tumors or when these drugs are
used to manage the secondary effects of brain tumors
in dogs receiving concurrent definitive treatments. The
high incidence of owner-perceived adverse effects associ-
ated with or confounded by phenobarbital treatment also
suggests that an anticonvulsant with less propensity to
cause sedation or ataxia, such as levetiracetam, should
be considered for use in dogs with brain tumors.\textsuperscript{15}

We recognize and wish to reinforce that interpre-
tation of the results of this study should occur with
knowledge of its limitations. Although we eliminated
misclassification of nonneoplastic lesions by requir-
ing histopathologic confirmation of neoplasia, we may
have introduced selection bias if the philosophical be-
lieds (or other factors) of owners or the clinical features

![Figure 1—Survival plot of 51 dogs with primary brain tumors by
neuroanatomic tumor location. Dogs with infratentorial tumors (n
= 18; solid line) had a significantly (P = 0.02) shorter survival time
than did dogs with supratentorial tumors (33; broken line). The
circles represent censored data points.](image-url)
of dogs that survive to discharge or are subjected to necropsy differ from those who do not. For example, in previous studies,\textsuperscript{20,21} including investigations at our institution, dogs with brain tumors have been commonly euthanized in immediate proximity to presumptive antemortem imaging diagnosis or have died prior to discharge. Thus, the survival times in this cohort may have been artificially inflated through selection bias of more mildly affected patients. However, we consider this unlikely because the overall survival time of 69 days in this population of dogs was similar to those reported in other canine studies,\textsuperscript{1,2,6} and dogs had the full range of severity of neurologic dysfunction possible within the grading scale used. In this study, 17 of 51 (33\%) dogs had moderate to severe neurologic dysfunction.

Alternatively, the survival times for patients in the present study may have been falsely abbreviated, considering that dogs treated with more aggressive palliative treatments were not included. Prednisone has been postulated to improve the clinical signs and quality of life in dogs with cancer through its anti-inflammatory, antiedema, and euphoric effects\textsuperscript{2,6} and perhaps could improve the clinical signs and quality of life in dogs with cancer through its anti-inflammatory properties.\textsuperscript{2,6}

Postmortem imaging diagnosis or have died prior to discharge. Thus, the survival times in this cohort may have been artificially inflated through selection bias of more mildly affected patients. However, we consider this unlikely because the overall survival time of 69 days in this population of dogs was similar to those reported in other canine studies,\textsuperscript{1,2,6} and dogs had the full range of severity of neurologic dysfunction possible within the grading scale used. In this study, 17 of 51 (33\%) dogs had moderate to severe neurologic dysfunction.

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Results indicated that dogs with primary intracranial tumors located in the caudal brainstem had a grave prognosis and were unlikely to survive > 2 months with palliative treatment following presumptive neuroimaging diagnosis. Although the prognosis for the entire cohort of dogs with brain tumors treated was poor, dogs with supratentorial tumors had prolonged survival times with palliative treatment.

\textsuperscript{a} OsiriX Imaging Software, version 3.9.2, OsiriX Foundation, Geneva, Switzerland.

\textsuperscript{b} Voxel Q Visualization Station, Philips Medical Systems, Bothell, Wash.

\textsuperscript{c} SAS, version 9.2, SAS Institute Inc, Cary, NC.

\textbf{References}