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STANDARD ARTICLE

Intracranial pressure monitoring in normal dogs using subdural and intraparenchymal miniature strain-gauge transducers

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Background: Monitoring of intracranial pressure (ICP) is a critical component in the management of intracranial hypertension. Safety, efficacy, and optimal location of microsensor devices have not been defined in dogs.

Hypothesis/Objective: Assessment of ICP using a microsensor transducer is feasible in anesthetized and conscious animals and is independent of transducer location. Intraparenchymal transducer placement is associated with more adverse effects.

Animals: Seven adult, bred-for-research dogs.

Methods: In a prospective investigational study, microsensor ICP transducers were inserted into subdural and intraparenchymal locations at defined rostral or caudal locations within the rostrotentorial compartment under general anesthesia. Mean arterial pressure and ICP were measured continuously during physiological maneuvers, and for 20 hours after anesthesia.

Results: Baseline mean ± SD values for ICP and cerebral perfusion pressure were 7.2 ± 2.3 and 78.9 ± 7.6 mm Hg, respectively. Catheter position did not have a significant effect on ICP measurements. There was significant variation from baseline ICP accompanying physiological maneuvers ($P < .001$) and with normal activities, especially with changes in head position ($P < .001$). Pathological sequelae were more evident after intraparenchymal versus subdural placement.

Conclusions and Clinical Importance: Use of a microsensor ICP transducer was technically straightforward and provided ICP measurements within previously reported reference ranges. Results support the use of an accessible dorsal location and subdural positioning. Transient fluctuations in ICP are normal events in conscious dogs and large variations associated with head position should be accounted for when evaluating animals with intracranial hypertension.

KEYWORDS

canine, Codman, intracranial hypertension, microsensor

1 | INTRODUCTION

Intracranial pressure (ICP) refers to the pressure exerted by the intracranial contents against an inelastic cranial vault and is a common consideration in patients with increased intracranial volume secondary to brain trauma, organic brain disease, or after intracranial surgery. Continuous measurement of prolonged intracranial hypertension (ICH) generally is recommended as standard of care in human medicine, particularly in cases of severe head trauma.[1–3] Monitoring ICP may provide timely information needed to guide early therapeutic intervention, assess response to treatment strategies, decrease indiscriminant treatments, and provide data relevant to prognosis.[1,3,4]

Abbreviations: CPP, cerebral perfusion pressure; CSF, cerebrospinal fluid; CVP, central venous pressure; $ETCO_2$, end-tidal partial pressure of carbon dioxide; HR, heart rate; ICH, intracranial hypertension; ICP, intracranial pressure; LS, least square; MAP, mean arterial pressure; $PaCO_2$, arterial carbon dioxide partial pressure; PaO_2, arterial oxygen partial pressure

Cerebral blood flow is critical for normal brain function and is a function of cerebral perfusion pressure (CPP), mean arterial pressure (MAP), and cerebral vascular tone or resistance. Although defining ICP is integral to monitoring CPP (CPP = MAP-ICP), measurement in veterinary practice has been limited because of expense, technical skills required for monitor placement, and compatibility of available monitoring systems with noncompliant animals. Intraventricular ICP devices, fluid-coupled to an external transducer, are considered the "gold standard" for measuring ICP in people,[5] and similar approaches have been reported from a study using experimental dogs.[6–13] However, marked variation in skull landmarks, variation in ventricular size and location, and the impracticalities of cerebellomedullary cistern placement together with the requirement to maintain the external transducer at head level limit the feasibility of ventricular placement in most clinical situations.

Nonfluid coupled ICP transducers using either catheter tip miniature strain gauges or fiber-optic-linked catheter tip diaphragm technology have distinct advantages over external transducers because these devices do not require "leveling" with the head, although they cannot be recalibrated to zero once they are inserted. Intracranial fiber-optic or solid state capacitance systems have been shown to provide accurate determination of ICP in both cats[14,15] and dogs,[10,16–22] but substantial limitations of this system include the necessity of anchoring the device to the skull by means of a subarachnoid bolt and the inherent fragility of the fiber optics.

Intracranial strain-gauge pressure-sensing devices consisting of a miniaturized silicon transducer enclosed in a titanium case and implanted in the tip of a flexible nylon-covered cable have been described for measurement of ICP in both awake and anesthetized horses under a variety of conditions.[23–29] Their flexibility, low profile design and ease of use mean they are more easily operated, less prone to breakage and better tolerated in the conscious animal. Data supporting optimal placement of strain-gauge ICP monitoring devices in dogs, in relation to consistency of readings and adverse effects, are not available.

The purposes of our study were to: (1) examine the feasibility of using a transducer-tip intracranial ICP monitor in dogs; (2) determine the variation in ICP measurements obtained from various anatomical locations (subdural versus intraparenchymal; rostral versus caudal) within the cranial vault; (3) evaluate the effects of general anesthesia, recovery, and behavior on ICP measurements in normal dogs; and (4) evaluate histologically the acute effects of subdural versus intraparenchymal catheter placement on intracranial tissues.

2 | MATERIALS AND METHODS

This was a prospective investigational study conducted at the University of California, Davis, School of Veterinary Medicine.

2.1 | Animals

Seven neurologically normal bred-for-research, spayed female hounds, ranging in age from 18 months to 8 years (mean, 5.2 years) were studied. Body weights varied from 27 to 35 kg (mean, 31 kg). For entry into the study, each dog was required to be healthy based on normal physical and neurological examinations and results of CBC, serum biochemistry panel, and urinalysis. Animals were cared for according to the principles outlined in the National Institutes of Health Guide for the Care and Use of Laboratory Animals, and the study protocol was approved by the Animal Care and Use Committee at the University of California, Davis.

2.2 | Anesthesia

After being fasted for 8 hours, each dog was premedicated with atropine (0.03 mg/kg SC), acepromazine (0.01 mg/kg), and butorphanol (0.4 mg/kg). Anesthesia was induced with thiopental (10 mg/kg) and maintained with isoflurane in oxygen. Animals were kept at a surgical plane of anesthesia during transducer placement by administration of isoflurane at 1.3-1.5 minimum alveolar concentration, and end-tidal CO_2 ($ETCO_2$) was maintained between 30 and 35 mm Hg using positive pressure ventilation. Heart rate, body temperature, MAP, and central venous pressure (CVP) were monitored continuously during anesthesia and recovery. Arterial blood gases were measured every 30 minutes during anesthesia.

2.3 | Surgical procedure

After anesthetic induction, an 18-gauge 2.5-inch catheter was inserted percutaneously into the right cephalic vein and a 20-gauge 70-cm drum catheter was inserted percutaneously into the right lateral saphenous vein and advanced to the level of the 10th rib. A urinary catheter was placed to monitor urine output during anesthesia and recovery.

The head was shaved, aseptically prepared, and draped with sterile surgical drapes. A standard dorsal midline surgical approach to the cranium was performed exposing the frontal, parietal, and occipital bones bilaterally. Masseter and temporalis muscles were retracted laterally. Rostral and caudal burr-hole craniectomies were performed on the left and right sides of the cranium using a high-speed pneumatic drill (Surgairtome, Linvatec Inc., Key Largo, Florida). Rostral sites were located 1 cm from the midline immediately caudal to the bregma, and caudal sites were located 1 cm from the midline, and 2 cm caudal to the rostral site. These sites resulted in transducer placements located over the boundary of the postcruciate and suprasylvian gyri rostrally, and ectomarginal gyrus caudally.[30,31] Codman MicroSensor ICP transducers (Codman Microsensor, Codman & Shurtleff Inc., Raynham, Massachusetts) were used, consisting of a miniature strain-gauge pressure sensor mounted in a titanium case at the end of a 100-cm flexible nylon covered cable (Figure 1). Functionality and accuracy of the transducers were verified preplacement by measuring pressure within a column of distilled water of known height. The absolute error of transducer readings was ≤1 mm Hg and was within manufacturer specifications. Before placement, transducers were electronically zeroed and calibrated in a shallow container of sterile saline (0.9% NaCl solution) as specified by the manufacturer. All transducers were inserted through 1 mm durotomy incisions. Intraparenchymal transducers were placed to a depth of 1.5 cm in the right rostral and caudal sites. For subdural placement, two 90° bends were made in the

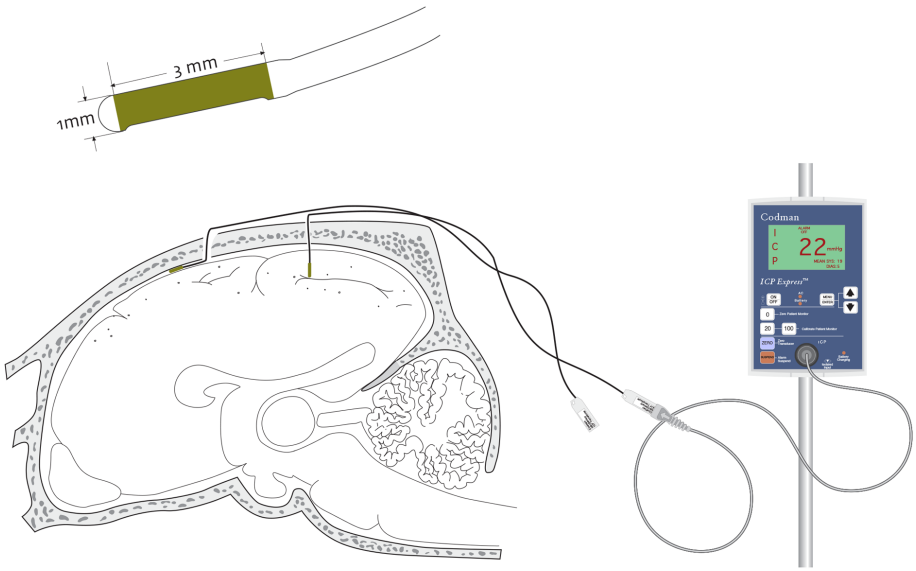

FIGURE 1 Schematic drawing illustrating the subdural and intraparenchymal placement of the Codman MicroSensor transducers. The nylon cable is purposefully kinked at 90° so that the opening in the pressure sensor in the strain gauge at the tip of the catheter lies in close contact with the surface of the brain. Bending and kinking the cable in this manner does not alter the function of the transducer. The cable interfaces with the Codman Intracranial Pressure (ICP) Express to display ICP measurement. The MicroSensor transducer (inset) measures 1.0 mm by 3.0 mm and consists of a microchip pressure sensor enclosed in a titanium case. The flexible nylon cable connecting the transducer to a pressure monitor measures 0.7 mm in diameter

transducer cable, 1 and 2 cm away from the strain gauge at the end of the transducer cable so that the strain gauge rested on the pia mater (Figure 1). Subdural transducers were placed in the left rostral and caudal sites. Transducers were secured in place with bone wax in the burr hole site and were connected to a cable that in turn connected to the Codman MicroSensor interface control unit (Codman ICP Express, Codman & Shurtleff Inc.). Occlusion of the jugular vein was used to test for in vivo functionality of all transducers resulting in detection of a consistent increase in ICP of at least 5-10 mm Hg within 15-30 seconds. The ICP catheters were sutured to the ventral aspect of the temporalis muscle alongside the external sagittal crest to exit the incision caudally, between sutures in the closure. The SC and subcuticular tissues were closed using 3-0 polydioxanone in a simple continuous pattern, and skin staples were used to close the skin with the ICP catheters exiting from the caudal aspect of the incision. Additional fixation for the nylon tubing was provided using adhesive tape encircling the catheters, affixed externally with skin staples between the tape and the skin over the dorsum of the neck.

2.4 | Data collection

Intracranial pressure was recorded continuously from all 4 catheters during anesthesia (4 hours), recovery, and after recovery for a total time period of 24 hours. During anesthesia, dogs were positioned in ventral recumbency. The MAP and CVP were measured by use of fluid-filled catheters attached to strain-gauge transducers positioned at the level of the heart and were calibrated against mercury and water manometers, respectively. Data were recorded by use of a multiple-channel chart recorder (Grass Model 7D, Statham Medical Instruments, Hato Rey, Puerto Rico) or digital output display (Model 90603A, Spacelabs Medical, Redmond, Washington). The pH, $PaCO_2$, and PaO_2 in blood samples, obtained every 30 minutes during anesthesia from the dorsal pedal artery, were determined by use of an automated blood gas analyzer (ABL 5, Radiometer America, Westlake, Ohio). Values were corrected on the basis of body temperature as measured by esophageal probe.

2.5 | ICP manipulations

Jugular occlusion was achieved by placing a blood pressure cuff circumferentially around the neck and slowly inflating the cuff to 90 mm Hg. Intracranial pressure, MAP, CVP, heart rate (HR), and body temperature were recorded at each 10 mm Hg increase in jugular occlusion pressure until occlusion pressures reached 90 mm Hg.

End-tidal CO_2 increase from 32 to 70 mm Hg was achieved by removing the soda lime canister from the anesthetic machine. Intracranial pressure, MAP, CVP, HR, and body temperature were recorded at each 10 mm Hg increase in $ETCO_2$.

During the recovery period, ICP, MAP, HR, and body temperature were recorded every 15 minutes. After recovery from anesthesia, ICP MAP, HR, and body temperature were recorded every 2 hours with dogs in sternal recumbency and in a standing position. Additionally, ICP measurements were recorded at least once in every 2 hours when dogs assumed various body postures including standing with head up and head down, and in sternal recumbency with head up and head down. Head down position was achieved by offering food placed at floor level, and elevation was achieved by holding food above the dog's head resulting in increase of the head level with an angle of the nasal plane >45° from horizontal. Intracranial pressure measurements also were recorded intermittently during ongoing normal activities such as drinking, eating, walking, stretching, and playing (moving head from side to side) during each 2-hour interval. Dogs were monitored continuously by a board-certified neurologist during the 24-hour recording period for any neurological deficits and for signs of

discomfort or apparent pain, including vocalization, rubbing, or scratching of the head or cable insertion sites and altered behavior including change in body posture, change in demeanor, altered reaction to touch, altered interaction with the investigator, altered mobility, and decreased appetite.

2.6 | Pathology

To verify transducer position and assess local effects of transducer placement on the brain parenchyma, all dogs were euthanized using pentobarbital/phenytoin (6 mg/kg IV; Beuthanazia-D, Merck Animal Health, Madison, New Jersey) at the conclusion of the 24-hour recording period and the brain was removed for pathological examination. The entire brain was examined for histopathological alterations created by the transducer, either from initial placement or movement of the transducer during normal activities in the recovering or awake dog. Intracranial pressure transducer cables were cut close to the point of insertion into the skull to preserve identification of the subdural or intraparenchymal location of the transducer. Then, methylene blue was used to mark transducer tracks in the ICP monitoring sites once the brain was removed. Brains and skulls were immersion fixed in 10% buffered formalin for a minimum of 7 days and brains were sectioned into 3-mm thick sections including the marked catheter insertion sites, and gross lesions were digitally photographed. Brain tissue sections were processed for routine paraffin embedding and 5-μm thick sections were routinely stained with hematoxylin and eosin.

2.7 | Statistical analysis

All data analyses were conducted using R (version 3.4.1, R Core Team, 2017. R Foundation for Statistical Computing, Vienna, Austria) with the R package lme4.[32] A series of linear mixed effects models was generated with dog subjects as random effects, and with the between-subject fixed effect of catheter placement and various physiological manipulations of ICP during the 3 separate experiments (variable jugular occlusion pressure, variable increases in $ETCO_2$, and variable position of head and body relative to the heart) as individual test variables. Visual inspection of residual plots for each model did not identify any large deviations from homoscedasticity or normality. Likelihood ratio tests were used to assess the importance of fixed effects. Pairwise comparisons of fixed effect levels were tested using Tukey's adjustment. Statistical significance was considered for P values <.05.

3 | RESULTS

All catheters (28 total) were placed in all dogs without immediate surgical complications. Post-anesthesia, all catheters were functional for the 24-hour period of the study and were tolerated well by all dogs with no subjective evidence of discomfort or apparent pain. No neurological deficits were observed during the 24-hour period. Necropsy of Dog 4 identified an intracranial meningioma and data from this dog was excluded from the analysis. Anesthesia technical issues resulted in $ETCO_2$ manipulation data being unavailable for 1 dog. All dogs were normocapnic and normoxemic without evidence of acid-base disturbances through the entire study, with the exception of data acquired during manipulation of $ETCO_2$ (Table 1).

3.1 | Effect of transducer location

Intracranial pressure measurements did not differ significantly across transducer sites (rostral, caudal, intraparenchymal, and subdural) measured at baseline time points before or after physiological manipulations (P = .19; Figure 2). Subsequent analysis of physiological maneuvers was performed on pooled data from all 4 transducer sites. Mean ICP and CPPs at baseline and maximal manipulation state are summarized in Table 1.

3.2 | Effect of physiological maneuvers

Significant increases in ICP were seen with both increasing jugular occlusion pressure (P < .001) and increasing $ETCO_2$ (P < .001), and the effect was true for each transducer placement. Pairwise comparisons showed significant differences in ICP with jugular occlusion pressure differences across 20, 40, and 80 mm Hg (P < .05 for all). The magnitude of effect on ICP was decreased at higher occlusion pressures. (Figure 3, Table 2). Significant differences in ICP with increasing $ETCO_2$ were seen across 40, 50, and 70 mm Hg (P < .05 for all). The magnitude of effect on ICP was decreased at higher $ETCO_2$ (Figure 4, Table 2).

3.3 | ICP recording in conscious dogs

Post-anesthesia ICP results were found to be significantly different based on both body and head position across time (P < .001; Figure 5). Pairwise comparisons showed significant differences in ICP among standing/head elevated, standing/head down, sternal/head elevated, and sternal/head down (P < .05 for all; Table 3). Mean values for ICP increased by >100% comparing head elevation to head down positions (Figure 5). Other activities that were noted to

TABLE 1 Mean physiological variables (±SD) of all dogs at baseline and maximal physiological manipulations

Time point	ICP (mm Hg) (n = 6)	MAP (mm Hg (n = 6)	CPP (mm Hg) (n = 6)	$PaCO_2$ (mm Hg) (n = 6)
Baseline 1	7.2 ± 3.8	86.1 ± 8.5	78.9 ± 7.6	39.8 ± 3.4
JUG 90	30.8 ± 10.4	101.0 ± 13.4	70.2 ± 13.4	39.4 ± 2.2
CO_2 70	20.1 ± 7.5	93.9 ± 23.4	73.8 ± 19.2	70.61 ± 5.0
Baseline 2	9.5 ± 3.3	113.8 ± 14.6	104.3 ± 13.9	40.1 ± 1.7

Abbreviations: IC, intracranial pressure; CPP, cerebral perfusion pressure; MAP, mean arterial pressure; $PaCO_2$, arterial carbon dioxide partial pressure.

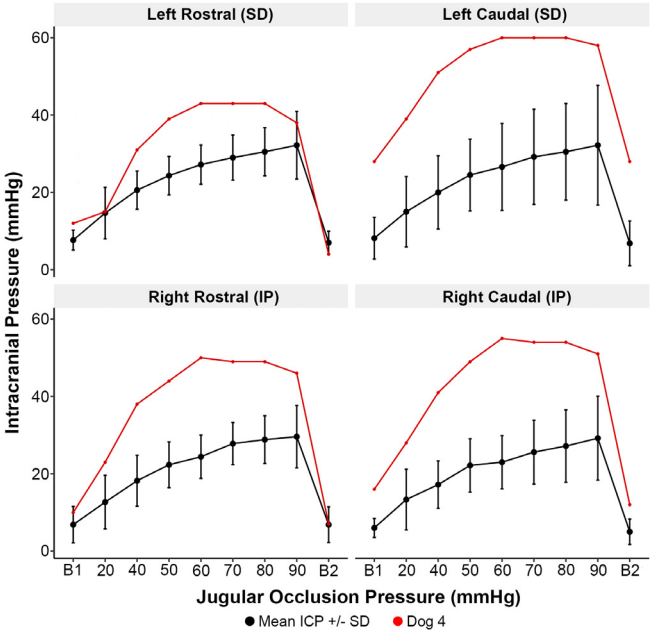

FIGURE 2 Mean ICP (±SD) for all dogs (black lines) recorded from all transducer locations after progressive restriction of cranial venous outflow secondary to jugular occlusion. ICP readings were similar for all transducer sites whether from rostral, caudal, intraparenchymal, or subdural locations. Recordings from Dog 4 (red lines) were excluded from analysis because of outlying data, subsequently attributed to the presence of an intracranial mass lesion (meningioma) found at necropsy. B1 and B2 are baseline time points before and after manipulation, respectively

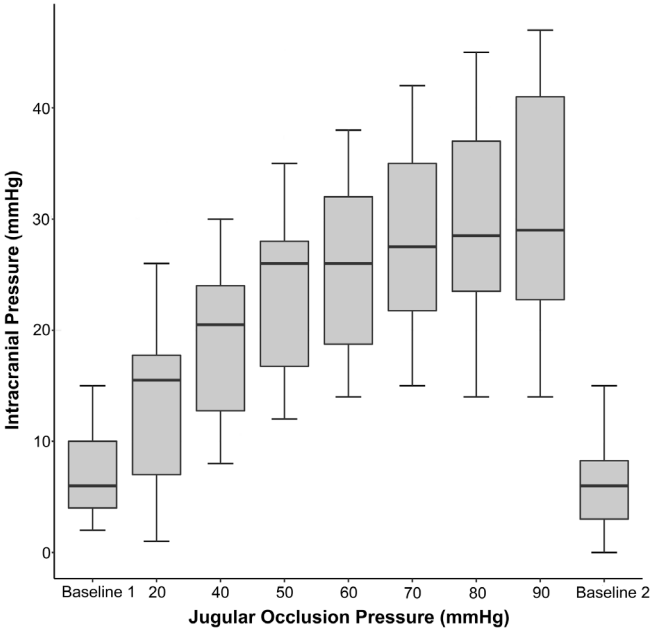

FIGURE 3 Boxplots of ICP at various jugular occlusion pressures. ICP values increase with increasing occlusion pressure. Whiskers represent ±1.5 interquartile ranges

consistently result in transient marked increases in ICP included stretching, head shaking, and large excursions of the head from side to side.

3.4 | Pathology

Grossly, after removal of the dura, the entire surface of the brain appeared normal in 4/7 dogs except for 1-mm holes in the right rostral and caudal locations where transducer cables had entered the brain as a consequence of intraparenchymal transducer placement. One of these dogs had a 0.5 cm × 0.8 cm mass arising from the dura 0.5 cm rostral to the left rostral catheter. Histological examination confirmed the mass to be a meningioma. In 3/7 dogs, gross abnormalities were noted on the surface of the brain; in 2/3 of these dogs, 3-4 mm focal hemorrhages were present at the point where some of the catheters penetrated the dura. A small amount of blood was evident in the tracks of IP transducers on the right cerebrum in both dogs. In the 3rd dog, a subdural hemorrhage was present, extending over much of the surface of the right cerebrum, including the location of the right caudal transducer. The hemorrhage had remained superficial and was not causing compression, swelling or edema of the cerebral hemisphere (Figure 6). Histological examination of the brain surrounding the area of microsensor placement showed a clearly demonstrable tract from IP transducer placement that in some instances had small areas of hemorrhage, especially where the tip of the sensor was located (Figure 6). No polymorphonuclear, lymphocytic, or macrophage infiltration was present in any of the dogs.

4 | DISCUSSION

The Codman MicroSensor has been shown to provide reliable and accurate ICP measurements in human patients compared to a variety of ICP monitoring techniques.[33–37] The results of our study support the use of the Codman MicroSensor in dogs as a simple method for

TABLE 2 Mean ICP results (±SD) in all dogs after physiological maneuvers of jugular occlusion (JUG) and increase in $ETCO_2$

Time point	ICP (mm Hg)	*P* value
JUG (mm Hg)		<.001
0	7.2 ± 3.8^{a}	
20	13.9 ± 7.2^{b}	
40	19.0 ± 6.6^{c}	
50	$23.3 \pm 6.6^{c,d}$	
60	$25.3 \pm 7.2^{d,e}$	
70	$27.9 \pm 7.9^{d,e,f}$	
80	$29.2 \pm 8.5^{e,f}$	
90	30.8 ± 10.4^{f}	
0	6.4 ± 4.1^{a}	
$ETCO_2$ (%)		<.001
0	6.4 ± 4.1^{g}	
40	10.0 ± 5.1^{g}	
50	15.2 ± 7.6^{h}	
60	$19.3 \pm 8.1^{h,i}$	
65	$17.2 \pm 5.6^{h,i}$	
70	20.1 ± 7.5^{i}	
0	9.3 ± 3.3^{g}	

Likelihood ratio test *P* value from the linear mixed models showed overall significant effects of both manipulations.

[a] ICP means that have no superscript in common were significantly different from each other (Tukey's honest significance difference test, $P < .05$).

obtaining continuous direct ICP measurements in anesthetized and awake animals in the clinical setting. Lack of cumbersome anchoring equipment and the ability to interface with a wide variety of patient monitoring systems were clinically relevant advantages. Removal of the transducer is relatively simple compared to bolt-based systems and can be done without surgery in the conscious dog. Moreover, the absence of significant differences in ICP readings based on rostral/caudal positioning or intraparenchymal versus subdural placement has implications for clinical use, because flexibility in the placement of the rostrotentorial burr hole may be advantageous in the clinical setting. Although clinically apparent neurological deficits were not documented, pathology associated with intraparenchymal placement was predictably more apparent (Figure 6), supporting the use of subdural placement. Additionally, the Codman MicroSensor has been shown to be safe, have acceptable function, and produce minimal imaging artifact during MRI as long as manufacturer's imaging guidelines are strictly followed.[38–40]

Choice of monitoring locations for our study was made considering assessment of potential clinical sequelae, including ease of placement, minimization of adverse effects, and reliability of readings. Noninvasive methods for ICP measurement would have been ideal, but these have not been shown to be sufficiently reliable for routine clinical use.[41,42] Intraparenchymal transducers are the mainstay for monitoring ICP in humans[5,43,44] and provided consistent readings in our study. Subdural transducer placement provided a less invasive option, but less data exist regarding the relative value and accuracy of subdural ICP monitoring devices, and subdural readings may be less accurate than intraparenchymal readings. However, our study found no difference between subdural and intraparenchymal transducer readings, consistent with a previous study in dogs using a bolt-based solid-state transducer[45] and subdural ICP device studies across several species.[25,36,37,45–51]

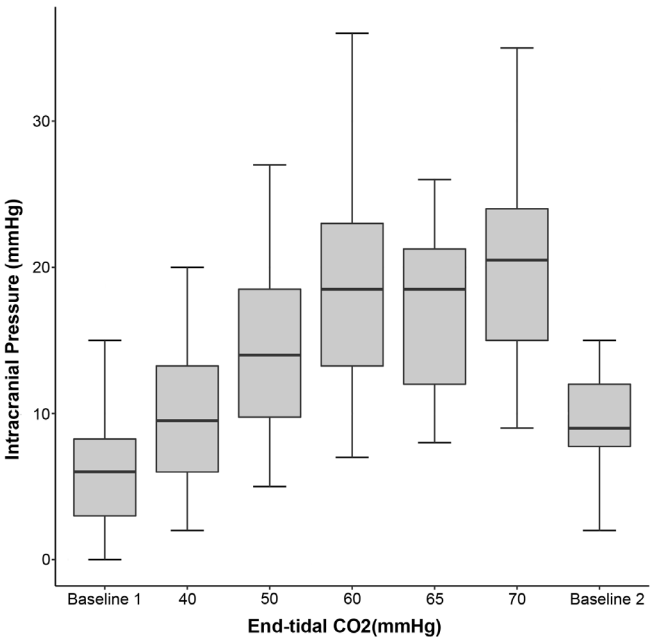

FIGURE 4 Boxplots of ICP at various end-tidal partial pressure of carbon dioxide ($ETCO_2$) manipulations. ICP values increase with increasing CO_2. Whiskers represent ±1.5 interquartile ranges

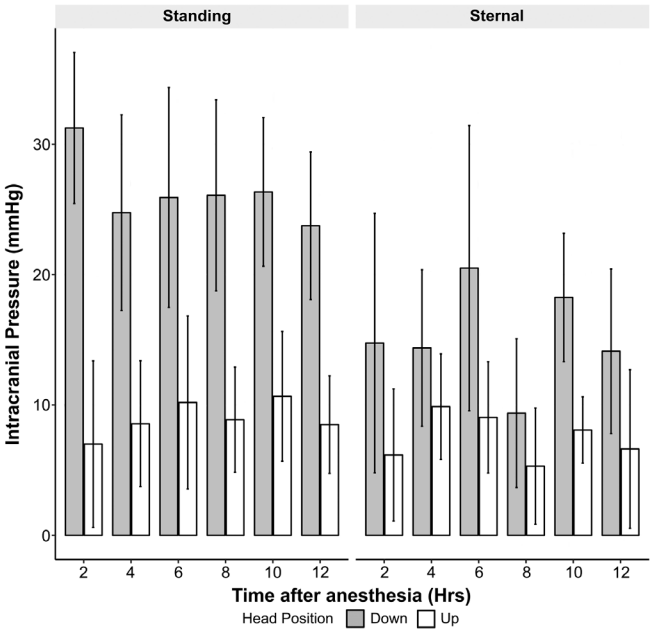

FIGURE 5 Mean ICP (±SD) associated with head and body position in conscious dogs measured every 2 hours post-anesthesia. Head down postures consistently resulted in increase of mean ICP values by 100%

The effects of head and body position in our study were notable in their magnitude and are relevant to ICP management in the clinical setting. An increase in ICP associated with head down positioning was seen in all dogs as predicted by ICH physiology, although head position was reported to have no effect on ICP in anesthetized dogs in a previous study.[17] The increase in mean ICP (approximately +7.6 mm Hg/+100%) with head down positioning in sternally recumbent conscious dogs in our study is more consistent with a previous report of similar maneuvers in conscious dogs in lateral recumbency.[10] The maximal recorded increase in mean ICP (approximately +16.5 mm Hg/+180%) occurred after head down positioning in standing dogs (Figure 5, Table 3) and likely reflects the higher potential for attaining a head position below the level of the heart compared to sternally positioned dogs. Optimal head elevation has been well documented to result in decreased ICP in human traumatic brain injury patients,[52] although effect on clinical outcome is less defined.[53] Large ICP variations with head position in our study in normal dogs similarly highlight

TABLE 3 Mean ICP results (±SD) in all dogs with altered body and head positions

Position	ICP (mm Hg)	*P* value
		<.001
Standing/up	9.2 ± 5.2^a	
Standing/down	25.7 ± 6.7^b	
Sternal/up	7.6 ± 8.1^c	
Sternal/down	15.2 ± 8.1^d	

Likelihood ratio test *P* value from the linear mixed model showed overall significant effects of changes in head and body position, adjusted for time elapsed after anesthesia.

[a] ICP means that have no superscript in common were significantly different from each other (Tukey's honest significance difference test, $P < .05$).

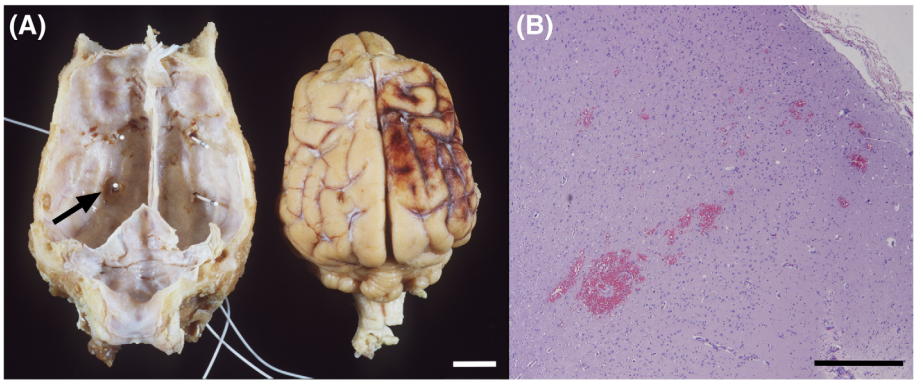

FIGURE 6 (A) Gross appearance of a subdural hemorrhage after transducer placement in 1 dog. There does not appear to be associated swelling, and ICP measurements obtained from the transducers on this side were not different from the contralateral transducers. The hemorrhage likely occurred by compromising a meningeal vessel while incising the dura during transducer placement. Bleeding from around the right caudal transducer (arrow) is likely the source of this hemorrhage. (B) Hematoxylin and eosin histological transverse section of brain showing hemorrhage within the neuropil associated with intraparenchymal transducer placement. Bar size: A = 1 cm, B = 500 μm

the likely importance of this basic nursing component on ICP management in dogs with ICH.

No clinically apparent adverse events were seen in the study dogs, although minor pathological sequelae were seen at necropsy. Adverse events associated with choice of monitoring device and location have been well documented in human patients for intraventricular devices with external ventricular drainage compared to intraparenchymal solid state sensors. Hemorrhage associated with intraventricular devices occurs in approximately 10% of cases although <1% is clinically relevant,[54] and infection rates have been reported in up to 20% of cases.[55] Reported complications for intraparenchymal devices are markedly lower[56,57] and are <0.5% in reported studies in humans for the Codman MicroSensor, similar to what was observed in our study.[34] Fewer data are available for subdurally placed sensors, however complications in our study and in humans with a subdural Codman MicroSensor also were uncommon.[37]

Limitations of our study include lack of measurement of ICP using an intraventricular "gold standard," no assessment of device "drift" (loss of accuracy of device readings because of change in scale zero over time), and assessment of ICP in normal dogs in the absence of defined structural brain lesions. However, physiological maneuvers known to increase ICP produced consistent and predictable responses in the anesthetized dogs (Figures 2–4), and baseline ICP results (approximately 7-12 mm Hg) consistent with the current findings in anesthetized normal dogs have been reported previously using gold standard direct ventricular CSF measurements,[6] and other intracranial locations (intraparenchymal, subarachnoid,[9,12,17,21] and at the cisterna magna[7,8]). Non-anesthetized dogs in sternal recumbency in our study also had mean ICP results in a range (7-15 mm Hg) consistent with previous reports in conscious dogs, suggesting a minimal effect of the anesthesia protocol in these animals.[9,10]

Drift in micro-strain-gauge devices, and specifically in the Codman MicroSensor, has been shown to be small in laboratory studies and in clinical studies in humans, even over several days,[34–36] and any effects on device location comparisons over 24 hours in our study were likely to be small. No differences were seen in ICP results in our study related to transducer placement within the cranial vault, but it is not possible to extrapolate these findings in normal dogs to a pathological setting. Craniospinal and supratentorial pressure gradients with intracranial lesions generally are accepted to exist, but debate still exists in human medicine regarding the presence of interhemispheric gradients, particularly with focal lesions.[58–60] After experimental epidural lesions in dogs,[45] no differences were seen in ICP using ipsilateral or contralateral measurements recorded from ventricular, intraparenchymal, or subdural transducer locations. Anecdotally, increased ICP results in the 1 dog (Figure 5) with an intracranial meningioma also were found to be similar for all catheter locations. Current recommendations in human medicine are to place devices ipsilateral to mass lesions when clinically appropriate.[61] Further study in dogs will be required to define optimal placement relative to specific lesions.

Based on the pathological findings in these dogs, the Codman MicroSensor ICP devices stayed secure throughout the course of the study and appeared to be tolerated well by the animals. Focal hemorrhages around the dural insertion site were seen in 2 of the dogs with more widespread hemorrhage over the majority of the cerebral hemisphere in an additional dog. All hemorrhage appeared to be self-limiting with no apparent effect on ICP or clinical signs, but compromise of dural vessels is a potential issue that may require a larger burr hole diameter to better visualize meningeal vasculature. Pathology after intraparenchymal placement was similar to that of previous reports after placement of other ICP devices in dogs in this location.[10,17] The lack of apparent clinical signs resulting from CNS pathology in these reports and the current study, together with extensive clinical data in humans, support the use of intraparenchymal placement of ICP sensors. Obvious histopathological sequelae are however present after intraparenchymal placement, and the lack of difference in ICP measurement between intraparenchymal and subdural locations for the Codman MicroSensor supports the placement in a subdural location.

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CONFLICT OF INTEREST DECLARATION

Authors declare no conflict of interest.

OFF-LABEL ANTIMICROBIAL DECLARATION

Authors declare no off-label use of antimicrobials.

INSTITUTIONAL ANIMAL CARE AND USE COMMITTEE (IACUC) OR OTHER APPROVAL DECLARATION

The experimental research protocol for use of bred-for research experimental dogs was approved by the University of California Davis IACUC. Protocol #8716.

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REFERENCES

1. Brain Trauma Foundation, American Association of Neurological Surgeons, Congress of Neurological Surgeons et al. Guidelines for the management of severe traumatic brain injury. VI. Indications for intracranial pressure monitoring. *J Neurotrauma*. 2007;24(Suppl 1): S37-S44.
2. Yuan Q, Wu X, Sun Y, et al. Impact of intracranial pressure monitoring on mortality in patients with traumatic brain injury: a systematic review and meta-analysis. *J Neurosurg*. 2015;122:574-587.
3. Carney N, Totten AM, O'Reilly C, et al. Guidelines for the management of severe traumatic brain injury, fourth edition. *Neurosurgery*. 2017; 80:6-15.
4. Garcia-Larrea L, Artru F, Bertrand O, Pernier J, Mauguiere F. The combined monitoring of brain stem auditory evoked potentials and intracranial pressure in coma. A study of 57 patients. *J Neurol Neurosurg Psychiatry*. 1992;55:792-798.
5. Brain Trauma Foundation, American Association of Neurological Surgeons, Congress of Neurological Surgeons et al. Guidelines for the management of severe traumatic brain injury. VII. Intracranial pressure monitoring technology. *J Neurotrauma*. 2007;24(Suppl 1):S45-S54.
6. Leonard JL, Redding RW. Effects of hypertonic solutions on cerebrospinal fluid pressure in the lateral ventricle of the dog. *Am J Vet Res*. 1973;34:213-219.
7. Novak G, Digel C, Burns B, James AE. Cerebrospinal fluid pressure measurements and radioisotope cisternography in dogs. *Lab Anim*. 1974;8:85-91.
8. Simpson ST, Reed RB. Manometric values for normal cerebrospinal fluid pressure in dogs. *J Am Anim Hosp Assoc*. 1987;23:629-632.
9. Verdura J, White RJ, Albin M. Chronic measurements of cerebrospinal-fluid pressure in the dog. A new method and results. *J Neurosurg*. 1964;21:1047-1050.
10. Kroin JS, McCarthy RJ, Stylos L, et al. Long-term testing of an intracranial pressure monitoring device. *J Neurosurg*. 2000;93:852-858.
11. Jakobsson KE, Thuomas KA, Bergstrom K, et al. Rebound of ICP after brain compression. An MRI study in dogs. *Acta Neurochir*. 1990;104: 126-135.
12. Grasberger RC, Spatz EL, Mortara RW, Ordia JI, Yeston NS. Effect of high-frequency ventilation versus conventional mechanical ventilation on ICP in head-injured dogs. *J Neurosurg*. 1984;60:1214-1218.
13. Palafox BA, Johnson MN, McEwen DK, et al. ICP changes following application of the MAST suit. *J Trauma*. 1981;21:55-59.
14. Dewey CW, Bailey CS, Haskins SC, Kass PH, Crowe DT. Evaluation of an epidural intracranial pressure monitoring system in cats. *J Vet Emerg Crit Care*. 1997;7:20-33.
15. Harrington ML, Bagley RS, Moore MP, Tyler JW. Effect of craniectomy, durotomy, and wound closure on intracranial pressure in healthy cats. *Am J Vet Res*. 1996;57:1659-1661.
16. Bagley RS, Harrington ML, Pluhar GE, et al. Effect of craniectomy/durotomy alone and in combination with hyperventilation, diuretics, and corticosteroids on intracranial pressure in clinically normal dogs. *Am J Vet Res*. 1996;57:116-119.
17. Bagley RS, Keegan RD, Greene SA, Harrington ML, Moore MP. Pathologic effects in brain after intracranial pressure monitoring in clinically normal dogs, using a fiberoptic monitoring system. *Am J Vet Res*. 1995;56:1475-1478.
18. Bagley RS, Keegan RD, Greene SA, Moore MP, Gavin PR. Intraoperative monitoring of intracranial pressure in five dogs with space-occupying intracranial lesions. *J Am Vet Med Assoc*. 1995;207: 588-591.
19. Keegan RD, Greene SA, Bagley RS, Moore MP, Weil AB, Short CE. Effects of medetomidine administration on intracranial pressure and cardiovascular variables of isoflurane-anesthetized dogs. *Am J Vet Res*. 1995;56:193-198.
20. Pluhar GE, Bagley RS, Keegan RD, et al. The effect of acute, unilateral transverse venous sinus occlusion on intracranial pressure in normal dogs. *Vet Surg*. 1996;25:480-486.
21. Packer RA, Simmons JP, Davis NM, Constable PD. Evaluation of an acute focal epidural mass model to characterize the intracranial pressure-volume relationship in healthy beagles. *Am J Vet Res*. 2011; 72:103-108.
22. Lopes PCF, Nunes N, Belmonte EA, et al. Two levels of the inspired oxygen fraction in propofol-anesthetized dogs with high intracranial pressure: cardiopulmonary function. *Arq Bras Med Vet Zoo*. 2014;66: 1351-1358.
23. Brosnan RJ, Esteller-Vico A, Steffey EP, LeCouteur RA, Liu IKM, Vaughan B. Effects of head-down positioning on regional central nervous system perfusion in isoflurane-anesthetized horses. *Am J Vet Res*. 2008;69:737-743.
24. Brosnan RJ, LeCouteur RA, Steffey EP, et al. Intracranial elastance in isoflurane-anesthetized horses. *Am J Vet Res*. 2004;65:1042-1046.
25. Brosnan RJ, LeCouteur RA, Steffey EP, et al. Direct measurement of intracranial pressure in adult horses. *Am J Vet Res*. 2002;63: 1252-1256.
26. Brosnan RJ, Steffey EP, LeCouteur RA, et al. Effects of duration of isoflurane anesthesia and mode of ventilation on intracranial and cerebral perfusion pressures in horses. *Am J Vet Res*. 2003;64:1444-1448.
27. Brosnan RJ, Steffey EP, LeCouteur RA, et al. Effects of body position on intracranial and cerebral perfusion pressures in isoflurane-anesthetized horses. *J Appl Physiol (1985)*. 2002;92:2542-2546.
28. Brosnan RJ, Steffey EP, LeCouteur RA, et al. Effects of ventilation and isoflurane end-tidal concentration on intracranial and cerebral perfusion pressures in horses. *Am J Vet Res*. 2003;64:21-25.
29. Kortz GD, Madigan JE, Goetzman BW, Durando M. Intracranial pressure and cerebral perfusion pressure in clinically normal equine neonates. *Am J Vet Res*. 1995;56:1351-1355.
30. Adrianov OS, Mering TA. *Atlas of the Canine Brain*. Ann Arbor, MI: Edwards Brothers, Inc.; 1964.
31. Lim RKS, Liu C, Moffitt RL. *A Stereotaxic Atlas of the Dog's Brain*. Springfield, IL: Charles C. Thomas; 1960.
32. Bates D, Maechler M, Bolker B, et al. Fitting linear mixed-effects models using lme4. *J Stat Softw*. 2015;67:1-48.
33. Lescot T, Reina V, Le Manach Y, et al. In vivo accuracy of two intraparenchymal intracranial pressure monitors. *Intensive Care Med*. 2011;37: 875-879.
34. Koskinen LO, Olivecrona M. Clinical experience with the intraparenchymal intracranial pressure monitoring Codman MicroSensor system. *Neurosurgery*. 2005;56:693-698. discussion 693-698.
35. Zacchetti L, Magnoni S, Di Corte F, et al. Accuracy of intracranial pressure monitoring: systematic review and meta-analysis. *Crit Care*. 2015; 19:420.

36. Gray WP, Palmer JD, Gill J, et al. A clinical study of parenchymal and subdural miniature strain-gauge transducers for monitoring intracranial pressure. *Neurosurgery*. 1996;39:927-931. discussion 931-922.
37. Hong WC, Tu YK, Chen YS, Lien LM, Huang SJ. Subdural intracranial pressure monitoring in severe head injury: clinical experience with the Codman MicroSensor. *Surg Neurol*. 2006;66(Suppl 2):S8-S13.
38. Newcombe VF, Hawkes RC, Harding SG, et al. Potential heating caused by intraparenchymal intracranial pressure transducers in a 3-tesla magnetic resonance imaging system using a body radiofrequency resonator: assessment of the Codman MicroSensor transducer. *J Neurosurg*. 2008;109:159-164.
39. Tanaka R, Yumoto T, Shiba N, et al. Overheated and melted intracranial pressure transducer as cause of thermal brain injury during magnetic resonance imaging: case report. *J Neurosurg*. 2012;117:1100-1109.
40. Macmillan CS, Wild JM, Andrews PJ, et al. Accuracy of a miniature intracranial pressure monitor, its function during magnetic resonance scanning, and assessment of image artifact generation. *Neurosurgery*. 1999;45:188-192. discussion 192-183.
41. Zhang X, Medow JE, Iskandar BJ, et al. Invasive and noninvasive means of measuring intracranial pressure: a review. *Physiol Meas*. 2017;38:R143-R182.
42. Ilie LA, Thomovsky EJ, Johnson PA, et al. Relationship between intracranial pressure as measured by an epidural intracranial pressure monitoring system and optic nerve sheath diameter in healthy dogs. *Am J Vet Res*. 2015;76:724-731.
43. Wiegand C, Richards P. Measurement of intracranial pressure in children: a critical review of current methods. *Dev Med Child Neurol*. 2007;49:935-941.
44. Berlin T, Murray-Krezan C, Yonas H. Comparison of parenchymal and ventricular intracranial pressure readings utilizing a novel multi-parameter intracranial access system. *Springerplus*. 2015;4:10.
45. Crutchfield JS, Narayan RK, Robertson CS, Michael LH. Evaluation of a fiberoptic intracranial pressure monitor. *J Neurosurg*. 1990;72:482-487.
46. Jallo J, Saetzler R, Mishke C, Young WF, Vasthare U, Tuma RF. A chronic model to simultaneously measure intracranial pressure, cerebral blood flow, and study the pial microvasculature. *J Neurosci Methods*. 1997;75:155-160.
47. Rahimifar M, Tator CH, Shanlin RJ, Sole MJ. Effect of blood transfusion, dopamine, or normal saline on neurogenic shock secondary to acutely raised intracranial pressure. *J Neurosurg*. 1989;70:932-941.
48. Zwienenberg M, Gong QZ, Lee LL, et al. ICP monitoring in the rat: comparison of monitoring in the ventricle, brain parenchyma, and cisterna magna. *J Neurotrauma*. 1999;16:1095-1102.
49. Schmitt M, Eymann R, Antes S, et al. Subdural or intraparenchymal placement of long-term telemetric intracranial pressure measurement devices? *Acta Neurochir Suppl*. 2012;113:109-113.
50. Poca MA, Sahuquillo J, Topczewski T, Peñarrubia MJ, Muns A. Is intracranial pressure monitoring in the epidural space reliable? Fact and fiction. *J Neurosurg*. 2007;106:548-556.
51. Barlow P, Mendelow AD, Lawrence AE, Barlow M, Rowan JO. Clinical evaluation of two methods of subdural pressure monitoring. *J Neurosurg*. 1985;63:578-582.
52. Jiang Y, Ye Z, You C, et al. Systematic review of decreased intracranial pressure with optimal head elevation in postcraniotomy patients: a meta-analysis. *J Adv Nurs*. 2015;71:2237-2246.
53. Alarcon JD, Rubiano AM, Okonkwo DO, et al. Elevation of the head during intensive care management in people with severe traumatic brain injury. *Cochrane Database Syst Rev*. 2017;12:CD009986.
54. Binz DD, Toussaint LG 3rd, Friedman JA. Hemorrhagic complications of ventriculostomy placement: a meta-analysis. *Neurocrit Care*. 2009;10:253-256.
55. Lozier AP, Sciacca RR, Romagnoli MF, Connolly ES Jr. Ventriculostomy-related infections: a critical review of the literature. *Neurosurgery*. 2002;51:170-181. discussion 181-172.
56. Anderson RC, Kan P, Klimo P, Brockmeyer DL, Walker ML, Kestle JR. Complications of intracranial pressure monitoring in children with head trauma. *J Neurosurg*. 2004;101:53-58.
57. Guyot LL, Dowling C, Diaz FG, Michael DB. Cerebral monitoring devices: analysis of complications. *Acta Neurochir Suppl*. 1998;71:47-49.
58. Sahuquillo J, Poca MA, Arribas M, Garnacho A, Rubio E. Interhemispheric supratentorial intracranial pressure gradients in head-injured patients: are they clinically important? *J Neurosurg*. 1999;90:16-26.
59. Bekar A, Taskapilioglu O, Yilmazlar S, Ender K, Aksoy K. Is supratentorial pressure difference clinically relevant? Analysis of 55 consecutive cases by bilateral intracranial pressure monitoring. *Neurol Res*. 2008;30:465-470.
60. Yano M, Ikeda Y, Kobayashi S, Otsuka T. Intracranial pressure in head-injured patients with various intracranial lesions is identical throughout the supratentorial intracranial compartment. *Neurosurgery*. 1987;21:688-692.
61. Chesnut R, Videtta W, Vespa P, et al. Intracranial pressure monitoring: fundamental considerations and rationale for monitoring. *Neurocrit Care*. 2014;21(Suppl 2):S64-S84.

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