Direct measurement of intracranial pressure in adult horses

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Objective—To develop a method for surgical placement of a commercial microsensor intracranial pressure (ICP) transducer and to characterize normal ICP and cerebral perfusion pressures (CPP) in conscious adult horses.

Animals—6 healthy castrated male adult horses (1 Holsteiner, 1 Quarter Horse, and 4 Thoroughbreds).

Procedure—Anesthesia was induced and maintained by use of isoflurane as the sole agent. Catheters were inserted percutaneously into the jugular vein and carotid artery. A microsensor ICP transducer was inserted in the subarachnoid space by means of right parietal craniotomy. The burr hole was then sealed with bone wax, the surgical incision was sutured, and the transducer was secured in place. Measurements were collected 1 hour after horses were able to stand during recovery from anesthesia.

Results—Mean ± SD values for ICP and CPP were 2 ± 4 and 102 ± 26 mm Hg, respectively.

Conclusions and Clinical Relevance—This report describes a relatively facile technique for obtaining direct and accurate ICP measurements for adult horses. The ICP values obtained in this study are within reference ranges established for other species and provide a point of reference for the diagnosis of abnormal ICP in adult horses. (*Am J Vet Res* 2002;63:1252–1256)

Cerebral blood flow (CBF) is a function of the fluid driving pressure to the brain (**cerebral perfusion pressure [CPP]**) and **cerebral vascular resistance(CVR)**; it can be expressed as a modification of Ohm's Law: CBF=CBP/CVR. In turn, CPP is equal to the difference between **mean arterial blood pressure in the arterial circle of the brain (MAP_{ACB})** and the pressure within the cranial vault (**intracranial pressure [ICP]**), as determined by use of the following equation: $CPP = MAP_{ACB} - ICP$. As a result, when constant arterial blood pressure is maintained, an increase in ICP causes a decrease in CPP. Similarly, when constant cerebral vascular resistance is maintained, a decrease in CPP causes a decrease in cerebral blood flow and may compromise cerebral oxygen delivery.

In healthy animals, cerebral blood flow changes in response to cerebral metabolic demand, not in response to CPP. However, this normal autoregulatory mechanism may be disrupted by intracranial disease. The space within the rigid calvarium is fixed and comprised of 3 volumes; namely, brain parenchyma, CSF, and blood. An increase in 1 of these volumes that is not compensated by a decrease in the other intracranial volumes must result in increased pressure within the calvarium. Space-occupying lesions (such as hematomas or tumors) and parenchymal edema (which can develop following trauma or infection) may progress and exceed the compensatory capacity within the calvarium, resulting in intracranial hypertension and cerebral ischemia.

Despite clinical indications for direct ICP measurements to diagnose and monitor the course of intracranial hypertension, descriptions for this technique in adult horses do not exist. Therefore, the objectives of the study reported here were to develop a method that can be used to accurately obtain direct subarachnoid ICP measurements in adult horses for use in clinical and research situations and to establish a reference range of ICP values in conscious standing horses.

Materials and Methods

Animals—Six castrated male horses were included in the study, which included 1 Holsteiner, 1 Quarter Horse, and 4 Thoroughbreds. Horses were 3 to 8 years old (mean ± SD, 4.5 ± 1.9 years) and weighed between 494 and 576 kg (518 ± 37 kg). Results of physical examination of all horses were unremarkable. This study protocol was approved by the Animal Care and Use Committee at the University of California, Davis.

Anesthesia—Food was withheld from all horses for 12 hours prior to the start of the study, but water was available ad libitum. Preanesthetic medications were not administered. Anesthesia was induced by use of isoflurane administered via a face mask, using a procedure that has been described elsewhere.[1-3] During intracranial placement of a pressure transducer for measurement of ICP, a surgical plane of anesthesia was maintained by administration of isoflurane at 1.3 to 1.5 **minimum alveolar concentration (MAC)**, as verified by analysis of end-tidal gases by use of a calibrated infrared gas analyzer[a]; gross or purposeful movement of horses was not observed. Although placement of the pressure transducer was accomplished in < 45 minutes, horses remained anesthetized as part of a separate anesthesia study for approximately 5 additional hours at a constant dose of 1.57% isoflurane, equivalent to 1.2 MAC for this species.[4]

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Insertion of intravascular catheters—Following anesthetic induction, the skin over the right jugular furrow was shaved and aseptically prepared. A 16-gauge, 13.1-cm catheter was percutaneously inserted into the right carotid artery such that the catheter tip was located close to the thoracic inlet. An 18-gauge, 110-cm polypropylene catheter was inserted percutaneously via the Seldinger technique and advanced into the right ventricle, as verified by continuous pressure waveform readings. This catheter was then gradually withdrawn until the ventricular waveform was lost as the tip moved into the right atrium, allowing measurement of central venous pressure. Both catheters were secured in place with nylon sutures and cyanoacrylate tissue glue.

Surgical procedure—A rectangular area of skin overlying the dorsal aspects of the parietal and frontal bones was shaved, scrubbed with povidone iodine and isopropyl alcohol, and covered with a sterile surgical drape. A point located 1 cm caudal to the midpoint on the median plane between the lateral canthus of both eyes and the external occipital protuberance served as the landmark (**Fig 1**). From this point, a curvilinear skin incision was made that extended 2 cm in the rostral and caudal directions and traced the origin of the right temporalis muscle. Care was taken to avoid a substantial cutaneous vein that consistently was found at the more rostral aspect of the incision.

With Weitlaner retractors in place, the right temporalis muscle was elevated from the underlying parietal bone. A high-speed nitrogen-driven drill[b] was used to create a 4-mm-diameter parietal craniotomy at a site 0.5 to 1 cm to the right of the midline of the aforementioned landmark. Use of this position was essential to avoid the dorsal sagittal sinus and several large meningeal vessels. A small channel also was cut into the caudal aspect of the craniotomy to allow smooth egress for the transducer cable.

A 1,000 × 0.7-cm nylon microsensor ICP[c] transducer was electronically zeroed and calibrated in a shallow container of sterile saline (0.9% NaCl) solution, as directed by the manufacturer. Two 90° bends away from the strain gauge were made 1 and 2 cm, respectively, from the end of the transducer cable. The end of the transducer cable was then inserted through a 1-mm durotomy into the subarachnoid space so that the strain gauge rested upon the pia mater (**Fig 2**). The craniotomy was sealed with bone wax with the transducer still in place. Jugular vein occlusion was used to test for normal functioning of the transducer and resulted in detection of a consistent increase in ICP of at least 4 to 5 mm Hg within 15 to 30 seconds.

Subcutaneous and subcuticular tissues were closed with 2-0 polydioxanone monofilament suture[d] on a reverse cutting needle with a simple-interrupted or simple-continuous pattern. Cutaneous cruciate mattress sutures of 3-0 nylon completed closure.

Accuracy of the ICP transducers was verified following each experiment by measuring pressure within a column of distilled water of known height. For calibration pressures between –20 and +30 mm Hg, the absolute error of transducer readings was always ≤ 1 mm Hg and was well within manufacturer specifications.

Data collection—Measurements were obtained from standing conscious horses 1 hour after they were able to stand during recovery from anesthesia. In another study,[5] investigators did not detect significant differences between cardiovascular variables in horses measured 1 hour following recovery from isoflurane-only anesthesia, compared with values obtained in unmedicated horses prior to anesthesia.

The pH, Pa_{O_2}, and Pa_{CO_2} in blood samples obtained from the carotid artery were determined by use of an automated blood gas analyzer.[e] These values were corrected on the basis of standard curves obtained through tonometry of equine blood and certified standard gas mixtures. Values also were corrected on the basis of body temperature, as measured by a rectal thermometer. Arterial and central venous blood pressures were measured by use of fluid-filled catheters attached to strain gauge transducers positioned at the intersection of the jugular furrow and thoracic inlet and were calibrated daily against mercury and water manometers, respectively. Data were recorded by use of a multiple-channel chart recorder[f] or digital output display.[g]

Height of the lateral canthus relative to the thoracic inlet

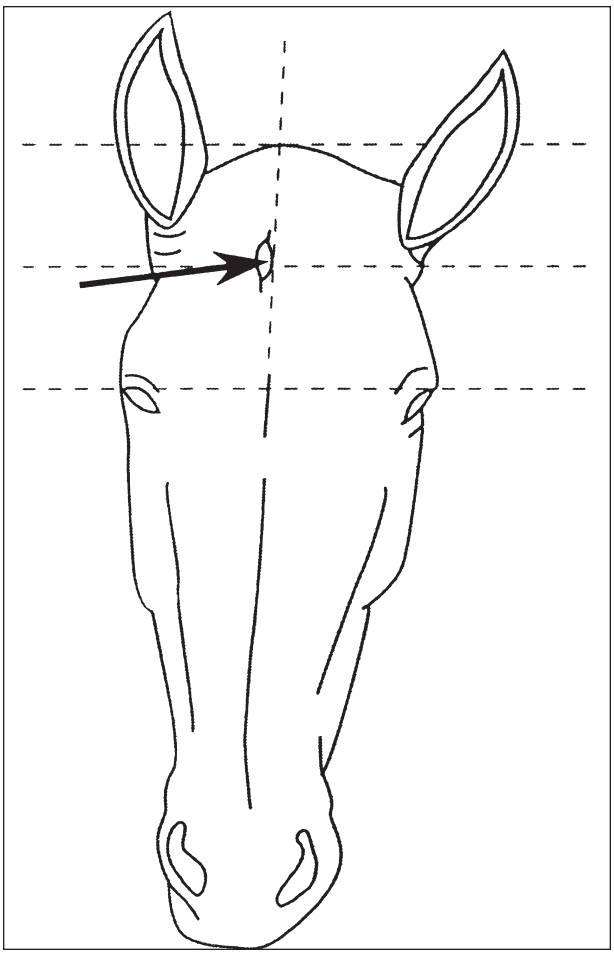

Figure 1—Diagram depicting surgical landmarks on the head of a horse to facilitate direct measurement of intracranial pressure. Craniotomy was performed at a point located 1 cm caudal to the midpoint between the lateral canthus and occipital protuberance and 0.5 cm to the right of the median plane (arrow).

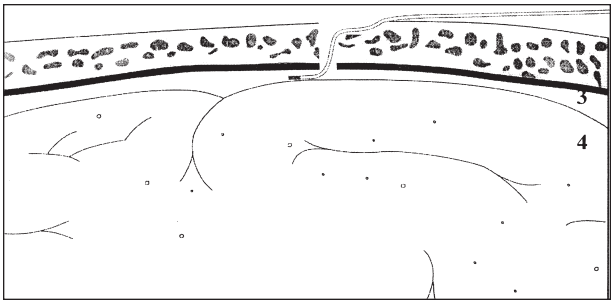

Figure 2—Cross-sectional diagram depicting placement of a subarachnoid transducer in the craniotomy site. 1 = Bone. 2 = Dura and arachnoid mater. 3 = Subarachnoid space. 4 = Brain.

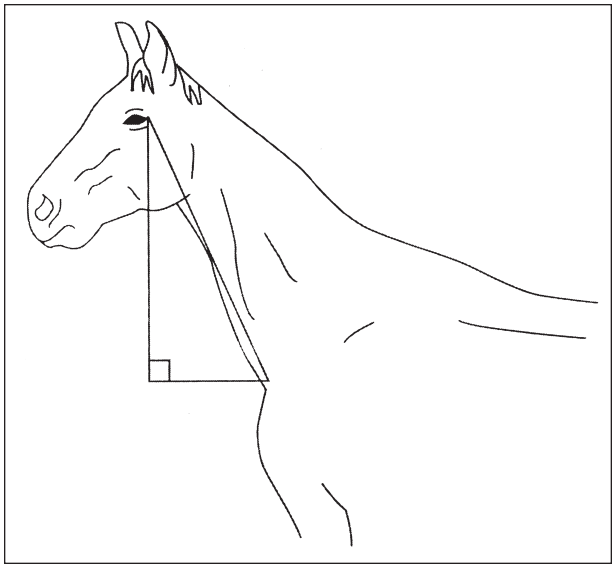

Figure 3—Diagram of the triangulation technique used to measure head elevation. The hypotenuse equals the distance between the lateral canthus and thoracic inlet. The horizontal leg of the triangle equals the shortest distance between the thoracic inlet and a weighted string dropped from the lateral canthus. The vertical leg of the triangle (head height) was calculated by use of the Pythagorean theorem.

was easily and rapidly triangulated, using the distance from the thoracic inlet to the lateral canthus as the hypotenuse and the horizontal distance from the thoracic inlet to a line dropped from the lateral canthus as a leg of the triangle[a] **(Fig 3)**. This vertical distance (head height) was used to closely approximate the hydrostatic gradient between the thoracic inlet and arterial circle of the brain (circle of Willis). Because a pressure of 1 mm Hg is equivalent to 1.36 cm H_2O, arterial pressure measurements could be used to estimate blood pressure within the arterial circle of the brain. Thus, for head positions above the thoracic inlet, pressures in the arterial circle of the brain were equal to measured arterial pressure minus head height with the difference divided by 1.36 cm. For head positions below the thoracic inlet, pressures in the arterial circle of the brain were equal to measured arterial pressure plus head height with the sum divided by 1.36 cm. The CPP was calculated as the difference between mean pressure in the arterial circle of the brain and ICP (ie, $CPP = MAP_{ACB} - ICP$).

Follow-up monitoring—Following data collection, the ICP transducer was removed in conscious horses by applying gentle traction on the cable. Nitrofurazone topical ointment was applied to the skin's surface to help reduce the risk of local infection. Systemic administration of antibiotics was not performed unless there was an indication of possible infection.

All horses received daily physical examinations for 1 week after the study. Treatment for postoperative complications was instituted when needed.

Statistical analysis—Means and standard deviations were calculated for physiologic responses. Data were analyzed by use of linear regression.

Results

Measurement of variables—Results from arterial blood gas analyses were only available for 3 horses **(Table 1)**. These horses were all normocapnic and normoxemic without evidence of any acid-base disturbances. All horses were similarly calm and eupneic when intracranial and hemodynamic measurements were obtained.

Table 1—Mean ± SD values for blood pressures, intracranial pressure (ICP), and arterial blood gas analysis in 6 conscious horses 1 hour after recovery from isoflurane-induced anesthesia

Variable	Mean ± SD	Range
Head height (cm)	41 ± 26	0–69
Pa_{O_2} (mm Hg)*	104 ± 30	80–134
Pa_{CO_2} (mm Hg)*	41 ± 3	37–43
pH*	7.39 ± 0.01	7.39–7.40
Carotid SAP (mm Hg)	182 ± 22	150–209
Carotid DAP (mm Hg)	112 ± 7	103–119
Carotid MAP (mm Hg)	133 ± 17	111–153
CVP (mm Hg)	4 ± 14	–16–25
Circle MAP (mm Hg)	103 ± 26	85–153
ICP (mm Hg)	2 ± 4	–3–7
CPP (mm Hg)	101 ± 22	78–152

*Represents values for only 3 horses.
SAP = Systolic arterial pressure. DAP = Diastolic arterial pressure. MAP = Mean arterial pressure. CVP = Central venous pressure. Circle = Arterial circle of the brain. CPP = Cerebral perfusion pressure.

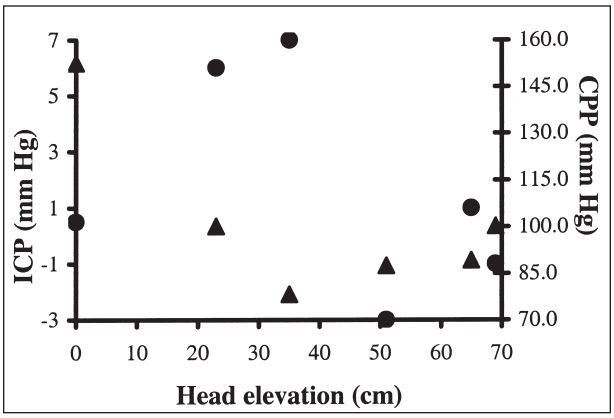

Figure 4—Graph of intracranial pressure (ICP [circles]) and cerebral perfusion pressure (CPP [triangles]) versus head elevation above the thoracic inlet for 6 conscious standing horses.

Arterial and central venous pressures and ICP responses were summarized (Table 1). There was a poor correlation between head elevation and ICP (r^2 = 0.14) as well as between mean arterial pressure and ICP (r^2 = 0.30). In 2 horses with elevated head positions, slightly subatmospheric ICP was recorded **(Fig 4)**. Additionally, a relatively wide range of CPP was observed (78 to 152 mm Hg).

Follow-up monitoring—For 24 to 48 hours after recovery, all horses tended to be quiet and less active in their stalls, often standing with their head down. However, the horses still ate and were alert.

One horse that received phenylbutazone for 36 hours to treat postrecovery peripheral neuropathy became febrile (39.7 C) 3 days after the experiment and was tachycardic and inappetent. The horse had been reintubated during the anesthetic procedure, and a small amount of mucous and short plant fibers were found in the endotracheal tube at that time. A CBC count revealed neutrophilia with a slightly toxic left shift and hyperfibrinogenemia. We did not detect evidence of cervical pain or discharge or inflammation at the surgical site. It was believed that pyrexia was most

likely the result of pneumonia, although thoracic radiographs were not obtained to confirm this tentative diagnosis. The horse was treated with 20,000 U of procaine penicillin G/kg administered IM every 12 hours for 1 week. Clinical signs resolved within the subsequent 24 hours and did not return following discontinuation of treatment.

Discussion

The study reported here has documented a method for obtaining direct ICP measurements in conscious standing adult horses. Furthermore, reference ranges obtained from this study are essential for future comparisons to values of horses with possible intracranial pathology.

Mean ICP values in adult horses are within the generally accepted reference range of ICP values obtained by direct measurement in humans[6] but tends to be lower than values reported for other animals (**Appendix**). Most notable are the small but significant increased ICP values reported in foals,[7] compared to adult horses. This difference may be the result of body position, because foals in that study were restrained in right lateral recumbency and, thus, had a decreased hydrostatic gradient between the right atrium and brain. Additionally, those foals were allowed to suckle prior to obtaining measurements; stomach distention, cranial displacement of the diaphragm, and increased visceral pressure on the vena cava caused by positioning in right lateral recumbency could possibly have caused increased central venous pressure, thereby decreasing cerebral venous drainage and increasing ICP. In contrast, arterial pressure changes are unlikely contributors to these ICP differences, because mean CPP was 20 to 25 mm Hg less in the foals than the adult horses and, therefore, would not be expected to contribute to increased cerebral blood volume given a constant cerebrovascular tone. Age-related changes in cerebrovascular autoregulation or parenchymal elastance in horses may be involved, although there is no evidence in the literature to support this.

A poor correlation between blood pressure and ICP implies active compensatory changes in cerebral vascular resistance to maintain constant cerebral blood volume and flow in the face of alterations in CPP. This is particularly important for animals such as horses that have large changes in hydrostatic pressure concomitant with changes in head position. Thus, it is important to recognize the potential for pathologic conditions (ie, intracranial disease, pulmonary CO_2 retention) and drugs (ie, anesthetics) to interfere with normal physiologic regulation and, hence, compromise cerebral blood flow.

The commercial microsensor ICP transducer used in the study[c] reported here can produce pressure measurements with high accuracy (within 4 mm Hg) and response frequency (approx 30 Hz) and low temporal (< 1 mm Hg/d) and temperature (< 0.03 mm Hg/1 C) drifts, compared with that of other commercially available monitoring devices.[8-14] Because the transducer is located at the cable tip, there is no need for a fluid column that may degrade the signal or become occluded. However, 1 primary disadvantage of implantable transducers is the inability to recalibrate them once they have been surgically placed.[15] Moreover, without an additional display device, numeric digital output from the commercial ICP monitor used in the study reported here precludes ICP waveform analysis.

Although intraventricular pressure is often regarded as the criterion-referenced standard site for ICP measurements, the subdural space provides much easier access with a decreased risk of parenchymal trauma, hemorrhage, and infection while still allowing for accurate quantitation of pressure, providing that pathologic changes are not compartmentalized within the calvarium.[16] Calculation of CPP also requires referencing of the arterial transducer to an external landmark to approximate pressure within the arterial circle of the brain. In recumbent animals with the head positioned level with the thoracic inlet, an adjustment is generally not necessary. However, elevating or lowering the head will lead to a respective overestimation or underestimation of CPP if the arterial pressure is referenced at the level of the heart. In humans, arterial pressure is referenced to the foramen of Munro and skull base, approximated externally by a point located 1 cm above and two thirds posteriorly along an imaginary line connecting the lateral canthus of the eye and the tragus of the ear.[17] In horses, the lateral canthus was used as a close external approximation of the base of the brain, and arterial circle pressure estimates could be obtained by adjusting mean arterial pressure measurements by a known vertical hydrostatic difference.

The horses reported here had minimal postoperative morbidity associated with placement of the intracranial transducer. The quiet deportment of horses up to 48 hours after the study may have been a behavioral manifestation of headache caused by loss of some CSF, resulting in decreased pressure and increased tension on pain-sensitive intracranial vessels.[16] Although the febrile episode observed in 1 horse in our study probably was caused by pneumonia, infection of the CNS remains an important risk when obtaining direct measurements. Retrospective analysis[18] of direct ICP monitoring in a human trauma center found a CSF infection rate of 7.4%; increased risk was associated with ventricular catheters, CSF leakage, systemic infections, and chronic or repeated ICP measurements. Antibiotic prophylaxis in instrumented patients remains controversial, although there is evidence to suggest that preemptive treatment does not reduce the incidence of infections and may actually contribute to increased morbidity from sepsis.[19]

Ultimately, measurement of ICP and CPP are important because these variables are determinants of cerebral blood flow. However, decreased CPP may suggest, but cannot confirm, decreased cerebral blood flow. It would be ideal to measure cerebral blood flow directly in horses, although anatomic considerations make this difficult. Methods involving arteriovenous differences of inert gases or tracers are generally only useful in research settings. Positron-emission tomography and other nuclear medicine techniques have clinical or investigative potential, but they are expensive, often unaccommodating to larger species, and not readily available in most veterinary facilities. Thus,

direct ICP monitoring provides a potentially valuable clinical tool in the management of cerebral disease in horses.

[a]LB2 anesthetic analyzer, Sensormedics Corp, Anaheim, Calif.
[b]Surgairtome, Linvatec Inc, Key Largo, Fla.
[c]Codman Microsensor, Codman & Shurtleff Inc, Raynham, Mass.
[d]PDS, Ethicon Inc, Somerville, NJ.
[e]ABL 5, Radiometer America, Westlake, Ohio.
[f]Grass Model 7D, Statham Medical Instruments, Hato Rey, Puerto Rico.
[g]Model 90603A, Spacelabs Medical, Redmond, Wash.
[h]ICP Express monitor, Codman & Shurtleff Inc, Raynham, Mass.
[i]Sturges BK, LeCouteur RA, Tripp LD. Intracranial pressure monitoring in clinically normal dogs using the Codman Microsensor ICP transducer (abstr), in *Proceedings*. 18th Annu Vet Med Forum, 2000;238.

Appendix

Intracranial pressure measurements (mean ± SD or range) in conscious unsedated animals

Animals	Measurement technique	ICP (mm Hg)
Horses (foals)	Subdural catheter[7]	5.8 ± 1.8 – 9.6 ± 1.6
Humans	Direct measurement[6]	0 – 15
Monkeys	Subdural transducer[20]	6.4 (mean)*
Dogs		
	Subdural transducer[i]	0 – 25
	Subdural catheter[21]	10 – 11 (mean)
Rats		
	Intraventricular transducer[22]	8.0 ± 3.4 – 10.4 ± 3.2
	Intraventricular catheter[23]	9.5 ± 0.6 – 13.3 ± 0.3*
	Intraventricular catheter[24]	7.0 ± 1.9*†
	Intraparenchymal transducer[25]	4.3 ± 2.9
Rabbits		
	Intraventricular bolt catheter[26]	5.2 ± 1.1
	Subarachnoid bolt catheter[27]	4.2 ± 0.7*
	Intraventricular catheter[28]	5.3 ± 1.9
Goats		
	Intraventricular catheter[29]	6 ± 6 – 8 ± 3
	Intraventricular catheter[30]	11.1 ± 4.5*

*Calculated conversion from millimeters or centimeters of H_20. †Data interpolated from graph.

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