Sonography for Determining the Optic Nerve Sheath Diameter With Increasing Intracranial Pressure in a Porcine Model

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Objectives—This study investigated whether it is feasible to use sonography to monitor changes in the optic nerve sheath diameter in a porcine model.

Methods—A fiber-optic intracranial pressure transducer was surgically placed through the frontal sinus directly into the brain parenchyma of adult Yorkshire pigs (n = 5). A second bolt was placed on the contralateral side for intraparenchymal fluid infusion. Optic nerve sheath diameter measurements were acquired by each of 2 ultrasound operators around the leading edge of the nerve, 3 to 5 mm distal from the origin of the optic nerve. To induce a change in diameter, intracranial pressure was manipulated by injecting normal saline into the intraparenchymal infusion catheter located in the symmetric contralateral position as the pressure-monitoring probe.

Results—Data from 1 pig were unusable because of a cerebrospinal fluid leak into the sinus and orbital fissure. Saline aliquots of 1 to 10 mL were able to generate intracranial pressures typically starting from 10 to 15 mm Hg and increasing to 75 to 90 mm Hg, which eventually evoked a Cushing response. Fluid injection was controlled to increase pressures by 60 mm Hg over a 15- to 20-minute period. Regression analysis of all animals showed that the optic nerve sheath diameter increased by 0.0034 mm/mm Hg of intracranial pressure; however, this slope ranged from 0.0025 to 0.0046, depending on the animal measured. There was no discernible effect of the ultrasound operator on the slope; however, measurements made by 1 operator were consistently higher than the others by about 8% of the overall diameter range.

Conclusions—These results suggest that the use of the optic nerve sheath diameter to noninvasively confirm acute changes in intracranial pressure over 1 hour is feasible in a porcine model. We recommend that this method be validated in humans using direct intracranial pressure measurement where possible to confirm it as a screening tool for acute and chronically increased diameters secondary to elevated pressure in clinical settings.

Key Words—elevated intracranial pressure; intracranial pressure; optic nerve; optic nerve sheath diameter; sonography

Since the first report of ultrasound imaging of the eye in 1956,1 a group of applications has evolved to offer crucial diagnostic information for many ocular disorders2–5 and, more recently, head injury.6–8 Sonography is especially helpful when visual or ophthalmologic inspection is impossible to perform or does not provide a definitive diagnosis. In ophthalmology, sonography, is used in 3 distinct clinical applications: sonographic biometry, which pursues precise distance measurements (A-mode);
sonographic biomicroscopy, which is largely limited to the anterior segment; and B-mode (2-dimensional) ocular imaging, which is primarily used for posterior segment diseases.

Multipurpose ultrasound systems are commonly used for first-line diagnostic and treatment support applications in emergency medicine,7 in the field and remote settings,10 and in isolated environments such as spacecraft.11-13 These systems also have shown excellent ophthalmic images when fitted with high-frequency probes.14-16 Most systems of this class use sophisticated focusing, image optimization, and Doppler capabilities that are different from “ophthalmic-only” ultrasound systems; their ability to image the eye through the eyelid give them the advantage of faster, safer examinations without the need for topical anesthesia. Linear and microconvex ultrasound transducers have been used to image trauma and primary diseases of the globe,17 as well as a multitude of secondary changes in the eye and orbital structures due to systemic and extracocular processes such as elevated intracranial pressure. Multipurpose devices have been used for assessment of altered flow patterns in the central retinal artery, suggesting carotid artery occlusion or dissection, venous slugging, suggesting hyperviscosity syndromes or peri-orbital gas secondary to facial trauma, and others. However, the growing popularity of ophthalmic sonography in emergency and critical care medicine is largely attributable to its ability to reveal or rule out characteristic intraorbital signs of elevated intracranial pressure.

All available bedside methods for determining elevated intracranial pressure are to some degree invasive, and none are rapid. Recent studies have implied a causal relationship between elevated intracranial pressure and optic nerve sheath diameter measured by sonography. Sonography of the eye in the emergency department is more commonly used to rule out any visual defect secondary to retinal detachment, vitreous detachment, or bleeding. With sonography becoming more affordable, portable, rugged, reliable, and ubiquitous in emergency departments and remote settings, this is rapidly mounting to support this efficient, noninvasive application for determining intracranial pressure in many medical environments.

As with any sonographic measurement, there is always a concern about interoperator variability. Ballantyne et al18 performed a study to quantify the observer variation in sonographic measurement of the optic nerve sheath diameter in healthy adults. Sixty-seven healthy adult volunteers underwent sonographic examination of both eyes by 3 independent observers using a 7-MHz sector probe. They concluded that sonographic measurement of the optic nerve sheath diameter is a readily learned, reproducible technique with low intraobserver and interobserver variation. The average interobserver variation (±0.2 mm) is comparable to the inherent measurement accuracy of the equipment used in the study.

The experiment described herein was part of a study sponsored by the National Aeronautics and Space Administration to develop sonographic procedures for long-duration space flight.19 Because the only imaging device currently flown in space is a multipurpose ultrasound system (HD1 5000; Philips Healthcare, Bothell, WA), procedures and protocols were sought to take utmost advantage of the capabilities of this device, should certain foreseeable medical conditions occur in space, including ophthalmic trauma and blunt head and body injury. Accordingly, this study was designed to investigate whether changes in the optic nerve sheath diameter, as detected at the bedside by sonography, could reliably correlate with manipulated intracranial pressure in a porcine model.

Materials and Methods

Adult female Yorkshire pigs (n = 5; average weight, 55 kg) received preoperative induction sedation of tiletamine/zolazepam (4 mg/kg), ketamine (2 mg/kg), and xylazine (2 mg/kg) by intramuscular injection. A deep surgical plane of anesthesia was maintained with pentobarbital infusion (0.2–1 mg/kg/min) as needed. In a follow-on study, this experimental animal model was also studied during the reduced gravity portions of parabolic flight, and inhalation anesthesia was not certified for that environment. Therefore pentobarbital infusion was also used in this pilot study. The animals were intubated and continuously ventilated with a model 754 ventilator (Impact Instrumentation, Inc, Caldwell, NJ) with a tidal volume of 600 to 750 mL and a rate of 10 to 14 per minute. Other experiments were performed on these animals at the same time (abdominal sinus20 sonographic protocol development for follow-on microgravity during parabolic flights), and our protocol was designed to have no impact on these studies. Strict adherence to the “Standard Anesthetic Regimen” protocols and the Guide for the Care and Use of Laboratory Animals (National Institutes of Health publication 86-23) was ensured for all experiments on these animals.

Hair on the animals’ head was removed with a depilatory agent and shaving, and a Foley catheter was placed in the bladder. Intravenous access for fluids and anesthesia was secured through catheters in a large vein on both ears. Direct cut-down was performed to secure access to the jugular vein and internal carotid artery lumina for the measurement of central venous pressure and arterial pressure,
respectively, using disposable semiconductor strain gauge transducers. All pressure transducers were zeroed to the same hydrostatic pressure of the mid-right atrium in each animal after placing them in a prone position. Rectal temperature and 5-lead electrocardiography were monitored (CS/3 anesthesia monitor with a specially modified video output for real-time recording of the monitor display; Datex Ohmeda Inc, Madison, WI).

The surgical placement of an intracranial pressure monitor is more straightforward in humans compared to the porcine model. The porcine frontal sinus extends posteriorly to overlie most of the cerebrum, which is small relative to the size of the head, which makes accurate placement of an intraparenchymal intracranial pressure transducer more challenging. We developed a surgical approach, which traverses the anterior and posterior walls of the frontal sinus, permitting advancement of the fiber-optic intracranial pressure transducer through the frontal sinus and directly into the brain parenchyma. An incision was made at the mid-pupillary line, about 2 cm from the midline and 10 cm superior to the superior orbital ridge. An access hole was drilled perpendicular to the plane of the cranium until the anterior frontal sinus was traversed. After drilling through the posterior wall, a blunt probe was used to open the dura. A fiber-optic intracranial pressure probe (Camino MPM–1; Integra Neurosciences, Plainsboro, NJ) was then inserted into the brain parenchyma, and its position was confirmed by the presence of the typical intracranial pressure waveform associated with respiration. The intraparenchymal location of the probe also was positively confirmed at autopsy.

A specially designed restraint system was used to secure the animal in a supine position yet ensure access to the dorsal surface of the head for access to the cranial bolts used for intracranial pressure measurement in one hemisphere and for intraparenchymal fluid infusion through a similarly placed catheter, in the other.

The sonographic approach was accomplished from either the upper or lower lid from a slightly anterolateral-to-medial approach depending on the gaze of the animal. The imaging plane was chosen to achieve the best longitudinal image of the optic nerve for cross-sectional diameter measurements. This plane sometimes included the lens in the image; however, no distortions were observed when this procedure was done. A LOGIQ Book XP portable ultrasound system (GE Healthcare, Milwaukee, WI) was used for this study, and the transducer selection choices were from 1 of 2 higher-frequency transducers: a 10 MHz linear array “hockey stick” or a 6- to 10-MHz tightly curved microconvex transducer. Still images were acquired digitally and stored on the hard drive of the system. High-quality still-frame images were obtained for each increment in volume administered. The same probe was used on the same eye throughout manipulation of intracranial volumes.

Sonography was performed on both eyes of each animal by an independent operator on each eye. The ultrasound operators declared successful data captures as the intracranial pressure was increased by each new aliquot of saline. During subsequent data analysis, additional sonographic videos were reviewed to ensure the best achievable image quality and probe positioning. Image analysis was performed retrospectively offline. Figure 1 illustrates how measurements were made on a perpendicular intersect from the leading edge to the leading edge of the nerve, 3 to 5 mm distal from the anterior and posterior sclera surfaces.

The intracranial pressure of each animal was manipulated by injecting normal saline into the intraparenchymal infusion catheter located in the symmetric contralateral position as the intracranial pressure-monitoring probe. The animals were euthanized while still in deep anesthesia immediately after experiment completion, using an American Veterinary Medical Association–approved method: a rapid intravenous potassium chloride bolus to induce cardiac arrest or by B-Euthanasia (Intervet/Schering-Plough Animal Health, Millsboro, DE) at 1 mL/10 lb of body weight.

For statistical analysis, a fixed effects regression model was used to estimate and compare slopes and offsets of the optic nerve sheath diameter versus intracranial pressure response between operators, pigs, and their interactions. Because of the small number of experimental animals studied and because optic nerve sheath diameter measurements were made by only 2 ultrasound operators, the data were treated as a pilot study modeling fixed animal and operator effects.

Figure 1. Diagram showing posterior orbit, optic nerve, and optic nerve sheath dimensions measured using various distances from the anterior and posterior sclera interface.
erator effects. In other words, findings comparing operators or pigs are intended to apply only to the specific animals and operators in the study, without making inferences to hypothetical populations of animals or potential astronaut operators in the way that a random-effects model could have done in a larger study.

Results

Saline aliquots of 1 to 10 mL were able to augment intracranial pressures from a baseline of approximately 10 to 15 to approximately 75 to 90 mm Hg, which eventually evoked a Cushing response due to an embarrassed cerebral perfusion pressure: cerebral perfusion pressure = arterial blood pressure – intracranial pressure. (A Cushing response is a central nervous system ischemic response that results from increased pressure in the cranial vault. When the cerebrospinal fluid [CSF] pressure rises to equal the arterial pressure, it compresses the arteries, causing global ischemia of the brain. This condition initiates a compensatory increase in arterial pressure and relative bradycardia.)

The rate of fluid injection was controlled to increase the intracranial pressure by 60 mm Hg over a 15- to 20-minute period. Further elevation of the intracranial pressure beyond this point required an increased volume of fluid, suggesting a failure of fluid containment within the brain parenchyma, either through direct dissection through the foramen magnum or into a venous conduit. In some cases, the injection pressure caused minor fluid leakage from the burr hole and associated mounting hardware. Unfortunately, major leakage of fluid in animal 5 prevented the needed buildup of intracranial pressure despite the large volume of fluid (>50 mL) injected over 10 minutes. Leakage took place through the burr hole and frontal sinus into the nasal passage. As a result, data from this animal were not used for subsequent statistical analysis.

Figure 2 is a typical image of a posterior porcine eye showing the lines measuring a perpendicular intersection from the leading edge to the leading edge of the optic nerve sheath, 3 to 5 mm distal from the anterior and posterior sclera interface. The optic nerve is easily shown and surrounded by a fluid-filled subarachnoid space extending to the nerve sheath. The subarachnoid space increases in size with increasing intracranial pressure. The 5.5-mm optic nerve sheath diameter on this animal coincided with an intracranial pressure of approximately 60 mm Hg.

Regression analysis was performed on 53 data points in 4 animals (6–8 intracranial pressure values per operator-animal combination) with optic nerve sheath diameter as the dependent variable, intracranial pressure as a continuous predictor, and fixed animal and operator effects. On average, we found that the optic nerve sheath diameter increased by $0.0034 \pm 0.0003 \text{mm/mm Hg}$ of intracranial pressure; however, the slope was somewhat animal dependent ($P = .05$) and ranged from 0.0025 to 0.0046 for specific animals. There was no discernible difference in slope between the 2 ultrasound operators, although measurements made by 1 operator were on average higher ($P = .014$) than the others by about 0.024 mm, a difference corresponding to about 8% of the diameter range (0.26–0.57 mm). For a given operator and animal, the root mean square error around the regression line was estimated at about 0.034 mm, corresponding to $R^2 = 0.78$.

Figure 3 shows the relationship between intracranial pressure and optic nerve sheath diameter in each animal by operator as measured 3 mm posterior to the posterior sclera interface. For all animals and both operators, Figure 3 shows a clear increasing linear response of the measured diameters with increasing pressure.

In an attempt to confirm that intracranial pressure can be increased by increasing central venous pressure only, we performed a single pilot experiment on a prone animal (porcine) in which we increased the central venous pressure from 5 to 16 mm Hg using a saline bolus over 10 minutes. The intracranial pressure and optic nerve sheath diameter were measured using the same techniques stated in the "Materials and Methods" section of this article. Figure 4 shows a clear increasing linear relationship between intracranial pressure and central venous pressure (0.9155 [slope], 14.555 [x-intercept], 0.9917 [r value]). This find-
ing has been well documented in the literature and was expected in this animal.21

Of interest was the lack of a response in the optic nerve sheath diameter to changes in central venous pressure (Figure 5). As a consequence, a change in diameter alone is likely a poor indicator of a change in intracranial pressure when central venous pressure is the underlying mechanism affecting intracranial pressure. Thus, the use of the optic nerve sheath diameter as a means to measure acute changes in intracranial pressure may be limited under those circumstances in which central venous pressure also fluctuates over a wide range, such as after dialysis in patients with end-stage renal disease. Increased central venous pressure most likely increases surrounding nerve sheath interstitial pressure, which would negate the distending affect of the increased intracranial pressure on the optic nerve sheath diameter. More animals would need to be studied to confirm these phenomena, but this individual case suggests that clinicians who may use optic nerve sheath diameter measurements to confirm changes in intracranial pressure may need to monitor central venous pressure also.

Discussion

Elevated intracranial pressure may be present in patients with head trauma, spontaneous intracranial hemorrhage, or any other space-occupying lesion and may present itself in a variety of signs and symptoms, including an altered level of consciousness.22,23 In most emergency settings, elevated intracranial pressure suggests a serious life-threatening condition requiring rapid intervention.22,23 Computed tomography is the primary modality for assessing traumatic intracranial injury and other space-occupying lesions, which raise the clinical suspicion for elevated intracranial pressure. Unfortunately, in many situations computed tomography is not readily available or competes for time with vital resuscitation efforts.

Figure 4. Intracranial pressure (ICP) plotted as a function of central venous pressure (CVP).

Figure 5. Optic nerve sheath diameter (ONSD) plotted as a function of intracranial pressure (ICP). The intracranial pressure was increased by fluid loading.
Diagnosis and treatment of elevated intracranial pressure are especially complicated in remote sites with no access to tertiary care facilities and in disaster and mass casualty scenarios, which require rapid triage of numerous patients. Sonography was performed as a primary triage procedure in 400 of 750 mass casualty patients with trauma admitted to a large hospital within the first 72 hours after the 1988 Armenian earthquake. A quick noninvasive intracranial pressure measurement may prove to be a useful triage method in the future for closed head injuries and blunt trauma.24

The adult cranial vault is essentially a rigid confined bony vessel composed of brain parenchyma, CSF, and blood and may also harbor a mass or other space-occupying lesion (SOL). Vcranial (constant) = Vbrain + VolCSF + Volblood + VolSOL. All of the components of the cranial vault are noncompressible; therefore, once the cranial vault is filled above its unstressed volume, the intracranial pressure rises. Substantial increases in intracranial pressure therefore impede the cerebral blood flow by reducing cerebral perfusion pressure. As an intracranial space-occupying lesion expands, minor compensation occurs as CSF and blood are displaced into the spinal canal and extracranial vasculature, respectively. After this small reserve is used up, further compensation is impossible, and intracranial pressure rises abruptly.25

The treatment of patients with traumatic brain injury with a Glasgow Coma Scale Score of 8 or less with the placement of an intracranial pressure-monitoring device may guide clinicians toward a number of medical and surgical therapeutic interventions. The direct measurement of intracranial pressure is accomplished by the placement of a pressure monitor by a neurosurgeon into the ventricles of the brain or directly into the parenchyma. Acute increases in intracranial pressure of greater than 20 mm Hg should be treated, but maintenance of cerebral perfusion pressure is probably more clinically important, especially in a hypotensive patient, because a small increase in intracranial pressure could embarrass cerebral perfusion in a brain-injured patient. Alternatively, elevation of mean arterial pressure may be protective for increased intracranial pressure. Intracranial pressure monitoring is now an integral component in the critical care of severe brain injury. Generally, all patients with Glasgow Coma Scale score of 8 or less, the high-risk subset of those with scores of 9 to 12, and those who cannot be followed with serial neurologic examinations (eg, anesthetized for other procedures) are considered for intracranial pressure monitoring. Accurate intracranial and arterial pressure measurement is necessary to determine the cerebral perfusion pressure. There may be circumstances in which direct intracranial pressure measurement is not immediately available but yet needs to be assessed and managed.

Numerous techniques have been devised to estimate relative and absolute changes in intracranial pressure. The eye has been investigated as a window for noninvasive intracranial pressure measurement. Under physiologic conditions, the pressure in the central retinal vein must be greater than the intracranial pressure to preserve patency because the CSF has contiguity with the optic nerve sheath as it drains into the cavernous sinus.26 Because the optic nerve is surrounded by a dural sheath filled with CSF, pressure in the retinal vein is influenced by intracranial pressure. Therefore, it is assumed that high intracranial pressure is associated with collapse of the retinal vein. On this basis, Motschmann et al25 used ophthalmodynamometry for assessment of intracranial pressure. This procedure is performed by exerting pressure on the sclera with a spring plunger while observing the vessels through an ophthalmoscope. The pressure is gradually increased until the central retinal vein just starts to pulsate. Evaluation in 31 patients showed a strong linear correlation with the invasively measured intracranial pressure value (r = 0.968). The ophthalmodynamometry technique, however, can be hazardous when performed in comatose patients and those with high myopia. Quantitative pupillometry also has been recently reported as a reliable and safe method identifying patients with intracranial pressure of greater than 20 mm Hg.29

Helmke and Hansen27,28 showed that a segment of the optic nerve sheath diameter approximately 3 mm behind the papilla showed maximal diameter fluctuations induced by gelatinous injections in postmortem preparations. This diameter landmark has been used in several clinical trials correlating elevated CSF pressure–derived intracranial pressure with optic nerve sheath diameter–derived intracranial pressure.5,8,17,29 Hansen and Helmke30 used B-mode sonography of the optic nerve sheath diameter during intrathecal infusion of Ringer’s lactate. They found a linear relationship between optic nerve sheath diameter and CSF pressure (R2 = 0.752). Optic nerve sheath dilatation depends on communication of the perineural CSF compartment around the optic nerve and the craniospinal subarachnoid space. There are numerous fibrous adhesions within the optic subarachnoid space, and studies by Hayreh31,32 indicated sufficient patency to allow the transfer of CSF. Furthermore, Hansen and Helmke30 reported an almost immediate effect of increased CSF pressure on the optic nerve sheath diameter, suggesting unrestricted patency.
Papilledema and the lack of venous pulsations in the central retinal vein have been used in the past as indications of elevated intracranial pressure, and the presence of these findings at the bedside only suggests that intracranial pressure is elevated, with no insight into cerebral perfusion pressure. Another limitation of these bedside findings is the latency of as much as several hours after the increase in intracranial pressure. The accuracy of funduscopic findings for papilledema and central retinal vein pulsations is quite limited for nonophthalmologists using stereoscopy and pupil dilation (sensitivity, 84.5%; specificity, 59.3%).

To obtain meaningful baseline data for this noninvasive intracranial pressure measurement, Newman et al measured the optic nerve sheath diameter by sonography during acute raised intracranial pressure in hydrocephalus. That study suggested that the upper limit of normal for optic nerve sheath diameter is 4.5 mm (measured at 3 mm behind the globe) in patients older than 1 year and 4.0 mm in children younger 1 year. The optic nerve sheath diameter correlated well in those patients after decompression procedures, as evidenced by the finding that patients with patent ventriculoperitoneal shunts had a mean optic nerve sheath diameter of 2.9 (SD, 0.5) mm compared to 5.6 (0.6) mm in those with raised intracranial pressure (P < .0001).

Measurements of the optic nerve sheath diameter using bedside sonography by Kimberly et al were also shown to correlate with clinical and radiologic signs and symptoms of increased intracranial pressure. They performed a prospective blinded observational study of adult participants in both the emergency department and the neurologic intensive care unit who had invasive intracranial monitors placed as part of their clinical care. Using receiver operating characteristic curve analysis, the authors confirmed the commonly used optic nerve sheath diameter threshold of greater than 5 mm for detecting intracranial pressure of greater than 20 cm H2O. That study directly correlated ventriculostomy intracranial pressure measurements with sonographic optic nerve sheath diameter measurements and provided further support for the use of optic nerve sheath diameter measurements as noninvasive tests for elevated intracranial pressure. These results are encouraging, yet a study by Le et al of 64 pediatric patients found that the optic nerve sheath diameter was a poor screening test for raised intracranial pressure in the emergency department because of very poor specificity. We have observed that the optic nerve sheath diameter measurement technique needs to taught extensively to all users, or poor sensitivity and specificity will result.

Sutherland et al investigated the association of optic nerve sheath diameter, as a correlate of intracranial pressure, with acute mountain sickness. They studied 13 mountaineers (10 men and 3 women aged 23–52 years) on a British expedition to climb Mount Everest. The optic nerve sheath diameter was measured by sonography, 3 mm behind the globe using B scans at sea level and 2000, 3700, 5200, and 6400 m, and acute mountain sickness scores using heart rate and oxygen saturation levels also were recorded. The authors concluded that the optic nerve sheath diameter increases at high altitudes, and this increase is associated with more severe symptoms of acute mountain sickness and possibly high-altitude cerebral edema. Fagenholz et al repeated this technique on 287 participants in Nepal (4240 m) and found a strong correlation between the acute mountain sickness score and optic nerve sheath diameter. Both of these studies concluded that given the linkage between optic nerve sheath diameter and intracranial pressure, these results strongly suggest that intracranial pressure plays an important role in the pathophysiologic mechanism of acute mountain sickness.

An interesting additional finding of the previous study was that the optic nerve sheath diameter does not change with patient positioning. The Trendelenburg position is often used in hypotensive patients, and the reverse Trendelenburg position (30° head up) is often used in patients with head injury to help decrease intracranial pressure. Romaguolo et al conducted a prospective case-control blinded study using healthy consenting adults. Three separate investigators measured the optic nerve sheath diameter in each eye of 10 separate volunteers in the supine, Trendelenburg, and reverse Trendelenburg positions with 30° angulation from horizontal. They concluded that the optic nerve sheath diameter measured by sonography in healthy individuals does not change appreciably with the Trendelenburg or reverse Trendelenburg position in comparison with the supine baseline.

In conclusion, the results of this study suggest that the use of the optic nerve sheath diameter to noninvasively confirm acute changes in intracranial pressure over 1 hour is feasible in a porcine model. We recommend that this method be validated in humans using a direct intracranial pressure measurement where possible to confirm it as a screening tool for acute and chronically increased diameters secondary to elevated pressure in clinical settings. This measurement may be especially important in emergency settings where the placement of a direct intracranial pressure monitoring device is not immediately possible, craniofacial trauma prevents it, or the patient’s clinical status is initially asymptomatic but eventually deteriorates. This
noninvasive technique may detect a gradual asymptomatic increase in intracranial pressure before serious life-threatening symptoms are manifested. We suggest that the results of this porcine study may be used to design a prospective study to correlate optic nerve sheath diameter changes for monitoring intracranial pressure changes over minutes to days in patients with clinically suspicious conditions.

References


