

## Ultrasonography Assessments of Optic Nerve Sheath Diameter as a Noninvasive and Dynamic Method of Detecting Changes in Intracranial Pressure

[Li-juan Wang](#), MD, PhD,<sup>1</sup> [Li-min Chen](#), MS,<sup>1</sup> [Ying Chen](#), MD,<sup>1</sup> [Li-yang Bao](#), MS,<sup>1</sup> [Nan-nan Zheng](#), MS,<sup>1</sup> [Yu-zhi Wang](#), MS,<sup>1</sup> and [Ying-qi Xing](#), MD, PhD<sup>1</sup>

[Author information](#) [Article notes](#) [Copyright and License information](#) [Disclaimer](#)

See commentary "[Correlation Between Noninvasive Ultrasonography and Dynamically Monitored Intracranial Pressure.](#)" in *JAMA Ophthalmol*,

doi: 10.1001/jamaophthalmol.2017.6558.

This article has been [cited by](#) other articles in PMC.

### Abstract

#### Introduction

---

The optic nerve is an outward form of the diencephalon during embryogenesis. It is wrapped by the nerve sheath, which is derived from 3 layers of meninges and protrudes toward the orbit. Thus, the cerebrospinal fluid moves freely between the intracranial and intraorbital subarachnoid spaces. The intraorbital subarachnoid space surrounds the optic nerve, and it is subject to the same pressure changes as the intracranial subarachnoid space.

The “criterion standard” method for monitoring intracranial pressure (ICP) includes using invasive, intraparenchymal, or intraventricular devices that are often only available in specialist neurocritical care units. These monitoring techniques can result in complications, such as bacterial colonization and hemorrhaging, and can be painful for the patient, especially if repeated evaluations are needed; thus, they are not ideal. Hence, noninvasive, repeatable, and simple methods of assessing ICP are urgently needed.

Recently, measuring the optic nerve sheath diameter (ONSD) via ultrasonography has become a popular approach for detecting elevated ICP. Previously, we confirmed that this noninvasive technique could be used to qualitatively and effectively identify elevations in ICP. However, whether ultrasonographic ONSD measurements can be used to dynamically monitor ICP changes and assess the efficacy of ICP management strategies remains unclear. Therefore, in this study, we evaluated the ONSD and its association to ICP in patients with elevated ICP both before and after treatment to determine whether ultrasonographic ONSD measurements could serve as a noninvasive surrogate marker of ICP changes.

#### Methods

---

##### Study Setting

This study was performed in the ultrasonography center in the Department of Neurology at the First Hospital of Jilin University, a general public hospital in China.

The protocol was designed from August 1, 2015, to October 31, 2015, and was approved by the ethics committee of the First Hospital of Jilin University on December 5, 2015 (approval number, 2015-208). The institutional review board of the First Hospital of Jilin University approved the enrollment of patients who were suspected of having elevated ICP and needed to undergo lumbar puncture (LP) to confirm the elevated ICP. The ultrasonographic ONSD was measured in each patient before LP. The ONSD measurements were compared with the LP findings. All methods adhered to the relevant guidelines and regulations. All participants provided written informed consent. They were informed that the ultrasonographic ONSD measurements obtained would be compared with their LP findings.

## Patients

We recruited patients who were suspected of having elevated ICP for various reasons between June 2016 and December 2016. The ONSD was measured in each patient before LP to confirm the elevated ICP. The ICP was considered to be elevated if the value was more than 200 mm H<sub>2</sub>O (1 mm H<sub>2</sub>O is approximately 0.074 mm Hg). According to their LP on admission, patients were divided into 2 groups: group 1 (200 < LP ≤ 300 mm H<sub>2</sub>O) and group 2 (LP > 300 mm H<sub>2</sub>O). Osmotherapy and etiological treatments were administered to patients with elevated ICP according to previously published recommendations. The patients underwent follow-up ONSD and LP measurements within 1 month. The exclusion criteria were as follows: age younger than 18 years, ophthalmic diseases (such as tumors or traumas), history of glaucoma, administration of medications that may have affected the ICP (such as diuretics, carbonic anhydrase inhibitors, and glucocorticoids), an ICP of 200 mm H<sub>2</sub>O or less, and/or did not undergo follow-up ONSD and/or LP measurements within 1 month. Additionally, the following patient data were recorded: age, sex, and body mass index (BMI) (calculated as weight in kilograms divided by height in meters squared).

## Measurements

The ONSD measurements were performed before the LP. The interval between these 2 examinations was fewer than 10 minutes. Ultrasonography examinations of the eye were performed in B-mode using the iU22 ultrasound system (Philips) and a 9-3 MHz linear array transducer (Philips). The acoustic output of the ultrasonography system was adjusted according to the “as low as reasonably achievable” principle and the requirements for orbital sonography to avoid damaging the retina and lens. Patients were examined in the supine position. The probe was placed lightly on the closed upper eyelid, which was covered with a thick layer of ultrasonography gel to prevent pressure from being exerted on the eye. The position of the probe was adjusted to clearly display the entry of the optic nerve into the globe. Two measurements were performed for each optic nerve; the first measurement was in the transversal plane with the probe in a horizontal position and the second measurement was in the sagittal plane with the probe in a vertical position, according to previous protocols. The

ONSD was assessed bilaterally at 3 mm posterior to the orbit. Each eye was examined twice, and the mean value of the 8 total measurements for both eyes was recorded. All participants were examined by 2 experienced observers who were blinded to the ultrasonography results. To minimize variability, the final ONSD measurement value for an individual was the average of all 16 values.

The opening cerebrospinal fluid pressure was obtained through an LP, which was performed by an experienced neurological resident blinded to the ultrasonography results. Patients were awake and placed in the left lateral position with their hips and knees flexed and their head as close to their knees as comfortably possible. The area around the lower back was prepared using the aseptic technique, and patients were asked to relax. Once the subarachnoid space (SAS) had been entered, the patients were asked to straighten their legs, after which the opening pressure was recorded and fluid samples were obtained. The ONSD and cerebrospinal fluid pressure measurements were obtained at admission and follow-up, and all values were entered into the database for analysis.

### Statistical Analysis

The required sample size was calculated to be 13 participants based on the assumptions that the correlation of ONSD and ICP was 0.758, with an  $\alpha$  error of .05 and a statistical power of 91%. For the sample size estimation, the PASS software (NCSS) was used. Statistical analyses were performed using a commercially available statistical software package (SPSS for Windows, version 18.0; SPSS Inc). The distribution of continuous variables was assessed using the Kolmogorov-Smirnov test. Normally distributed variables were summarized using the mean and the standard deviation. A Pearson correlation analysis was used to compare the measurements between 2 eyes and 2 observers. A Bland-Altman analysis was used to determine interobserver reliability. The difference in the ONSD from admission to follow-up was assessed with a paired  $t$  test. The differences in age, BMI at admission, and ONSD at follow-up between the 2 groups were assessed with an independent-samples  $t$  test. A  $\chi^2$  test was used to compare the sex distribution. The correlation between the ONSD and ICP was evaluated with a Pearson correlation analysis. The correlation between the changes in ONSD and ICP was evaluated with a Pearson correlation analysis in which changes ONSD and ICP were the respective changes in the ONSD and ICP from admission to follow-up. We further estimated the correlation between ONSD and ICP and changes in ONSD and ICP using a partial correlation analysis. Covariates (age, cause of elevated ICP, and comorbidities) were included in the partial correlation analysis. The 95% CIs and 2-tailed  $P$  values were calculated. A  $P$  value of  $<.05$  was considered to be statistically significant.

[Go to:](#)

## Results

---

Ultrasonography ONSD and LP measurements were performed in 60 patients (Han nationality; mean [SD] age, 36.20 [12.04] years; range, 20–62 years; 29 [48%] female) on admission, 37 (62%) of whom had elevated ICP (>200 mm H<sub>2</sub>O) (Table 1). The mean (SD) ONSD and ICP of the 60 patients were 4.18 (0.61) mm (range, 3.40–6.0 mm) and 244 (87.49) mm H<sub>2</sub>O (range, 100–400 mm H<sub>2</sub>O), respectively. For the 60 patients on admission, the ONSD and ICP values were strongly correlated, with an *r* of 0.798 (95% CI, 0.709–0.867; *P* < .001). Twelve patients were excluded from further analysis because of a lack of follow-up ONSD and/or LP measurements. We found no evidence of differences in sex, age, BMI, ONSD, or ICP between the patients lacking follow-up and the patients enrolled. Thus, 25 patients with elevated ICP were included in the final analysis (Han nationality; mean age [SD], 35.16 [12.45] years; range, 20–62 years; 12 [48%] female). The mean (SD) BMI of the patients was 23.61 (3.66) kg/m<sup>2</sup> (range, 17.6–33.2 kg/m<sup>2</sup>). No evidence of difference was found in sex, age, or BMI between group 1 and group 2 (Table 1). The causes of the elevated ICP included cerebral infections (*n* = 18, 72%), cerebrovascular disease (*n* = 5, 20%), and hydrocephalus (*n* = 2, 8%). One patient had comorbidities (pneumonia). Pearson correlation coefficient between the 2 eyes for observer 1 was 0.794 in the transversal plane and 0.800 in the sagittal plane, and for observer 2, it was 0.790 in the transversal plane and 0.782 in the sagittal plane. Pearson correlation coefficient between the 2 observers for the left eye was 0.969 in the transversal plane and 0.977 in the sagittal plane, and for the right eye, it was 0.968 in the transversal plane and 0.976 in the sagittal plane. A Bland-Altman analysis yielded a mean (SD) difference of measurements of 0.014 (0.125) mm for the right and –0.009 (0.118) mm for the left ONSD. Limits of agreement (mean ± 1.96 times of SD) were 0.259 and –0.231 mm for the right and 0.222 and –0.240 mm for the left ONSD. The ICP and ONSD values obtained at admission and follow-up are shown in Table 2. The elevated ICPs and dilated ONSDs had returned to normal in the follow-up evaluations. We found that the ONSD and ICP values obtained on admission were strongly correlated, with an *r* of 0.724 (95% CI, 0.470–0.876; *P* < .001). The mean (SD) changes in ICP and ONSD were 126.64 (52.1) mm H<sub>2</sub>O (range, 20–210 mm H<sub>2</sub>O; 95% CI, 109.24–146.07) and 1.00 (0.512) mm (range, 0.418–2.37 mm; 95% CI, 0.83–1.20), respectively. The change in ONSD was strongly correlated with the change in ICP according to Pearson correlation analysis, with an *r* of 0.702 (95% CI, 0.425–0.870; *P* < .001) (Figure 1). The greatest disconnects in changes in ONSD and ICP were in a patient with a change in ONSD of 0.58 mm and a change in ICP of 190 mm Hg. The partial correlation analysis showed that, after controlling for the covariates (age, cause of elevated ICP, and comorbidities), the ONSD and ICP values obtained on admission were correlated, with an *r* of 0.736 (95% CI, 0.486–0.886; *P* < .001). The changes in the ONSD and ICP values were correlated, with an *r* of 0.669 (95% CI, 0.343–0.868; *P* < .001). Pearson correlation coefficients between the change in ONSD and the change in ICP in just 1 eye and a separate one with just 1 observer ranged from 0.630 to 0.692 (Table 3). We found no evidence of a difference in mean (95% CI) ONSDs between group 1 and group 2 (3.49 mm [3.34–3.62 mm], 3.51 mm [3.44–3.59 mm], respectively; *P* = .778) at follow-up.

**Table 1.****Patient Characteristics<sup>a</sup>**

Variables	Total Patients (n = 60)	Group 1 (n = 13)	Group 2 (n = 12)
Age, mean (SD), y	36.20 (12.04)	35.46 (12.12)	34.83 (13.33)
Female, No. (%)	29 (48)	6 (46.2)	6 (50.0)
BMI, mean (SD), kg/m <sup>2</sup>	23.68 (3.43)	23.44 (3.65)	23.81 (3.82)

Abbreviation: BMI, body mass index (calculated as weight in kilograms divided by height in meters squared).

<sup>a</sup>The differences in age and BMI between the 2 groups were assessed with an independent-samples *t* test. A  $\chi^2$  test was used to compare the sex distribution. Patients with elevated intracranial pressure were divided into group 1 (200 < lumbar puncture  $\leq$  300 mm H<sub>2</sub>O) and group 2 (lumbar puncture > 300 mm H<sub>2</sub>O).

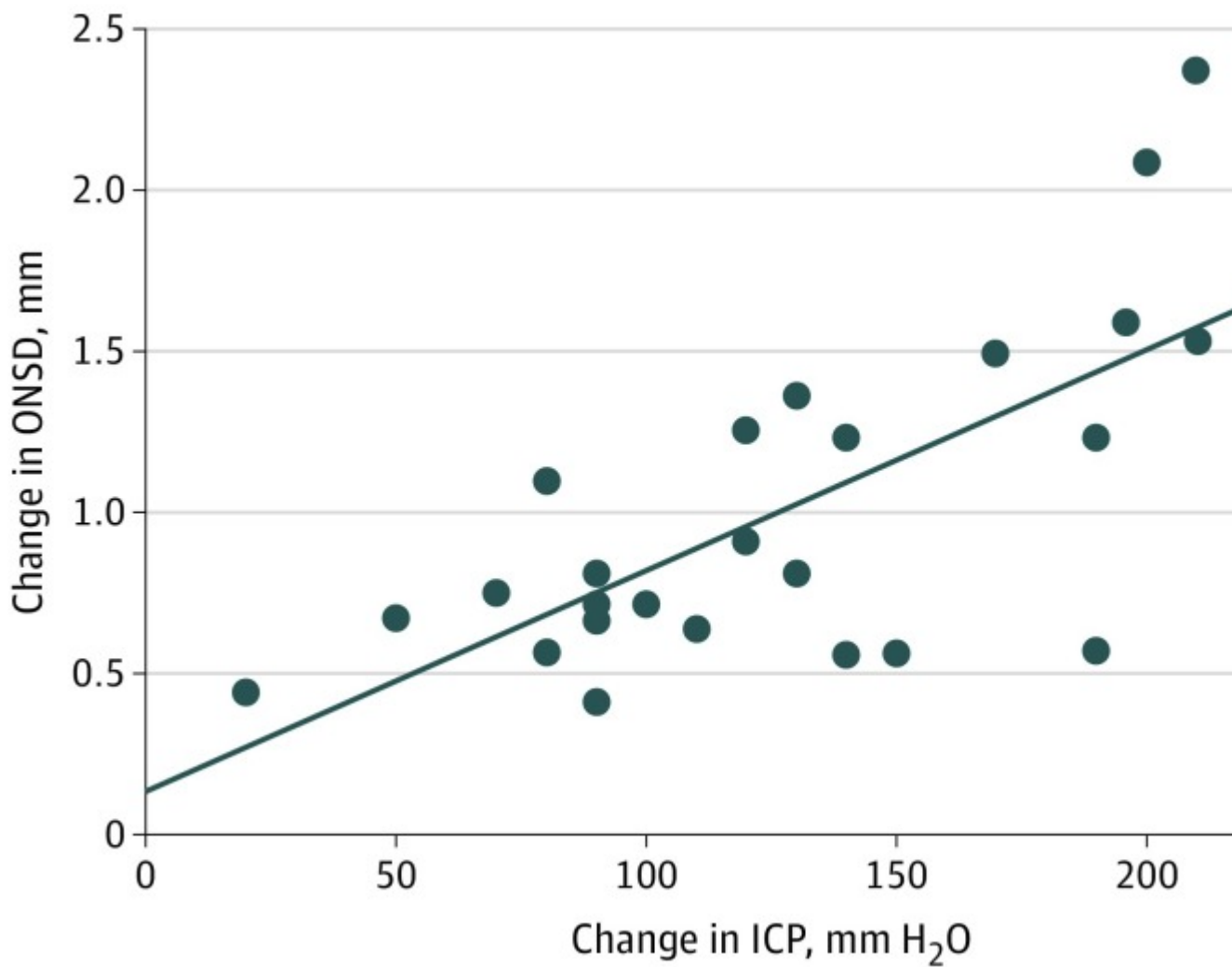
**Table 2.****ICP and ONSD Values Obtained at Admission and Follow-up**

Values	Admission	Follow-up	Difference (95% CI)
Range of ONSD, mm	4.17–6.0	3.09–3.95	NA
ONSD, mean (SD), mm <sup>a</sup>	4.50 (0.54)	3.50 (0.21)	1.00 (0.79–1.21)
Range of ICP, mm H <sub>2</sub> O	220–400	140–200	NA

Values	Admission	Follow-up	Difference (95% CI)
ICP, mean (SD), mm H <sub>2</sub> O <sup>a</sup>	302.40 (54.26)	175.76 (15.85)	126.64 (104.97–148.32)

Abbreviations: ICP, intracranial pressure; NA, not applicable; ONSD, optic nerve sheath diameter.

<sup>a</sup>The sample size is n = 25.



**Figure 1.**  
**Correlation Between Change in Intracranial Pressure (ICP) and Change in Optic Nerve Sheath Diameter (ONSD)**

From admission to after reducing the ICP (follow-up).

Table 3.

## Correlations Between the Changes in ONSD and ICP From Admission to Follow-up<sup>a</sup>

Values	Observer 1		Observer 2	
	Left Eye	Right Eye	Left Eye	Right Eye
<i>r</i>	0.672	0.646	0.692	0.630
95% CI	0.415–0.856	0.340–0.849	0.486–0.845	0.278–0.839
<i>P</i> value	<.001	<.001	<.001	.001

Abbreviations: ICP, intracranial pressure; ONSD, optic nerve sheath diameter.

<sup>a</sup>The sample size is n = 25.

[Go to:](#)


## Discussion

In this study, we measured the ONSD and ICP in patients with elevated ICP and found that the ICP and ONSD values obtained on admission were strongly correlated with an *r* of 0.724 (95% CI, 0.470- 0.876; *P* < .001). Additionally, ultrasonographic ONSD measurements were successfully used to dynamically assess the changes in ICP that occurred from admission to follow-up.

Our previous cross-sectional study found that ultrasonographic ONSD was correlated with ICP on admission and suggested that this noninvasive method could be potentially useful for identifying patients with elevated ICP. The results of this study were consistent with those of the previous one with different samples. Furthermore, this study focused on the follow-up of ONSD and ICP. Therefore, this study both confirmed the ONSD as a predictor of elevated ICP on admission and found that the change in ONSD was strongly correlated with the change in ICP according to Pearson correlation analysis, with an *r* of 0.702 (95% CI, 0.425-0.870; *P* < .001). Thus, it was suggested that ONSD could dynamically detect the changes in ICP in this study.

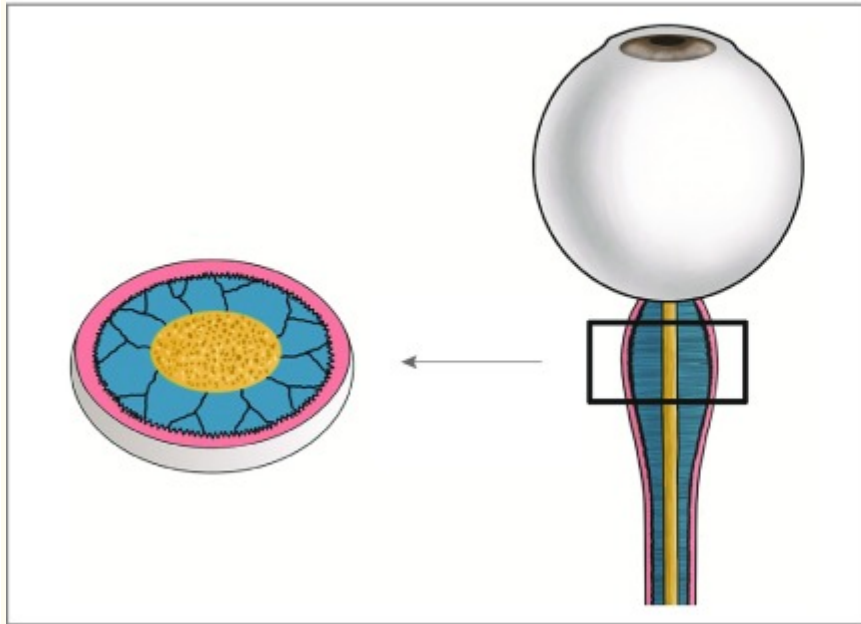
While many clinical studies have reported this dilation response, it was unclear whether the ONSD would decrease after the ICP decreased in vivo. Hansen et al demonstrated such ONSD changes in vitro through the controlled application of

incremental and decreased pressure steps in the SAS. Specifically, they isolated human optic nerve preparations that were obtained from autopsies and found that following submission to pressure, the ONSD increased. Similarly, reducing the pressure in the SAS invariably resulted in a decrease in the ONSD. However, to our knowledge, few clinical studies have estimated the ONSD variations that occur following treatment for elevated ICP. In one study of 13 patients with acute brain injury, Launey et al found a significant correlation between the ICP and ONSD measurements obtained before and after mannitol infusion. Likewise, our clinical study showed that the dilated ONSD decreased following treatment to reduce the elevated ICP. We speculate that the pressure-dependent behavior of the optic nerve sheath is associated with its exceptional elastic properties. Killer et al reported that the SAS of the human optic nerve contains various trabeculae, septa, and stout pillars that are arranged between the arachnoid and pia layers of the nerve meninges. Therefore, when the ICP is elevated, the presumably folded trabeculae likely stretch, allowing the optic nerve sheath to dilate. Similarly, on reduction of the ICP, the stretched trabeculae refold and the dilated optic nerve sheath shrinks. Our findings support that ONSD examinations can be used to dynamically assess variations in the ICP because of the elasticity of the optic nerve sheath ([Figure 2](#)).





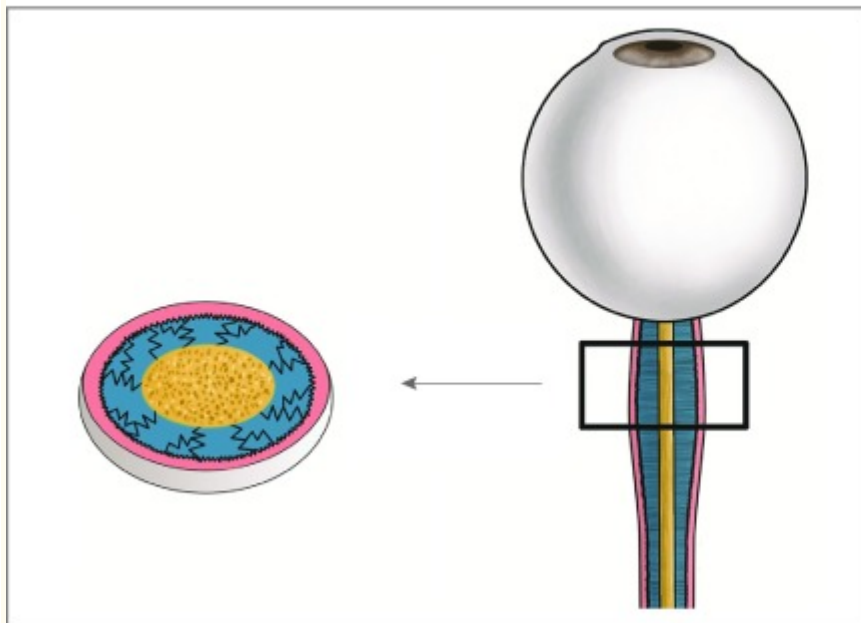
**A** Elevated ICP



**B** ONSD pretreatment



**C** Reduced ICP



**G** ONSD posttreatment



Figure 2.

**Illustrations and Ultrasonography Images of the Changes in Optic Nerve Sheath Diameter (ONSD)**

A, The folded trabeculae are stretched and the optic nerve sheath is dilated when the intracranial pressure (ICP) is elevated. B, The ONSD, as measured with transorbital ultrasonography, was 0.600 cm in this patient (a man in his early 40s) with elevated ICP on

admission. C, The stretched trabeculae are refolded and the dilated optic nerve sheath shrinks when the ICP is reduced. D, The ONSD decreased to 0.374 cm after treating the elevated ICP.

Other studies have also shown that the optic nerve sheath is quite elastic. For instance, it was recently reported that the ONSD significantly increases with hypercapnia and subsequently reverts to baseline values once normocapnia is established. Moreover, Maissan et al reported that during tracheal stimulation, both the ICP and ONSD increased simultaneously, while after the procedure, the ONSD returned to the baseline diameter at the same rate as the ICP. It has been suggested that the changes in ONSD may reflect a temporary and reversible increase in ICP because of acute elevations in the intraabdominal pressure. However, the effective range of the optic nerve sheath's elastic properties remains unclear. In vitro experiments of the ONSD showed that the diameter only recovered to baseline when the SAS pressure was less than 35 mm Hg, while residual dilatation remained on decompression from higher pressure levels ( $\geq 45$  mm Hg). In the present study, after treating the elevated ICP, the enlarged ONSD decreased to 3.09 to 3.95 mm, which was within the normal range for healthy Chinese adults. In addition, we found no evidence of difference in mean (95% CI) ONSDs between group 1 and group 2 (3.49 mm [3.34-3.62 mm], 3.51 mm [3.44-3.59 mm], respectively;  $P = .778$ ) at follow-up, indicating that the ONSD was able to return to normal after treatment regardless of the initial ICP value on admission (maximum ICP value, 400 mm H<sub>2</sub>O, approximately 29.4 mm Hg). Such findings imply that when the ONSD is within the range of reversible dilation, its structure may not undergo plastic deformation or trabecular damage-like overdistention. Hence, when in this effective range, the dilated ONSD can completely recover to normal, as was observed in our study.

Pearson correlation coefficients between the change in ONSD and change in ICP in 1 eye and those assessed by just 1 observer ranged from 0.630 to 0.692, which is similar to the overall  $r$  value (0.702). The results showed relatively good sensitivity. This study showed that ultrasonographic ONSD is a reproducible technique with high interobserver reliability. A previous study also confirmed that this technique was easy to learn and with high intra- and interobserver reliability. Therefore, it may be extensively applicable in clinical settings. Clinicians, such as intensivists or neurosurgeons, could easily master this technique and have confidence in the reliability of the measurements.

### Limitations

Although this study showed that this noninvasive technique may be a useful, simple tool for dynamically evaluating ICP, it was limited by its modest sample size. Thus, studies with larger samples should be conducted in the future to validate our findings for the implementation of the technique to the general population. Another possible limitation is that the study took place in a single center. Hence, there remains a possibility of incorrect inference if we only use this noninvasive technique instead of LP in some cases. In addition, the maximum ICP value in our study was 400 mm H<sub>2</sub>O; thus, the accuracy of the technique in evaluating cases with even higher ICP is not

clear. Additional clinical studies with patients who have an ICP of more than 400 mm H<sub>2</sub>O are needed. Moreover, with regard to promoting the clinical applicability of the technique, the change in ONSD cutoff point for identifying the change in ICP or a mathematical function to noninvasively and quantitatively estimate the change in ICP with the ultrasonographic measurement of change in ONSD should be investigated. Further, optic nerve swelling, papilla prominence, and optical coherence tomography could also be used to evaluate elevated ICP. Recently, Swanson et al reported that spectral domain optical coherence tomography measurements were correlated with ICP in children. However, there were some factors that limited the generalizability of that study. In addition, this study did not focus on the axial length and refraction of the 2 groups. Thus, we plan to investigate these issues in a future study; we will also look into the correlation of optic nerve swelling with changes in ICP. The *r* values that resulted from comparing the pre-LP ICD and ONSD values (0.724), and from comparing the changes in ICP and ONSD values (0.702) showed that there may be other factors that could be influencing these 2 measures. These influencing factors should be investigated in a future study. Finally, whether treatment could have an independent effect on ONSD is not clear; future studies should investigate any related mechanisms.

[Go to:](#)

## Conclusions

---

We showed that the dilated ONSDs that were observed in patients with increased ICP decreased along with the ICP reduction. Although further studies are required, this study suggests that ultrasonographic ONSD measurements may assist in monitoring patients with elevated ICP, as fluctuations in the ONSD approximate the respective ICP variations. This finding may be important because existing invasive ICP monitoring techniques can be painful and result in complications, such as bacterial colonization and hemorrhaging.

[Go to:](#)

## References

---

1. Hansen HC, Helmke K. The subarachnoid space surrounding the optic nerves: an ultrasound study of the optic nerve sheath. *Surg Radiol Anat.* 1996;18(4):323-328. [\[PubMed\]](#) [\[Google Scholar\]](#)
2. Hayreh SS. Pathogenesis of oedema of the optic disc. *Doc Ophthalmol.* 1968;24(2):289-411. [\[PubMed\]](#) [\[Google Scholar\]](#)
3. Citerio G, Bakker J, Bassetti M, et al. Year in review in Intensive Care Medicine 2014: I: cardiac dysfunction and cardiac arrest, ultrasound, neurocritical care, ICU-acquired weakness, nutrition, acute kidney injury, and miscellaneous. *Intensive Care Med.* 2015;41:179-191. doi:10.1007/s00134-015-3665-9 [\[CrossRef\]](#) [\[Google Scholar\]](#)

4. Amini A, Kariman H, Arhami Dolatabadi A, et al. Use of the sonographic diameter of optic nerve sheath to estimate intracranial pressure. *Am J Emerg Med*. 2013;31(1):236-239. [[PubMed](#)] [[Google Scholar](#)]
5. Dubourg J, Messerer M, Karakitsos D, et al. Individual patient data systematic review and meta-analysis of optic nerve sheath diameter ultrasonography for detecting raised intracranial pressure: protocol of the ONSD research group. *Syst Rev*. 2013;2:62. [[PMC free article](#)] [[PubMed](#)] [[Google Scholar](#)]
6. Geeraerts T, Launey Y, Martin L, et al. Ultrasonography of the optic nerve sheath may be useful for detecting raised intracranial pressure after severe brain injury. *Intensive Care Med*. 2007;33(10):1704-1711. [[PubMed](#)] [[Google Scholar](#)]
7. Moretti R, Pizzi B, Cassini F, Vivaldi N. Reliability of optic nerve ultrasound for the evaluation of patients with spontaneous intracranial hemorrhage. *Neurocrit Care*. 2009;11(3):406-410. [[PubMed](#)] [[Google Scholar](#)]
8. Major R, Girling S, Boyle A. Ultrasound measurement of optic nerve sheath diameter in patients with a clinical suspicion of raised intracranial pressure. *Emerg Med J*. 2011;28(8):679-681. [[PubMed](#)] [[Google Scholar](#)]
9. Soldatos T, Chatzimichail K, Papathanasiou M, Gouliamos A. Optic nerve sonography: a new window for the non-invasive evaluation of intracranial pressure in brain injury. *Emerg Med J*. 2009;26(9):630-634. [[PubMed](#)] [[Google Scholar](#)]
10. Hansen HC, Helmke K. Validation of the optic nerve sheath response to changing cerebrospinal fluid pressure: ultrasound findings during intrathecal infusion tests. *J Neurosurg*. 1997;87(1):34-40. [[PubMed](#)] [[Google Scholar](#)]
11. Launey Y, Nessler N, Le Maguet P, Mallédant Y, Seguin P. Effect of osmotherapy on optic nerve sheath diameter in patients with increased intracranial pressure. *J Neurotrauma*. 2014;31(10):984-988. [[PubMed](#)] [[Google Scholar](#)]
12. Wang L, Feng L, Yao Y, et al. Optimal optic nerve sheath diameter threshold for the identification of elevated opening pressure on lumbar puncture in a Chinese population. *PLoS One*. 2015;10(2):e0117939. [[PMC free article](#)] [[PubMed](#)] [[Google Scholar](#)]
13. Kimberly HH, Shah S, Marill K, Noble V. Correlation of optic nerve sheath diameter with direct measurement of intracranial pressure. *Acad Emerg Med*. 2008;15(2):201-204. [[PubMed](#)] [[Google Scholar](#)]
14. Grape S, Ravussin P. PRO: osmotherapy for the treatment of acute intracranial hypertension. *J Neurosurg Anesthesiol*. 2012;24(4):402-406. [[PubMed](#)] [[Google Scholar](#)]
15. Ertl M, Barinka F, Torka E, et al. Ocular color-coded sonography—a promising tool for neurologists and intensive care physicians. *Ultraschall Med*. 2014;35(5):422-431. [[PubMed](#)] [[Google Scholar](#)]
16. Tayal VS, Neulander M, Norton HJ, Foster T, Saunders T, Blaivas M. Emergency department sonographic measurement of optic nerve sheath diameter to detect findings of increased intracranial pressure in adult head injury patients. *Ann Emerg Med*. 2007;49(4):508-514. [[PubMed](#)] [[Google Scholar](#)]

17. Romagnuolo L, Tayal V, Tomaszewski C, Saunders T, Norton HJ. Optic nerve sheath diameter does not change with patient position. *Am J Emerg Med*. 2005;23(5):686-688. [[PubMed](#)] [[Google Scholar](#)]
18. Wang LJ, Yao Y, Feng LS, et al. Noninvasive and quantitative intracranial pressure estimation using ultrasonographic measurement of optic nerve sheath diameter. *Sci Rep*. 2017;7(7):42063. [[PMC free article](#)] [[PubMed](#)] [[Google Scholar](#)]
19. Hansen HC, Lagrèze W, Krueger O, Helmke K. Dependence of the optic nerve sheath diameter on acutely applied subarachnoidal pressure—an experimental ultrasound study. *Acta Ophthalmol*. 2011;89(6):e528-e532. [[PubMed](#)] [[Google Scholar](#)]
20. Killer HE, Laeng HR, Flammer J, Groscurth P. Architecture of arachnoid trabeculae, pillars, and septa in the subarachnoid space of the human optic nerve: anatomy and clinical considerations. *Br J Ophthalmol*. 2003;87(6):777-781. [[PMC free article](#)] [[PubMed](#)] [[Google Scholar](#)]
21. Dinsmore M, Han JS, Fisher JA, Chan VW, Venkatraghavan L. Effects of acute controlled changes in end-tidal carbon dioxide on the diameter of the optic nerve sheath: a transorbital ultrasonographic study in healthy volunteers. *Anaesthesia*. 2017;72(5):618-623. [[PubMed](#)] [[Google Scholar](#)]
22. Maissan IM, Dirven PJ, Haitsma IK, Hoeks SE, Gommers D, Stolker RJ. Ultrasonographic measured optic nerve sheath diameter as an accurate and quick monitor for changes in intracranial pressure. *J Neurosurg*. 2015;123(3):743-747. [[PubMed](#)] [[Google Scholar](#)]
23. Dip F, Nguyen D, Rosales A, et al. Impact of controlled intraabdominal pressure on the optic nerve sheath diameter during laparoscopic procedures. *Surg Endosc*. 2016;30(1):44-49. [[PubMed](#)] [[Google Scholar](#)]
24. Wang L, Feng L, Yao Y, et al. Ultrasonographic evaluation of optic nerve sheath diameter among healthy Chinese adults. *Ultrasound Med Biol*. 2016;42(3):683-688. [[PubMed](#)] [[Google Scholar](#)]
25. Potgieter DW, Kippin A, Ngu F, McKean C. Can accurate ultrasonographic measurement of the optic nerve sheath diameter (a non-invasive measure of intracranial pressure) be taught to novice operators in a single training session? *Anaesth Intensive Care*. 2011;39(1):95-100. [[PubMed](#)] [[Google Scholar](#)]
26. Ballantyne SA, O'Neill G, Hamilton R, Hollman AS. Observer variation in the sonographic measurement of optic nerve sheath diameter in normal adults. *Eur J Ultrasound*. 2002;15(3):145-149. [[PubMed](#)] [[Google Scholar](#)]
27. Bäuerle J, Lochner P, Kaps M, Nedelmann M. Intra- and interobserver reliability of sonographic assessment of the optic nerve sheath diameter in healthy adults. *J Neuroimaging*. 2012;22(1):42-45. [[PubMed](#)] [[Google Scholar](#)]
28. Swanson JW, Aleman TS, Xu W, et al. Evaluation of optical coherence tomography to detect elevated intracranial pressure in children. *JAMA Ophthalmol*. 2017;135(4):320-328. [[PMC free article](#)] [[PubMed](#)] [[Google Scholar](#)]
29. Brodsky MC, Chen JJ, Wetjen NM. Optical coherence tomography for the noninvasive detection of elevated intracranial pressure a new role for the

ophthalmologist? JAMA Ophthalmol. 2017;135(4):329-330. [[PubMed](#)] [[Google Scholar](#)]