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# *The Effect of Prone Positioning on Intraocular Pressure in Anesthetized Patients*

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***Background:*** **Ocular perfusion pressure is commonly defined as mean arterial pressure minus intraocular pressure (IOP). Changes in mean arterial pressure or IOP can affect ocular perfusion pressure. IOP has not been studied in this context in the prone anesthetized patient.**

***Methods:*** **After institutional human studies committee approval and informed consent, 20 patients (American Society of Anesthesiologists physical status I–III) without eye disease who were scheduled for spine surgery in the prone position were enrolled. IOP was measured with a Tono-pen® XL handheld tonometer at five time points: awake supine (baseline), anesthetized (supine 1), anesthetized prone (prone 1), anesthetized prone at conclusion of case (prone 2), and anesthetized supine before wake-up (supine 2). Anesthetic protocol was standardized. The head was positioned with a pinned head-holder. Data were analyzed with repeated-measures analysis of variance and paired *t* test.**

***Results:*** **Supine 1 IOP (13 ± 1 mmHg) decreased from baseline (19 ± 1 mmHg) ($P < 0.05$). Prone 1 IOP (27 ± 2 mmHg) increased in comparison with baseline ($P < 0.05$) and supine 1 ($P < 0.05$). Prone 2 IOP (40 ± 2 mmHg) was measured after 320 ± 107 min in the prone position and was significantly increased in comparison with all previous measurements ($P < 0.05$). Supine 2 IOP (31 ± 2 mmHg) decreased in comparison with prone 2 IOP ($P < 0.05$) but was relatively elevated in comparison with supine 1 and baseline ($P < 0.05$). Hemodynamic and ventilatory parameters remained unchanged during the prone period.**

***Conclusions:*** **Prone positioning increases IOP during anesthesia. Ocular perfusion pressure could therefore decrease, despite maintenance of normotension.**

IN a recent survey of 801 anesthesiologists by the Anesthesia Patient Safety Foundation, blindness due to anesthetic technique was ranked 11th highest among a total of 53 patient-safety concerns.[1] Numerous reports of visual loss after spine surgery in the prone position exist in the literature.[2–6] Most of these episodes do not appear to be related to direct pressure to the eye but rather to a change in the hemodynamics affecting optic nerve perfusion. Ocular perfusion pressure is commonly defined as the difference between mean arterial pressure (MAP) and intraocular pressure (IOP).[7] This simple equation has led some authors to advocate maintenance of intraoperative MAP in the normal to high–normal range during these procedures. However, ocular perfusion pressure is also indirectly related to IOP, which has not been studied in this context.

Because an increase in IOP can lower ocular perfusion pressure despite the maintenance of normal MAP, it is important to understand what happens to IOP in the prone anesthetized patient. In a study of awake volunteers, IOP increased from 13.5 ± 2.01 mmHg in the sitting position to 20.0 ± 3.27 mmHg in the prone position,[8] suggesting that prone positioning intraoperatively may also increase IOP. However, the use of general anesthesia has been shown to decrease IOP in the supine position.[9] The balance between the opposing effects of general anesthesia and prone positioning likely plays an important role in the net ocular perfusion pressure. The purpose of the current study was to examine the combined effects of general anesthesia and the prone position on IOP in patients undergoing spine surgery.

This article is featured in "This Month in Anesthesiology." Please see this issue of ANESTHESIOLOGY, page 5A.

## Materials and Methods

After approval by the human studies committee, informed consent was obtained from 20 patients (American Society of Anesthesiologists physical status I–III), aged 18–80 yr, scheduled for spine surgery in the prone position. Patients with preexisting eye disease or previous eye surgery, allergy to tetracaine, or allergy to latex were not enrolled in the study. Before commencement of the study, intravenous catheters and standard anesthetic monitors (*i.e.*, blood pressure cuff, electrocardiograph, and pulse oximeter) were placed for all patients.

Both eyes were topically anesthetized with 0.5% tetracaine hydrochloride drops, and baseline IOP was measured in the supine position before premedication (baseline) with a Tono-pen® XL handheld tonometer (Mentor, Norwell, MA). The tonometer operates on the principle of the Imbert–Fick law: P = F/A, where P = intraocular pressure, F = the amount of force exerted by the tonometer to flatten a specific area of the eye, and A = the area flattened.

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**Table 1. Demographic Data**

| Patient No. | Procedure | Age (yr) | Weight (kg) | Height (cm) | Gender |
|---|---|---|---|---|---|
| 1 | Posterior lumbar decompression transthoracic corpectomy | 59 | 70 | 163 | M |
| 2 | Posterior lumbar interbody fusion L3–L5 | 54 | 52 | 152 | F |
| 3 | Posterior cervical stabilization C1–C2 | 64 | 65 | 160 | F |
| 4 | Posterior laminectomy decompression C2–C7 | 54 | 91 | 183 | M |
| 5 | Posterior decompression and fusion C2–C3 | 65 | 89 | 178 | M |
| 6 | Posterior lumbar interbody fusion L4–S1 | 43 | 101 | 188 | M |
| 7 | Posterior lumbar interbody fusion L3–L4 | 67 | 78 | 173 | M |
| 8 | Posterior lumbar interbody fusion L4–S1 | 53 | 79 | 175 | M |
| 9 | Posterior lumbar interbody fusion L5–S1 | 36 | 63 | 168 | F |
| 10 | Posterior cervical laminectomy C3–C6 | 57 | 113 | 173 | M |
| 11 | Transarticular screw fixation C1–C2 | 40 | 60 | 157 | F |
| 12 | Lumbar decompression | 59 | 74 | 170 | F |
| 13 | Posterior lumbar interbody fusion L5–S1 | 29 | 79 | 160 | F |
| 14 | Posterior lumbar interbody fusion L4–L5 | 56 | 52 | NA | M |
| 15 | Excision C1–C2 schwannoma | 52 | 81 | 188 | M |
| 16 | Posterior lumbar interbody fusion | 40 | 84 | 193 | M |
| 17 | Posterior lumbar interbody fusion | 62 | 72 | 155 | F |
| 18 | Posterior lumbar interbody fusion | 64 | 134 | 180 | M |
| 19 | Posterior lumbar interbody fusion | 67 | 59 | 175 | M |
| 20 | Posterior laminectomy C3–C6 | 62 | 96 | 170 | F |

NA = not available.

The Tono-pen® XL contains a strain gauge that converts IOP measurements to an electrical signal. The tonometer averages four successful readings and displays the mean and SD. Measurements were retaken if the range was greater than 5%. At the time of each tonometer reading, the following data set was collected: MAP, heart rate, end-tidal carbon dioxide, end-tidal isoflurane, percent inspired oxygen, and peak inspiratory pressure.

Anesthesia protocol was standardized for all study patients. After the baseline IOP measurement, the patients were given midazolam (0.7 mg/kg). Anesthesia induction consisted of administration of pentothal (3–5 mg/kg) and rocuronium (1 mg/kg) to facilitate endotracheal intubation. After endotracheal intubation, anesthesia was maintained with isoflurane ($< 0.5\%$), 50% nitrous oxide in oxygen, fentanyl infusion (1–2 $\mu g \cdot kg^{-1} \cdot h^{-1}$), and rocuronium as needed. MAP was kept within 20% of awake value, and ventilation was adjusted to keep end-tidal carbon dioxide in the range of 30–35 mmHg throughout the intraoperative period. Ten minutes after intubation, the IOP was measured with the patient anesthetized and supine (supine 1).

All patients were turned prone and their heads positioned in the neutral position with a pinned head-holder to prevent any extraocular pressure. Neck flexion and extension were limited to less than 15 degrees from the horizontal. IOP was measured at the following times and positions: before incision in the prone position (prone 1), at conclusion of surgery in the prone position (prone 2), and supine before pharmacological reversal of muscle relaxants and emergence from anesthesia (supine 2). Surgery proceeded as usual. All fluids and blood products administered were recorded, and estimated blood loss and urine output were measured. The length of time in the prone position was noted. Hematocrit was recorded for each patient preoperatively and postoperatively. In the recovery room, patients were asked about any vision changes or eye discomfort.

*Statistical Analysis*

The data were analyzed with repeated-measures analysis of variance, followed by paired *t* tests for comparisons across successive time points. Spearman rank coefficients were used to determine correlations. Data are reported as mean ± SD. Data were analyzed with use of the Statistical Analysis System (SAS Institute, Cary, NC).

## Results

Informed consent was obtained from 20 patients (American Society of Anesthesiologists physical status I–III; 12 males and 8 females) without a history of eye disease or previous eye surgery. Demographic data are shown in table 1. The mean patient weight was 80 ± 20 kg, and the mean height was 173 ± 13 cm. No statistical difference was found between the measurements from the right and left eyes, so measurements from the right eye were used for statistical analysis. The hypothesis of constant IOP was rejected on the basis of repeated-measures analysis of variance ($P < 0.0001$). Supine 1 IOP (13 ± 1 mmHg) was significantly decreased from baseline IOP (19 ± 1 mmHg; $P < 0.05$).

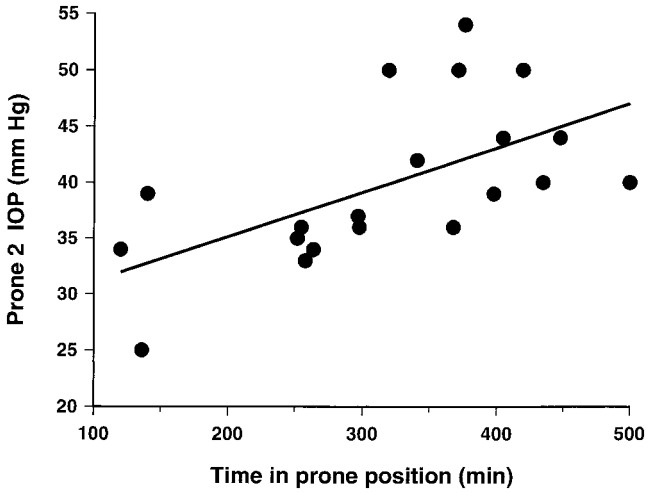


**Fig. 1. Intraocular pressure (IOP) at conclusion of prone positioning (prone 2) is correlated with total time spent in the prone position, in minutes ($P < 0.05$; $r^2 = 0.6$; n = 20). Prone 2 IOP ranged from 25 to 54 mmHg, and total time in the prone position ranged from 120 to 500 min.**

Prone 1 IOP (27 ± 2 mmHg) was significantly higher than both supine 1 IOP ($P < 0.05$) and baseline IOP ($P < 0.05$). Prone 2 IOP (40 ± 2 mmHg) was measured after 320 ± 107 min in the prone position and was significantly higher than all previous measurements ($P < 0.05$ for all). Supine 2 IOP (31 ± 2 mmHg) remained significantly elevated in comparison with baseline and supine 1 IOP ($P < 0.05$ for both). Longer time in the prone position was associated with higher prone 2 IOP ($P < 0.05$; $r^2 = 0.6$; fig. 1). During the period between prone 1 and prone 2 measurements, MAP, heart rate, end-tidal isoflurane, end-tidal carbon dioxide, and peak inspiratory pressure values did not significantly change ($P > 0.05$; table 2). Mean fluid balance, defined as [crystalloid (ml) + colloid (ml) + blood products (ml)] − [urine output (ml) + estimated blood loss (ml)], was positive (3,292 ± 1,120 ml).

## Discussion

The current study is the first to measure IOP in prone anesthetized patients. Confirming previous data,[10] our study demonstrated a decrease in IOP with the addition of general anesthesia in the supine position. Upon prone positioning, the IOP significantly increased in the anesthetized patients. There was a further significant increase after 320 ± 107 min of surgery. Similar results were reported by Lam *et al.*[8] in awake volunteers. They found that IOP after 8 min in the prone position was increased (20.0 ± 3.27 mmHg) when compared to the supine position (14.1±1.92 mmHg; $P < 0.05$). Interestingly, compared to the Lam study, our measurements of IOP in prone position were higher despite the presence of general anesthesia. This difference is likely due to the fact that our measurements were made after a longer average time in prone position.

The most accurate method of measuring IOP over a period of time is invasive and continuous. This type of recording was reported in a series of two patients who had their IOP continuously measured for up to 96 h *via* a probe implanted in the anterior chamber of the eye.[11] Because of the associated risks involved, this method of IOP measurement is impractical to propose for patients undergoing spine surgery. We opted instead to use the hand-held Tono-pen® XL tonometer, which can be used in any position and requires contact with the eye. This is the same equipment used by ophthalmologists to screen patients for glaucoma. Setogawa validated the methodology in rabbits by comparing IOP measurements made by an intraocular needle transducer *versus* the Tono-pen® XL and found a good correlation.[12] A larger, more expensive option is the hand-held noncontact tonometer, which uses an air pulse to measure IOP. A proposed noninvasive method of measuring IOP continuously is use of a contact lens with a pressure transducer, which is currently in development.[13]

Our study design had a flaw in that the prone 2 measurement was made at a uniform clinically relevant event (*i.e.*, end of surgery) rather than at a uniform time point (*e.g.*, 2 h in prone position). Because of the concerns with possible corneal injury, the number of IOP measurements in the prone position were limited to two in this pilot study. We chose the end of surgery to study the effect of length of surgery. We found a direct correlation between the amount of time spent in the prone position

**Table 2. Hemodynamic and Ventilatory Parameters**

| Time | MAP (mmHg) | HR (beats/min) | $ET_{CO_2}$ (mmHg) | $ET_{iso}$ (%) | PIP (mmHg) |
|---|---|---|---|---|---|
| Baseline | 98 ± 18 | 76 ± 16 | NA† | NA† | NA† |
| Supine 1 | 72 ± 7* | 76 ± 17 | 31 ± 2 | 0.34 ± 0.12 | 26 ± 6 |
| Prone 1 | 75 ± 9 | 67 ± 17 | 30 ± 2 | 0.34 ± 0.12 | 29 ± 6 |
| Prone 2 | 84 ± 11 | 70 ± 16 | 32 ± 2 | 0.32 ± 0.08 | 29 ± 5 |
| Supine 2 | 91 ± 11 | 76 ± 15 | 35 ± 6 | 0.23 ± 0.10 | 29 ± 6 |

* Statistically different *versus* baseline ($P < 0.05$). † Patient not intubated at time of measurement.

MAP = mean arterial pressure; HR = heart rate; $ET_{CO_2}$ = end-tidal carbon dioxide; $ET_{iso}$ = end-tidal isoflurane; PIP = peak inspiratory pressure; NA = not applicable; Supine 1 = after intubation, patient anesthetized, supine; Prone 1 = before incision, patient anesthetized, prone; Prone 2 = at conclusion of surgery, patient anesthetized, prone; Supine 2 = before emergence, patient anesthetized, supine.

and the magnitude of the last prone IOP measurement, which suggests a linear relation, although data from only two points are insufficient for discerning this relation. Future studies will include IOP measurements made at more frequent time-based units because there were no tonometer-related complications in this series of 20 patients.

There are reports in the literature of blindness in the prone position, due to increased extraocular pressure resulting from using a cushion or horseshoe headrest to position the head.[14,15] To eliminate the effects of any extraocular pressure in this study, all of our patients were positioned in neutral position with a pinned headholder. It is surprising that our data demonstrated IOP increases in the prone position in the headholder, suggesting that a factor other than extraocular pressure (*e.g.*, a horseshoe headrest) is responsible for this increase.

Deliberate hypotension has been advocated for spine surgery to decrease intraoperative blood loss.[16] However, hypotension itself may decrease ocular perfusion pressure (*i.e.*, ocular perfusion pressure = MAP − IOP). Some authors have suggested that deliberate hypotension may decrease IOP,[10] but this was not demonstrated in a porcine model.[17] MAP was maintained within the normal range in our protocol without significant differences between prone 1 and prone 2 measurements, so we were unable to observe the effect of hypotension on IOP in the prone position.

The prone position may increase peritoneal pressure and in turn central venous pressure, peak inspiratory pressure, and IOP. In patients without glaucoma, IOP did not increase during short laparoscopic surgery requiring pneumoperitoneum in the lithotomy position.[18] However, a study by the same group demonstrated a significant increase in IOP when anesthetized rabbits with glaucoma were placed in a head-down position.[19] In our study, there were no significant changes in the peak inspiratory pressure during the prone position study period, and central venous pressure was not measured. Therefore, the effects of peak inspiratory pressure and central venous pressure on IOP in the prone position were not assessed.

IOP has been shown to increase in anesthetized patients in the supine head-down (Trendelenberg) position[10] and in awake vertically inverted volunteers.[20] The mechanism for this increase may be related to higher episcleral venous pressure. In a study of inverted humans, Friberg *et al.*[21] found a 1-mmHg increase in IOP for every 0.83 ± 0.21 mmHg increase in episcleral venous pressure. To optimize the operative site, a slight head-down position may be used, which may contribute to a further increase in the IOP in the prone position. Therefore, a head-neutral or head-up position may attenuate the observed IOP increase in the prone position.

Increased arterial carbon dioxide tension can contribute to an increase in IOP during general anesthesia in the supine position.[22] There is evidence that although arterial carbon dioxide tension remains unchanged when moving an anesthetized patient to the prone position,[23] the arterial carbon dioxide tension–end-tidal carbon dioxide gradient can increase[24]. The end-tidal carbon dioxide value remained constant in our study. However, it may be possible that the arterial carbon dioxide tension was slightly higher at the end of surgery, which could contribute to the observed increase in IOP.

Increased IOP may also be related to the observed positive intraoperative fluid balance. Supporting this hypothesis is evidence in healthy volunteers that acute oral water loading (14 ml/kg) increased IOP,[25] whereas exercise-induced dehydration reduced IOP.[26] Decreased serum osmolality during dialysis increased IOP in patients with renal failure.[27] In another report, three severely burned patients were found to have IOPs in the range of 37.2–81.7 mmHg that were due to extreme orbital congestion related to large amounts of intravenous fluid.[28] Similarly, IOP increased in anesthetized patients immediately after cardiopulmonary bypass was started, while arterial perfusion pressure and hematocrit levels concurrently decreased.[29] Prospective controlled studies are needed to discern if there is a relation between fluid balance and IOP in the prone position.

## Conclusion

This study represents an initial attempt to elucidate a probable mechanism for the recently recognized problem of visual loss after spine surgery. We did not intend to establish a cause-and-effect relation between IOP changes and visual loss. Rather, our goal was to isolate and examine the IOP piece of the ocular perfusion pressure puzzle. Our findings show that the prone position increases IOP in the anesthetized patient, suggesting that a concurrent drop in MAP could be deleterious to the eye. Because we were fortunate that none of the patients suffered postoperative visual loss, we cannot make any conclusions regarding the role of IOP and postoperative visual loss. Further work needs to be done to determine the time course, etiology, and possible treatment of the IOP increase in prone anesthetized patients undergoing spine surgery.

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# Causes of Elevated Intraocular Pressure during Prone Spine Surgery

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To the Editor:—

In the article by Cheng *et al.*[1] on the effect of prone positioning on intraocular pressure (IOP) during spinal operations, they reported an increase in IOP from 19 ± 1 mmHg supine baseline to 40 ± 2 mmHg at the end of the procedure in the prone position. The range of IOPs at the end of the case in the prone position was 25 to 54 mmHg. Despite an approximate 47% reduction in ocular perfusion pressure (MAP − IOP) from baseline to the end of the case, and an average duration of 320 ± 107 min, no visual deficits were reported. The very high IOP in this study is difficult to accept at face value.

As the authors pointed out, Lam and Douthwaite [2] reported that IOP increased in awake volunteers from 13.5 ± 2.01 mmHg baseline supine to 20.0 ± 3.27 mmHg in the prone position. Why was the initial prone value of IOP (27 mmHg) so much higher in the study of Cheng *et al.* with anesthetized patients compared to the study of Lam and Douthwaite with awake volunteers? Our studies have also shown lower initial prone values of 18.1 ± 0.8 mmHg, with peak prone IOP values of 24.6 ± 1.1 mmHg. [3] It seems that that technical error might be responsible for this difference. One of the most important causes of spuriously high recordings of IOP is inadvertent pressure on the globe while retracting the eyelids. This problem can be difficult to avoid when there is significant periorbital/conjunctival swelling, particularly in the prone position. In addition, contact of the tonometer with the globe must be made at a 90° angle. Failure to perform IOP measurements with these guidelines will result in erroneous values.

Although the authors discuss the possibility that an increased arterial carbon dioxide tension ($Paco_2$) may increase the IOPs, it is unlikely that this would result in IOPs up to 54 mmHg. We have found that the measurement of IOPs during emergence (unpublished data) results in greatly elevated IOPs, similar to normal awakening. [4,5] In the study of Cheng *et al.*, although reportedly not statistically significant, there is a trend toward increasing mean arterial pressure (MAP) at the end of the case (“prone 2” MAP, 84 ± 11 mmHg, and “supine 2” MAP, 91 ± 11 mmHg) compared to the initial supine and prone MAP measurements (“supine 1” and “prone 1” MAPs of 72 ± 7 and 75 ± 9 mmHg, respectively). This is consistent with “lightening” of anesthesia and perhaps early emergence from anesthesia. It is plausible that partial emergence from anesthesia, in conjunction with the technical challenge of retracting edematous eyelids, contributed to the extremely high IOPs observed in this study at the end of the procedure in the prone

position.

Another explanation for increased IOP is the effect of fluid administration. With increasing duration of surgery, one would expect greater fluid requirements. The significantly elevated IOP (31 mmHg, “supine 2”) even after return to supine at the conclusion of surgery, suggests this mechanism may be operative. Our studies also demonstrated a statistically significant increase in IOPs at the end of the case (average duration, 450 min) in the supine position (21 ± 1.1 mmHg, SEM), compared to baseline values (12 ± 0.7 mmHg), with very large average estimated blood loss and intravenous fluid administration. [3] Unfortunately, this study lacks a control supine group to evaluate this possibility in isolation. Elective supine cases should be matched for duration, estimated blood loss, and quantity of intravenous fluid administration. Given that those cases are difficult to find, it may be acceptable to study IOP in either supine operations of long duration or with comparable estimated blood loss and large intravenous fluid administration. A control group would allow changes in IOP caused solely by position to be evaluated independently of the other factors. The study results, while interesting and relevant, must be interpreted in the absence of these necessary controls.

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# Intraocular Pressure in Pediatric Patients During Prone Surgery

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**BACKGROUND:** Intraoperative intraocular pressure (IOP) in the prone position and IOP changes over time have not been evaluated in pediatric surgical patients. We sought to determine time-dependent changes in IOP in children undergoing surgery in prone position.
**METHODS:** Thirty patients undergoing neurosurgical procedures in prone position were included. Using a pulse-mode pneumatonometer, IOP was measured in supine position after induction and before emergence of anesthesia and in prone position before the start and after the end of surgery. IOP changes over time in the prone position were assessed with a linear mixed model (i.e., random slope and intercept model) to adjust for the within-patient correlation.
**RESULTS:** IOP in prone position increased by an average of 2.2 mm Hg per hour ($P < 0.001$). Sixty-three percent of patients (95% confidence interval [CI], 46%–81%) had at least 1 IOP value exceeding 30 mm Hg, and 13% (95% CI, 1%–25%) had at least 1 IOP value exceeding 40 mm Hg while prone. Mean IOP increased 7 mm Hg (95% CI, 6–9) during the position change from supine to prone ($P < 0.001$) and decreased 10 mm Hg (95% CI, 9–12) after changing the position from prone back to supine ($P < 0.001$).
**CONCLUSIONS:** Changing position from supine to prone significantly increases IOP in anesthetized pediatric patients. Moreover, the IOP continued to increase during surgery and reached potentially harmful values, especially when combined with low mean arterial blood pressures that are common during major surgery. (Anesth Analg 2013;116:1309–13)

Vision loss is a devastating perioperative complication[1,2] that has been reported as a complication of cranial vault reconstruction,[3] spine surgery,[4] and orbital surgery.[5] A United States national study estimated the overall incidence of perioperative visual loss to be 2.4 per 10,000 cases (0.02%), but that the risk is 0.03% for spinal fusion and 0.09% for cardiac surgery.[6]

An unexpected finding from analysis of the Nationwide Inpatient Sample was an alarmingly high risk of pediatric patients developing postoperative visual loss after all surgical procedures (odds ratio 6.9 versus adults). The odds ratio for developing visual loss in patients younger than 18 years after spinal fusion surgery was 5.8,[7] whereas the odds ratio of young patients to develop cortical blindness versus adults across all procedures was 64.[6] The reason for the increased visual loss risk in pediatric patients is not clear, but an embolic mechanism seems more likely than stroke (which is uncommon in children).[6] There are nonetheless only sporadic published reports of postoperative visual loss in pediatric patients.[3–5,8–10]

The causes of vision loss after spine surgery in prone position remain poorly understood, but appear to be multifactorial and may include impaired perfusion of the eye, occlusion of retinal vessels, or an "eye compartment syndrome" caused by increased orbital pressure and decreased perfusion secondary to use of large amounts of crystalloids.[11] Inadequate ocular perfusion pressure can cause retinal ischemia and may contribute to postoperative visual loss.[12,13] Ocular perfusion pressure is commonly defined as the difference between mean arterial blood pressure and intraocular pressure (IOP).[14] At a given mean arterial pressure, retinal perfusion pressure is determined by IOP. Factors that influence perioperative IOP are thus of considerable interest. IOP can be influenced by general anesthesia, fluid balance, and end-tidal carbon dioxide partial pressure. Aqueous humor flow, choroidal blood volume, central venous pressure, and extraocular muscle tone also contribute.[15] Positioning is yet another factor that influences IOP during surgery.[16] For example, IOP is increased by prone[17,18] and deep Trendelenburg[19] positions, with the increases being comparable with and without general anesthesia.[19,20] IOP also continues to increase over time in the prone position,[16,19–21] an effect that is thought to result from continued production of aqueous fluid by the ciliary body inside the eye[19] or to the accumulation of edema in the orbit.[11] With only a single exception,[18] all studies have found a time-dependent increase in IOP in adults.

The normal distribution of IOP is well established in unanesthetized, pediatric subjects.[22] However, intraoperative IOP and the extent to which it changes over time have

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yet to be evaluated fully in pediatric patients. Furthermore, the effect of prolonged prone positioning remains unknown in pediatric patients. We thus sought to determine time-dependent changes in IOP in children undergoing surgery in the prone position.

## METHODS

With approval of the University of Texas Southwestern IRB and written consent from parents, we enrolled consecutive patients from newborn to 18 years of age who were scheduled for neurosurgery in prone position with an expected duration exceeding 2 hours. Patients with a history of increased IOP or glaucoma, known visual impairment, heart failure, or ASA physical status scores >3 were excluded.

Both induction and maintenance of anesthesia were left to the discretion of the anesthesiologist, but typically included propofol (1.5–3.0 mg/kg), fentanyl (1–2 mcg/kg), vecuronium (0.1 mg/kg), and sevoflurane or isoflurane at approximately 1 minimum alveolar concentration. All patients were given dexamethasone 0.5 mg/kg[1] shortly after induction. Arterial blood pressure was monitored from an arterial catheter. Mechanical ventilation was adjusted to provide an end-tidal $Pco_2$ near 35 mm Hg. Anesthetic administration was adjusted as necessary to maintain mean arterial blood pressure and heart rate about 20% below preinduction values.

As is routine in these cases, the patient's head was secured with skull pins which allowed free access to the eyes while avoiding any direct mechanical pressure to the globe. The patient's head was elevated $10^0$ to reduce venous stasis.

Patients were given 5 to 7 mL/kg lactated Ringer's solution in the immediate postinduction period, which was followed by 5 mL/kg/h maintenance hydration. Additional lactated Ringer's solution was given as necessary to replace blood loss (usually in a 3:1 ratio) and to maintain mean arterial blood pressure about 20% below the preinduction value, heart rate within 20% of the preoperative value, and urine output ≥0.5 mL/kg/h. Blood was transfused as necessary to maintain a hematocrit ≥30%.

Morphometric and demographic characteristics were recorded, along with mean arterial blood pressure, blood loss, fluid administration, urine output, and the duration of surgery. IOP was measured with a Model 30 Classic Tm pulse-mode pneumatonometer (Reichert Technologies, Depew, NY). The pneumatonometer is self-calibrating and records 40 values per second; we thus made a single measurement for each eye at each time point. All measurements were performed by the same investigator (RBP). This system is well validated in pediatric patients.[23]

IOP was recorded first with patients supine 15 minutes after anesthetic induction but before the head was positioned in pins; second, 15 minutes after patients were turned prone; third, at the end of surgery while the patient was still in prone position; and fourth, 10 minutes after patients were turned supine at the end of surgery before tracheal extubation. When possible, IOP was determined in each eye at each measurement interval. Anesthesia was discontinued only after the final IOP measurements in supine position.

IOP changes over time in the prone position were assessed using a linear mixed model (i.e., a random slope and intercept model) with an unstructured covariance matrix to adjust for the within-patient correlation. This model assumes that patient effects (intercepts) and time effects (slopes—IOP changes over time) are random (i.e., differ among patients). The average IOP change per hour in the prone position was estimated with 95% confidence interval (CI). In addition, percentages of patients who had at least 1 IOP in the prone position exceeding 30 and 40 mm Hg were reported along with Wald confidence limits.

We assessed the IOP change from supine to prone position by comparing the initial measurement in the supine position and the first measurement in the prone position. Similarly, the IOP change from prone back to supine position was also assessed by comparing the final measurement in the prone position and the measurement in the supine position after changing back from the prone position. Pressures were compared with paired Student *t* tests. The corresponding mean (95% CI) of the IOP changes were estimated.

A total sample size of 26 was required to be able to detect a change of 2 mm Hg or more per hour in IOP at the 0.05 significance level and 90% power, assuming an SD of 3 mm Hg and a correlation of 0.5 based on previous experience. A total sample size of 30 patients was thus selected. SAS statistical software 9.2 for Windows (SAS Institute, Cary, NC) was used for all analyses.

**Table 1. Demographics Baseline and Intraoperative Characteristics (*N* = 30)**

| Variables | Statistics[a] |
|---|---|
| Age, mo | 104 (58) |
| Weight, kg | 38 (25) |
| Length, cm | 131 (33) |
| Gender (male), % | 40 |
| ASA physical status class | 1.7 (0.6) |
| Type of surgery, % | |
| Lumbar laminectomy for tethered cord | 43 |
| Craniotomy | 13 |
| Chiari decompression | 43 |
| Volatile anesthetic used, % | |
| Isoflurane | 38 |
| Desflurane | 4 |
| Sevoflurane | 58 |
| Length of surgery, h | 4.3 (1.3) |

[a]Summary statistics presented as mean (SD) or % of patients.

## RESULTS

Thirty pediatric patients were included in the study. Table 1 provides the summary of the demographics baseline and intraoperative characteristics. Blood loss was minimal in all patients, and none required blood replacement.

The change of IOP over time during the prone position did not vary by eye side ($P = 0.19$, assessment of interaction). We thus averaged IOP for the left and right sides when both were available at a given time point, or used the nonmissing IOP measurement when only 1 was available.

IOP changed approximately linearly over time in patients with >2 prone measurements (Figs. 1 and 2); a random slope and intercept model was therefore used to assess IOP change over time during the prone position. The estimated average slope was 2.2 (95% CI, 1.5–2.9) mm Hg per hour, indicating an average of 2.2 mm Hg increase in

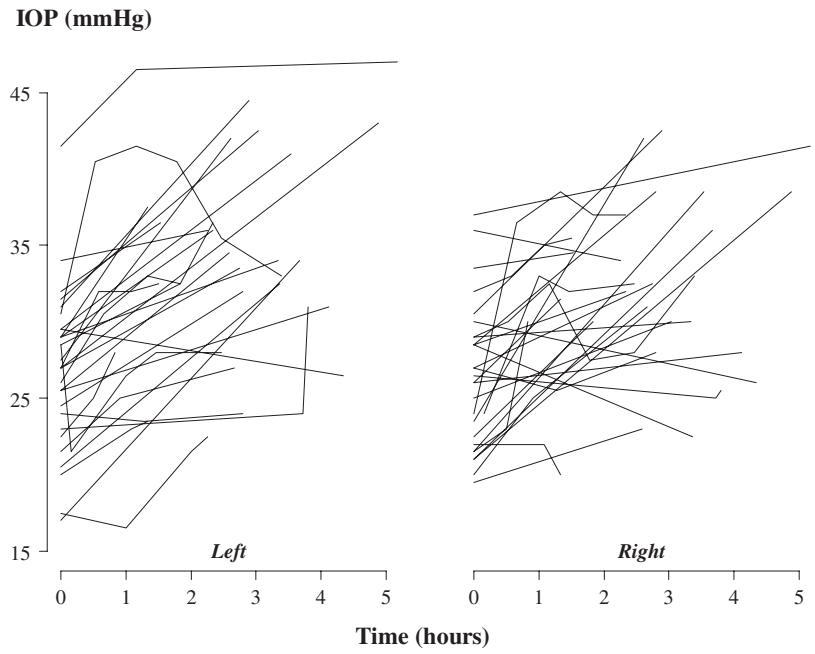


**Figure 1.** Left and right eye intraocular pressure (IOP) for 30 pediatric patients undergoing surgery in prone position. Zero on x-axis refers to the first IOP measurement in prone position. Each line represents IOP measurements for each patient.

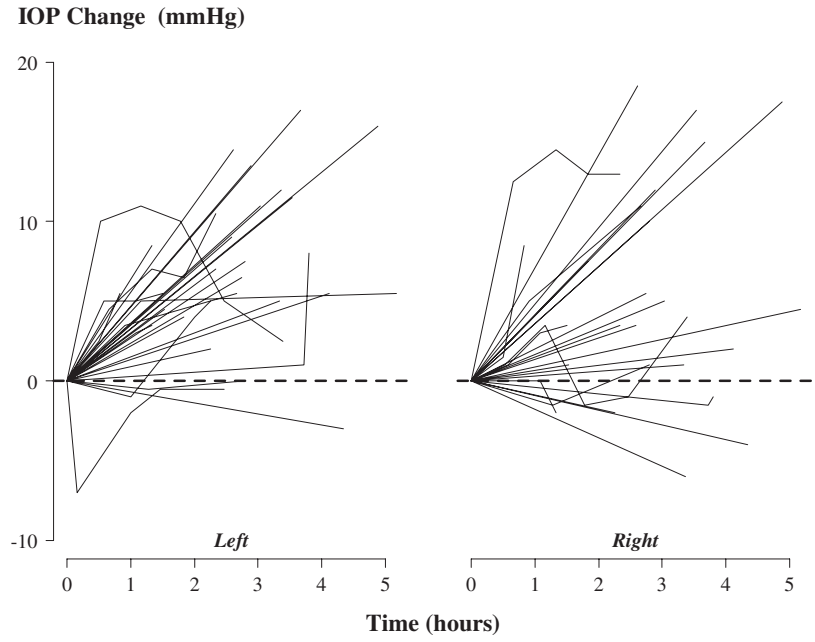


**Figure 2.** Changes in intraocular pressure (IOP) over time from first prone position (left and right eye) for 30 pediatric patients undergoing surgery in prone position. Zero on x-axis refers to the first IOP measurement in prone position; zero on y-axis (dashed line) refers to no change in IOP. Each line represents changes in IOP for each patient.

IOP per hour in prone position ($P < 0.001$; Fig. 3, middle panel).

Sixty-three percent of patients (95% CI, 46%–81%) had at least 1 IOP value exceeding 30 mm Hg, and 13% (95% CI, 1%–25%) had at least 1 IOP value exceeding 40 mm Hg while prone.

The observed mean (SD) of IOP was 19 (3) mm Hg for the initial measurement in the supine position and 27 (5) mm Hg for the first measurement in the prone position (Fig. 3, left panel). Mean IOP thus increased 7 (95% CI, 6–9) mm Hg during the position change from supine to prone ($P < 0.001$).

The observed mean (SD) of IOP was 32 (6) mm Hg at the last IOP measurement in the prone position and 22 (4) mm Hg in the supine position after changing back from the prone position (Fig. 3, right panel). Mean IOP thus decreased 10 (95% CI, 9–12) mm Hg after changing the position from prone back to supine ($P < 0.001$). One patient did not have IOP measured in supine position after changing back from prone position; thus 29 patients were included in this analysis.

## DISCUSSION

Our results indicate that in pediatric patients mean IOP increased 7 (95% CI, 6–9) mm Hg during the position change from supine to prone ($P < 0.001$) and decreased 10 (95% CI, 9–12) mm Hg after changing the position from prone back

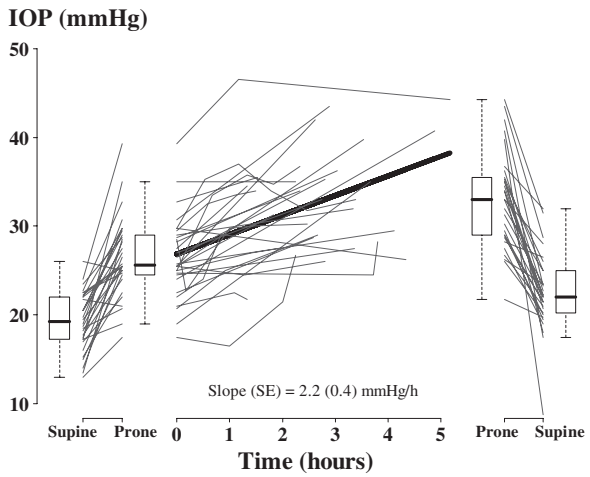


**Figure 3.** Average (left and right eye) intraocular pressure (IOP) for 30 pediatric patients undergoing surgery in prone position. Left panel: Box plots of IOP in the initial supine position and the first measurement in the prone position, and a plot of individual changes from supine to prone position. Middle panel: Plot of the IOP over time from the first IOP measurement in prone position. Right panel: Box plots of the final IOP measurement in the prone position and IOP in the final supine position, and a plot of individual changes from prone back to supine position. Each line represents changes in IOP for each patient. The middle, upper, and lower edges of the box indicate the 50th, 75th, and 25th percentile of the data. The ends of the vertical lines indicate 1.5 times the interquartile range.

to supine ($P < 0.001$). IOP in prone position increased by an average of 2.2 mm Hg per hour ($P < 0.001$). These results are comparable with those in adults.[4,5]

IOP in children is generally low, reaching adult pressures at about 12 years of age. For example, IOP in a normal pediatric population ranges from a mean (SD) of 8 (3) mm Hg in infants younger than 1 year to 15 (3) mm Hg in children aged 11 to 12 years.[5,22] These values were slightly lower than the 19 (3) mm Hg we observed in our patients while supine after induction of anesthesia. The discrepancy might result from differing approaches to IOP determination because previous measurements were made with conventional tonometry rather than applanation pneumotonometry as in our study. The distinction is important as Eisenberg et al.[23] found that conventional tonometry significantly underestimates IOP in pediatric patients; in contrast, applanation pneumotonometry remained accurate.

Sugata et al.[24] showed that the choice of anesthetic (sevoflurane or propofol) does not have significant effect on IOP changes during short periods of prone surgery; however, changes in body position have a noticeable effect. IOP increased from 19 (3) mm Hg while supine to 27 (5) mm Hg after patients were initially turned prone. IOP then decreased from 32 (6) mm Hg while prone at the end of surgery to 22 (4) mm Hg in the supine position. The effect of changing position was thus similar at the beginning and end of surgery.

As in adults, IOP increased over time in the prone position. However, the slope was only 2.2 (0.4) mm Hg/h. Consequently, mean pressure was 32 (6) mm Hg at the end of procedures lasting 4.3 (1.3) hours. We found that 63% of our pediatric patients (95% CI, 46%–81%) had IOP exceeding 30 mm Hg, and 13% (95% CI, 1%–25%) had IOP exceeding 40 mm Hg while prone. Sustained increases in IOP over time have been reported to have negative effects both in animal and humans studies.[12,13] It is thus plausible that IOPs exceeding 40 mm Hg in our patients could have put them at risk of visual loss.

Blood flow to the optic nerve head is regulated and thus remains relatively constant despite changes in IOP.[20] The IOP at which autoregulation fails in pediatric patients is unknown, but in adult volunteers IOP remained nearly constant until ocular pressures reached 40 mm Hg.[25] Even if 40 mm Hg were the safe threshold in pediatric patients, 13% of our patients exceed this pressure. However, it is conceivable that blood flow in the optic nerve is lower in infants and approaches adult values in older children. It is thus concerning that IOP exceeded 30 mm Hg in more than half of the patients we evaluated during prone surgery.

Grant et al.[21] evaluated the anatomy of the posterior optic nerve in volunteers laying supine or prone for 5 hours by using ultrasound imaging. In the prone position only, there was a thickening of the choroid layer which progressed over time, along with an increase in optic nerve diameter. These results support the hypothesis that time-dependent increases in IOP result at least partially from orbital venous congestion and its effect on episcleral venous congestion.

That being said, the clinical implications of increased IOP remain poorly understood. Thus, while pressures exceeding 40 mm Hg are certainly concerning, it is unknown whether relatively brief periods (i.e., hours) at such pressures actually provoke visual loss. Ocular perfusion pressure, by definition, depends on mean arterial blood pressure, but blood pressure is often low during surgery which presumably aggravates risk. We also note that it is difficult to accurately assess visual ability in infants and children and that much postoperative visual loss may never be detected clinically or even in studies. Finally, IOP, and changes in IOP during surgery, varied considerably from patient to patient. A consequence is that the average values we report poorly predict individual pressures; a corollary is that without individual IOP measurements, it will be difficult to predict a given patient's pressure at any particular time.

Reported differences in IOP among studies may result from various methods used to position patients' heads and from various methods for measuring IOP. The head was supported by scalp pins in all our patients; consequently, there was no direct pressure on the eyes at any time.

Our study was far too small to establish a cause and effect relation between IOP changes and visual loss, and that was never among our goals. Instead, we sought to determine time-dependent changes in IOP in children undergoing surgery in prone position. Due to technical difficulties with IOP assessment during prone position, we measured IOP only before and after surgery in most patients. We were thus unable to fully characterize the shape of the IOP curve over time and have assumed based on limited data that it is approximately linear. The mean duration of surgery was 4.3 (1.3) hours. We do not know whether IOP would continue to increase during longer operations, or if it would reach a plateau.

In summary, changing from supine to the prone position significantly increases IOP in anesthetized pediatric patients. Moreover, IOP progressively increased during surgery and often reached potential harmful values. ■

## DISCLOSURES

**Name:** Peter Szmuk, MD.
**Contribution:** This author helped design and conduct the study and write the manuscript.
**Attestation:** Peter Szmuk has seen the original study data, reviewed the analysis of the data, approved the final manuscript, and is the author responsible for archiving the study files.
**Name:** Jeffrey W. Steiner, DO.
**Contribution:** This author helped conduct the study and write the manuscript.
**Attestation:** Jeffrey W. Steiner has seen the original study data, reviewed the analysis of the data, and approved the final manuscript.
**Name:** Radu B. Pop, MS.
**Contribution:** This author helped conduct the study.
**Attestation:** Radu B. Pop has seen the original study data and approved the final manuscript.
**Name:** Jing You, MS.
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**Attestation:** Jing You has seen the original study data, reviewed the analysis of the data, and approved the final manuscript.
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**Attestation:** David R. Weakley has seen the original study data and approved the final manuscript.
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**Contribution:** This author helped design and conduct the study.
**Attestation:** Dale M. Swift has seen the original study data and approved the final manuscript.
**Name:** Daniel I. Sessler, MD.
**Contribution:** This author helped design the study, analyze the data, and write the manuscript.
**Attestation:** Daniel I. Sessler has seen the original study data, reviewed the analysis of the data, and approved the final manuscript.
**This manuscript was handled by:** Peter J. Davis, MD.

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