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

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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.

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.

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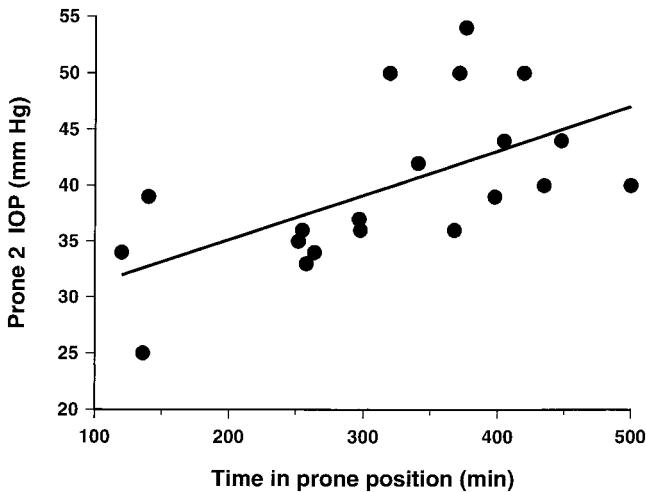
Fig. 1. Intraocular pressure (IOP) at conclusion of prone positioning (prone 2) is correlated with total time spent in the prone position, in minutes ($P < 0.05$; $r^2 = 0.6$; $n = 20$). Prone 2 IOP ranged from 25 to 54 mmHg, and total time in the prone position ranged from 120 to 500 min.

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 ($3,292 \pm 1,120$ 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 Tonopen® 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)	ETco$_2$ (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; ETco$_2$ = 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.

CHENG ET AL.

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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