Original Article

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Intraocular Pressure and Optic Nerve Sheath Diameter during Prone-position Thoracolumbar Spine Surgery: A Prospective Observational Study

Anaesthesia Section

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ABSTRACT

Introduction: Patients undergoing prone-position spine surgery risk raised Intraocular Pressure (IOP) and, rarely, Postoperative Visual Loss (POVL). The temporal behaviour of IOP, Ocular Perfusion Pressure (OPP) and Optic Nerve Sheath Diameter (ONSD), a non-invasive surrogate of Intracranial Pressure (ICP), and the relationship between these ocular and intracranial parameters, remain incompletely characterised.

Aim: To evaluate the magnitude and temporal pattern of changes in IOP, OPP, ONSD and Mean Arterial Pressure (MAP) during prone-position thoracolumbar spine surgery, to identify factors associated with IOP elevation, and to examine the correlation between IOP and ONSD.

Materials and Methods: This prospective observational study was conducted in the Department of Anaesthesiology and Critical Care Medicine, Institute of Medical Sciences, Banaras Hindu University, Varanasi, Uttar Pradesh, India. A total of 37 American Society of Anaesthesiologists (ASA) I-II patients undergoing thoracolumbar spine fixation were enrolled. Bilateral IOP, OPP, ONSD and MAP were measured at eight time points: postintubation (baseline), hourly for up to six hours, and before extubation. Data were analysed with the Friedman test, paired t-tests and Spearman rank correlation.

Results: Thirty-seven patients completed the study. IOP rose significantly from baseline (right eye 13.46±1.95 mmHg) to 22.65±2.50 mmHg at 5 hours and 23.40±1.52 mmHg at 6 hours, p-value <0.001, and remained elevated before extubation (16.06±2.82 mmHg, p-value <0.001). ONSD increased during prone positioning and returned towards baseline by extubation. IOP and ONSD correlated positively across intraoperative measurements (pooled Spearman ρ=0.299, p-value <0.001, n=486). Age (ρ=0.543) and baseline MAP (ρ=0.533) correlated with baseline IOP (both p-value=0.001). OPP remained above 50 mmHg in every patient. No patient developed visual impairment postoperatively or at 48 hours.

Conclusion: Prone-position spine surgery causes significant, progressive IOP elevation that persists until extubation. The parallel rise in ONSD and its correlation with IOP suggest a concurrent increase in ICP, an eye-brain axis response. OPP was nonetheless preserved and no POVL occurred, indicating that surgery of this duration was tolerated by ASA I-II patients. Advanced age and higher baseline MAP confer greater risk, supporting perioperative IOP monitoring in high-risk patients.

Keywords: Intracranial pressure, Ischaemic optic neuropathy, Ocular perfusion pressure, Ocular ultrasonography, Postoperative visual loss, Tonometry

INTRODUCTION

POVL following non-ocular surgery is one of the most devastating complications in modern anaesthesia practice [1]. Its reported incidence ranges from 0.0002% to 0.2%, with significantly higher rates after spine surgery performed in the prone position [2]. The prone position, while essential for surgical access, creates physiological challenges that can compromise ocular perfusion and raise IOP. The dependent position of the head increases episcleral venous pressure, impairs aqueous-humour outflow and promotes choroidal engorgement; raised central venous pressure, positive-pressure ventilation and prolonged surgical duration further exacerbate these changes [3,4]. IOP can rise within minutes of assuming the prone position, with progressive elevation that correlates with duration [5]. OPP determines blood flow to the optic nerve head and is calculated as the difference between MAP and IOP [6]. Elevated IOP reduces OPP, potentially compromising optic nerve perfusion and increasing the risk of ischaemic optic neuropathy, the commonest cause of POVL [7]. The American Academy of Ophthalmology defines normal IOP as 10-21 mmHg, with higher pressures increasing the risk of glaucomatous damage [8].

The ONSD has emerged as a reliable non-invasive surrogate marker for ICP [9,10]. The optic nerve sheath is a direct extension of the dural meninges, and cerebrospinal fluid within it communicates with the intracranial compartment, so raised ICP causes measurable distension of the sheath that is detectable with bedside ultrasound [11]. Several studies including systematic reviews and meta-analyses have demonstrated correlations between ONSD and invasively measured ICP [9,11,12]. The POVL Study Group identified male sex, increased Body Mass Index (BMI), prolonged surgical duration (>6 hours) and substantial intraoperative blood loss as factors associated with ischaemic optic neuropathy after spine surgery [13], emphasising the importance of understanding individual risk factors for IOP elevation. A spectrum of less severe ocular complications can also occur during prone positioning, including subconjunctival haemorrhage, corneal abrasion, acute angle-closure glaucoma and Horner's syndrome [14-16]. The prone position may also influence ICP, although neurocritical-care studies have produced inconsistent results, with some reporting increases and others stability or even decreases [17,18]. Evidence from laparoscopic and other specialties indicates that positioning-related IOP elevation is not unique to spine surgery [19], and positioning modifications such as reverse

Trendelenburg and head elevation can mitigate it [20,21]. Cheng MA et al., were among the first to document IOP changes during prone positioning [22], Sugata A et al., compared anaesthetic agents [23], and other work has shown that IOP changes are position-dependent and potentially modifiable. Despite this, the precise temporal pattern of IOP change, the relationship between IOP and ONSD, and the patient factors predicting significant IOP elevation remain incompletely characterised. The aim of this study was to evaluate the magnitude and temporal pattern of changes in IOP, OPP, ONSD and MAP during prone-position thoracolumbar spine surgery. The primary objective was to characterise the time course of these four parameters from baseline to extubation. The secondary objectives were: (i) to identify the demographic and haemodynamic factors-age, sex, BMI, ASA class, baseline MAP, heart rate and intraoperative blood loss, associated with IOP elevation; and (ii) to examine the correlation between IOP and ONSD as an index of a shared ocular-intracranial (eye-brain axis) response to positioning.

MATERIALS AND METHODS

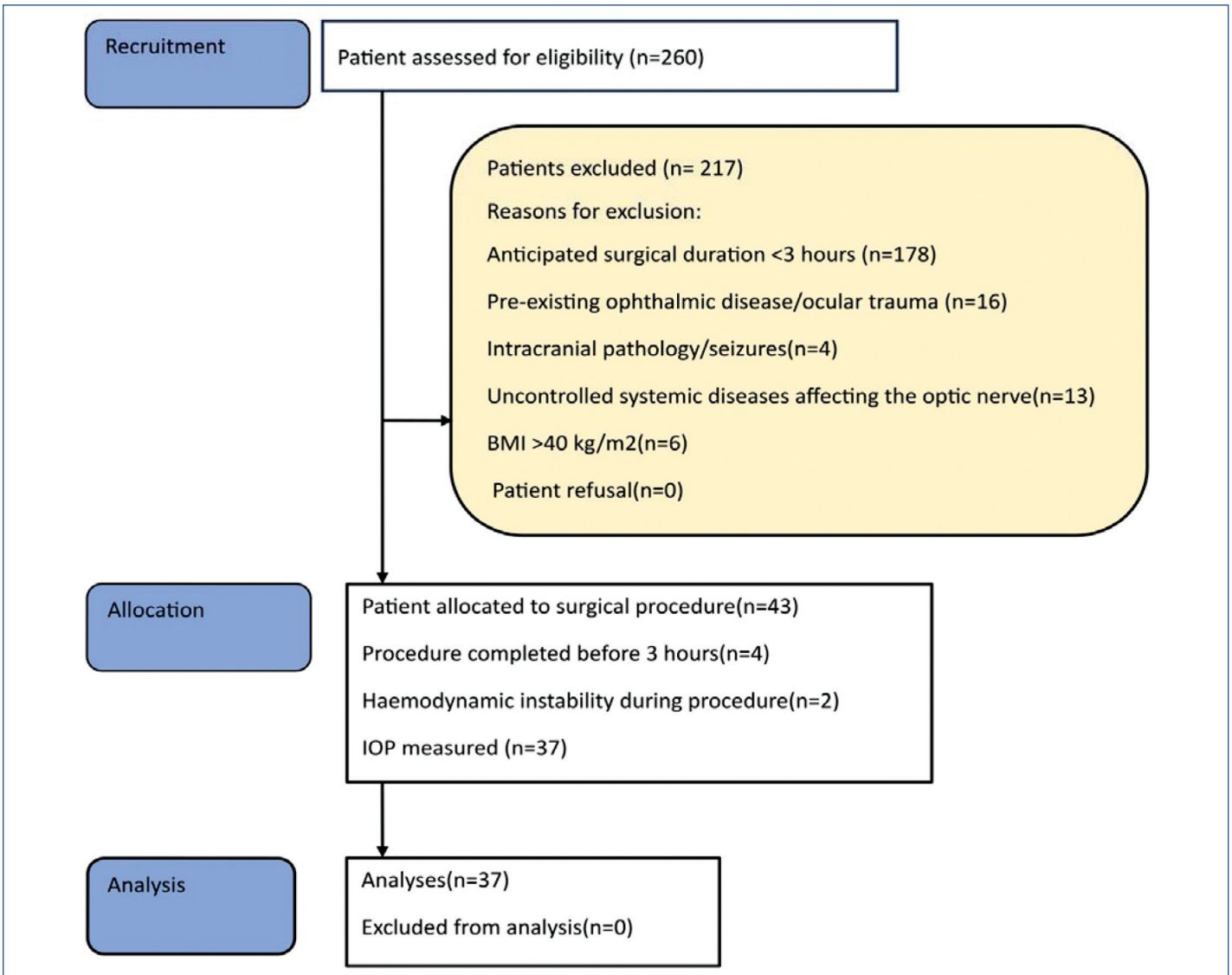
This prospective observational study was conducted in the Department of Anaesthesiology and Critical Care Medicine, Trauma Centre, Institute of Medical Sciences, Banaras Hindu University, Varanasi, Uttar Pradesh, India, over eight months from May 2023 to December 2023. The study was approved by the Institutional Ethics Committee (No. Dean/2022/EC/4067, dated 15 April 2023) and conducted in accordance with the Declaration of Helsinki (1975, revised 2013); written informed consent was obtained from all participants. As this was an observational study, a consecutive (convenience) sampling method was used: all patients who met the inclusion criteria during the study period were enrolled, so a formal a priori sample-size calculation was not undertaken.–The bilateral measurements at eight time points additionally provided 486 paired IOP-ONSD observations, conferring substantial power for the correlation and repeated-measures analyses.

Inclusion criteria: Patients aged 18-80 years with ASA physical status I-II scheduled for elective thoracolumbar spine fixation requiring prone positioning were included in the study.

Exclusion criteria: Patients with any of the following were excluded from the study: pre-existing ophthalmic disease or ocular trauma; intracranial pathology or seizures; anticipated surgical duration <3 hours; uncontrolled systemic disease affecting the optic nerve; BMI >40 kg/m^2; and patient refusal. Of 260 patients assessed for eligibility, 217 were excluded and 43 were allocated; six were subsequently not analysed (four whose surgery finished within three hours and two with intraoperative haemodynamic instability), leaving 37 patients with complete data [Table/Fig-1].

Study Procedure

All patients were kept fasted according to standard ASA preoperative fasting guidelines (a minimum of six hours for solids and two hours for clear fluids). All patients received standardised general anaesthesia induced with propofol (2-2.5 mg/kg), fentanyl (2 µg/kg) and rocuronium (0.6 mg/kg), and maintained with sevoflurane (1-1.5 MAC) in an oxygen–air mixture. Patients were positioned prone on a Wilson frame with the head neutral and the eyes free from direct pressure. Mechanical ventilation was adjusted to maintain end-tidal



[Table/Fig-1]: Flow chart showing the recruitment process of patients for the study.

CO_2 at 30-35 mmHg; this target maintains arterial normocapnia, because end-tidal CO_2 reads approximately 2-5 mmHg below arterial $PaCO_2$ and this gradient widens in the prone position, so an end-tidal value of 30-35 mmHg corresponds to a $PaCO_2$ of about 35–40 mmHg (the measured mean end-tidal CO_2 was 33.9±1.7 mmHg).

Tight normocapnia was maintained deliberately because carbon dioxide independently alters ONSD and ICP, so uncontrolled hypocapnia or hypercapnia would have confounded the ocular-intracranial measurements [24]. Standard monitoring comprised electrocardiography, non-invasive blood pressure, pulse oximetry, continuous capnography and inspired/expired oxygen and anaesthetic-agent gas monitoring.

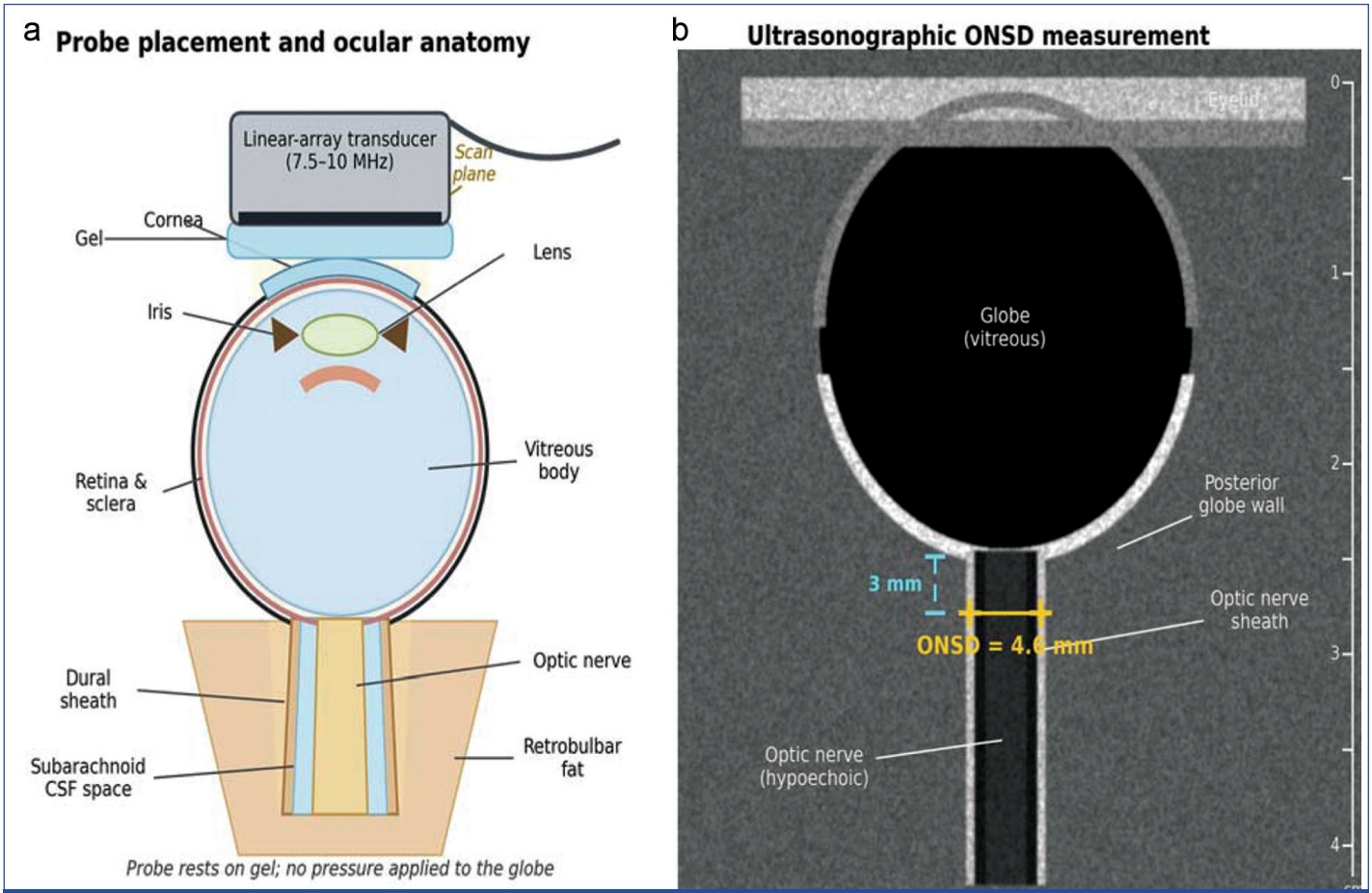
Bilateral IOP was measured with a Tono-Pen AVIA (Reichert Inc., NY, USA) applanation tonometer after instillation of a topical anaesthetic; the device was calibrated per the manufacturer's instructions, and at each time point readings were accepted only when the displayed coefficient of variability was ≤5%. ONSD was measured by ocular ultrasonography using a Fujifilm SonoSite M-Turbo system with a 7.5-10 MHz linear-array transducer (FUJIFILM SonoSite Inc., Bothell, WA, USA). With the eyes closed, ultrasound gel was applied to the upper eyelid and the transducer rested gently on the gel without pressure on the globe; ONSD was measured 3 mm behind the posterior globe margin, perpendicular to the nerve axis, using on-screen callipers, and three readings per eye were averaged at each time point (the technique is illustrated in [Table/Fig-2]). All ONSD measurements were performed by a single anaesthesiologist with more than eight years' experience in perioperative ocular ultrasonography, to eliminate inter-observer variability; to reduce measurement bias, this operator was blinded to the simultaneously recorded IOP values, and anaesthesia and positioning were standardised across all patients. Bilateral IOP, OPP, ONSD and MAP were recorded at eight time points: postintubation in the supine position (baseline); hourly after prone positioning for up to six hours; and immediately before extubation after return to the supine position. OPP was calculated for each eye as OPP=MAP - IOP; because the eye lies close to heart level in the prone anaesthetised patient, the two-thirds hydrostatic correction used for upright subjects was not applied. Intraoperative estimated blood loss was also recorded. All patients were assessed clinically for visual symptoms on emergence in the post-anaesthesia care unit and again at 48 hours, including direct questioning regarding blurring of vision, visual-field disturbance, diplopia, eye pain or subjective visual loss.

STATISTICAL ANALYSIS

Data were analysed with IBM Statistical Package for the Social Sciences (SPSS) Statistics v25.0 (IBM Corp., Armonk, NY, USA). Continuous variables were presented as mean±SD and categorical variables as frequencies, and normality was assessed with the Shapiro-Wilk test. Changes across the eight time points were tested with the Friedman test, with Wilcoxon signed-rank (Bonferroni-corrected) or paired t-tests for specific paired comparisons as appropriate. Associations between continuous variables, including IOP and ONSD, were assessed with the Spearman rank correlation coefficient- used as the single correlation test throughout because IOP, ONSD and MAP were non-normally distributed- at each time point and in a pooled analysis. Group comparisons used the independent-samples t-test or Mann-Whitney U test, and a two-tailed p-value <0.05 was considered significant.

RESULTS

Thirty-seven patients completed the study; their demographic and baseline characteristics are summarised in [Table/Fig-3]. The mean



[Table/Fig-2]: Technique of Optic Nerve Sheath Diameter (ONSD) measurement (original illustration): (a) Sagittal schematic of the closed eye showing the linear-array transducer (7.5-10 MHz) resting on the eyelid over ultrasound gel without pressure on the globe, with the relevant ocular and orbital anatomy labelled- cornea, lens, iris, vitreous body, retina and sclera, the optic nerve with its dural sheath and subarachnoid cerebrospinal fluid space, and retrobulbar fat; (b) Corresponding B-mode ultrasonographic appearance: the anechoic globe, hyperechoic posterior globe wall and hypoechoic optic nerve are seen; the ONSD is measured 3 mm behind the posterior globe margin, perpendicular to the nerve axis, from the outer borders of the hyperechoic dural sheath using electronic callipers (representative value 4.6 mm).

Parameter	Value
Total patients (N)	37
Age, years	42.5±11.9 (range 19–60)
BMI, kg/m²	27.1±4.6 (range 18.1–36.0)
Sex (male/female)	21 (56.8%)/16 (43.2%)
ASA class (I/II)	22 (59.5%)/15 (40.5%)
Baseline MAP, mmHg	83.1±8.5
Heart rate, bpm	84.2±10.9
SpO_2, %	98.8±1.2
End-tidal CO_2, mmHg	33.9±1.7
Blood loss, mL	524±223 (range 200-1250)

[Table/Fig-3]: Patient demographics and baseline characteristics (N=37).
ASA: American Society of Anaesthesiologists; BMI: Body mass index; MAP: Mean arterial pressure

age was 42.5±11.9 years, 21 (56.8%) were male and 22 (59.5%) were ASA I.

IOP rose progressively and highly significantly throughout prone surgery [Table/Fig-4]. From a baseline of about 13.46 mmHg, it increased by roughly 44% within the first hour and peaked at 5-6 hours at 22-25 mmHg (a 68-70% rise), exceeding the upper normal limit of 21 mmHg in prolonged cases. After return to the supine position IOP fell but remained significantly elevated before extubation (16.06±2.82 mmHg, right eye; +19.7%; paired t=-6.94, p-value <0.001). The number of patients contributing data fell at the later time points (34 at four hours, 20 at five hours and five at six hours) because measurements were recorded only while surgery was ongoing; patients whose operation finished earlier simply had no later time points, so these are not missing data but reflect the natural variation in surgical duration.

Time point	n	Right IOP (mean±SD)	Left IOP (mean±SD)	IOP, median (IQR)
Postintubation (baseline)	37	13.46±1.95	13.76±2.30	14.0 (12.0-15.0)
1 hour	37	19.32±2.01	19.46±2.23	19.5 (18.0-20.5)
2 hours	37	20.00±2.38	20.38±2.79	20.0 (18.5-21.5)
3 hours	37	20.68±2.51	21.22±2.92	20.5 (19.0-22.5)
4 hours	34	21.38±2.55	21.74±2.96	21.2 (19.5-23.0)
5 hours	20	22.65±2.50	22.80±1.96	23.0 (21.5-24.0)
6 hours	5	23.40±1.52	25.20±1.64	24.5 (24.5-25.0)
Before extubation	36	16.06±2.82	16.28±2.42	16.5 (14.0-18.1)

[Table/Fig-4]: Intraocular Pressure (IOP, mmHg) at the eight study time points.
IQR: Interquartile range. Friedman test across post-intubation to 4 hours (n=34): right eye χ^2=110.3, left eye χ^2=109.4, both p<0.001. The decreasing n at the later time points reflects variable surgical duration: measurements were taken only while surgery was ongoing, so patients whose operation finished earlier did not contribute later time points (these are not missing data).

OPP showed the inverse pattern [Table/Fig-5], declining by about 9% to a nadir of 61-63 mmHg at 5-6 hours but remaining above 50 mmHg in every patient (lowest individual value 53 mmHg) and recovering to baseline by extubation. ONSD increased significantly during prone positioning, peaking at six hours (right eye 4.86±0.31 mm), and- unlike IOP- returned essentially to baseline by extubation, with no significant post-intubation-to-extubation difference (paired t-test p-value=0.063 and p-value=0.634 for the right and left eyes).

One patient who developed intraoperative haemodynamic instability was not extubated on the operating table and was transferred, intubated, to the intensive care unit for delayed extubation; this patient therefore had no before-extubation measurement, so the paired pre- versus post-comparison comprises the 36 patients with complete paired data. Paired comparison of postintubation and pre-extubation values [Table/Fig-6] confirmed that only IOP (both eyes, p-value <0.001) and MAP (p-value=0.015) differed significantly between these time points, whereas OPP and ONSD did not, indicating that the OPP reduction and ONSD distension had resolved by the end of surgery while the IOP elevation persisted.

IOP and ONSD were positively correlated at the early intraoperative time points, from postintubation through four hours (Spearman ρ 0.33–0.42, all p-value <0.01), with a positive but non-significant trend at five and six hours where fewer patients contributed; across all measurements the correlation was highly significant (pooled ρ=0.299, p-value <0.001, n=486 paired measurements) [Table/Fig-7]. This indicates that eyes with higher IOP also showed greater optic nerve sheath distension and that the two parameters rose and fell in parallel during prone positioning. Among baseline characteristics, only older age (ρ=0.543, p-value=0.001) and higher baseline MAP (ρ=0.533, p-value=0.001) were significantly associated with baseline IOP; sex, BMI, heart rate and blood loss were not [Table/Fig-7a,b]. Older age and higher baseline MAP also clustered together: ASA II patients were significantly older than ASA I patients and had higher BMI and baseline MAP, although baseline IOP did not differ significantly between the ASA groups [Table/Fig-8].

No patient developed clinically detectable visual impairment. On emergence and at 48 hours, none reported blurring of vision, visual-field disturbance, diplopia, eye pain or subjective visual loss; gross confrontation testing of acuity and fields was normal in all 37 patients, and none required ophthalmological referral. Thus, despite substantial intraoperative IOP elevation and prone durations beyond six hours in some patients, no episode of POVL occurred in these ASA I-II patients.

DISCUSSION

This study characterises the temporal behaviour of IOP, OPP, ONSD and MAP during prone-position thoracolumbar spine surgery. In the current study IOP rose from a baseline of about 13.46 mmHg to a peak of 22-25 mmHg at 5-6 hours- a 68-70% increase- and remained about 2.6 mmHg (19.7%) above baseline at extubation. This magnitude was consistent with previous reports: Cheng MA et al., observed a mean IOP increase of about 13.4 mmHg in prone anaesthetised patients [22], Emery SE et al., demonstrated substantial IOP rises during lumbar spine fusion [25], and Yoshimura K et al., reported elevations of 7-10 mmHg and identified longer duration and greater body weight as predictors [5], partially mirroring the present study findings for age and MAP. Sugata A et al., showed

Time point	n	Right OPP	Left OPP	Right ONSD	Left ONSD	MAP
Postintubation	37	68.97±6.80	68.89±6.98	4.61±0.29	4.55±0.26	82.4±7.7
1 hour	37	64.86±6.16	64.73±6.21	4.62±0.30	4.58±0.29	84.2±6.6
2 hours	37	64.54±5.62	64.27±5.77	4.63±0.29	4.59±0.28	84.9±6.6
3 hours	37	64.41±5.99	63.86±5.64	4.65±0.30	4.61±0.29	84.8±6.7
4 hours	34	63.29±4.28	62.94±4.18	4.66±0.32	4.62±0.29	84.7±5.3
5 hours	20	62.90±5.82	62.75±5.54	4.66±0.32	4.69±0.32	85.6±6.1
6 hours	5	62.80±4.02	61.00±4.74	4.86±0.31	4.80±0.38	86.2±5.3
Before extubation	36	68.31±6.38	68.19±5.92	4.56±0.24	4.56±0.24	84.5±6.8

[Table/Fig-5]: Ocular Perfusion Pressure (OPP, mmHg), Optic Nerve Sheath Diameter (ONSD, mm) and Mean Arterial Pressure (MAP, mmHg) at the eight time points (mean±SD).
Friedman test (post-intubation to 4 hours, n=34): OPP right χ^2=31.0, left χ^2=29.4; ONSD right χ^2=34.6, left χ^2=30.2; MAP χ^2=12.6; all p≤0.014. OPP remained above 50 mmHg in every patient (lowest individual value 53 mmHg).

Parameter	Postintubation	Before extubation	Mean diff.	t	p-value
Right IOP (mmHg)	13.42±1.96	16.06±2.82	+2.64	–6.94	<0.001*
Left IOP (mmHg)	13.64±2.22	16.28±2.42	+2.64	–7.02	<0.001*
Right OPP (mmHg)	68.81±6.82	68.31±6.38	–0.50	0.58	0.567
Left OPP (mmHg)	68.81±7.05	68.19±5.92	–0.61	0.65	0.518
Right ONSD (mm)	4.60±0.29	4.56±0.24	–0.04	1.92	0.063
Left ONSD (mm)	4.55±0.26	4.56±0.24	+0.01	–0.48	0.634
MAP (mmHg)	82.22±7.66	84.47±6.78	+2.25	–2.56	0.015*

[Table/Fig-6]: Paired comparison of bilateral IOP, OPP, ONSD and MAP at postintubation versus before extubation (paired t-test, n=36).
* Statistically significant (p<0.05). Baseline (post-intubation) values in this table are for the 36 patients with complete paired data (one patient without a pre-extubation measurement was excluded); they therefore differ marginally from the n=37 baseline in [Table/Fig-4].

(a) Variable vs baseline IOP		Spearman ρ	p-value
Age (years)		0.543	0.001*
Baseline MAP (mmHg)		0.533	0.001*
BMI (kg/m²)		0.153	0.365
Heart rate (/min)		0.231	0.169
Blood loss (mL)		0.102	0.550
(b) Time point	**n (eyes)**	**Spearman ρ**	**p-value**
Postintubation	74	0.422	<0.001*
1 hour	74	0.369	0.001*
2 hours	74	0.397	<0.001*
3 hours	74	0.325	0.005*
4 hours	68	0.407	<0.001*
5 hours	40	0.251	0.118
6 hours	10	0.403	0.249
Before extubation	72	0.235	0.047*
Pooled (all time points)	486	0.299	<0.001*

[Table/Fig-7]: Correlations (Spearman ρ): a) Association of demographic and haemodynamic variables with baseline IOP; b) correlation between IOP and ONSD at each time point and pooled.
*Statistically significant (p<0.05). Sex showed no significant association with baseline IOP (Mann-Whitney p-value=0.266).

Variable	ASA I (n=22)	ASA II (n=15)	p-value
Age (years)	36.73±11.20	51.07±6.89	<0.001*
BMI (kg/m²)	25.75±4.67	28.99±3.85	0.033*
Baseline MAP (mmHg)	80.14±6.88	87.33±9.12	0.010*
Baseline IOP, right (mmHg)	13.05±1.84	14.07±2.02	0.120

[Table/Fig-8]: Comparison of baseline characteristics between ASA I and ASA II groups (independent-samples t-test).
*Statistically significant (p<0.05). ASA II patients were significantly older, with higher BMI and baseline MAP, whereas baseline IOP did not differ significantly between the groups.

that IOP increases regardless of whether propofol or sevoflurane is used [23], indicating that positioning rather than anaesthetic choice is the primary determinant. Because the prone-related rise in episcleral and central venous pressure and choroidal congestion is cumulative, IOP climbs progressively rather than plateauing early; in the present study data the 21 mmHg threshold was first crossed between four and five hours, so longer prone duration translates into both higher absolute IOP and a longer period above the threshold associated with reduced perfusion and glaucomatous risk [8], the mechanism by which prolonged surgery becomes a risk factor for ocular complications.

OPP followed the inverse course, falling by about 9% to a nadir of 61-63 mmHg but remaining above 50 mmHg in every patient, with the lowest individual value 53 mmHg. This is reassuring, since an OPP above roughly 50 mmHg is generally considered adequate for optic-nerve-head perfusion [6]; nonetheless, the reduction is meaningful and might be insufficient in patients with compromised circulation or in whom MAP is allowed to fall. The recovery of OPP to baseline by extubation paralleled the recovery of MAP and the resolution of the ONSD changes, suggesting that the perfusion compromise during prone positioning was transient in this healthy cohort.

The ONSD increased significantly during prone positioning and returned to baseline by extubation and was positively correlated with IOP at every early intraoperative time point and in the pooled analysis (Spearman ρ=0.299, p-value <0.001, n=486). Because the optic nerve sheath communicates with the intracranial subarachnoid space, ONSD is a validated non-invasive surrogate for ICP [9,10,12], and its rise here suggests a concurrent, positionally driven increase in ICP.

This parallel behaviour of the ocular and intracranial compartments is the quantitative basis for the eye-brain axis: both respond to the same upstream stimulus- raised episcleral and central venous pressure, impaired orbital and cranial venous drainage, and positive-pressure ventilation- rather than one causing the other. The correlation weakened by extubation, explained by the faster normalisation of ONSD (and, by inference, ICP) than of IOP once the patient was returned supine. The present study findings extend recent prone-position work: Demir U et al., found that prolonged prone positioning raises ONSD in a time-dependent manner and Rahangdale A et al., reported an approximately 21% intraoperative ONSD increase that resolved postoperatively [26,27]. Notably, Adas RH et al., in 59 prone-surgery patients, found that IOP and ONSD both rose but were not correlated [28]; the significant correlation in the present study, derived from repeated bilateral measurements at multiple time points, may reflect the greater temporal resolution of the present protocol and supports IOP and ONSD as parallel markers of the positional venous response. Neurocritical-care studies have produced mixed results, with Roth C et al. reporting no consistent ICP rise in brain-injured patients [17] and Thelandersson A et al., reporting stable ICP in patients with reduced intracranial compliance [18], underlining that the ICP response to prone positioning is patient-specific. Because carbon dioxide itself alters ONSD [24], the present study maintained tight normocapnia so that the observed ONSD changes reflect positioning rather than ventilation; direct invasive ICP measurement, though confirmatory, is neither feasible nor ethical in elective surgical patients.

Among baseline characteristics, only older age (ρ=0.543) and higher baseline MAP (ρ=0.533) were significantly associated with baseline IOP, whereas sex, BMI, heart rate and blood loss were not. The age association was consistent with the age-related decline in aqueous-humour outflow facility [29], and the MAP association suggests that intraoperative blood-pressure management may influence ocular pressure. These findings echo the POVL Study Group's identification of prolonged duration and substantial blood loss as risk factors for ischaemic optic neuropathy [13]. Fluid management is also relevant: Yang XY et al., showed in a randomised trial that a liberal fluid regimen increased both IOP and ONSD compared with a restrictive regimen during prone surgery [30], supporting judicious fluid administration in long prone cases. These observations have practical implications. Patients undergoing prolonged prone surgery (>4 hours), and older patients or those with higher baseline MAP, should be regarded as higher-risk, and periodic intraoperative IOP monitoring together with meticulous neutral head positioning, avoidance of orbital pressure and judicious fluid therapy is advisable. Positioning modifications may help: Grant GP et al., and Carey TW et al., showed that reverse Trendelenburg positioning reduces IOP during prone surgery in a dose-dependent manner [20,21], and attention to venous drainage and operative efficiency may further limit IOP elevation [13,19].

Limitation(s)

This study had several limitations. It was a single-centre study with a moderate sample size. ONSD is an indirect surrogate for ICP rather than a direct measurement. Fewer patients contributed data at the

later time points, reflecting the natural variation in surgical duration. Visual assessment was clinical and limited to 48 hours, so subtle or delayed changes could have been missed. Finally, the cohort was restricted to ASA I-II patients, so the findings may not extend to higher-risk patients with reduced OPP reserve.

CONCLUSION(S)

Prone-position thoracolumbar spine surgery causes a significant, progressive rise in IOP that peaks at five to six hours and does not fully resolve before extubation. ONSD rises in parallel and is positively correlated with IOP throughout surgery, indicating a concurrent, positionally driven increase in ICP- a measurable eye-brain axis response. Despite these changes, OPP was preserved above the critical 50 mmHg threshold and no patient developed POVL, suggesting that prone surgery of this duration was tolerated by ASA I-II patients. Advanced age and higher baseline MAP identified the patients at greatest risk of IOP elevation. Periodic intraoperative IOP monitoring and meticulous neutral positioning are therefore advisable in older patients and in prolonged (>4 hour) prone cases. Larger multicentre studies with formal ophthalmological follow-up are needed to confirm these findings and to evaluate positioning and fluid strategies that may protect vision.

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