



**CLINICAL RESEARCH**

## The effect of robotic surgery on intraocular pressure and optic nerve sheath diameter: a prospective study



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Received 14 May 2019; accepted 8 February 2021

Available online 21 March 2021

### KEYWORDS

Intraocular pressure;  
Optic nerve sheath  
diameter;  
Robotic-assisted  
laparoscopic  
prostatectomy;  
Trendelenburg  
position;  
Ultrasonography

### Abstract

**Background and objectives:** To investigate the effect of the steep Trendelenburg position (35° to 45°) and carbon dioxide (CO2) insufflation on optic nerve sheath diameter (ONSD), intraocular pressure (IOP), and hemodynamic parameters in patients undergoing robot-assisted laparoscopic prostatectomy (RALP), and to evaluate possible correlations between these parameters.

**Methods:** A total of 34 patients were included in this study. ONSD was measured using ultrasonography and IOP was measured using a tonometer at four time points: T1 (5 minutes after intubation in the supine position); T2 (30 minutes after CO2 insufflation); T3 (120 minutes in steep Trendelenburg position); and T4 (in the supine position, after abdominal exsufflation). Systolic and diastolic arterial pressure, heart rate, and end-tidal CO2 (etCO2) were also evaluated.

**Results:** The mean IOP was 12.4 mmHg at T1, 20 mmHg at T2, 21.8 mmHg at T3, and 15.6 mmHg at T4. The mean ONSD was 4.87 mm at T1, 5.21 mm at T2, 5.30 mm at T3, and 5.08 at T4. There was a statistically significant increase and decrease in IOP and ONSD between measurements at T1 and T4, respectively. However, no significant correlation was found between IOP and ONSD. A significant positive correlation was found only between ONSD and diastolic arterial pressure. Mean arterial pressure, heart rate, and etCO2 were not correlated with IOP or ONSD.

**Conclusions:** A significant increase in IOP and ONSD were evident during RALP; however, there was no significant correlation between the two parameters.

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## Introduction

With the advent of the Da Vinci Surgical System (Intuitive Surgical Inc., Sunnyvale, CA, USA), robot-assisted laparoscopic prostatectomy (RALP) has been increasingly used in recent years. This procedure requires different patient positions to facilitate operative exposure. After the initial supine position, the patient is placed in the lithotomy position to create pneumoperitoneum through abdominal carbon dioxide (CO<sub>2</sub>) insufflation, followed by the steep Trendelenburg position (STP). These three positions can cause hemodynamic alterations that may have an effect on intraocular pressure (IOP) and intracranial pressure (ICP).<sup>1,2</sup>

IOP is regulated by aqueous humor production and outflow. While the production of aqueous humor is usually stable, outflow of aqueous humor to the venous system is affected by choroidal blood volume, vitreous humor volume, extraocular muscle tone, and auto-regulation.<sup>3</sup> During surgical procedures, possible blood loss combined with increased IOP can cause a decrease in optic nerve perfusion and, eventually, postoperative vision loss. Avoiding increases in IOP is important in preventing optic nerve damage, particularly in high-risk patients.

The optic nerve sheath is the extension of meninges and subarachnoid dura mater and is dilated through increased ICP.<sup>4</sup> Noninvasive measurements of the dilated optic nerve sheath diameter (ONSD) using ultrasonography have been demonstrated to be directly associated with the ICP, which may increase while patients are in the STP.<sup>5</sup>

In the present study, we investigated the effect of different operational positions for RALP on ONSD and IOP. More specifically, we aimed to evaluate the effect of arterial pressures, heart rate (HR), and end-tidal CO<sub>2</sub> (etCO<sub>2</sub>) on IOP and ONSD. Additionally, we aimed to examine whether there is any correlation between IOP and ONSD.

## Methods

This study included 34 patients aged 50 to 80 years who were scheduled for RALP at Health and Sciences University Bakırkoy Doctor Sadi Konuk Training and Research Hospital between September 2017 and August 2018. Patients with an intracranial lesion, previous intracranial operation, glaucoma, previous eye surgery, cardiac, hepatic, renal, or neurological disease increasing the American Society of Anesthesiologists (ASA) physical status  $\geq$  III, and those who did not provide consent to participate were excluded from the study. All patients included in the study were fully informed about the purposes of the study, and written consent was obtained from each. The study was approved by the ethics committee of Health and Sciences University Bakırkoy Doctor Sadi Konuk Training and Research Hospital Hospital and adhered to the tenets of the Declaration of Helsinki (Decree No. 2017/212, July 31, 2017).

All patients received standard anesthesia induction and maintenance. No preoperative premedication was administered. Standard anesthesia induction was performed using midazolam 3 mg (intravenous [IV]), fentanyl 1.5  $\mu\text{g} \cdot \text{kg}^{-1}$  (IV), propofol 2  $\text{mg} \cdot \text{kg}^{-1}$  (IV), and rocuronium bromide 0.6  $\text{mg} \cdot \text{kg}^{-1}$ . Orotracheal intubation was performed. Before surgery, gastric emptying was achieved using an oroga-

tric tube and free drainage was established. All patients underwent mechanical ventilation in the pressure-regulated volume control mode. The standard mechanical ventilation setting was applied as follows: fraction of inspired oxygen (FiO<sub>2</sub>) 40%; tidal volume 6 to 8  $\text{mL} \cdot \text{kg}^{-1}$ ; respiratory rate 12 to 15 breaths/min (intervention in the presence of etCO<sub>2</sub> > 40 mmHg); inspiration/expiration 1/2; and positive end-expiratory pressure 6 to 7 cmH<sub>2</sub>O. Maintenance of anesthesia was achieved using sevoflurane: minimum alveolar concentration 1 with fresh gas flow (3 L·min<sup>-1</sup>) and remifentanil 0.05–0.5  $\mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ . Surgical muscle relaxation was maintained using repeat doses of rocuronium bromide 0.10 mg/kg<sup>-1</sup>.

Arterial blood pressure was kept constant at a maximal decline of 20% compared to the pre-induction value. All procedures were performed by a single experienced urologist. Before termination of vesicoureteral anastomosis, crystalloid fluid was controlled at a maximum dose of 100 mL.

Patients were placed supine, and a Veress needle was inserted into the infra-umbilical region and CO<sub>2</sub> insufflation was started, with pressure preset to 12 mmHg. Patients were then placed in the STP (35° to 45° horizontally), which was the maximal Trendelenburg angle of the surgical table (Maquet, Maquet Vertrieb und Service, Deutschland, GmbH, Germany).

IOP measurement was performed by one single trained anesthesiologist using a rebound tonometer (iCare PRO, iCare Finland Oy, Helsinki, Finland). This tool is designed to measure IOP according to induction-based rebound. A lightweight tonometer probe is accelerated against the patient's cornea, and the velocity of the rebound is measured using a specially designed coil from which IOP is calculated.<sup>6</sup> After six consecutive measurements, the average of each set is displayed.

All ONSD measurements were performed by one single anesthesiologist using a linear, multi-frequency ultrasound system (GE Vivid model 12L, GE Medical Systems, Madison, WI, USA). Using the visual axis technique, which is the most common method for ONSD measurement, a linear probe was placed on the upper eyelid, and ONSD was measured 3 mm behind the globe.<sup>7,8</sup> The mean of all measurements in four planes from the right eye was calculated.

ONSD and IOP were measured at four time points: T1, 5 minutes after intubation in the supine position; T2, 30 minutes after CO<sub>2</sub> insufflation, when the intra-abdominal pressure reached 12 to 14 mmHg; T3, 120 minutes in STP (35° to 45°), when the intra-abdominal pressure reached 10 mmHg; and T4, in the supine position after desufflation of pneumoperitoneum.

Data including age, weight, and height, HR, systolic blood pressure (SBP), and diastolic blood pressure (DBP), mean arterial pressure (MAP), oxygen saturation (SpO<sub>2</sub>), and etCO<sub>2</sub>. IOP and ONSD measured at four time points, duration of anesthesia, duration of surgery, duration of pneumoperitoneum, and duration of STP were also recorded.

Statistical analysis was performed using SPSS version 22.0 software (IBM Corp., Armonk, NY, USA). Descriptive data are expressed as mean  $\pm$  standard deviation (SD), median (minimum–maximum) values, or number and frequency. The Kolmogorov-Smirnov test was used to determine whether the data were normally distributed. The Wilcoxon test was used to analyze dependent quantitative variables.

**Table 1** Demographic and clinical characteristics of patients.

Variable	Min-Max	Mean $\pm$ SD
Age, year	52.0–75.0	62.6 $\pm$ 5.4
Anesthesia duration, min	195–330	245 $\pm$ 32.0
Surgery duration, min	160–275	205.3 $\pm$ 44.3
CO <sub>2</sub> insufflation, min	135–245	176 $\pm$ 27.0
Duration of Trendelenburg positioning, min	130–225	176 $\pm$ 27.0
Weight, kg	52–115	80 $\pm$ 10.8
Height, cm	160–185	173 $\pm$ 5.6
Body mass index, kg.m <sup>-2</sup>	21–33	29.2 $\pm$ 4.4

SD, standard deviation; CO<sub>2</sub>, carbon dioxide.

Spearman's correlation analysis was performed to examine correlations between the variables. Differences with  $p \leq 0.05$  were considered statistically significant. The sample size was estimated using the free software G\*Power version 3.1.9.2 (Franz Faul, University of Kiel, Kiel, Germany).<sup>9</sup> With a power of 80%, a statistical level of significance of 0.05, and effect size of 0.8, the sample size was calculated to be 34.

## Results

The mean ( $\pm$  SD) age of the participants was  $62.6 \pm 5.4$  years (range, 52–75 years). The mean duration of anesthesia was  $245.1 \pm 32.0$  min, and the mean duration of surgery was  $205.3 \pm 44.0$  min. There were no significant differences in demographic or clinical characteristics among the patients. Baseline demographic and clinical characteristics of the patients are summarized in Table 1.

There was a significant increase in IOP measured at T2 compared with T1, at T3 compared with T1, and at T3 compared with T2 ( $p < 0.05$ ). There was a significant decrease in IOP at pre-extubation compared with T3 ( $p < 0.05$ ) (Table 2).

There was a significant increase in ONSD at T2 compared with T1, and at T3 compared with T2 ( $p < 0.05$ ). However, there was no significant difference in ONSD between T3 and T1, pre-extubation and T1, and the pre-extubation and T3 ( $p > 0.05$ ) (Table 2).

There was a significant decrease in SBP at T2 compared with T1 ( $p < 0.05$ ). However, there was no significant change in the SBP between T3 and T1, and between pre-extubation and T1 ( $p > 0.05$ ). There was a significant increase in SBP at T3 compared with T2 ( $p < 0.05$ ) (Table 2).

There was no significant difference in DBP between T1 and T2, T1 and T3 ( $p > 0.05$ ), and between pre-extubation and T1 ( $p > 0.05$ ). Although a significant increase in DBP was observed at T3 compared with T2 ( $p > 0.05$ ), there was no significant difference in DBP between pre-extubation and T3 ( $p > 0.05$ ) (Table 2).

There was no significant difference in MAP between T1 and T2, T1 and T3, and between pre-extubation and T1 ( $p > 0.05$ ). Although a significant increase in MAP was observed at T3 compared with T2 ( $p > 0.05$ ), there was no significant difference in MAP between pre-extubation and T3 ( $p > 0.05$ ) (Table 2).

There was a significant decrease in HR at T2 compared with T1 ( $p < 0.05$ ), at T3 compared with T1, and at

pre-extubation compared with T1 ( $p < 0.05$ ). A significant increase was observed in HR at T3 compared with pre-extubation ( $p < 0.05$ ). There was no significant difference in HR between T2 and T3 ( $p > 0.05$ ) (Table 2).

There was a significant decrease in etCO<sub>2</sub> at T2 compared with T1, at pre-extubation compared with T1, and at T3 compared with T2 ( $p < 0.05$ ). There was no significant difference in etCO<sub>2</sub> between T1 and T3 ( $p > 0.05$ ). On the other hand, a significant increase was observed in etCO<sub>2</sub> at pre-extubation compared with T3 ( $p < 0.05$ ) (Table 2).

Correlation analysis revealed a significant positive correlation only between ONSD and DBP ( $p < 0.05$ ). There was no significant correlation between IOP and ONSD, SBP, DBP, MAP, HR, and etCO<sub>2</sub> ( $p > 0.05$ ). Finally, there was no significant correlation between ONSD and SBP, MAP, HR, and etCO<sub>2</sub> ( $p > 0.05$ ) (Table 3).

## Discussion

Many studies in the literature have described IOP variation(s) during RALP. The studies reviewed by Ackerman et al. reported a direct association between STP and increased IOP.<sup>10</sup> Only one study reported MAP, which is also a risk factor for posterior ischemic optic neuropathy in addition to IOP.<sup>11</sup> There has been only one study that evaluated etCO<sub>2</sub> and its relationship with IOP. The researchers found that etCO<sub>2</sub> and surgical duration were significant predictors of increased IOP while patients were in STP.<sup>12</sup>

In the present study, we aimed to evaluate changes in both IOP and ONSD during RALP, and to examine whether there was any correlation between IOP and ONSD. Furthermore, we aimed to investigate the effect of SBP and DBP, HR, and etCO<sub>2</sub> on IOP and ONSD.

The mean IOP was found to be 20 mmHg after CO<sub>2</sub> insufflation and 21.8 mmHg while in STP. The Trendelenburg position is associated with increased intra-abdominal pressure and partial pressure of CO<sub>2</sub>, leading to a simultaneous increase in cerebral blood flow, ICP, and IOP.<sup>13</sup> Molloy et al. reported that 32.5% of patients who underwent laparoscopic surgery experienced increased IOP to  $> 40$  mmHg.<sup>14</sup> In contrast, the highest IOP recorded in our study cohort was 32 mmHg.

Furthermore, we observed a significant increase in ONSD (by 0.34 mm) following CO<sub>2</sub> insufflation and by 0.34 mm in STP compared with baseline during RALP. However, Blecha et al. reported no significant increase in ONSD compared with baseline (maximal increase of 3.4% [0.2 mm]).<sup>15</sup> In another study, a 12.5% (0.6 mm) increase in ONSD was evident following CO<sub>2</sub> insufflation and in the STP in 20 patients undergoing RALP.<sup>16</sup> The mean increase in ONSD was higher in our study compared with the study by Blecha et al., but lower than the latter study. The discrepancy in results may be attributed to the fact that variations in ONSD primarily depend on demographic characteristics of the patients and body position.

Whiteley et al. demonstrated a direct correlation between increased ONSD and MAP, which was not found in our study.<sup>17</sup> We found significant positive correlation between ONSD and DBP, which may have resulted from decreasing DBP lowering cerebral perfusion pressure. Although both IOP and ONSD increased in STP, we did not

**Table 2** Measurements of IOP, ONSD and hemodynamic parameters at prespecified time points.

	Mean $\pm$ SD (Min-Max)	$P^a$	$P^b$
<b>IOP, mmHg</b>			
T1	12,4 $\pm$ 3,1 (3,2–17,3)		
T2	20,0 $\pm$ 4,4 (11,9–30,7)	0,000 <sup>c</sup>	
T3	21,8 $\pm$ 4,7 (14,3–32)	0,000 <sup>c</sup>	0,003 <sup>c</sup>
T4	15,6 $\pm$ 5,0 (6,6–30,8)	0,001 <sup>c</sup>	0,000 <sup>c</sup>
<b>ONSD, mm</b>			
T1	4,87 $\pm$ 0,34 (4,3–5,5)		
T2	5,21 $\pm$ 0,31 (4,5–5,9)	0,000 <sup>c</sup>	
T3	5,30 $\pm$ 0,33 (4,50–6,1)	0,000 <sup>c</sup>	0,000 <sup>c</sup>
T4	5,08 $\pm$ 0,32 (4,30–5,7)	0,000 <sup>c</sup>	0,000 <sup>c</sup>
<b>SBP, mmHg</b>			
T1	111,0 $\pm$ 21,2 (71–172)		
T2	103,6 $\pm$ 14,3 (76–126)	0,042 <sup>c</sup>	
T3	105,6 $\pm$ 13,1 (84–132)	0,105 <sup>c</sup>	0,259 <sup>c</sup>
T4	107,5 $\pm$ 27,0 (14,7–162)	0,829 <sup>c</sup>	0,459 <sup>c</sup>
<b>DBP, mmHg</b>			
T1	66,3 $\pm$ 15,3 (35–99)		
T2	70,6 $\pm$ 9,3 (54–85)	0,060 <sup>c</sup>	
T3	71,3 $\pm$ 9,3 (55–91)	0,092 <sup>c</sup>	0,523 <sup>c</sup>
T4	66,1 $\pm$ 10,8 (42–85)	0,696 <sup>c</sup>	0,050 <sup>c</sup>
<b>MAP, mmHg</b>			
T1	81,3 $\pm$ 18,3 (54–134)		
T2	81,8 $\pm$ 11,5 (60–101)	0,830 <sup>c</sup>	
T3	83,3 $\pm$ 11,7 (61–105)	0,549 <sup>c</sup>	0,115 <sup>c</sup>
T4	81,2 $\pm$ 15,1 (51–115)	0,945 <sup>c</sup>	0,514 <sup>c</sup>
<b>HR, bpm</b>			
T1	68,2 $\pm$ 9,7 (48–84)		
T2	53,8 $\pm$ 7,9 (44–79)	0,000 <sup>c</sup>	
T3	54,4 $\pm$ 6,4 (43–74)	0,000 <sup>c</sup>	0,589 <sup>c</sup>
T4	62,3 $\pm$ 10,5 (40–80)	0,027 <sup>c</sup>	0,001 <sup>c</sup>
<b>etCO<sub>2</sub>, mmHg</b>			
T1	34,7 $\pm$ 3,8 (26–41)		
T2	32,7 $\pm$ 4,6 (24–40)	0,002 <sup>c</sup>	
T3	32,0 $\pm$ 4,7 (25–41)	0,000 <sup>c</sup>	0,044 <sup>c</sup>
T4	34,7 $\pm$ 5,2 (25–43)	0,749 <sup>c</sup>	0,001 <sup>c</sup>

T1, at 5 min, post-intubation; T2, at 30 min post-intubation; T3, at 120 min, post-intubation; T4, desufflation with supine position; IOP, intraocular pressure; ONSD, optic nerve sheath diameter; SD, standard deviation; SBP, systolic blood pressure; DBP, diastolic blood pressure; MAP, mean arterial pressure; HR, heart rate; etCO<sub>2</sub>, end-tidal carbon dioxide.

<sup>a</sup> *p* value, compared to T1.

<sup>b</sup> *p* value, compared to previous measurement.

<sup>c</sup> Wilcoxon test.

**Table 3** Spearman's rank order correlation analysis of IOP and ONSD measurement.

Variable	ONSD (mm)	SBP (mmHg)	DBP (mmHg)	MAP (mmHg)	HR (bpm)	etCO <sub>2</sub> , mmHg
<b>IOP</b>						
r	0,178	0,036	0,073	0,164	0,117	-0,149
p	0,322	0,841	0,685	0,362	0,515	0,409
<b>ONSD</b>						
r		0,262	0,408	0,321	-0,112	0,031
p		0,141	0,018	0,069	0,536	0,865

IOP, intraocular pressure; ONSD, optic nerve sheath diameter; SBP, systolic blood pressure; DBP, diastolic blood pressure; MAP, mean arterial pressure; HR, heart rate; etCO<sub>2</sub>, end-tidal carbon dioxide.

\* Spearman correlation analysis.

*r*, Spearman's rho correlation coefficient.

*p*<0,05 is significant.

observe any significant correlation between the two parameters. This could be explained by the different mechanisms that increase IOP and ONSD. Moreover, we did not find any correlation between IOP and SBP, and DBP, HR, or etCO<sub>2</sub>.

In the present study, we used the B-scan technique, which is the most commonly used method to measure ONSD. Some researchers have suggested the use of the Standardized A-scan technique, a blooming effect-free ultrasound method for ONSD measurement.<sup>18,19</sup> Additionally, performing a "30-degree test", which enables discrimination between an increase in ONSD caused by raised ICP, and those associated with other diseases, such as optic neuritis or meningioma, is possible by using A-scan. However, the A-scan measurement technique is not used generally in critical care ultrasonography; therefore, we preferred the B-scan technique, which is more familiar to anesthesiologists and used in the vast majority of previous studies.

There were some limitations to the present study, the first of which were its small sample size and second, it was not a randomized controlled trial. Moreover, we were unable to measure IOP and ONSD before intubation and after extubation due to limited capabilities of the facility. Therefore, we did not evaluate the exact effect of anesthesia and intubation/extubation on the outcomes. Nevertheless, our results demonstrated that the STP with pneumoperitoneum was associated with a significant increase in IOP and ONSD during RALP; however, there were no significant correlations between IOP and ONSD. We also demonstrated a positive correlation between DBP and ONSD. As such, in the presence of increased DBP, operators should be aware of the risk for increased ICP. Nevertheless, further studies are needed to validate these findings.

## Conclusions

The STP with pneumoperitoneum was associated with a significant increase in IOP and ONSD during RALP. Ultrasonographic measurement of ONSD and measurement of IOP is a simple, cost-effective, and reproducible method and can be useful for anesthesiologists in high-risk patients to evaluate IOP and ICP in the operating room. Based on these findings, we suggest that intraoperative measurement of IOP, and ONSD can be useful in patients at high-risk for increased IOP during laparoscopic surgery requiring Trendelenburg positioning.

## Conflict of interests

The authors declare no conflicts interest.

## Acknowledgements

Statistical analysis were performed by Prin Statistical Data Analysis Service. Proof reading were supported by Gokhan Demirayak.

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