

COMPARISON OF THE IMPACT OF THE ANESTHESIA INDUCTION USING THIOPENTAL AND PROPOFOL ON CARDIAC FUNCTION FOR NON-CARDIAC SURGERY

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BACKGROUND: Thiopental and propofol have been widely used for general anesthesia induction, but their impacts on cardiac function have not been well described. A recent study speculated that anesthesia induction using propofol 2 mg/kg transiently reduced left ventricular (LV) contraction by analyzing tissue Doppler-derived imaging (TDI) during induction phase. The purpose of this study was to analyze and to compare the impacts of propofol- and thiopental-induction on LV function.

METHODS: Twenty-four female patients with normal LV function undergoing non-cardiac surgery were randomly administered intravenous bolus thiopental (5 mg/kg, Thiopental-group, n = 12) or propofol (2 mg/kg, Propofol-group, n = 12) for anesthesia-induction. TDI of septal mitral annular velocity during systole (S'), early diastole (e') and atrial contraction (a') were determined by transthoracic echocardiography before and 1, 3, and 5 minutes after thiopental/propofol administration (T0, T1, T2, and T3, respectively).

RESULTS: The bispectral index and systolic blood pressure declined significantly during anesthesia induction in both groups, however, more depressed in Thiopental-group compared with those in Propofol-group at T2 and T3 (all, $p < 0.05$). Among TDI two parameters demonstrated a significant inter-group difference: the S' in propofol was lower than that in Thiopental-group at T3 ($p = 0.002$), and a' velocities were persistently lower in Propofol-group, compared with same time values in Thiopental-group (T1, T2, and T3: $p = 0.025, 0.007, \text{ and } 0.009$, respectively).

CONCLUSION: Anesthesia induction using propofol revealed a more persistent and profound decline of LV and atrial contraction than that using thiopental. Further studies are needed to understand the clinical implication.

KEY WORDS: Intraoperative · Echocardiography · Doppler · Thiopental · Propofol.

INTRODUCTION

Evaluating and avoiding perioperative risk factors compromising myocardial performance is important to enhance post-operative outcomes and reduce perioperative morbidity and mortality in adult non-cardiac surgery.¹⁻³⁾ For preoperative risk evaluation, traditionally the revised cardiac risk index⁴⁾ and American College of Cardiology and American Heart Association guidelines⁵⁾ are applied. Unfortunately, the traditional predictive tools do not integrate purely anesthesia-related factors for routine surgeries, presumably in the belief that the so-

phistication of modern anesthesia practice renders it devoid of relevant risks in such cases.

Anesthesia induction is commonly initiated by an intravenous (IV) administration of hypnotics, such as thiopental or propofol, for abruptly bringing wakeful patients into unresponsiveness to strong adrenergic stimuli, including tracheal intubation and surgical procedures. However, hemodynamic instability in various degrees is commonly associated during the anesthesia induction phase using these hypnotics: 9% of patients experienced severe hypotension during and after anes-

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thetia induction in clinical practice.⁶⁾ As hypotension often occurs after IV thiopental and propofol, in patients with left ventricular (LV) dysfunction, etomidate is sometimes preferred as an alternative.⁷⁾

Unfortunately, even though LV systolic function is one risk factor for anesthesia induction events, and it is well known that induction has significant hemodynamic and cardiac effects, the kind and magnitude to the effects on LV myocardium function of anesthesia induction, including the particular agents used, have not been thoroughly studied. This may be due to the routineness and apparent harmlessness of the effects as observed casually and also a lack of monitoring tools and procedures.

Doppler tissue imaging (DTI) non-invasively assesses myocardial tissue motion to evaluate both systolic and diastolic myocardial function with the advantage of less load-dependence than conventional flow pulsed wave (PW) Doppler techniques.⁸⁾ Intraoperative tissue Doppler monitoring during anesthesia induction can be helpful to provide immediate insight into hemodynamic changes, giving an idea of loading conditions as well as myocardial performance during this vulnerable phase. However, only a few previous studies have used DTI during anesthesia induction and evaluated propofol's impact on cardiac function.^{9,10)}

Considering that thiopental and propofol are the two most popular and widely used IV anesthesia induction agents, we sought to analyze and to compare the impacts of clinical dosage of thiopental and propofol for anesthesia induction on cardiac function by using intraoperative transthoracic DTI.

METHODS

STUDY POPULATION

After Institutional Review Board approval, we prospectively enrolled 24 adult patients who were scheduled for elective non-cardiac surgery and had low-risk as indicated by a revised cardiac risk index of 0,⁴⁾ normal sinus rhythm, normal LV function (LV ejection fraction $\geq 60\%$ and mitral septal annular $e' \geq 8$ cm/s),¹¹⁾ no regional wall motion abnormality, and no structural heart diseases. Patients with any of the following conditions were excluded in the operation room: unfavorable airway or facemask fit, intractable coughing, hiccups, or hypotension [mean blood pressure (BP) < 60 mmHg] requiring IV positive inotrope or vasoconstrictors during the study period.

ANESTHESIA INDUCTION

Pre-medication accorded with our routine practice: 10 mL/kg/hour of Ringer's lactate solution, a balanced crystalloid solution (Plasma Solution-A™, CJ Bio and Pharmaceutical, Seoul, Korea), 0.2 mg of glycopyrrolate, and 0.03 mg/kg of midazolam. Once in the operation room, we started to monitor electrocardiography, non-invasive BP, heart rate (HR), pulse oximeter oxygen saturation, and the bispectral index (BIS). To

begin anesthesia induction, patients spontaneously inhaled oxygen (8 L/min) through a transparent facemask and a breathing circuit with a reservoir bag. Either bolus thiopental (5.0 mg/kg) (Pentotal™ sodium, JW Pharmaceutical, Seoul, Korea; Thiopental group) or propofol (2.0 mg/kg) (Pofol™, Dongkuk Pharmaceutical, Seoul, Korea; Propofol group) was administered intravenously for 10 seconds, after which assisted and controlled ventilation followed. After 5 minutes, complete induction was confirmed via a lack of train-of-four response and followed by tracheal intubation for maintenance anesthesia.

INTRAOPERATIVE TRANSTHORACIC ECHOCARDIOGRAPHY

After positioning the patient supine on the operation table, serial transthoracic echocardiography (TTE), two-dimensional imaging of the apical 4-chamber view and PW Doppler imaging of mitral inflow, was performed with a portable GE Vivid Q platform (General Electric, Milwaukee, WI, USA), before (T0) and 1, 3, and 5 minutes after the thiopental bolus injection (T1, T2, and T3, respectively) along with hemodynamic recordings at the same times (BP, HR, and BIS). DTI was recorded at the septal mitral annulus from the apical 4-chamber view to determine longitudinal annular velocities with a sweep of 66.7 mm/s. Each set of images required less than 30 seconds.

The LV ejection fraction was determined from the 2-dimensional apical 4-chamber imaging by the modified Simpson's method. The early diastolic velocity (E), late atrial filling velocity (A), and a deceleration time of E were assessed from the mitral inflow PW Doppler imaging. Tissue Doppler-derived indices of systolic (S'), early diastolic (e'), and late diastolic (a') velocities were measured from mitral septal annular DTI from an average 3 beats.

STATISTICAL ANALYSIS

Sample size estimation was based on the previous prospective studies: Gauss et al.¹²⁾ (n = 10 for propofol; n = 10 for etomidate; n = 10 for thiopental), Mulier et al.¹³⁾ (n = 10 for each group), and Wodey et al.¹⁴⁾ (n = 10 for thiopental; n = 10 for propofol), taking potential drop-outs during the measurement (20%) due to insufficient echocardiographic window into account.

Demographic data were presented as median values (interquartile range) or number of patients. Continuous variables were expressed as mean (standard deviation) or median (interquartile range). For comparison of demographic data between groups, the Mann-Whitney test was used. For statistical comparisons of serial changes within a group, the Friedman test with multiple comparisons was applied. For comparison of cardiovascular data between thiopental and propofol group, the Wilcoxon-Mann-Whitney test was used. Statistical analysis was performed using dBSTAT 5.0 for Windows (dBSTAT, Seoul, Korea). A p -value < 0.05 (two-sided) was considered as statisti-

cally significant.

RESULTS

A total of 24 consecutive patients were enrolled (12 for thiopental-, and 12 for propofol-based anesthesia induction). The induction phase was uneventful and without exclusion. The clinical characteristics are compared in Table 1. No significant difference was noted between the two groups.

The serial hemodynamic and echocardiographic changes in thiopental and propofol group are compared in Table 2.

Within the thiopental group, the BIS declined significantly after thiopental injection (T1, T2 vs. T0, $p < 0.0001$ and $p = 0.0006$, respectively); and then it recovered at T3 (T0, T1 vs. T3, $p = 0.106$ and 0.004 , respectively). Similar pattern was observed in mean BP (T1, T2, T3 vs. T0: 78.0, 85.5, 85.5 vs. 97.0 mmHg, $p = 0.0006$, 1.000, and 0.239, respectively; T1 vs. T3, $p = 0.026$). The LV ejection fraction decreased significantly from baseline, at T1 and T2 ($p = 0.005$ and 0.026 , respectively), and recovered at T3 ($p = 0.928$). Even though the LV ejection fraction reduced temporarily, the quartile range was within normal systolic function [T1: 62.0% (56.1–65.2), T2: 63.3% (58.3–66.4)]. The median value of mitral inflow E, and deceleration time of E decreased from the baseline (T1, T2, T3 vs. T0: 69.5, 66.7, 68.2 vs. 84.5 cm/s, $p = 0.056$, 0.001, and 0.004; 180, 179, 183 vs. 160 ms, $p = 0.034$, 0.003, and 0.011, respectively), but no significant changes on late diastolic atrial contraction velocity ($p = 0.332$). Tissue Doppler-derived S' velocity declined significantly at T1 and T2 compared with T0 ($p = 0.003$ and 0.001), however, it also slightly recovered at T3 ($p = 0.072$). Early diastolic annular e' reduced significantly compared with T0 (all, $p < 0.05$), but no changes in the late diastolic a' velocities which were corresponding to the atrial contraction ($p = 0.140$).

Within the propofol group, the BIS and systolic BP, declined from the baseline persistently throughout the induction phase (all, $p < 0.05$). The mitral inflow E decreased significantly at T1, T2, and T3 compared with T0 ($p = 0.002$, 0.001, and 0.001, respectively), combined with the reduced corresponding A velocities ($p = 0.191$, 0.008, and < 0.0001 , respectively). Tissue Doppler-derived S' velocity declined persistently at T1, T2, and T3 compared with T0 ($p = 0.001$, 0.004, and 0.001, respectively). The mitral septal annular early diastolic e' velocities also decreased at T1, T2, and T3 compared with T0 ($p = 0.010$, 0.079, and 0.006, respectively), with the reduced corresponding a' velocities ($p = 0.079$, 0.004, and < 0.0001 , respectively).

COMPARISON BETWEEN THIOPENTAL- AND PROPOFOL-BASED ANESTHESIA INDUCTIONS

Comparison of the impact on cardiac function between thiopental and propofol group is presented in Table 2. The hemodynamic comparison between groups is illustrated in Fig. 1. Both groups showed declines of BIS and systolic BP after IV anesthesia injection (T0 vs. T1, all, $p < 0.05$); however, the decline and recovery pattern of BIS and systolic BP showed significant inter-group difference. In BIS, the baseline value was similar between the two ($p = 0.553$), so as the first drop at T1 ($p = 0.069$), however, after that thiopental group showed more recovered and higher level of BIS compared with those of propofol [T2: 57.5 (52.0–64.8) vs. 44.0 (37.5–50.8), $p = 0.009$; T3: 63.5 (62.0–66.5) vs. 45.0 (41.3–52.0), $p < 0.001$] (Fig. 1A). A related pattern was observed in systolic BP: the baseline and initial decline were similar between the two groups ($p = 0.193$ and 0.214); however, after that thiopental group tended to recover and significantly higher level of systolic BP compared with those of propofol [T2: 121.0 (111.3–136.8) vs.

Table 1. Clinical characteristics

	Thiopental (n = 12)	Propofol (n = 12)
Age (years)	42 (40–46)	42 (38–47)
Female/male	12/0	12/0
Weight (kg)	53.2 (51.1–55.3)	53.5 (52.3–61.7)
BSA (m ²)	1.54 (1.50–1.57)	1.57 (1.51–1.65)
Hypertension on medication	0	0
Diabetes mellitus	0	0
Chronic renal failure	0	0
History of coronary heart disease	0	0
NYHA functional class > I	0	0
Diagnosis for operation		
Breast cancer	8	5
Ovarian neoplasm	1	3
Thyroid cancer	3	1
Uterine	0	3

Data are expressed as median (interquartile range) or n. Thiopental: patients administered bolus thiopental 5.0 mg/kg, Propofol: patients administered bolus propofol 2.0 mg/kg. BSA: body surface area by Mosteller equation, NYHA: New York Heart Association

Table 2. Cardiovascular data in Thiopental and Propofol groups

	Group	T0	T1	T2	T3	<i>p</i> within group
BIS (0-100)	Thiopental	95.5 (92.3-97.8)	40.5 (33.3-51.3)* [†]	57.5 (52.0-64.8)* [‡]	63.5 (62.0-66.5) ^{†‡}	< 0.0001
	Propofol	95.5 (95.0-98.0)	34.5 (20.3-40.5)*	44.0 (37.5-50.8)* [‡]	45.0 (41.3-52.0)* [‡]	< 0.0001
SBP (mmHg)	Thiopental	140.0 (125.3-149.3)	110.5 (102.3-116.0)*	121.0 (111.3-136.8) [‡]	117.0 (106.0-136.8)* [‡]	0.0001
	Propofol	133.0 (120.3-140.0)	107.0 (92.3-111.0)*	106.5 (95.3-115.0)* [‡]	99.0 (91.0-110.0)* [‡]	< 0.0001
DBP (mmHg)	Thiopental	73.0 (60.5-78.5)	58.0 (55.3-69.5)*	61.0 (60.0-79.3) [‡]	67.0 (58.0-72.8)	0.0078
	Propofol	71.0 (61.5-84.5)	58.5 (53.3-68.5)	58.0 (55.0-61.5)* [‡]	57.0 (49.0-62.0)*	0.0001
HR (/min)	Thiopental	68.5 (63.5-78.0)	76 (66.0-80.8)	77.0 (66.3-80.3)	74.5 (66.3-80.8)	0.9796
	Propofol	73.5 (64.3-80.0)	69.0 (64.5-77.5)	65.0 (63.0-75.0)	63.0 (61.0-76.0)	0.1466
LV EF (%)	Thiopental	65.4 (63.1-70.1)	62.0 (56.1-65.2)	63.3 (58.3-66.4)*	64.2 (62.9-66.7)	0.0037
	Propofol	66.1 (65.1-68.3)	65.8 (63.8-66.9)	65.1 (63.0-68.1)	64.5 (62.3-69.8)	0.0872
E (cm/s)	Thiopental	84.5 (78.9-91.6)	69.5 (62.2-79.1)	66.7 (62.3-77.4)*	68.2 (62.3-81.0)*	0.0005
	Propofol	83.7 (78.8-95.3)	69.8 (60.2-81.8)*	69.8 (61.5-84.0)*	70.3 (65.5-79.0)*	0.0001
A (cm/s)	Thiopental	46.8 (42.8-59.3)	49.2 (39.1-58.0)	43.9 (39.0-54.9)	42.4 (38.8-54.5)	0.3342
	Propofol	57.4 (46.2-63.7)	44.8 (36.8-52.5)	40.4 (36.5-45.8)*	40.5 (36.0-42.8)*	0.0002
DT (ms)	Thiopental	159.5 (153.8-162.3)	180.0 (160.8-197.0)*	178.5 (166.9-189.4)*	183.0 (161.0-186.0)*	0.0015
	Propofol	167.0 (157.9-180.4)	179.3 (167.6-182.8)	177.9 (161.1-185.1)	173.0 (164.5-189.7)	0.0707
S' (cm/s)	Thiopental	7.66 (7.23-7.95)	6.28 (5.38-7.01)*	6.35 (5.60-7.03)*	6.50 (6.41-6.93) [‡]	0.0005
	Propofol	7.63 (7.12-8.10)	5.95 (4.99-6.21)*	5.70 (5.01-6.45)*	5.65 (4.80-5.90)* [‡]	0.0001
e' (cm/s)	Thiopental	12.04 (10.88-12.91)	10.67 (9.21-11.19)*	10.64 (8.88-11.65)*	10.69 (9.29-11.55)*	0.0006
	Propofol	11.30 (10.11-12.14)	10.03 (9.08-10.67)*	9.98 (9.40-11.23)	9.80 (9.00-10.95)*	0.0029
a' (cm/s)	Thiopental	8.02 (6.95-8.56)	7.44 (6.51-7.73) [‡]	7.01 (6.41-7.77) [‡]	6.79 (6.40-8.13) [‡]	0.1402
	Propofol	8.00 (7.45-8.53)	6.39 (5.50-6.68) [‡]	5.53 (5.05-6.33)* [‡]	5.40 (4.90-6.15)* [‡]	0.0001
E/e'	Thiopental	7.08 (6.02-8.30)	7.02 (5.47-8.09)	6.92 (5.60-7.97)	7.28 (5.76-7.83)	0.1072
	Propofol	7.96 (7.24-8.34)	7.37 (6.29-8.17)	6.89 (6.24-7.71)*	7.14 (6.29-8.22)	0.0161

Baseline and follow-up data are expressed as median (interquartile range). Serial parameters before and 1, 3, and 5 minutes after intravenous bolus thiopental or propofol (T0, T1, T2, and T3, respectively). Thiopental: patients administered bolus thiopental 5.0 mg/kg, Propofol: patients administered bolus propofol 2.0 mg/kg. **p* < 0.05: vs. T0 within the group, [†]*p* < 0.05: T1 vs. T3 within the group by Friedman's test with multiple comparisons, [‡]*p* < 0.05: compared with same-time value in the other group by the Wilcoxon-Mann-Whitney test. BIS: bispectral index, SBP: systolic blood pressure, DBP: diastolic blood pressure, HR: heart rate, LV: left ventricular, EF: ejection fraction, E: mitral inflow peak early filling velocity, A: mitral inflow peak late filling velocity, DT: deceleration time of E velocity, S': mitral septal annular systolic myocardial velocity, e': mitral septal annular early diastolic myocardial relaxation velocity, a': mitral septal annular velocity associated with atrial contraction

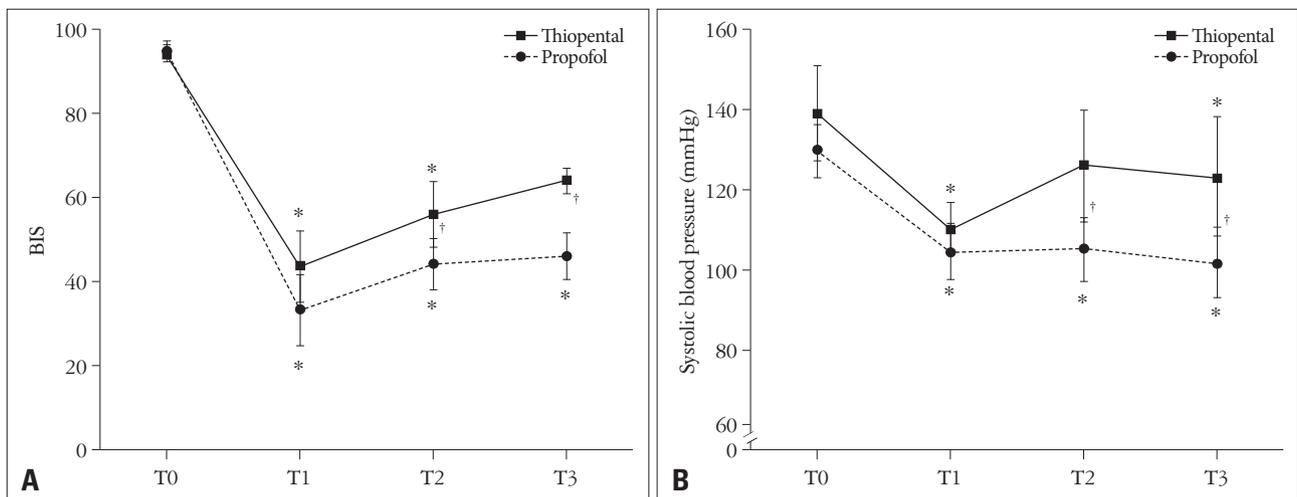


Fig. 1. Comparison of serial hemodynamic changes between Thiopental and Propofol group. Serial bispectral index (BIS) (A), and systolic blood pressure (B) in the thiopental and propofol group, before and 1, 3, and 5 minutes after injection of anesthesia (T0, T1, T2, and T3, respectively). Thiopental: patients administered bolus thiopental 5.0 mg/kg, Propofol: patients administered bolus propofol 2.0 mg/kg. Values were median (interquartile). **p* < 0.05 vs. T0 within the group by Friedman's test with multiple comparisons, [†]*p* < 0.05, between the group by the Wilcoxon-Mann-Whitney test. A significant group difference is noted 3 to 5 minutes after anesthesia injection.

106.5 (95.3–115.0) mmHg, $p = 0.014$; T3: 117.0 (106.0–136.8) vs. 99.0 (91.0–110.0) mmHg, $p = 0.016$] (Fig. 1B).

Among the tissue Doppler-derived indices, significant inter-group difference was found in S' and a' velocities (Fig. 2). In S' velocity, the baseline median value was the almost identical, and initial decline at T1, and T2 did not show revealed significant inter-group differences; however, at T3, propofol group revealed significantly lower S' velocity than the thiopental group [6.50 (6.14–6.93) vs. 5.65 (4.80–5.90) cm/s, $p = 0.002$] (Fig. 2A). In a' velocity, the baseline median was similar; however, there were persistent inter-group differences at T1, T2, and T3 ($p = 0.025, 0.007, \text{ and } 0.009$, respectively), the propofol group showed significantly depressed atrial contraction after anesthesia, which was not observed in the thiopental group (Fig. 2B).

DISCUSSION

The present study showed a clinical dosage of thiopental or propofol for anesthesia induction commonly revealed a significant and immediate decline of S' and e'. Considering its result with the significant decline of a', these results corresponded well to those of the previous studies showing propofol-induced declines of S', e', and a' in TTE during anesthesia induction.^{9,10} The present study found a significant difference in drug-specific depressive impacts on cardiac function—propofol-group revealed a more profound decline of S' at T3 ($p = 0.002$), and a' velocities at T1, T2, and T3, than thiopental-based one ($p = 0.025, 0.007, \text{ and } 0.009$, respectively).

During this short and dynamic anesthesia induction period, Doppler monitoring was successful in 22 of 24 patients (92%). In 2 cases (8%), the velocity measurement was limited by premature beats or tachycardia after thiopental injection. There-

fore, the feasibility of DTI monitoring during the induction anesthesia was 100% in propofol and 83% in the thiopental group.

Hemodynamic effects of anesthesia induction using thiopental or propofol have been reported in several clinical studies^{6,12-15} for various ages or American Society of Anesthesiologists' (ASA) statuses.¹⁶ Reich et al.⁶ reported from retrospective anesthesia records that a clinically significant hypotension [mean arterial pressure (MAP) < 60 mmHg; or MAP decrease > 40% and < 70 mmHg] within 5 minutes after thiopental injection was 0% in ASA I-II, and 2.3% in ASA III-IV; after propofol injection was 3.0% in ASA I-II, and 4.8% in ASA III-IV. In the present study, all the subjects were carefully selected as ASA I, therefore, either group has any clinical consequential hypotensive event during the study period.

Sørensen et al.¹⁵ reported that at a clinical dosage of thiopental and propofol, there was a shorter onset time with thiopental, and deeper BIS and mean arterial BP declines with propofol. That pattern was similar to our clinical observation of more persistent declines in BP and BIS with propofol compared with thiopental. On the other hand, changes of HR after thiopental or propofol injections have been reported inconsistently. With thiopental, it has been reported mostly as increases^{12,13} but with no change at lower doses or in infants^{13,14} and decreases in the elderly population.¹⁵ With propofol no changes have been reported.¹⁷ In this study, neither thiopental nor propofol revealed significant changes during the induction period, although two cases of abrupt tachyarrhythmia occurred only in the thiopental group.

Cardiovascular depressive effects of thiopental or propofol have been explained from direct myocardial effects^{18,19} as well

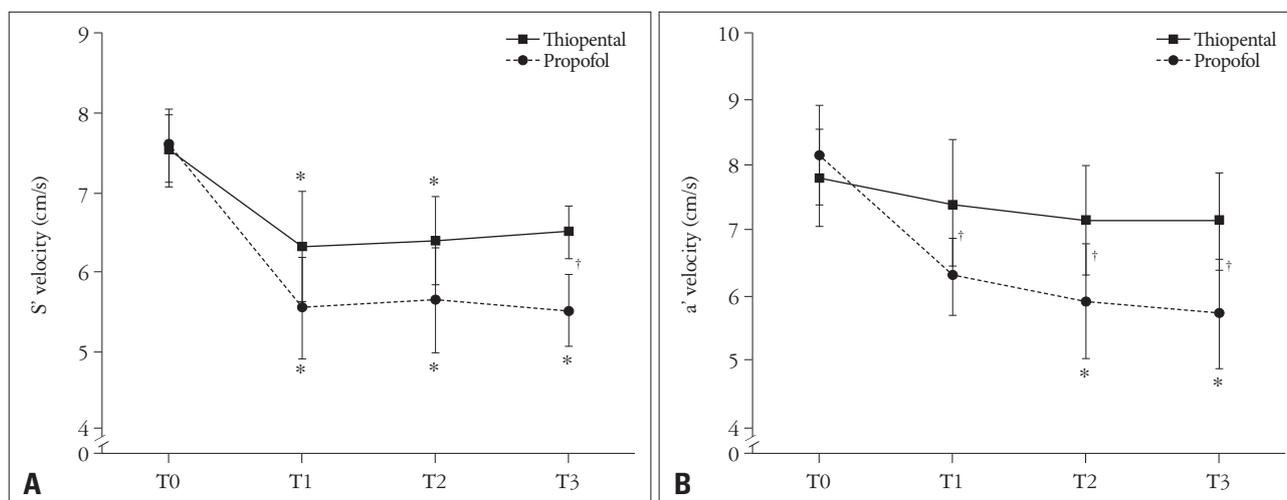


Fig. 2. Comparison of tissue Doppler-derived indices of septal mitral antrical velocity during systole (S') and late diastole atrial contraction (a'). Serial S' velocities (A), and a' velocities (B) in Thiopental and Propofol group, before and 1, 3, and 5 minutes after injection of anesthesia (T0, T1, T2, and T3, respectively). Thiopental: patients administered bolus thiopental 5.0 mg/kg, Propofol: patients administered bolus propofol 2.0 mg/kg. Values were median (interquartile). * $p < 0.05$, vs. T0 within the group by Friedman's test with multiple comparisons, † $p < 0.05$, between the group. Persistent decline of S' (compared with T0) is noted at T3 in the propofol group, which is recovered in the thiopental. At T3, the S' is lower in propofol than in thiopental ($p = 0.002$). Declining of a' velocities from T0 is significant throughout the study period in the propofol group; however, those changes are not observed in the thiopental group ($p < 0.05$).

as indirect effects on the neuronal system.²⁰ Dose-dependent myocardial depression has been reported by many experimental studies.¹⁸⁾⁽¹⁹⁾⁽²¹⁾ However, only a few clinical studies could be found regarding comparison of the myocardial depressive effect of thiopental versus propofol anesthesia induction.¹²⁻¹⁴ Gauss et al.¹² reported that fractional shortening dropped about 14% after thiopental induction, versus no change after propofol. Mulier et al.¹³ concluded that the cardio-depressant effects of propofol are more pronounced and more prolonged than those of equipotent doses of thiopental when given as a single bolus, by measuring LV volume using intraoperative transesophageal echocardiography. Wodey et al.¹⁴ demonstrated that myocardial contractility decreased significantly 5 minutes after induction with both thiopental and propofol in infants, measuring LV diameters using TTE. These previous clinical studies¹²⁻¹⁴ provided reasonable concepts of myocardial depressive effects of induction anesthesia. However, their evaluation tool of M-mode dimension or volume, and fractional shortening would not sufficiently monitor (give an idea from beat to beat) live in this short induction period. In this study, we adopted live monitoring of intraoperative transthoracic DTI to evaluate the impact of IV anesthesia induction on myocardial performance. Intraoperative transthoracic DTI evaluation seems promising to provide immediate insight into hemodynamic changes as well as myocardial performance.

Regarding systolic function, in this study, the LV ejection fraction was temporarily decreased at T1 and T2 compared with T0, in thiopental; there were no significant changes in propofol. These results are similar to the aforementioned study by Gauss et al.¹² on fractional shortening.¹² However, even though there was a statistically significant decrease of LV ejection in the thiopental group, the clinical significance of the roughly 5% decrease may in itself be low. The present study is somewhat unique in directly monitoring S' by DTI to compare systolic function between two commonly used induction anesthesia. In both anesthesia inductions, S' significantly decreased from T0 within a group; however, at T3, the thiopental group tended to recover. Therefore, the between group difference was significant [6.50 (6.14–6.93) vs. 5.65 (4.80–5.90) cm/s, $p = 0.002$]. It may support the previous findings¹⁵ that with a clinical dosage of thiopental and propofol, thiopental has a shorter onset time and shorter depressive effect on myocardium during the induction period. The clinical impact of such a degree of decline of S' has not been tested.

A more interesting finding was between-group differences on diastolic function. The early relaxation velocities (e') declined consistently in both groups; the impact on atrial contraction (a') was significantly different between groups. There was a significant decrease of a' with propofol but not with thiopental ($p < 0.05$). Therefore, we can speculate that thiopental may compromise atrial contraction less. Further studies are needed to discern the precise clinical impact.

This study has several limitations. First, it reflects only a

single center with a relatively small and strictly female population. Non-parametric analysis would overcome the small sample size. Performing studies in gynecologic surgery is limited to the female population, further study could include either gender or higher-risk patients. Second, for reasons of practicality, it did not measure sophisticated indices of preload or afterload. To include all the desired parameters may require more time, and turn to non-continuous monitoring as a point of single parameter-likely therefore, missing closely-timed variations. Therefore, in this study we focused on clinically applicable DTI parameters, which seem relatively less load-dependent and easy to perform. Third, even though we limited parameters to those with DTI, the measuring was not completely continuous manner since that would require multiple simultaneous Doppler scanners.

Furthermore, this study was an observational, non-interventional design, to see changes of tissue Doppler-derived indices during routine clinical practice using either IV anesthesia induction agent. Considering that BIS decreased more profoundly and persistently with propofol than thiopental ($p < 0.001$); further studies may be warranted controlling for BIS level to determine BIS-independent effects of the induction agents on cardiac function. Future studies would also usefully expand to include other clinical populations.

In conclusion, using intraoperative transthoracic DTI imaging, we examined impacts on cardiac function from the two most commonly applied IV anesthesia induction agents, thiopental and propofol—the propofol-based anesthesia group revealed a more persistent and profound decline of S' and a' velocities than the thiopental-based group. This suggests a specific drug-dependent impact on myocardial performance during the anesthesia induction. Further studies are warranted to understand the clinical implications—these may affect the choice of induction anesthesia agent during pre-operative patient evaluation.

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