原 著

Comparism of Hemodynamic and Catecholamine Responses between Propofol and Thiamylal Used in a Rapid Sequence Induction

Tetsuya Hara*, Makoto Fukusaki*, Toshiaki Nakamura*, Sadayo Niiya*, Koji Sumikawa**

Abstract

Authors evaluated hemodynamic and catecholamine responses during rapid sequence induction with propofol compared to those with thiamylal without any influences of other anesthetic agent. After institutional approval and informed consent, 20 ASA physical status I and II patients were randomly divided into two groups. Anesthesia was induced with either thiamylal, 5 $mg \cdot kg^{-1}$, (n=10) or propofol, 2 $mg \cdot kg^{-1}$, (n=10). Trachea was intubated under succinylcholine, 1.5 $mg \cdot kg^{-1}$, 1 min after induction followed by artificial ventilation with pure oxygen until the end of the study. Hemodynamic parameters and plasma catecholamine concentrations were measured as appropriate during the study period. Systolic blood pressure, heart rate and rate pressure product showed significant increases after intubation in both groups ($p<0.01$). However, these increases were greater ($p<0.05$) in thiamylal group compared to propofol group. The trend of ST-segment in electrocardiogram showed no ischemic change in either group. Plasma epinephrine showed no significant change in either group. Plasma norepinephrine showed a significant increase after intubation in thiamylal group ($p<0.05$) but not in propofol group. Three patients in thiamylal group had arrhythmias after intubation. The results suggest that propofol would be more useful for rapid sequence induction compared with thiamylal regarding hemodynamic and catecholamine responses in normotensive patients.

Key words: Catecholamines, Hemodynamics, Propofol, Rapid sequence induction

Introduction

Rapid sequence induction (RSI) with thiamylal and succinylcholine is useful to prevent aspiration in patients with full stomach[1], while significant hemodynamic and catecholamine changes associated with tracheal intubation were reported[2].

Although some studies concerning hemodynamic and catecholamine responses to tracheal intubation were reported previously[3~5], there have been few reports that evaluated these responses in RSI with propofol.

We evaluated hemodynamic and catecholamine responses during RSI with propofol compared to thiamylal supplemented with no other anesthetic agents in normotensive patients.

Methods

The subjects of this investigation were 20 ASA physical status I and II normotensive patients aged 21-64 and weighing 51 - 88 kg, who were scheduled elective surgery. Patients treated with antihypertensive drugs were excluded in this study. The protocol was

*Department of Anesthesiology, Nagasaki Rosai Hospital, Nagasaki, Japan
**Department of Anesthesiology, Nagasaki University School of Medicine, Nagasaki, Japan

approved by Nagasaki Rosai Hospital Ethical Committiee, and written informed consent was obtained from each patient.

Premedication consisted of hydroxyzine, 1 $mg \cdot kg^{-1}$, and atropine, 0.01 $mg \cdot kg^{-1}$, given intramusculary 30 min before anesthesia. Radial arterial catheter was inserted for continuous monitoring of arterial blood pressure and for obtaining blood samples. Five minutes after precurarization with vecuronium, 0.015 $mg \cdot kg^{-1}$ and inhaling oxygen at a flow rate of 5 $L \cdot min^{-1}$, anesthesia was induced with either intravenous thiamylal, 5 $mg \cdot kg^{-1}$, (group T; n=10) or propofol, 2 $mg \cdot kg^{-1}$, (group P; n=10) for 40 min. Before injection of thiamylal or propofol, lidocaine, 20 mg, was injected into the vein with proximal occlusion of the vein to minimize injection pain. Tracheal intubation was facilitated with intravenous succinylcholine, 1.5 $mg \cdot kg^{-1}$, 1 min after administration of thiamylal or propofol by same staff anesthesiologist. End-tidal CO_2 tension was maintained at 35 mmHg (Capnomac, Datex, Helsinki, Finland) by manual ventilation with pure oxygen until the end of the study.

Measurements included hemodynamic and catecholamine responses and arterial blood gas analysis. Systolic blood pressure (SBP), heart rate (HR), rate pressure product (RPP) and ST-segment change in electrocardiogram (ST) were measured (Bioview, Nippon Koden, Tokyo, Japan) before induction of anesthesia, immediately after intubation, and 1, 2 and 3 min after intubation. Plasma epinephrine (E) and norepinephrine (NE) concentrations were determined in a fully automated high-performance liquid chromatography-fluorometric system (model HLC-8030 Catecholamine Analyzer, Tosoh, Tokyo, Japan) and arterial blood gas analysis were performed (ABL-4, Radiometer Corp., Copenhagen, Denmark) at before induction of anesthesia, and 1 and 3 min after tracheal intubation.

The data were expressed as mean±SEM. Student's t-test for unpaired data was used for statistical analysis of the differences between two groups. Differences among repeated measures were analyzed by analysis of variance using Scheffe's F test. A p value<0.05 was considered to be significant.

Results

The two groups were similar in demographic characteristics (gender, age, weight and height) (Table 1). There was no difference in results of arterial blood gas analysis between two groups (Table 2). Pa_{O_2},

Table 1 Patient's Demographic Data (mean±SEM)

	group T	group P
N	10	10
Gender (male / female)	7 / 3	8 / 2
Age (yr)	43±4	42±5
Weight (kg)	60±3	58±2
Height (cm)	163±3	165±2

Values are given as mean±SEM
group T = rapid sequence induction with thiamylal;
group P = rapid sequence induction with propofol.

Table 2 Changes in Arterial Blood Gas in group T and group P (mean±SEM)

	group	T1	T2	T3
pH	T	7.42±0.02	7.41±0.03	7.43±0.04
	P	7.41±0.04	7.41±0.04	7.43±0.04
Pa_{CO_2} (mmHg)	T	40±4	41±4	38±3
	P	39±3	40±4	38±4
Pa_{O_2} (mmHg)	T	321±44	462±51**	522±76**
	P	340±72	487±40**	526±31**

Values are given as mean±SEM
**p<0.01 vs T1.

$PaCO_2$ and pH were within normal range in both groups.

The changes of SBP and HR are shown in Fig. 1 RPP and plasma E and NE are shown in Fig. 2 and 3, respectively. The change of ST is shown in Table 3. In group T, SBP, HR and RPP showed a significant increase after tracheal intubation ($p<0.01$). ST showed no significant change throughout the time course. Plasma E showed no change throughout the time course. Plasma NE showed a significant increase ($p<0.05$) immediately after intubation. Three patients in group T had transient arrhythmias (atrial paroxysmal contraction and ventricular paroxysmal contraction) after tracheal intubation.

In group P, SBP, HR and RPP showed significant increases only immediately after tracheal intubation ($p<0.01$), whereas the values after intubation were significantly lower than those in group T ($p<0.05$). ST showed no significant change throughout the time course. Plasma E and NE showed no change throughout the time course, and NE immediately after tracheal

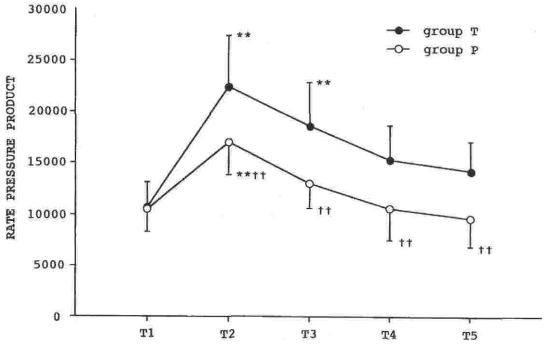

Fig. 2 Time course of changes of rate pressure product in group T and group P (mean±SEM, n=10 for each point). **p<0.01 vs T1, ††p<0.01 vs group T. T1=before induction of anesthesia, T2=immediately after intubation, T3, T4 and T5=1, 2 and 3 min after intubation respectively.

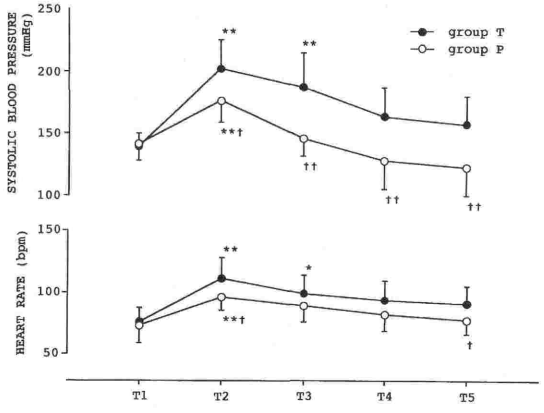

Fig. 1 Time course of changes of systolic blood pressure and heart rate in group T and group P (mean ± SEM, n=10 for each point). *p<0.05 vs T1, **p<0.01 vs T1, †p<0.05 vs group T, ††p<0.01 vs group T. T1=before induction of anesthesia, T2=immediately after intubation, T3, T4 and T5=1, 2 and 3 min after intubation respectively.

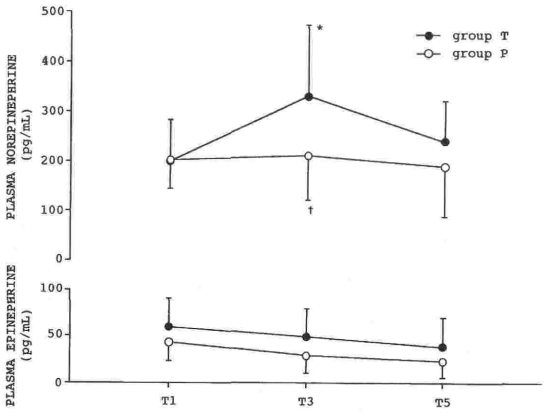

Fig. 3 Time course of changes of plasma epinephrine and norepinephrine concentrations in group T and group P (mean±SEM, n=10 for each point). *p<0.05 vs T1, †p<0.05 vs group T. T1=before induction of anesthesia, T3 and T5=1 and 3 min after intubation respectively.

Table 3 Change in ST-segmment in ECG in group T and group P (mean±SEM)

	T1	T2	T3	T4	T5
group T (mV)	0.01±0.02	0.01±0.05	−0.01±0.06	−0.06±0.07	−0.10±0.08
group P (mV)	0.03±0.02	0.06±0.05	−0.03±0.04	0.04±0.06	−0.01±0.07

intubation was significantly lower than that in group T ($p<0.05$).

Discussion

Authors evaluated efficacy of propofol as anesthetic induction agent in RSI in normotensive patients. The present results show that propofol attenuates the hemodynamic and catecholamine responses in RSI more effectively than does thiamylal. Although SBP, HR and RPP increased after endotracheal intubation with either drug, the increments of those parameters were significantly higher in patients who received thiamylal. Plasma NE showed a significant increase, and arrhythmias were observed in patients with thiamylal but not in patients with propofol.

We adopted the induction doses of propofol and thiamylal according to the study by Naguib et al.[6]. They reported that the potency ratio of propofol to thiopentone for abolition of response to eyelash stimulation were 1 : 2.35 one min after administration. In the present study, patients were intubated 1 min after receiving either propofol, 2 $mg \cdot kg^{-1}$ or thiamylal, 5 $mg \cdot kg^{-1}$.

Some investigators have studied hemodynamic and catecholamine responses after tracheal intubation in comparison between propofol and barbiturate. Harris et al.[3] reported that in the case of propofol, the arterial pressure did not increase above the control level after intubation, while it increased significantly above control level in the case of thiopentone. In their patients, the lungs were ventilated with 70% nitrous oxide in oxygen after induction of anesthesia, and plasma catecholamines were not measured. Lindgren et al.[4] reported that the increase in SBP and plasma E were greater with thiopentone than with propofol after tracheal intubation. However, the induction method in their study was not RSI and the intubation was facilitated with vecuronium. Brossy MJ et al.[5] reported that propofol suppressed increases in catecholamines more effectively after tracheal intubation than thiopentone. In their study, patients were ventilated with 50% nitrous oxide in oxygen supplemented with halothane after tracheal intubation. The changes in E and NE in our study are similar to the results of Coley et al.[7]. They compared hemodynamic and catecholamine responses to endotracheal intubation after induction of propofol or thiopentone supplemented with fentanyl and nitrous oxide. Plasma NE increased only with thiopentone. No change in plasma E occurred with either agent.

There have been a few studies that compared hemodynamic and catecholamine responses in RSI in comparison between propofol and barbiturate without any other anesthetics or analgesics. It was reported that plasma NE level increased immediately after the intravenous administration of succinylcholine[8]. In the present study, propofol suppressed the increase in plasma E and NE after tracheal intubation, whereas thiamylal could not suppress the increase in NE. No increase in E suggests that adrenomedullarly response to tracheal intubation might be suppressed with both propofol and thiamylal. In patients who received thiamylal, the increase in NE 1 min after intubation would be related to the increases in SBP and HR.

Hypoxemia or hypercapnia increases sympathetic nerve activity resulting in the release of catecholamines. In the present study, no patient had hypoxemia or hypercapnia. Thus respiratory factors did not influence the hemodynamic and catecholamine responses.

We gave lidocaine 20 mg before thiamylal or propofol injection to minimize pain during injection. While lidocaine has been reported to limit airway reactivity as measured by cough reflex, the dose required is a minimum of 1.5 $mg \cdot kg^{-1}$ [9]. Thus lidocaine could not have a significant influence on the present results.

In conclusion, propofol in doses for induction would be more useful for RSI compared with thiamylal regarding hemodynamic and catecholamine responses in normotensive patients.

References

1) Selick BA : Cricoidpressure to control regurgitation of stomach contents during induction of anesthesia. Lancet 2 : 404-406, 1961

2) Derbyshire DR, Chmielewski A, Fell D, et al : Plasma catecholamine responses to tracheal intubation. Br J Anaesth 55 : 855-860, 1983

3) Harris CE, Murry AM, Anderson JM, et al : Effects of thiopentone, etomidate and propofol on the haemodynamic response to tracheal intubation. Anaesthesia 43S : 32-36, 1988
4) Lindgren L, Yli-hankala A, Randell T, et al : Haemodynamic and catecholamine responses to induction of anaesthesia and tracheal intubation: comparison between propofol and thiopentone. Br J Anaeth 70 : 306-310, 1993
5) Brossy MJ, James MFM, Janicki PK, et al : Haemodynamic and catecholamine change after induction of anaesthesia with either thiopentone or propofol with suxamethonium. Br J Anaeth 72 : 596-598, 1994
6) Naguib M, Sari-kouzel A, Seraj M, et al : Induction dose-response studies with propofol and thiopentone. Br J Anaesth 68 : 308-310, 1992
7) Coley S, Mobley KA, Bone ME, et al : Haemodynamic changes after induction of anaesthesia and tracheal intubation following propofol or thiopentone in patients of ASA grade I and III. Br J Anaesth 63 : 423-428, 1989
8) Nigrovic V, McCullough LS, Wajskol A, et al : Succinylcholine-induced increase in plasma catecholamine levels in humans. Anesth Analg 62 : 627-632, 1983
9) Yukioka H, Hayashi M, Terai T, et al : Intravenous lidocaine as a suppressant of coughing during tracheal intubation in elderly patients. Anesth Analg 77 : 309-312, 1993

(Circ Cont 21 : 32～36, 2000)