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Effects of Esmolol on Hemodynamic Responses to Laryngoscopy and Tracheal Intubation in Diabetic Versus Non-Diabetic Patients

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Aim: We aimed to investigate the efficiency of esmolol, a short-acting ß-blocker, in preventing the hemodynamic response to laryngoscopy and endotracheal intubation in diabetic patients.

Materials and Methods: Eighty diabetic or non-diabetic patients with ASA physical status I-II scheduled for noncardiac surgery were included in this study. They were divided randomly into 4 groups (Non-diabetic control: NDC, Non-diabetic esmolol: NDE, Diabetic control: DC, Diabetic esmolol: DE). Blood glucose analyses were measured in the preoperative period and at the 10th min of the study. Prior to anesthetic induction, 1 mg/kg esmolol to Groups NDE and DE and saline to Groups NDC and DC were administered in 1 min by slow infusion. After 2 mins, systolic and diastolic arterial blood pressures (SBP, DBP), heart rate (HR), bispectral index (BIS) and peripheral oxygen saturation (SpO_2) were recorded in all groups. Laryngoscopy and endotracheal intubation were performed after induction. SBP, DBP, HR, SpO_2 and BIS values were recorded every minute during 10 mins after intubation.

Results: In Groups NDE and DE, SBP, DBP and HR values were significantly lower after drug administration than the values obtained before drug administration ($p<0.05$). In Groups NDC, NDE and DC, SBP, DBP and HR values were significantly higher in the first minute of the intubation compared to before drug administration ($p<0.05$), but were significantly low in subsequent measurements ($p<0.05$). Blood glucose analyses were found significantly higher in Group NDE than Group NDC ($p<0.05$).

Conclusions: We propose that esmolol might be used effectively to control hemodynamic response to tracheal intubation in diabetic patients. We also determined that esmolol causes no difference in blood glucose levels.

Key Words: Esmolol, laryngoscopy, intubation, diabetics

Diabetik ve Diabetik Olmayan Olgularda Laringoskopi ve Trakeal Entübasyona Hemodinamik Yanıtlara Esmololün Etkisi

Amaç: Diabetik hastalarda kısa etkili bir ß-bloker olan esmololün, laringoskopi ve trakeal entübasyona hemodinamik yanıtı önlemedeki etkinliğini araştırmayı amaçladık.

Yöntem ve Gereç: Non-kardiyak cerrahi planlanan ASA I-II, diabetik veya diabetik olmayan 80 olgu çalışmaya dahil edildi. Olgular randomize olarak dört gruba ayrıldı.(Nondiyabetik kontrol; NDC, Nondiyabetik esmolol; NDE, Diabetik kontrol; DC, Diyabetik esmolol; DE) . Preoperatif periyotta ve çalışmanın 10. dakikasında kan şekeri ölçümleri yapıldı. İndüksiyondan önce NDE ve DE gruplarına 1 mg/kg esmolol, NDC ve DC gruplarına ise salin 1 dk içinde yavaş infüzyonla uygulandı. Bu esmolol veya salin uygulamasından 2 dk sonra, tüm olguların sistolik arter basıncı (SAB), diastolik arter basıncı (DAB), kalp atım hızı (KAH), bispektral indeks (BIS) ve periferik oksijen satürasyonu (SpO_2) değerleri kaydedildi. İndüksiyonu takiben laringoskopi ve endotrakeal entübasyon uygulandı. Entübasyon sonrası olguların SAP, DAP, HR, SpO_2 ve BIS değerleri 10 dk boyunca her 1 dk'da bir ölçüldü ve kaydedildi.

Bulgular: Grup NDE ve DE'de ilaç uygulaması sonrası SAB, DAB ve KAH değerlerinin ilaç uygulaması öncesi değerlere göre anlamlı olarak düşük olduğu saptandı ($p<0.05$). NDC, NDE ve DC gruplarında entübasyon sonrası 1. dakika SAB, DAB ve KAH değerleri ilaç uygulaması öncesi değerlere göre anlamlı olarak yüksek bulunurken ($p<0.05$), bundan sonraki ölçümlerde anlamlı olarak düşük bulundu ($p<0.05$). Kan şekeri değerleri NDE grubunda NDC grubuna göre anlamlı olarak yüksek saptandı ($p<0.05$).

Sonuç: Esmolol diabetik hastalarda da laringoskopi ve trakeal entübasyona hemodinamik yanıtı etkili şekilde kontrol etmek amacıyla kullanılabilir. Aynı zamanda esmololün kan şekeri seviyelerinde değişikliğe yol açmayacağı sonucuna vardık.

Anahtar Sözcükler: Esmolol, Laringoskopi, Entübasyon, Diabetes Mellitus

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Introduction

Increases in heart rate and blood pressure are the principal changes in the cardiovascular system during laryngoscopy and tracheal intubation. Stimulus of the laryngeal and tracheal tissues may also cause increases in both sympathetic and sympathoadrenal reflex activity (1,2). Hemodynamic changes are generally temporary without any sequelae. However, these changes can facilitate and accelerate the development of myocardial ischemia, arrhythmia, infarction and cerebral hemorrhage in patients with coronary artery disease, hypertension or cerebrovascular disease (3,4). Different pharmacologic agents like lidocaine, vasodilator agents inhibiting sympathoadrenal response, α- and ß- adrenergic blockers, and opioids can be administered prior to tracheal intubation in order to prevent hemodynamic responses (5-8).

ß blockers are used to reduce the necessity of anesthetic drugs by their sedative properties and reduce unwanted hemodynamic responses. Esmolol is an ultra-short-acting, $ß_1$ cardioselective, ß blocking agent with a short half-life (9 min). This agent has been used recently to reduce the increase in heart rate and blood pressure in response to tracheal intubation (9,10).

Increased cardiovascular instability during anesthesia and abnormal cardiovascular responses to intubation and induction of anesthesia have been described in patients with diabetic autonomic neuropathy (11,12). Both attenuated (11,12) and enhanced (13,14) pressure responses to intubation have been reported. These altered responses have been attributed directly to autonomic neuropathy (11-14).

No studies of esmolol in diabetic patients exist in the literature. The purpose of this study is to show the effectiveness of esmolol, a short-acting ß blocker agent, on blocking the hemodynamic response to laryngoscopy and tracheal intubation in non-diabetic versus diabetic patients who are at high risk for silent myocardial ischemia.

Materials and Methods

The study was approved by the Ethical Committee of the University. Patients were given information about the procedure and informed consent was obtained. Eighty patients with ASA I-II physical status, between 18-70 years of age, and diabetic (using insulin or oral anti-diabetic drugs) or non-diabetic who were scheduled to undergo elective operations under general anesthesia were included in this study. Patients with unstable coronary arterial disease, atrial or ventricular tachyarrhythmia, 2°-3° heart block, sinusal bradycardia, blood pressure not between 100/50 – 160/110 mmHg, morbid obesity, uncontrolled asthma, chronic ß blocker and opioid usage, severe liver and renal insufficiency, or with difficult intubation or prolonged intubation longer than 20 secs were excluded from the study.

The patients were randomly divided into four groups as non-diabetic control (NDC), non-diabetic esmolol (NDE), diabetic control (DC) and diabetic esmolol (DE). All patients received 10 mg of oral diazepam 1 h before the operation. Blood glucose levels were measured prior to the operation. After standard monitoring (noninvasive blood pressure, pulse oximeter, esophageal temperature and bispectral index) in the operating suite, electrocardiogram baseline systolic blood pressure (SBP), diastolic blood pressure (DBP), heart rate (HR), bispectral index (BIS), and peripheral oxygen saturation (SpO_2) were recorded. Before induction of general anesthesia, esmolol 1 mg/kg to Groups DE and NDE and normal saline solution to Groups DC and NDC were infused slowly in 1 min. Two mins later, SBP, DBP, HR, BIS and SpO_2 values of all patients were recorded. All patients received fentanyl 2 µg/kg, propofol 2 mg/kg and rocuronium 0.6 mg/kg for the induction of general anesthesia. Intubation was done when BIS level was <60. SBP, DBP, HR, BIS and SpO_2 values were recorded every minute after intubation for 10 min. During this period, onset of surgery was not allowed. Blood glucose levels were measured at 10^{th} min after intubation. After tracheal intubation, anesthesia was maintained with 65% nitrous oxide in oxygen and 2% sevoflurane in order to provide a BIS level under 60. Normothermia was maintained during the whole procedure. Hypotension and bradycardia were defined as SBP less than 90 mmHg and HR less than 45 beats/min, respectively. Those patients experiencing hypotension or bradycardia were treated with ephedrine 5 mg or atropine 0.5 mg, respectively.

Statistical Analysis

Demographic data and duration of surgery according to group were compared by Kruskal Wallis test and dual comparisons among the groups by quantitative Mann-Whitney U test. Gender and types of the operations

among the groups were compared by X^2 test. Plasma glucose change analysis was made by using one-way ANOVA and dual comparisons among the groups by paired t test. Hemodynamic data at different times in each group were compared using repeated measures ANOVA. Quantitative data are shown as mean ± SD. P<0.05 was considered as statistically significant.

Results

Variables including demographic data and duration of surgery and diabetes in the diabetic group were similar and no statistically significant differences were found (p>0.05) (Table 1). Plasma glucose levels of Group NDE measured after intubation were significantly higher when compared to Group NDC (p<0.05) (Table 2).

Comparison of the groups with respect to SBP demonstrated that SBP at the 1st min after intubation was significantly higher in Group NDC (p<0.05) with respect to SBP before esmolol infusion, but all SBP measures from the 3rd min after intubation were found significantly low (p<0.05). SBP measures after esmolol infusion in Group NDE were significantly lower than SBP measures before infusion (p<0.05). In the same group, SBP measurements at the 1st min after intubation were significantly lower than before infusion (p<0.05) and measurements at the 2nd and subsequent minutes after intubation were also significantly low (p<0.05). In Group DC, all SBP measurements in the 2nd and following minutes after intubation were significantly low (p<0.05). In Group DE, SBP measurements at the 2nd min after intubation were significantly higher than those of group

Table 1. Demographic data of diabetic groups (mean ± SD).

	Diabetic Control (n=20)	Diabetic Esmolol (n=20)	p values
Age (year)	57.6 ± 10.0	54.6 ± 8.4	0.31
Sex (M/F)	7/13 (35%/65%)	7/13 (35%/65%)	0.62
Height (cm)	162.1 ± 6.2	165.0 ± 6.9	0.16
Weight (kg)	73.5 ± 10.7	74.4 ± 11.9	0.80
Duration of surgery (min)	106.5 ± 45.9	110.0 ± 47.2	0.81
Duration of diabetes (year)	8.0 ± 6.2	7.0 ± 5.3	0.60
Pre-op blood glucose (mg/dl)	122.1 ± 37.1	110.2 ± 26.9	0.25
Post-intubation blood glucose (mg/dl)	126.6 ± 37.1	125.5 ± 41.8	0.93

Table 2. Demographic data of nondiabetic groups (mean ± SD).

	Diabetic Control (n=20)	Diabetic Esmolol (n=20)	p values
Age (year)	40.8 ± 11.6	43.1 ± 9.9	0.49
Sex (M/F)	7/13 (35%/65%)	5/15 (25%/75%)	0.36
Height (cm)	165.4 ± 5.8	163.2 ± 5.9	0.24
Weight (kg)	74.5 ± 13.4	70.4 ± 9.9	0.27
Duration of surgery (min)	113.0 ± 56.4	109.0 ± 34.8	0.78
Pre-op blood glucose (mg/dl)	91.7 ± 17.8	82.9 ± 20.5	0.1
Post-intubation blood glucose (mg/dl)	91.2 ± 17.6	91.5 ± 24.1	0.95

DC (p<0.05). SBP measurements after infusion in the same group were significantly lower than the measurements before infusion (p<0.05). In Group DE, SBP measurements were significantly low (p<0.05) after the 3rd and subsequent minutes. SBP measurements of the groups are shown in Table 3.

In Group NDC, DBP measurements in the 1st min after intubation were significantly higher (p<0.05) than DBP measurements before esmolol infusion. However, all DBP measurements in the 5th and following minutes after intubation were significantly low (p<0.05). In Group NDE, DBP measurements after infusion were significantly lower than DBP measurements before intubation (p<0.05). In the same group, DBP measurements at the 1st min after intubation were significantly higher than DBP before infusion (p<0.05), but all DBP measurements at the 3rd and following minutes after intubation were significantly low (p<0.05). In Group DC, DBP measurements at the 1st min after intubation were higher than DBP before infusion (p<0.05), but all DBP measurements after the 3rd min were significantly low (p<0.05). In Group DE, DBP measurements after infusion were lower than DBP measurements before infusion (p<0.05). In the same group, DBP measurements at the 1st min after intubation were lower than DBP measurements before infusion (p<0.05), but all DBP measurements at the 4th and following minutes after intubation were significantly low (p<0.05). DBP measurements of the groups are given in Table 4.

In Group NDC, HR measurements after drug infusion were significantly lower than HR measurements before drug infusion (p<0.05). In the same group, HR measurements at 1st min to 4th min after intubation were significantly higher than HR measurements before drug infusion (p<0.05), but HR measurements from the 5th min onwards after intubation and HR measurements before drug infusion demonstrated no significant different (p>0.05). In Group NDE, HR measurements after drug infusion and from 1st to 5th min after intubation were significantly lower than values in NDC (p<0.05). In the same group, HR measurements after drug infusion were significantly lower than the measurements before drug infusion (p<0.05). HR measurements at the 1st and 2nd mins after intubation were significantly high (p<0.05), but no significant difference was obtained between HR measurements from the 3rd min onwards following intubations and before drug values (p>0.05). In group DC, HR measurements after drug infusion were significantly lower than before drug infusion (p<0.05). HR measurements from the 1st

Table 3. Systolic blood pressure (SBP) of groups according to time (min) (mean ± SD).

	Non-diabetic Control (n=20)	Non-diabetic Esmolol (n=20)	Diabetic Control (n=20)	Diabetic Esmolol (n=20)
Before induction	133.8 ± 18.0	144.8 ± 14.7	146.3 ± 19.0	154.1 ± 21.6
After induction	131.2 ± 18.6	136.4 ± 15.7*	145.1 ± 19.1	147.8 ± 19.1*
1	151.3 ± 23.9*	159.4 ± 15.6*	152.4 ± 24.8	164.3 ± 36.5
2	130.4 ± 21.4	135.3 ± 14.7*	135.3 ± 22.3	151.4 ± 32.2†
3	116.7 ± 19.1*	122.5 ± 14.8*	122.9 ± 19.7*	134.4 ± 26.9*
4	112.2 ± 19.9*	113.8 ± 10.7*	117.0 ± 16.4*	123.1 ± 23.5*
5	107.1 ± 19.6*	109.5 ± 11.3*	108.4 ± 15.2*	117.5 ± 17.9*
6	105.6 ± 16.8*	105.4 ± 10.5*	102.5 ± 15.5*	108.1 ± 18.8*
7	104.4 ± 17.9*	104.0 ± 10.9*	100.8 ± 15.7*	107.0 ± 15.5*
8	101.7 ± 17.1*	101.6 ± 10.2*	97.9 ± 15.5*	102.9 ± 15.8*
9	99.9 ± 15.6*	100.5 ± 9.6*	99.4 ± 17.8*	101.0 ± 16.3*
10	99.4 ± 16.1*	99.3 ± 9.6*	98.0 ± 17.0*	99.4 ± 15.0*

(*) p<0.05: when compared with before induction SBP measurements.

Table 4. Diastolic blood pressure (DBP) of groups according to time (mean ± SD).

	Non-diabetic Control (n=20)	Non-diabetic Esmolol (n=20)	Diabetic Control (n=20)	Diabetic Esmolol (n=20)
Before induction	75.3 ± 10.2	83.1 ± 9.1	86.2 ± 9.5	87.6 ± 10.8
After induction	76.9 ± 12.3	79.4 ± 9.8*	87.2 ± 10.9	84.3 ± 11.6*
1	97.2 ± 15.3*	98.1 ± 12.6*	95.0 ± 16.0*	101.8 ± 19.5*
2	81.4 ± 12.9	83.6 ± 10.5	86.3 ± 12.0	91.2 ± 17.4
3	74.7 ± 14.2	74.2 ± 11.3*	78.9 ± 11.2*	84.5 ± 15.8
4	70.6 ± 15.3	65.4 ± 8.3*	73.0 ± 12.1*	71.3 ± 15.9*
5	65.5 ± 15.7*	61.7 ± 8.1*	69.3 ± 10.2*	71.6 ± 16.0*
6	64.3 ± 13.8*	61.2 ± 9.4*	64.8 ± 11.5*	65.5 ± 12.5*
7	62.6 ± 14.0*	58.8 ± 8.3*	63.0 ± 10.9*	67.5 ± 12.5*
8	61.7 ± 12.7*	57.5 ± 9.4*	61.3 ± 11.6*	61.4 ± 18.6*
9	61.2 ± 12.9*	56.7 ± 8.0*	62.3 ± 12.2*	64.6 ± 12.0*
10	59.5 ± 13.5*	55.4 ± 7.5*	61.0 ± 12.2*	64.3 ± 12.6*

(*): $p<0.05$ when compared with before induction DBP measurements.

to 3rd min after intubation were significantly higher than HR measurements before drug ($p<0.05$), but HR measurements from the 4th min onwards after intubation and HR measurements before drug showed no significant difference ($p>0.05$). In Group DE, HR measurements after drug infusion were significantly lower than measurements before drug. There was no significant difference between all HR measurements from the 1st min of intubation and before drug administration ($p>0.05$). HR measurements of the groups are given in Table 5.

Discussion

Cardiovascular changes that appear during tracheal intubation are stress effects of manipulation of the epiglottis and increase in SBP and HR. This effect usually lasts less than 5 mins, but can be seen after nearly all intubations. Under normal conditions in healthy people, this can be well tolerated, but in patients with hypertension, ischemic heart disease, increased intracranial pressure, and eye injury, there is an increased high risk of morbidity and mortality (1). In the present study, we evaluated the hemodynamic response to laryngoscopy and tracheal intubation following esmolol in diabetic patients. We observed that BP and HR values of the esmolol groups were significantly low after drug administration compared to values obtained before drug administration.

The cardiovascular changes and catecholamine discharge seen during laryngoscopy and tracheal intubation appear in two phases. The effects of laryngoscopy should be distinguished from effects seen while the endotracheal tube is placed through the trachea. Shribman et al. (15) showed the differences between these two events. Even with stable anesthesia, laryngoscopy alone without intubation can cause a supraglottic stimulus. As a result, both SBP and DBP increase in contrast to the measurements before induction. However, no significant increase in HR occurs during laryngoscopy. Increase in BP is due to norepinephrine, while HR increase is due to epinephrine discharge. Infraglottic stimulus caused by placing the endotracheal tube occurs in phase two. In this situation, an extra cardiovascular response and catecholamine discharge occur. Stress response increases at this stage and both SBP and DBP measurements increase by 36-40% in contrast to control levels. HR levels increase more than 20% with tracheal intubation in contrast to laryngoscopy (15).

Amar et al. (16) administered 0.15 mg/kg of labetalol for induction and 0.25-0.3 mg/kg for maintenance of

Table 5. Heart rate (HR) of groups according to time (min) (mean ± SD).

	Non-diabetic Control (n=20)	Non-diabetic Esmolol (n=20)	Diabetic Control (n=20)	Diabetic Esmolol (n=20)
Before induction	81.7 ± 12.8	77.7 ± 9.9	81.7 ± 17.2	82.5 ± 15.9
After induction	86.1 ± 14.5*	69.2 ± 8.4#*	83.9 ± 17.2*	74.4 ± 13.8*
1	99.9 ± 11.6*	88.6 ± 13.7#*	96.0 ± 16.8*	92.1 ± 13.8
2	96.7 ± 11.9*	85.1 ± 12.0#*	91.3 ± 15.3*	89.8 ± 15.8
3	91.8 ± 11.0*	82.7 ± 10.9#	90.5 ± 14.3*	86.6 ± 13.9
4	89.6 ± 11.4*	80.4 ± 10.2#	86.4 ± 13.7	83.3 ± 12.6
5	86.4 ± 11.5	78.6 ± 9.9#	84.0 ± 12.8	83.2 ± 14.6
6	85.7 ± 14.1	77 ± 10.5	81.8 ± 13.1	80.5 ± 12.4
7	84.2 ± 14.8	75.6 ± 10.8	80.9 ± 12.9	80.4 ± 14.0
8	82.4 ± 13.5	74.9 ± 10.9	79.1 ± 12.9	80.1 ± 13.9
9	81.3 ± 13.3	74.4 ± 10.9	79.1 ± 13.0	78.9 ± 12.9
10	82.65 ± 11.6	74.1 ± 10.8	79.1 ± 13.0	78.7 ± 12.5

(*): $p<0.05$ when compared with before induction HR measurements.

(#): $p<0.05$, when compared with Group NDC.

anesthesia in a study investigating its effects on perioperative stress. Increases in HR and mean arterial pressure (MAP) at intubation in the placebo group were 33% and 52%, respectively, and in the labetalol group, 7.3% and 21.3%, respectively. Plasma noradrenalin concentrations increased in both groups after intubation and the relation between increased concentration and cardiovascular responses was shown.

Esmolol is a suitable beta-blocking agent for controlling hemodynamic response with its short half-life (9-12 min) and ability of titration during laryngoscopy and tracheal intubation. The first study on this subject was published by Gold et al. in 1984 (17), who evaluated the effects of esmolol after ketamine induction. According to meta-analysis from 1984 until 2001, 38 randomized controlled studies have been done (18). There are also other studies where esmolol is compared with other agents.

Helfman et al. (5) compared 80 patients in four groups who received lidocaine 200 mg, fentanyl 200 µg, esmolol 150 mg and placebo. In this study, 150 mg of esmolol was found consistent and reliable for preventing increase in both SBP and HR accompanying laryngoscopy and tracheal intubation, but lidocaine 200 mg and fentanyl 200 µg were found to be protective only against the increase in SBP.

In a meta-analysis made by Figueredo (18), esmolol was compared with placebo at different doses. In most of the studies, esmolol has been given before the induction. However, in the same study, esmolol given before or after the induction did not cause significant changes in these parameters. In our study, the purpose of giving esmolol before induction was to evaluate the changes independent from the control measurements.

Sintetos et al. (19) reported that esmolol should be given 2 min before intubation for effective results because the maximum effect of esmolol on HR occurred in the 1st and on BP in the 2nd min. In our study, hemodynamic measurements were recorded 2 mins after esmolol injection. Induction and intubation were done within 2 mins after esmolol administration.

Esmolol is used in different bolus and infusion doses to prevent hemodynamic changes in response to intubation and laryngoscopy. Figueredo et al. (18) reported that an infusion of esmolol 200 and 300 µg/kg/min after a loading dose of 500 µg/kg/min for 4 mins was more effective. However, in a multicenter study Miller et al. (20), they reported that such an infusion at induction complicated the induction because of the possibility of mistakes in preparing the infusion doses. Therefore, Miller et al. (20) suggested that one selected dose of esmolol would be easier and more effective. In

our study, 1 mg/kg of esmolol was infused in 1 min.

Tachycardia causes more stress effect on the heart than increases in BP. This effect can be due to the increase in myocardial oxygen requirement, decreased diastolic filling, and reduction in the time needed for effective coronary circulation. Tachycardia accompanied with hypertension increases the existing ischemia risk in patients with coronary artery disease (21). This is especially important in diabetic patients with silent ischemia. In a study in which esmolol 1 mg/kg was compared with nicardipine (22), SBP and DBP decreased significantly in 2 min after esmolol administration. Similar to results in our study, in Groups NDE and DE, SBP and DBP decreased significantly before induction. But in the 1st min after intubation, SBP did not significantly increase in Group DE, whereas in the other three groups SBP increased significantly. In Groups NDE and DE, SBP and DBP measurements returned to control values 2 mins after intubation and measurements until the 10th min were significantly lower than before drug infusion because of anesthesia maintenance.

Even though esmolol is effective in controlling the chronotropic response to intubation, it is not as effective as in controlling the BP. Atlee et al. (22) in their study reported that HR decreased significantly after esmolol and this decrease lasted for 1 min after intubation. In our study, in Groups NDE and DE, HR decreased significantly after esmolol but in Group NDE an increase was determined after intubation until the 2nd min. Nevertheless, in Group DE, HR showed no change after intubation compared to baseline measurements.

Beta-adrenergic blocking agents can impair glucose tolerance, mask the stimulus, and block the glycemic response to hypoglycemia in diabetic patients (23). In our study, esmolol caused increase in plasma glucose levels in the diabetic and non-diabetic groups, but the only statistically significant increase was found in the non-diabetic group.

Esmolol may cause bradycardia, atrioventricular block and bronchospasm as adverse effects (20). In our study, these adverse effects were not observed before or after intubation with esmolol 1 mg/kg.

In conclusion, we suggest that esmolol may be used to control hemodynamic response to laryngoscopy and tracheal intubation in diabetic patients with high resting HR and several day-time fluctuations without causing a difference in blood glucose values.

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