Original Article/*Özgün Araştırma*

doi: 10.54875/jarss.2023.47560

The Effect of Pre-Intubation Esmolol and Dexmedetomidine on Ischemia Modified Albumin Levels: A Prospective Randomized Trial

Entübasyon Öncesi Yapılan Esmolol ve Deksmedetomidinin İskemi Modifiye Albumin Seviyeleri Üzerine Etkisi: Randomize Prospektif Bir Çalışma

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ABSTRACT

Objective: Laryngoscopy and intubation cause hypertension, tachycardia and arrhythmia. Ischemia modified albumin (IMA) is formed secondary to endothelial or extracellular hypoxia, acidosis and free oxygen radicals and can be detected in the early stages of ischemia. In this study, we aimed to compare the effects of two agents on preventing hypertension and tachycardia by measuring IMA levels.

Methods: Following ethics approval and randomization, dexmedetomidine 0.5 μg kg^{-1} in Group D (n=21) or esmolol 0.5 mg kg^{-1} in Group E (n=21) diluted in 20 mL saline was given as infusion for 5 minutes prior to induction. Patients in Group C (n=21) received only 20 mL saline infusion. An automated perfusor was set at 4 mL min^{-1} for the delivery of study solutions. Blood samples for IMA measurement were taken following monitorization and at 10^{th} and 20^{th} minutes after intubation.

Results: There was no statistically significant difference concerning IMA levels at 10 minutes following intubation. However, IMA levels in Group C were significantly lower compared to Group E and Group D (p=0.025 and 0.015 respectively) at 20 minutes. There was no significant difference between Group E and Group D (p=0.980). In Group D and E, a significant drop in systolic, diastolic and mean arterial pressures compared to baseline was observed after the study drugs were given. Groups were similar in terms of heart rate in the first 5 minutes however Groups E and D had lower heart rates compared to Group C starting from the 6^{th} minute until the end of study period.

Conclusion: Inhibiton of hemodynamic response to intubation may have negative results, and pharmacological interventions for this should be carried out with caution in critical patients and close hemodynamic monitorization should be kept in mind.

Keywords: Dexmedetomidine, esmolol, ischemia-modified serum albumin

ÖZ

Amaç: Laringoskopi ve entübasyonun hipertansiyon, taşikardi ve aritmiye sebep olduğu bilinmektedir. İskemi modifiye albumin (İMA); endotelyal veya ekstraselüler hipoksi, asidoz ve serbest oksijen radikallerine bağlı oluşur ve iskeminin erken dönemlerinde saptanabilir. Bu çalışmada İMA seviyelerinin ölçümü üzerinden hipertansiyon ve taşikardinin önlenmesinde kullanılan iki ajanın etkisinin karşılaştırılması amaçlanmıştır.

Yöntem: Etik onam alınması ve randomizasyonun ardından Grup D'de (n=21) 0,5 μg kg^{-1} deksmedetomidin, Grup E'de (n=21) ise 0,5 mg kg^{-1} esmolol 20 mL serum fizyolojik içerisinde sulandırılarak indüksiyondan 5 dak önce infüzyon şeklinde verildi. Grup C'deki (n=21) hastalara ise yalnızca 20 mL serum fizyolojik verildi. Çalışma solüsyonlarının verilmesinde 4 mL dak^{-1} hıza ayarlanmış otomatize perfüzör kullanıldı. İskemi modifiye albumin ölçümü için kan örnekleri monitörizasyondan sonra ve entübasyondan sonraki 10 ve 20. dakikalarda alındı.

Bulgular: Entübasyondan sonraki 10. dakikada ölçülen İMA değerleri açısından bir fark bulunmadı. İskemi modifiye albumin seviyeleri 20. dakikada Grup C'de Grup E ve D ile karşılaştırıldığında anlamlı olarak düşüktü (sırasıyla p=0,025 ve 0,015). Grup E ve D arasında anlamlı bir fark saptanmadı (p=0,980). Grup D ve E'de sistolik, diyastolik ve ortalama arter basınçlarında çalışma ilaçları verildikten sonra başlangıca göre anlamlı düşüş gözlendi. Gruplar ilk 5 dakika boyunca kalp hızı açısından benzerdi fakat 6. dakikadan başlayarak çalışma periyodunun sonuna kadar Grup E ve D'de kalp hızı düşük izlendi.

Sonuç: Sonuç olarak, entübasyona hemodinamik yanıtın inhibisyonunun negatif sonuçları olabileceği düşünülerek buna yönelik farmakolojik müdahaleler özellikle kritik hastalarda dikkatli yapılmalı ve yakın hemodinamik monitörizasyonun önemi akılda tutulmalıdır.

Anahtar sözcükler: Deksmedetomidin, esmolol, iskemi-modifiye albumin

Received/Geliş tarihi : 31.08.2022
Accepted/Kabul tarihi : 05.04.2023
Publication date : 28.04.2023

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Cite as: Yilmaz H, Kazbek BK, Koksoy UC, Ekmekci P, Candar T, Yavuzekinci MA, Tuzuner F. The effect of pre-intubation esmolol and dexmedetomidine on ischemia modified albumin levels: A prospective randomized trial. JARSS 2023;31(2):143-149.

INTRODUCTION

Laryngoscopy and intubation cause a mechanical stimulus in respiratory and cardiovascular systems which results in a reflex response that peaks in 1 minute and lasts for 5-10 minutes as well as a chemical stimulus that results in hypertension, tachycardia and arrhythmia due to increased catecholamine release. Blood pressure rises in 5 seconds following laryngoscopy and peaks within 2 minutes (1). Narcotic agents, alfa-2 agonists, vasodilators, beta blockers, calcium channel blockers and local anesthetics have been used in order to attenuate the effects caused by laryngoscopy and endotracheal intubation however, these methods have their own drawbacks (2). Esmolol is a cardioselective beta-1 adrenergic blocker which inhibits beta-2 receptors found in bronchi and vessel walls in a dose-dependent manner and has a rapid onset as well as a short half-life. Dexmedetomidine is a selective alpha-2 adrenergic agonist with more prominent cardiovascular effects (3).

Ischemia modified albumin (IMA) is formed when the binding capacity of the albumin N-terminal for metals (i.e. Cobalt) decreases as a result of endothelial or extracellular hypoxia, acidosis or free oxygen radicals (4). Serum IMA levels increases within minutes following ischemia and returns to normal within 6-12 hours (5). Peripheral vascular disease, exercise induced skeletal muscle ischemia, end stage renal disease requiring hemodialysis, acute stroke and calf muscle ischemia can also elevate serum IMA levels (4). To the best of our knowledge, there is no study investigating the relationship between hemodynamic effects caused by drugs used to control the sympathetic response induced by intubation and IMA levels in the literature. We aimed to predict the effects of two different agents which are used to prevent hemodynamic changes by measuring IMA levels and to show that IMA can be used as an early warning biomarker in addition to physiologic parameters.

MATERIAL and METHODS

Following the approval of ethics committee, 63 ASA I-II patients between 18-65 years old who were scheduled to undergo grade I-II elective surgeries were included in the study (Date: 18.09.2015; No: 20/02). Exclusion criteria were patient refusal, body mass index >30, Mallampati score 3-4, severe systemic disease, severe chronic obstructive pulmonary disease, serum albumin level <3 or >5.5 g dL^{-1}, history of β-blocker or antihypertensive medication usage. Patients with difficult ventilation or intubation and known allergic reaction to study drugs were removed from the study. The patients were randomly allocated to dexmedetomidine (Group D, n=21), esmolol (Group E, n=21) and control (Group C, n=21) using closed envelope technique after obtaining written informed consent.

Routine monitorization (electrocardiography, noninvasive blood pressure, peripheral oxygen saturation) was applied to all patients in the operating room. Oxygen saturation, systolic, diastolic and mean arterial pressure in addition to heart rate were measured and recorded before study drugs were given. Dexmedetomidine 0.5 μg kg^{-1} in Group D or esmolol 0.5 mg kg^{-1} in Group E diluted in 20 mL normal saline was given as infusion for 5 minutes prior to induction. Same volume of normal saline was given before induction to the patients in the control group. An automated perfusor was set at 4 mL min^{-1} for the delivery of study solutions. The infusion solutions were prepared by an anesthesiologist who was not responsible for data collection. The anesthesiologist responsible for the clinical care of the patient was blinded to the study drugs. Vital signs were recorded and blood samples for baseline IMA measurement were obtained before induction. Induction was carried out using 1-2 mg kg^{-1} propofol, 1 μg kg^{-1} fentanyl and 0.6 mg kg^{-1} rocuronium and all patients were intubated within 90 seconds. Patients who could not be intubated within 15-20 seconds of laryngoscopy were removed from the study.

Sevoflurane (1 MAC) and 50%/50% oxygen-nitrous oxide mixture was used for maintenance. Additional blood samples for IMA measurement were taken at 10^{th} and 20^{th} minutes after intubation. Surgery was commenced 10 minutes following intubation. Systolic, diastolic and mean arterial pressure values in addition to SpO_2 and heart rate values were recorded every minute after the study drugs were given. The rate of fluid infusion was increased if systolic arterial pressure dropped more than 20% of the basal values or below 90 mmHg. Volatile anesthetic concentration was reduced and intermittent 4 mg boluses of ephedrine was given if necessary. Hypertension was defined as more than 20% increase in systolic blood pressure compared to the baseline and anesthetic depth was increased by increasing the end-tidal concentration of the volatile anesthetic, 0.1 mg nitroglycerin bolus was given if hypertension persisted and recorded. Bradycardia was defined as heart rate below 45 beats min^{-1} and 0.5 mg atropine was given and recorded.

Blood samples were centrifuged as soon as received by the laboratory and the separated serum was stored at -80°C until assayed. IMA concentration analysis was carried via the measurement of the complex composed of cobalt and dithioerthreitol unbound to albumin by the colorimetric method in a spectrophotometer. 50 μL cobalt chloride (Sigma Aldrich, St Louis, MO, USA) and 200 μL of patient's serum was mixed glass tubes. This mixture was incubated at room temperature for two minutes. Finally, 1000 mL of 0.9% sodium chloride was added. A blank specimen was prepared using distilled water for control. Spectrophotometric analyses (Human Humalyzer 2000, Germany) were carried at 470 nm and results were expressed as absorbance units (ABSU).

Statistical Analysis

Power analysis revealed that at least 63 patients (21 for each group) were necessary in order to detect 0.1 (SD=0.1) units change in IMA levels with 80% power and 5% type I error. SPSS for Windows v25 was used for data analysis. The quantitative parameters were compared using ANOVA test and repeated measure ANOVA test was used for comparison of repetitive variables. Qualitative variables were compared using the χ^2 test. Bonferroni test was utilized for binary comparisons. A p value lower than 0.05 was considered significant.

RESULTS

Sixty-seven patients were enrolled in the study. One patient in Group C was excluded due to difficult ventilation, 2 patients in Group D were excluded due to difficult intubation and 1 patient in Group E was excluded due to an allergic reaction following induction was excluded from the study. The study was completed with 63 patients. The groups were similar concerning demographic variables (Table I).

Baseline IMA values were 0.232±0.009 in Group C, 0.255±0.078 in Group D and 0.284±0.048 ABSU in Group E (p=0.121) (Table II). There was no statistically significant difference concerning IMA levels at 10 minutes following intubation. However, IMA levels in Group C were significantly lower compared to Group E and Group D (p=0.025 and 0.015 respectively) at 20 minutes (Table III). There was no significant difference between Group E and Group D.

Groups were similar in terms of systolic, diastolic and mean arterial pressures in addition to heart rate before induction. However, a significant drop was observed in systolic, diastolic and mean arterial blood pressure values in group D and E following the administration of study drugs where intragroup changes were concerned (Figures 1, 2 and 3). Groups were similar in terms of heart rate in the first 5 minutes however Groups E and D had lower heart rates compared to Group C starting from the 6th minute until the end of study period (Figure 4). There were no hypotension, bradycardia or ECG changes that required treatment in any patients in 20 minutes.

DISCUSSION

This study has showed that serum IMA levels increased as a result of lower heart rate and blood pressure due to dexmedetomidine and esmolol utilization for the attenuation of hypertension and tachycardia caused by intubation.

Dexmedetomidine is a selective and specific alfa-2 agonist which lowers the central sympathetic outflow and decreases serum epinephrine and norepinephrine levels in a dose dependent manner. In addition to being a potent sedative, it also lowers anesthetic requirements (6). The cardiovascular effects of dexmedetomidine appear in a short, biphasic and

Table I. Demographic Data

	Group C (n=21)	Group E (n=21)	Group D (n=21)	p
Age (years)	38.8 ± 13.3	42.9 ± 18.2	40.1 ± 15.6	0.692
Weight (kg)	74.2 ± 13.3	69.3 ± 9.8	69.2 ± 11.1	0.273
Height (cm)	166.7 ± 10.4	165.8 ± 7.1	164.6 ± 5.0	0.688
BMI (kg m^{-2})	26.7 ± 3.8	25.3 ± 4.0	25.5 ± 3.9	0.493
Sex (M/F)	7/14	9/12	16/5	0.121
ASA I/II	4/17	4/17	5/16	0.901
Baseline SpO_2 (%)	98.7 ± 1.3	97.1 ± 4.1	97.8 ± 2.1	0.185
Baseline HR (beats min^{-1})	88.9 ± 12.4	91.1 ± 11.2	83.3 ± 10.5	0.082
Baseline MAP (mmHg)	97.3 ± 20.9	104.9 ± 17.6	98.1 ± 18.2	0.365

BMI: Body-mass index, **M:** Male, **F:** Female, **ASA:** American Society of Anesthesiologists, **SpO_2:** Peripheral oxygen saturation, **HR:** Heart rate, **MAP:** Mean arterial pressure.

Table II. Ischemia Modified Albumin Values at Baseline, 10th and 20th Minutes Following Intubation

	Baseline	Intubation +10 min	Intubation +20 min	p
Group C	0.2326 ± 0.098	0.2452 ± 0.082	0.2269 ± 0.096	**0.015***
Group E	0.2554 ± 0.078	0.2739 ± 0.083	0.2914 ± 0.058	**0.025****
Group D	0.2842 ± 0.048	0.2730 ± 0.049	0.2871 ± 0.049	0.980***

*: p value at 20th minute between Groups C and E, **: p value at 20th minutes between Groups C and D, ***: p value at 20th minutes between Groups D and E.

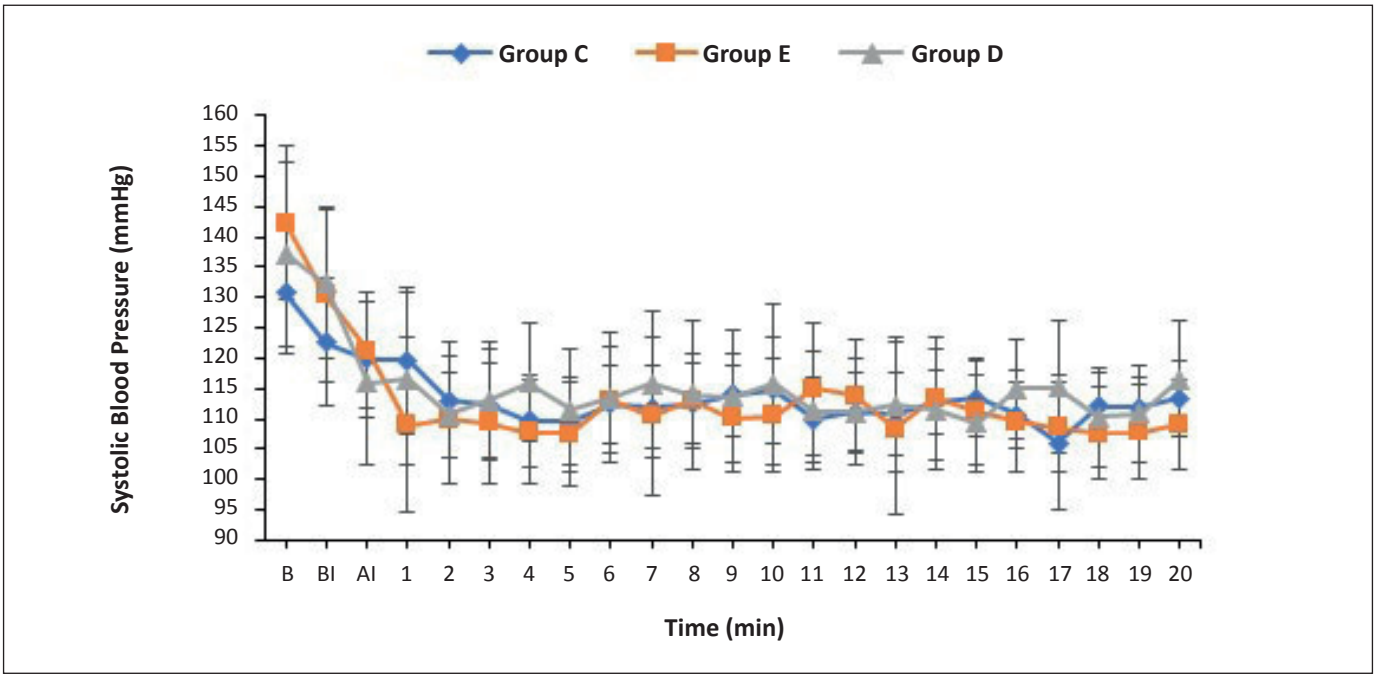

Figure 1. Systolic blood pressure (mmHg). **B:** Basal values, **BI:** Before intubation, **AI:** After intubation.

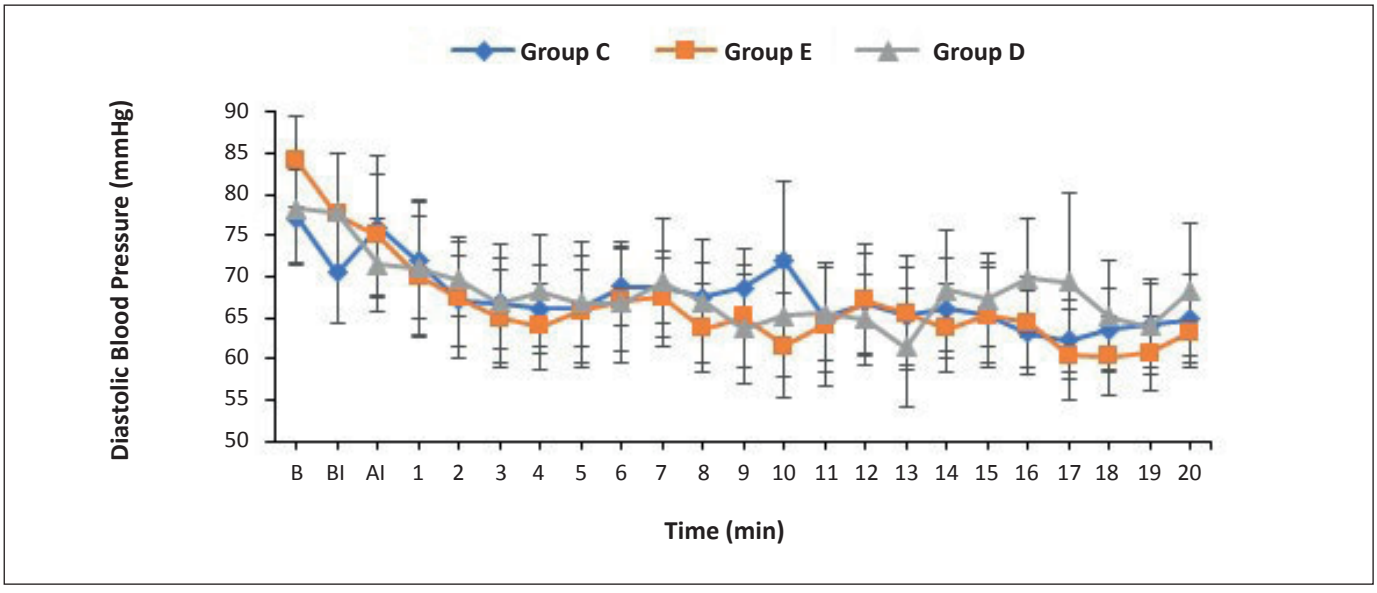

Figure 2. Diastolic blood pressure (mmHg). **B:** Basal values, **BI:** Before intubation, **AI:** After intubation.

dose dependent manner following administration. Different doses of dexmedetomidine have been used in studies aiming to prevent hemodynamic responses. Mudassir et al. have compared the effects of 0.5 μg kg^{-1} and 1 μg kg^{-1} dexmedetomidine given 10 minutes before intubation on hemodynamic response and have reported that 0.5 μg kg^{-1} dexmedetomidine given as slow bolus was as effective as 1 μg kg^{-1} in protecting hemodynamics and they have not encountered a drop in blood pressure or heart rate. (7). Demiri et al. in their meta-analysis, have reported that perioperative dexmedetomidine less than 0.5 μg kg^{-1} as bolus or infusion does not cause intraoperative hypotension or postoperative bradycardia (8). Similarly, Mahiswar et al. have compared 0.5 μg kg^{-1} dexmedetomidine and 2 μg kg^{-1} fentanyl given before intubation and have reported that they had similar efficiency where prevention of hemodynamic response to intubation was concerned and no patients in the study required rescue medication (9). Roy et al. have compared 0.5 μg kg^{-1} dexmedetomidine given as a bolus in 10 minutes to 0.5 mg kg^{-1} esmolol and have found dexmedetomidine to be more

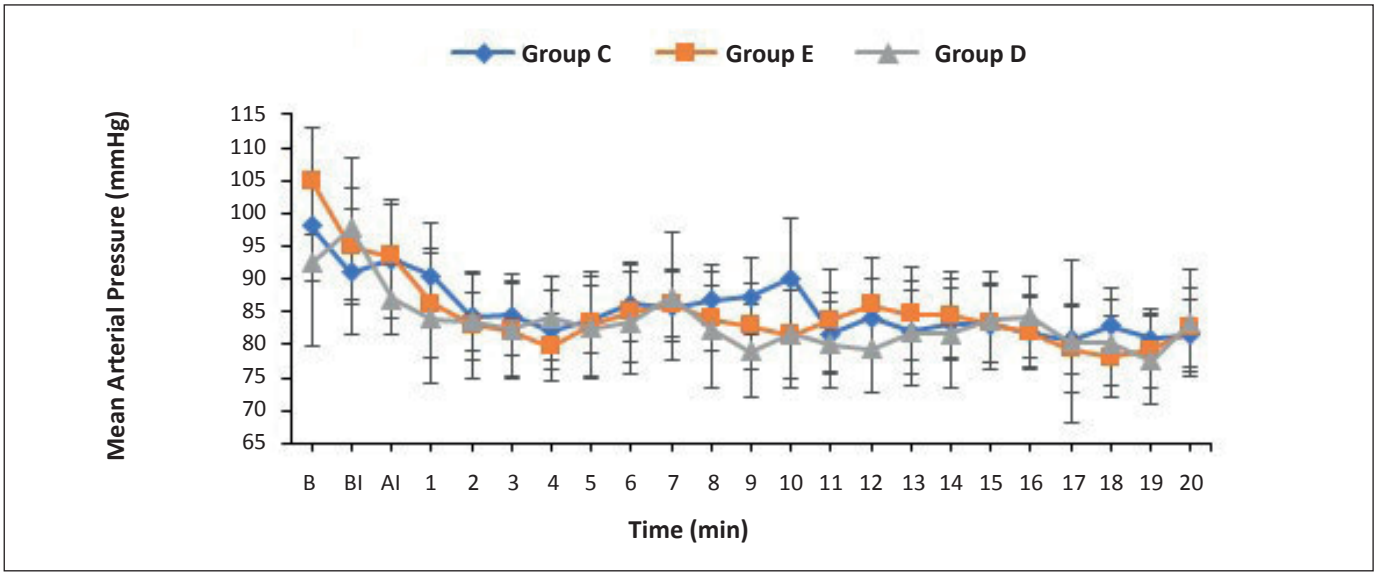

Figure 3. Mean arterial pressure (mmHg). **B:** Basal values, **BI:** Before intubation, **AI:** After intubation

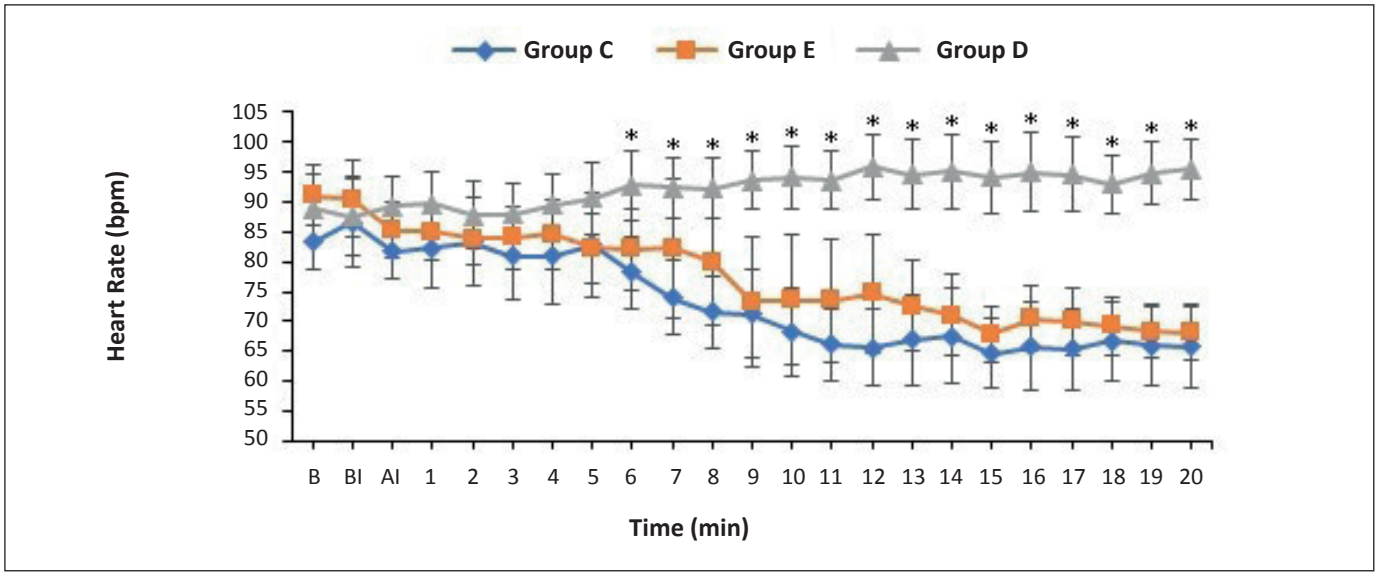

Figure 4. Heart rate (beats per minute). **B:** Basal values, **BI:** Before intubation, **AI:** After intubation. ***** Statistically significant differences have been marked.

effective in protecting hemodynamic stability (10). However, Basar et al. have applied dexmedetomidine 0.5 µg kg^{-1} as slow bolus over 60 seconds, 10 minutes before induction and have reported a significant drop in blood pressure and heart rate following bolus application, during intubation and 10 minutes following intubation (11). Sulaiman et al. have compared the application of 0.5 µg kg^{-1} dexmedetomidine as slow bolus 10 minutes before intubation to placebo and have reported lower heart rate and systolic blood pressures following intubation throughout the study period (12). We observed a significant drop in heart rate and blood pressure following 0.5 µg kg^{-1} dexmedetomidine infusion compared to control in regard to baseline values although there was no difference between esmolol and dexmedetomidine, and none of the patients in our study required any intervention for hemodynamic status which indicates that these doses are sufficient for controlling the hemodynamic response.

Esmolol is a selective ß1 adrenergic receptor blocker with a very short duration of effect. It has a distribution half-life of 2 minutes and an elimination half-life of 9 minutes. Although there are various studies evaluating the effect of esmolol suppressing hemodynamic response, there is no consensus on the ideal timing and dosage. Song et al have stated that both

0.5 and 1 mg kg^{-1} esmolol administered before intubation effectively suppressed hemodynamic response to intubation (13). Similarly, Erbesler et al. have stated that 0.5 mg kg^{-1} esmolol and 0.5 µg kg^{-1} dexmedetomidine given 10 minutes before induction resulted in similar hemodynamic outcomes (14). Selvaraj and Manoharan have investigated the effects of 1 µg kg^{-1} dexmedetomidine and 0.5 mg kg^{-1} esmolol on response to intubation and reported that esmolol given as a single bolus was more effective at providing a stable hemodynamic status compared to 1 µg kg^{-1} dexmedetomidine (15). This study shows that esmolol was effective even if infusion was not continued after induction. We found a drop in systolic, diastolic and mean arterial pressures compared to baseline when 0.5 mg kg^{-1} esmolol loading dose was given over 5 minutes as slow infusion before intubation but these changes did not necessitate any pharmacological interventions which indicates that these patients had a hemodynamically stable course.

An overview of past studies on IMA reveals that it has been mostly used as a marker of myocardial ischemia in situations such as acute coronary syndrome (16). Additionally, the rise in IMA levels following strenuous exercise where the workload of the myocardium is increased has showed that ischemic situations can arise in healthy subjects when oxygen delivery is decreased (17). Based on these points, we believe that IMA can be used as an early warning factor when oxygen delivery is weakened as a result of the suppression of sympathetic response during intubation.

Laryngoscopy and tracheal intubation cause significant alterations in heart rate and blood pressure which in turn disrupts the myocardial oxygen supply-demand equilibrium. Since tachycardia causes more myocardial ischemia compared to hypertension, beta-adrenergic antagonists have been subjects of interest in inhibiting the cardiac responses. The degree of reflex response to laryngeal stimulation depends on the difficulty and duration of intubation in addition to patient-related factors such as diabetes or cardiovascular diseases. Although the suppression of hemodynamic responses to intubation is a desired endpoint, sympatholytic agents used for this end could cause hypotension, bradycardia and myocardial depression when combined with anesthetic agents and opioids (18).

There is no study which evaluates the normal reference values for IMA as the majority of literature compares ischemic patients to a healthy control population. Türedi et al. report average IMA levels in the control group as 0.160 ABSU while this value is reported as 0.172 by Gunduz et al. (19,20). The cut-off value for IMA in ischemic conditions has been reported as 0.400 ABSU (4,21). In our study, the average baseline IMA values in ASA I-II patients without a known ischemic disease was 0.257 and IMA levels showed a significant rise at 20 minutes after intubation due to lower heart rate and blood pressure in addition to a drop in myocardial oxygen supply as a result of the synergistic effect of anesthetic and sympatholytic agents. IMA measurement was performed in the after monitorization, 10^{th} and 20^{th} minutes following intubation. The rationale behind this was the fact that the hemodynamic changes caused by intubation were most frequently observed in the first 30 minutes following intubation (22). We aimed to detect the effect of intubation and hemodynamic changes on IMA levels by performing the measurement at these time points. This shows that the transient effects of the combination of sympatholytic and anesthetic agents can cause ischemia even in a healthy population. The lack of hemodynamic deterioration can be expected when the study population and their ASA status are considered. Although IMA levels at the 20th minute alone can not indicate organ hypoperfusion, we believe that perioperative fluctuations can be regarded as a warning for the myocardium.

The limitations of our study are the lack of 24^{th} hour IMA levels and cardiac output or other advanced hemodynamic monitoring techniques used at the measurement time points. Additionally, the lack of postoperative ECG and Troponin levels can be regarded as a limitation.

As a result, the inhibition of hemodynamic response to intubation can have negative results and pharmacological interventions to this end should be carried out carefully in critical patients and the importance of close hemodynamic monitorization should be kept in mind.

CONCLUSION

Future studies aiming to elucidate ischemic conditions which can be caused by sympatholytic agents used to suppress hemodynamic response to intubation should focus on optimal dosage, technique and timing using advanced hemodynamic monitorization techniques and tissue specific biomarkers.

AUTHOR CONTRIBUTIONS

Conception or design of the work: HY, TC

Data collection: HY, PE, TC, MAY

Data analysis and interpretation: HY, PE, BKK

Drafting the article: HY, BKK, UCK

Critical revision of the article: PE, FT

Other (study supervision, fundings, materials, etc): HY, BKK, UCK, PE, TC, MAY, FT

All authors (HY, BKK, UCK, PE, TC, MAY, FT) reviewed the results and approved the final version of the manuscript.

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