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Optimal Dose of Dexmedetomidine for Attenuation of Endotracheal Intubation Pressor Response Using Cardiometry: A Randomized, Double-Blinded Study

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Abstract:

Background: Dexmedetomidine (DEX) effectively counteracts the hypertension induced by laryngoscopy and endotracheal intubation (ETI), minimizing the hemodynamic stress associated with these procedures. The study aimed to determine the optimal dose of dexmedetomidine for attenuating the hemodynamic pressor response to laryngoscopy and ETI. **Methods**: This randomized, prospective study, double-blinded study enrolled 60 patients between the ages of 18 and 60, including both sexes, with a type I or II physical state as defined by the American Society of Anesthesiology, having elective surgery while under general anesthesia while receiving ETI. Patients were randomized into two equal groups. Group A received 0.5 mcg/kg DEX, while Group B received 1 µg/kg DEX. DEX was diluted in 50 ml of normal saline and administered over 10 minutes as a single dose. **Results**: Group B had a much lower heart rate (HR) and cardiac output (CO) compared to group A at six measurements after induction and before laryngoscopy, and in ten readings following ETI ($P<0.05$). The systolic (SBP) and diastolic blood pressure (DBP) readings were considerably lower in group B compared to group A during three readings after induction and before laryngoscopy, as well as five readings following ETI ($P<0.05$). Stroke volume (SV) measurements were comparable between both groups. **Conclusions**: DEX 1 mcg/kg is superior to 0.5 mcg/kg in the hemodynamic pressor response to laryngoscopy and intubation attenuation as evidenced by lowering HR, SBP, DBP, and CO without a significant difference in SV.

Keywords: Cardiometery, Dexmedetomidine, Endotracheal intubation, Hemodynamic stress.

1. Introduction

Endotracheal intubation (ETT) and direct laryngoscopy trigger hemodynamic changes caused by increased sympathetic nervous system activity, resulting in potential episodes of hypertension and tachycardia. While these hemodynamic fluctuations are usually transient, they may lead to negative consequences, including hypertensive emergencies, arrhythmias, myocardial ischemia, or elevated intracranial pressure, posing a particular risk for patients with pre-existing cardiac conditions. (1)

Several drugs have been investigated for their potential to alleviate the stress response with variable results. These drugs include opioids, local anesthetics, calcium channel, and beta blockers. (2, 3, 4, 5)

Dexmedetomidine is an alpha-2 receptor agonist that is gaining widespread popularity in perioperative use. The pharmacodynamics of dexmedetomidine promote its usage as an anesthetic adjuvant as it decreases the analgesic requirements, and has amnesic properties, and sympatholytic properties. (6). Also, these properties enable it to blunt the stress response associated with laryngoscopy and endotracheal intubation. It was shown that it may lower the HR (heart rate), MAP (mean arterial blood pressure), and CO (cardiac output) in response to laryngoscopy and ETT. (7) Also, its sedative properties decrease the anesthetic requirements in the perioperative time. (8)

Electrical cardiometry is a non-invasive cardiovascular monitoring device that measures beat-by-beat changes in cardiac output (CO). It assesses various hemodynamic variables, including CO, stroke volume (SV), and systemic vascular resistance. Electrical cardiometry provides accurate and reliable measurements of CO with high sensitivity and specificity. (9)

Even though different doses of dexmedetomidine have been shown to effectively decrease the hemodynamic pressor response in adult patients. (10,11,12). Yet, upon reviewing the literature, the absence of including CO monitoring devices to detect minimal fluctuations of hemodynamics during laryngoscopy and ETT was noticed.

This study aims to find out whether the dose of dexmedetomidine (0.5 μg/kg or 1.0 μg/kg) had a more stable hemodynamic profile during laryngoscopy and intubation by using electrical cardiometery.

Sample size calculation:

The program G*Power 3.1.9.2 from the University of Kiel in Germany was used to calculate the sample size. A pilot study was conducted, enrolling five participants in each group, and the mean (± standard deviation) HR recorded one-minute post-intubation (the primary outcome) was 76.4 ± 5.17 in group A and 70.4 ± 8.9 in group B. The sample size was computed using the following parameters: effect size of 0.824, 95% confidence level, 80% study power, a 1:1 group ratio, and an extra five individuals in each group to cover any possible attrition. Subsequently, 30 patients were joined in each group.

Methods:

This is a prospective, randomized double-blind control study that was conducted in the Department of Anesthesia and Surgical Intensive Care Unit at Theodor Bilharz Research Institute after approval by the research ethics committee (PT 811) and patient informed consent. The trial was registered at ClinicalTrials.gov ID: NCT06592027.

This study enrolled 60 patients between the ages of 18 and 60, including both sexes, with an ASA (American Society of Anesthesiologists) I or II undergoing elective surgery that required general anesthesia with ETT. Patients were excluded from the study if they were undergoing emergency surgery, had full stomach, pregnant females, had preexisting renal or hepatic diseases, and those on regular use of calcium channel or beta blockers. Patients with a history of difficult intubation or suspected difficult airway such as obese patients with body mass index (BMI) ≥ 30 kg/m^2, large neck circumference, and limited cervical movement

were also excluded from the study. Additionally, patients with known dexmedetomidine hypersensitivity or contraindications such as known psychiatric, neuromuscular, or neurological disorders were also excluded.

The randomization process was done using computer-generated numbers which were employed to allocate patients into two groups in a parallel manner. The allocation ratio was 1:1 with each patient's group assignment kept in a sealed opaque envelope. For double blinding, two investigators participated in this study; an anesthesiologist who was not part of the study, was responsible for drug preparation and the other anesthesiologist who was unaware of group allocation was responsible for the data collection and analysis.

Patients were allocated to either group A which received a single dose of 0.5 µg/ kg IV dexmedetomidine in 50 ml normal saline over 10 minutes, while group B received 1 µg/kg IV dexmedetomidine in 50 ml normal saline over 10 minute.

Anesthesia Technique:

A preoperative assessment, including a history, physical examination, review of laboratory data, and assignment of ASA classification was performed on all patients before the procedure. Anesthesia and procedural consent were obtained.

Upon arrival to the operating room, basic monitoring as Electrocardiography (ECG), Non-invasive Blood Pressure (NIBP) monitor and pulse oximetry (Spo2), neuromuscular monitor (TOF) were applied and baseline readings were recorded.

Hemodynamic monitoring system ICON ® (Osyka Medical GmbH, Berlin, Germany, model C3) manufactured by ICON Cardiotronics, Inc. of La Jolla, CA 92307 was applied for continuous SV and CO monitoring. One electrical cardiometer sensor was placed 5 cm from the base of the neck, another on the base of the neck, a third at the level of the xiphoid process in the lower thorax, and a fourth 5 cm below the third electrode at the anterior axillary.

After obtaining hemodynamic baseline readings, dexmedetomidine infusion was started according to the group allocation. Group A received IV infusion of dexmedetomidine 0.5 µg /kg in 50 ml normal saline over 10 minutes and Group B received IV infusion of dexmedetomidine 1µg /kg in 50 ml normal saline over 10 minutes. At the end of dexmedetomidine infusion, HR, SBP, DBP, MAP, SV, and CO were recorded.

All patients were preoxygenated with four or five breaths of 100% oxygen. Induction of general anesthesia was done using IV 1 mg/kg propofol,1 µg/kg fentanyl, and 0.5 mg/kg atracurium. After 3 minutes of mask ventilation with 1 MAC (minimum alveolar concentration) of sevoflurane, Endotracheal intubation (ETT) was performed by an experienced anesthesiologist. Patients who experienced prolonged laryngoscopy for more than 15 seconds or developed bronchospasm or laryngospasm were excluded from the study.

If SBP decreased to below 90 mmHg or the MAP decreased by 20% from baseline, an IV ephedrine bolus of 5-10 mg was administered. If HR dropped to levels below 50 beats/minute, IV 0.5 mg atropine was given.

The HR, CO, and SV measurements were recorded at baseline, after drug infusion, six readings (T1:T6) after induction, before laryngoscopy, and a period of 30 seconds beginning with the baseline reading and continuing for 5 minutes after the end of the ETI. Systolic (SBP) and diastolic blood pressure (DBP), mean arterial pressure (MAP), SV, and CO were documented at baseline, after drug infusion, three readings after induction, and before laryngoscopy (T1:T3) and 5 minutes after ETI at 1-minute intervals.

The pressor response, defined as an increase in HR, CO, and SBP of 20% or more from baseline, was assessed after ETI for 5 minutes.

The primary outcome was the measurement of HR taken one minute after intubation. The secondary outcomes included HR, CO, SV, SBP, and DBP at other times.

Statistical analysis

We used IBM's SPSS 27 (Armonk, NY, USA) statistical software to conduct the analysis. The Shapiro-Wilk test was employed to check data distribution normality, which was also visualized using histograms. Parametric quantitative data were represented by the mean and standard deviation (SD), and we used the unpaired t-test to compare the groups. Qualitative data were shown as frequencies and %, and the chi-square test was used for analysis, with Fisher's exact test applied in cases of small sample sizes. For statistical purposes, a two-tailed p-value less than 0.05 was deemed significant.

Results:

In the current study, 74 patients were initially evaluated for participation. However, nine patients were not eligible for inclusion, and five declined to participate. As a result, 60 patients were divided equally between the two groups and were subsequently followed up and continued with statistical analysis. (Figure 1)

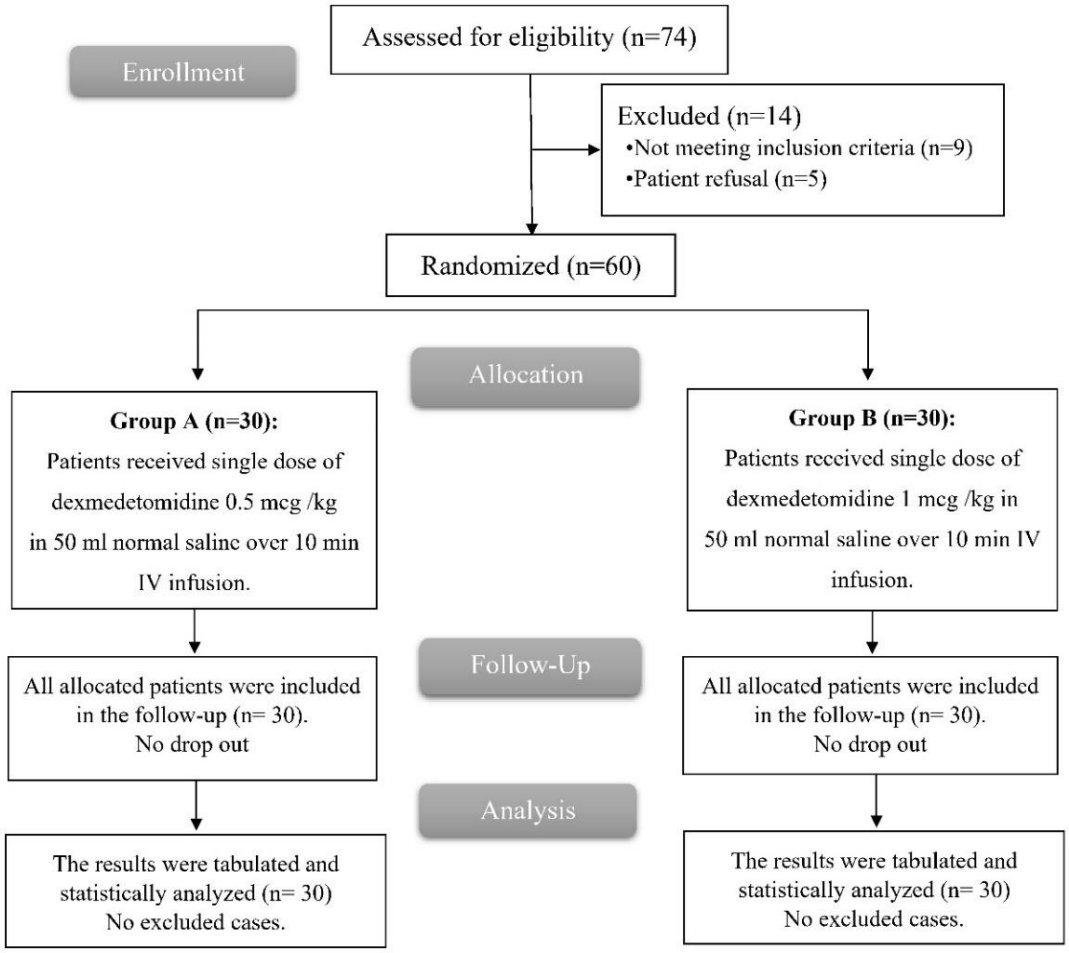

Figure 1: CONSORT flowchart of the enrolled patients.

The demographic data, ASA physical status, and duration of surgery were insignificantly different between the two groups. **Table 1**

Table 1: Demographic data and duration of surgery of the studied groups

		Group A (n=30)	Group B (n=30)	P value
Age (years)		39.77 ± 9.5	41.57 ± 8.11	0.433
Sex	**Male**	17 (56.67%)	13 (43.33%)	0.302
	Female	13 (43.33%)	17 (56.67%)	
Weight (kg)		73.9 ± 9.02	75.13 ± 6.79	0.552
Height (cm)		168.07 ± 6.77	169.1 ± 6.19	0.540
BMI (kg/m^2)		26.28 ± 3.78	26.35 ± 2.82	0.932
ASA physical status	**I**	12 (40%)	10 (33.33%)	0.592
	II	18 (60%)	20 (66.67%)	
Duration of surgery (min)		99.33 ± 17.8	95.67 ± 19.51	0.450

Data are presented as mean ± SD or frequency (%). BMI: Body mass index, ASA: American Society of Anesthesiologists.

Analysis of HR measurements showed the following. HR measurements were insignificantly different at baseline and immediately after dexmedetomidine infusion between the two groups. The HR measurements were significantly lower at the 6-time points from the induction of general anesthesia till laryngoscopy and ETT in group B than in group A with a p-value < 0.05. Also, there was a significant decrease in HR measurements in the 10 time points after ETT in group B than in group A with a p-value <0.05. **Table 2**

Table 2: Heart rate measurements of the studied groups

		Group A (n=30)	Group B (n=30)	P value
Baseline		77.43±8.84	77.9±7.72	0.828
After drug infusion		73±8.8	70.9±8.39	0.348
After induction and before laryngoscopy	**T1**	74.93±8.75	67.33±8.47	**0.001***
	T2	76±8.99	68.87±8.37	**0.002***
	T3	77.27±9.04	69.4±8.3	**0.001***
	T4	77.9±9.04	69.9±8.26	**0.001***
	T5	77.73±8.64	70.5±8.17	**0.002***
	T6	79±9.06	70.97±8.24	**0.001***
After ETI	**0.5min**	79.3±8.91	71.43±8.39	**0.001***
	1min	76.97±8.82	70.37±8.71	**0.005***
	1.5min	77.97±8.72	70.83±8.69	**0.002***
	2min	77.93±8.89	71.33±8.84	**0.006***
	2.5min	78.27±8.71	70.83±8.87	**0.002***
	3min	77.93±9.02	70.5±8.93	**0.002***
	3.5min	76.83±9.48	70.6±8.49	**0.010***
	4min	76.9±8.9	68.67±8.36	**<0.001***
	4.5min	74.8±8.85	66.63±8.25	**<0.001***
	5min	73.5±8.85	65.1±8.32	**<0.001***

Data are presented as mean ± SD. *: Significant as p value <0.05. ETT: Endotracheal intubation. T1:T6 are six readings after induction and before laryngoscopy.

Concerning the SBP and DBP measurements. They were comparable at baseline reading and immediately after dexmeditomidine infusion. The readings of SBP and DBP were significantly lower at the 8 recorded time points from induction of anesthesia till 5 minutes after ETT insertion in group B than in group A with a p-value <0.05. Table 3 and Table 4.

Table 3: Systolic blood pressure measurements of the studied groups

		Group A (n=30)	**Group B (n=30)**	**P value**
Baseline		129.1±4.44	130.47±6.59	0.350
After drug infusion		127.17±4.55	126.67±6.57	0.733
After induction and before laryngoscopy	**T1**	125.8±4.6	122.93±6.08	**0.044***
	T2	131.3±4.47	125.13±5.97	**<0.001***
	T3	131.23±4.17	127.17±6.04	**0.004***
After ETI	**1min**	128.9±4.47	125.5±6.11	**0.017***
	2min	127.17±4.36	123.1±6.12	**0.004***
	3min	127.77±4.38	122.57±5.93	**<0.001***
	4min	127.57±4.61	123.17±6.66	**0.004***
	5min	127.63±4.48	123.7±6.65	**0.009***

Data are presented as mean ± SD. *: Significant as p value <0.05.ETT: Endotracheal intubation. T1:T6 are three readings after induction and before laryngoscopy.

Table 4: Diastolic blood pressure measurements of the studied groups

		Group A (n=30)	**Group B (n=30)**	**P value**
Baseline		129.1±4.44	130.47±6.59	0.350
After drug infusion		127.17±4.55	126.67±6.57	0.733
After induction and before laryngoscopy	**T1**	125.8±4.6	122.93±6.08	**0.044***
	T2	131.3±4.47	125.13±5.97	**<0.001***
	T3	131.23±4.17	127.17±6.04	**0.004***
After ETI	**1min**	128.9±4.47	125.5±6.11	**0.017***
	2min	127.17±4.36	123.1±6.12	**0.004***
	3min	127.77±4.38	122.57±5.93	**<0.001**
	4min	127.57±4.61	123.17±6.66	**0.004***
	5min	127.63±4.48	123.7±6.65	**0.009***

Data are presented as mean ± SD. *: Significant as p value <0.05. ETT: Endotracheal intubation. T1:T6 are three readings after induction and before laryngoscopy.

The electrical cardiometry measured variables showed the following: the SV measurements were insignificantly different at baseline reading and immediately after dexmetomidine infusion between both groups. Also, the recorded SV readings at 16 different time points from induction of general anesthesia till 5 minutes after ETT insertion were insignificantly different between the studied groups. Table 5

Table 5: Stroke volume measurements of the studied groups

		Group A (n=30)	Group B (n=30)	P value
Baseline		69.87±6.16	72.63±11.53	0.251
After drug infusion		66.23±6.26	65.17±11.38	0.654
After induction and before laryngoscopy	**T1**	69.23±6.2	65.17±11.27	0.089
	T2	70.27±6.01	65.63±11.8	0.060
	T3	71.53±6.44	66.93±11.59	0.062
	T4	71.93±6.45	67.13±11.75	0.055
	T5	72.27±6.58	68.97±11.85	0.188
	T6	73.57±6.65	69.6±11.82	0.115
After ETI	**0.5min**	74.03±6.45	70.2±11.85	0.125
	1min	74±6.07	70.8±11.89	0.194
	1.5min	74.23±6.23	70.23±12.07	0.112
	2min	74.17±6.14	69.7±12.05	0.076
	2.5min	74.97±6.34	71.17±12.15	0.134
	3min	74.63±5.95	70.73±12.11	0.119
	3.5min	73.83±6.61	69.27±12.2	0.077
	4min	71.87±6.32	67.3±12.26	0.075
	4.5min	72.6±6.14	68.5±12.29	0.107
	5min	71.83±6.23	67.97±12.32	0.131

Data are presented as mean ± SD. ETI: Endotracheal intubation. T1:T6 is the readings after induction and before laryngoscopy.

The second measured variable by the electrical cardiometry was CO and it showed the following. The CO measurements were insignificantly different at baseline reading and immediately after dexmedetomidine infusion between the two groups. However, the following 16-time point measurements of CO from induction of general anesthesia till 5 minutes after insertion of ETT were significantly lower in group B than in group A with a p-value < 0.05. Table 6

Table 6: Cardiac output measurements of the studied groups

		Group A (n=30)	Group B (n=30)	P value
Baseline		5408.97±777.08	5641.83±959.29	0.306
After drug infusion		4833.87±736.34	4604.1±886.24	0.279
After induction and before laryngoscopy	**T1**	5187.03±760.42	4374.53±874.34	**<0.001***
	T2	5341.73±793.93	4497.27±866.69	**<0.001***
	T3	5525.33±810.7	4638.43±881.53	**<0.001***
	T4	5603.5±830.19	4626.57±877.73	**<0.001***
	T5	5618.73±817.19	4840±889.13	**0.001***
	T6	5807.23±826.66	4915.67±890.67	**<0.001***
After ETI	**0.5min**	5867.23±806.44	4989.33±896.22	**<0.001***
	1min	5698.8±835.09	4954.87±895.6	**0.002***
	1.5min	5785.83±798.3	4947.07±907.75	**<0.001***
	2min	5781±829.48	4944.4±919.76	**<0.001***
	2.5min	5865.93±814.96	5014.43±933.51	**<0.001***
	3min	5818.5±836.25	4961.8±939.15	**<0.001***
	3.5min	5670.33±857.76	4865.47±919.77	**0.001***
	4min	5522.57±780.15	4596.37±890.52	**<0.001***
	4.5min	5427.73±778.11	4538.7±866.84	**<0.001***
	5min	5279.57±790.4	4398.27±857.73	**<0.001***

Data are presented as mean ± SD. *: Significant as p value <0.05.; ETT: Endotracheal intubation. T1:T6 are six readings after induction and before laryngoscopy.

Discussion:

Dexmeditomidine provides a range of benefits, including sedation, analgesia, sympatholysis, and cardiovascular stability, all while minimizing the risk of respiratory depression. (13) While the evidence suggested that dexmedtomidine successfully decreases the stress response to intubation, the optimal dose remains unclear.

In the current study, we compared a single IV infusion of 2 different doses of dexmedetomidine (0.5 and 1 µg/kg) over 10 minutes. This previously mentioned approach was investigated in the previous research and was proven to avoid the biphasic response of dexmedetomidine when it is rapidly infused. (14,15)

Our study showed that HR, CO, SBP, and DPB measurements recorded at different time points from induction of general anesthesia till 5 minutes after ETT were notably decreased in the 1 µg/kg group compared to the 0.5 µg/kg group. However, the SV measurements recorded at the same time points were comparable between both groups.

Vashisht et al. reported the same results and demonstrated that 1 µg of dexmeditomidine significantly decreased HR, SBP, and DPB measurements than 0.5 µg. (16) . Also, Jain et al. noticed that HR, SBP, and DPB measurements were significantly lower with dexmedetomidine 1 µg/kg than with dexmedetomidine 0.5 µg. (10)

Also, a previous study by Silpa et al. stated that 1 µg/kg dexmeditomidine was superior to 0.5 µg/kg in blunting the intubation-related hemodynamic stress response during cardiac surgeries. (17) Moreover, Keniya et al. reported that dexmedtomidine 1 µg/kg significantly decreased the hemodynamic reaction to intubation and laryngoscopy as compared to the control group. (18) Similarly, Bajwa et al. found that dexmedtomidine at 1 µg/kg was more effective than fentanyl in reducing the stress response related to ETT. (19)

The significant decrease in HR and blood pressure noticed in the 1 µg/kg dexmedetomidine group aligns with the known pharmacological effects of dexmedetomidine. As noted by Afonso and Reis, dexmedetomidine's action on α_2-adrenergic receptors leads to decreased sympathetic outflow and increased vagal activity, which results in bradycardia and hypotension. (20)

The use of electrical cardiometry in the current study allowed for continuous, non-invasive CO and SV monitoring, providing a more comprehensive assessment of hemodynamic changes during ETI. This approach offers advantages over traditional monitoring methods, as highlighted by Peyton and Chong, who emphasized the importance of continuous CO monitoring in perioperative care. (21)

Interestingly, while CO was significantly lower in the 1 µg/kg dexmedtomidine group, SV remained relatively constant between the two groups. This suggests that the reduction in CO was primarily due to the decrease in HR rather than a change in contractility. This observation agreed with the trial of Snapir and colleagues, which showed that DEX primarily affects HR without significantly impacting myocardial contractility. (22) This is supported by Lee et al., who showed no differences in biventricular systolic and diastolic function between the dexmedetomidine and saline groups. (23) Moreover, they found that the dexmedetomidine and saline groups exhibited no substantial differences in stroke volume. (23)

However, the small sample size and renal or hepatic impairments exclusion make it challenging to apply these results universally. The study focused only on the immediate hemodynamic responses without assessing potential long-term effects or adverse reactions associated with the different dexmedetomidine dosages. Given these findings and their limitations, it is recommended that future research should incorporate longer-term outcomes and side effect profiles.

Conclusions:

Dexmedetomidine as 1 μg/kg is superior to 0.5 μg/kg for attenuation of hemodynamic pressor response to laryngoscopy and ETT, as evidenced by lower HR, SBP, DBP, and CO.

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