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Ketamine Is Not Associated With More Post-Intubation Hypotension Than Etomidate In Patients Undergoing Endotracheal Intubation

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Abstract

Introduction: Emergency department (ED) patients undergoing emergent tracheal intubation often have multiple physiologic derangements putting them at risk for post-intubation hypotension. Prior work has shown that post-intubation hypotension is independently associated with increased morbidity and mortality. The choice of induction agent may be associated with post-intubation hypotension. Etomidate and ketamine are two of the most commonly used agents in the ED, however, there is controversy regarding whether either agent is superior in the setting of hemodynamic instability. The goal of this study is to determine whether there is a difference in the rate of post-intubation hypotension who received either ketamine or etomidate for induction. Additionally, we provide a subgroup analysis of patients at pre-existing risk of cardiovascular collapse (identified by pre-intubation shock index (SI) > 0.9)to determine if differences in rates of post-intubation hypotension exist as a function of sedative choice administered during tracheal intubation in these high-risk patients. We hypothesize that there is no difference in the incidence of post-intubation hypotension in patients who receive ketamine versus etomidate.

Methods: A retrospective cohort study was conducted on a database of 469 patients having undergone emergent intubation with either etomidate or ketamine induction at a large academic health system. Patients were identified by automatic query of the electronic health records from 1/1/2016 - 6/30/2019. Exclusion criteria were patients <18-years-old, tracheal intubation performed outside of the ED, incomplete peri-intubation vital signs, or cardiac arrest prior to intubation. Patients at high risk for hemodynamic collapse in the post-intubation period were

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identified by a pre-intubation SI > 0.9. The primary outcome was the incidence of post-intubation hypotension (systolic blood pressure < 90 mmHg or mean arterial pressure < 65 mmHg). Secondary outcomes included post-intubation vasopressor use and mortality. These analyses were performed on the full cohort and an exploratory analysis in patients with SI > 0.9. We also report adjusted odds ratios (aOR) from a multivariable logistic regression model of the entire cohort controlling for plausible confounding variables to determine independent factors associated with post-intubation hypotension.

Results: A total of 358 patients were included (etomidate: 272; ketamine: 86). The mean pre-intubation SI was higher in the group that received ketamine than etomidate, (0.97 vs. 0.83, difference: −0.14 (95%, CI −0.2 to −0.1). The incidence of post-intubation hypotension was greater in the ketamine group prior to SI stratification (difference: −10%, 95% CI −20.9% to -0.1%). Emergency physicians were more likely to use ketamine in patients with SI > 0.9. In our multivariate logistic regression analysis, choice of induction agent was not associated with post-intubation hypotension (aOR 1.45, 95% CI 0.79 to 2.65). We found that pre-intubation shock index was the strongest predictor of post-intubation hypotension.

Conclusion: In our cohort of patients undergoing emergent tracheal intubation, ketamine was used more often for patients with an elevated shock index. We did not identify an association between the incidence of post-intubation hypotension and induction agent between ketamine and etomidate. Patients with an elevated shock index were at higher risk of cardiovascular collapse regardless of the choice of ketamine or etomidate.

Keywords

ketamine; etomidate; post-intubation hypotension; shock index; rapid sequence intubation

1. Introduction

The ideal induction agent during tracheal intubation remains controversial. Etomidate and ketamine are the most widely used induction agents for rapid sequence intubation (RSI) in the ED, due in part to their superior hemodynamic stability [1-3]. Etomidate, the most frequently used induction agent in RSI, is a GABAergic compound while ketamine displays a diverse pharmacodynamic profile including NMDA antagonism [2, 4-5]. These agents have been the subject of controversy regarding their side effects [6-16]. Etomidate is a reversible inhibitor of 11-beta-hydroxylase and has been found to cause transient adrenocortical suppression. Ketamine has sympathomimetic properties secondary to the indirect release of catecholamines that may increase blood pressure [17]. Particularly in hypotensive patients, ketamine may offer added hemodynamic support and thus has seen an increase in utilization for RSI induction [1, 4, 18-24]. However, ketamine may cause direct myocardial depression in certain patients and has been reported to cause hypotension and cardiac arrest [16, 18-19, 25-29].

Post-intubation hypotension following the administration of a sedative during RSI is well-described. The proposed mechanism for post-intubation hypotension includes decreased preload from positive pressure ventilation, vasoplegia, myocardial depression, and catecholamine blunting due to the sedative induction agent [30-32]. Recent data has shown

that post-intubation hypotension is independently associated with peri-intubation cardiac arrest and increased mortality, making it a significant marker for poor outcomes [30-34]. Consequently, determining which induction agent, if any, best maintains hemodynamic stability during tracheal intubation is of great value to the emergency medicine physician.

Recently, a study from the National Emergency Airway Registry (NEAR) showed that ketamine was associated with a greater risk of post-intubation hypotension in normotensive ED patients [26], although the authors did not include patients with pre-existing hemodynamic instability. There is a paucity of literature regarding the selection of RSI induction agent in ED patients at high risk of post-intubation hypotension and cardiovascular collapse. To address this further, this study investigates whether there is a significant difference in the rate of post-intubation hypotension in patients who receive ketamine or etomidate for induction in the ED. Additionally, we perform an exploratory analysis in patients with pre-existing risk of cardiovascular collapse (identified by pre-intubation shock index > 0.9) to investigate induction agent effect in patients at high risk of post-intubation hypotension. We also compare secondary outcomes related to post-intubation hypotension, including post-intubation vasopressor use, incidence of peri-intubation cardiac arrest, and mortality. We hypothesize that in patients undergoing RSI there is no difference in the incidence of post-intubation hypotension in patients who receive ketamine versus etomidate.

2. Methods

2.1. Study Design and Setting

This is a retrospective cohort study examining post-intubation hypotension in patients who underwent emergent intubation in the ED using either etomidate or ketamine at a large academic medical system from January 2016 to June 2019. The medical system includes two separate hospitals; one functions as an urban safety-net hospital, the other as a quaternary care center. Together, these two sites have an annual census of approximately 95,000 ED visits. We obtained institutional review board approval with waiver of informed consent (IRB #191355). We prepared the manuscript in accordance with STROBE guidelines.

2.2. Inclusion and Exclusion

We included adults (at least 18 years old) who were intubated in the ED using either etomidate or ketamine. Patients who underwent endotracheal intubation were identified through automated electronic query by the presence of an intubation order, neuromuscular blockade order, mechanical ventilation order, or documentation of endotracheal intubation within the study period. Patients were excluded if they did not have mechanical ventilator settings recorded in the ED, if they suffered cardiac arrest prior to intubation, if they were missing vital sign data documentation less than 20 minutes before and after intubation, or if they received more than one induction agent prior to intubation. Patients with hypotensive periods prior to intubation were included. All of these cases had normotensive data points before intubation as well. Exclusion of these cases would disproportionately reduce the size of the ketamine group and affect the observed relationship between pre-existing hemodynamic instability, induction agent, and post-intubation adverse events.

2.3. Data Collection, Measurement, and Outcomes

Data for all cases were abstracted via automated query from the electronic medical record (EMR) using a reporting clinical database supporting the EPIC information system (Clarity). Abstracted data included age, gender, race, weight, height, body mass index (BMI), means of arrival, vital signs, medications administered, and hospital discharge disposition. Baseline vital sign data was defined as the first vital sign recording upon ED arrival. Factors related to the study's outcomes included: pre-intubation and post-intubation vital signs [heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), oxygen saturation (SpO2), and temperature], use of vasoactive medication, use of neuromuscular blocking agent medication (succinylcholine, rocuronium, or none) prior to intubation, use of an antibiotic agent, induction agent, lactate level, illness severity via the sequential organ failure assessment (SOFA) score, and hospital discharge disposition. Our institution's EMR began automatically calculating SOFA scores in 2017. Patients who underwent RSI prior to this did not have SOFA scores and were excluded from analyses specific to SOFA scores. All data elements were thus abstracted automatically, and no manual data abstraction occurred. All subjects were reviewed by a graduating medical student and study author (MF), who was not blinded to the study hypothesis, to confirm time of intubation with a standardized data collection sheet. A second study author and attending physician (GW) reviewed a random 10% of charts with a similar data collection sheet. Agreement via Cohen's kappa score was 100%.

The primary outcome was the development of post-intubation hypotension, defined as SBP less than 90 mmHg or mean arterial pressure (MAP) less than 65 mmHg recorded less than 20 minutes after intubation. This definition was selected based on several prior investigations [31-33]. Mean arterial pressure was calculated using abstracted SBP and DBP data. Given some patients had multiple vital sign data in the immediate post-intubation period, the lowest value during the defined period was included. A decrease in SBP greater than 20% from baseline was not used to define post-intubation hypotension in this study. In evaluating cases that met this percent change criterion but did not meet the absolute SBP or MAP criteria, most cases were hypertensive at baseline and remained hypertensive following intubation. Therefore, this measure in isolation was found to not truly reflect hemodynamic instability in our cohort.

Secondary outcomes included post-intubation vasopressor administration and in-hospital mortality. Additionally, the data was reviewed for incidence of peri-intubation cardiac arrest by identifying patients who received cardiopulmonary resuscitation (CPR) within 20 minutes of endotracheal intubation [34].

We conducted a planned secondary analysis of patients at pre-existing risk of cardiovascular collapse in the post-intubation phase by selecting patients with a pre-intubation SI ≥ 0.9, using the same outcome measures [27, 31-34, 35-37]. SI is defined as pulse rate divided by systolic blood pressure. This metric is advantageous in its ability to identify at-risk patients whose blood pressure would otherwise be considered within normal limits and is easily available to providers at the time of intubation [35]. The SI was obtained using vital sign data abstracted from the medical record.

2.4 Data analysis

Demographic, baseline, and outcome variables were analyzed following categorization into ketamine or etomidate treatment groups. Descriptive statistics and frequency distributions were used to compare patient characteristics. Categorical variables were compared using the chi-squared test where appropriate. Continuous variables were compared using the independent two-sample t-test as indicated. Data normality was assessed by examining kurtosis and skewness and inspection of histograms. The increase in incidence of post-intubation hypotension following subgrouping by SI > 0.9 was calculated within each induction agent group using effect size difference. A p-value < 0.05 was considered statistically significant in all analyses. Odds ratios and 95% confidence intervals are presented when appropriate.

Patients were then stratified by pre-intubation SI ≥ and the groups were analyzed as previously described. The outcome differences between the etomidate and ketamine groups were then compared in the high-SI groups and the unstratified groups.

We then performed a multivariate logistic regression analysis with post-intubation hypotension as the primary outcome, as previously defined. We selected variables based on biologic plausibility, including age, weight, serum lactate, induction agent (ketamine versus etomidate), and pre-intubation shock index. These covariates were included without overfitting the model. Adjusted odds ratios with 95% confidence intervals were calculated from these models. Multiple models were assessed for error and fit using the Akaike information criterion. We calculated the concordance statistic for these models to assess discriminatory capability. Further logistic regression modeling was used to compare the odds of ketamine use as an induction agent as a function of pre-intubation shock index. Models and figures were constructed using R statistical software (version 4.0.5; R Core Team, Vienna).

3. Results

3.1 Demographics and Patient Characteristics

A total of 469 cases were assessed for inclusion from the automatic query. Of these, 358 patients were included in the final study population. Patients were excluded for age < 18 (2, 0.4%), intubation outside of the ED (12, 2.6%), cardiac arrest prior to intubation (24, 5.1%, and incomplete peri-intubation vital signs (73, 15.6%). Of the 358 patients included in the study, 272 were in the etomidate treatment group and 86 were in the ketamine treatment group. When patients induced with ketamine were compared to those treated with etomidate, there was no significant difference in age (55 vs 56) or gender (66% vs 70% male), as seen in Table 1. Initial vital signs and SOFA scores as well as neuromuscular blocking agent and antibiotic administration for the two groups are listed in Table 2. Baseline body temperature, oxygen saturation, and heart rate were similar between the groups. A greater proportion of patients in the ketamine group had at least one hypotensive data point during the pre-intubation period (23% vs. 5%). Patients receiving ketamine had a higher mean pre-intubation SI (0.97 vs 0.83) and a higher proportion (59% vs 33%) of these patients met our criteria (SI > 0.9) for elevated risk of cardiovascular collapse (Table 2).

3.2. Primary and Secondary Outcomes

Prior to stratification by SI, post-intubation hypotension occurred in 28% of ketamine inductions and 18% of etomidate inductions (Difference in proportion −10%, 95% CI −20.9% to −0.1%). In the subgroup analysis of patients with SI > 0.9, ketamine administration was no longer associated with a higher rate of post-intubation hypotension. Patients sedated with ketamine had a post-intubation hypotension incidence of 33% and the etomidate group had an incidence of 30% (Difference in proportion: −3%, 95% CI −20.1% to 14.1%). A similar pattern was found when analyzing vasopressor use before and after subgrouping by SI. In the full cohort, the ketamine group was more likely to receive vasoactive medication (Difference in proportion: −11%, 95% CI −21.8% to −1.6%). After subgrouping by SI, this difference was no longer statistically significant (Difference in proportion: −6%, 95% CI −22.6% to 10.6%).

Of note, the ketamine group had a greater proportion of cases with at least one hypotensive blood pressure data point prior to intubation (23% vs 11%) (Table 2). Furthermore, in the ketamine group, 50% of patients with a pre-intubation hypotensive data point went on to meet criteria for post-intubation hypotension, which accounted for 42% of all ketamine post-intubation hypotension cases. Comparatively, 52% of etomidate patients with a pre-intubation hypotensive data point met criteria for post-intubation hypotension, which accounted for 31% of all etomidate post-intubation hypotension. Of those patients who remained normotensive prior to intubation, the incidence of post-intubation hypotension was 21% in the ketamine group and 14% in the etomidate group (Table 3).

There was no association between induction agent administered and hospital mortality, irrespective of SI (Table 3). Only two patients in our cohort suffered cardiac arrest that met criteria for peri-intubation cardiac arrest as most patients who experienced a cardiac arrest did so beyond 20 minutes post-intubation. Therefore, statistical analyses were not performed. Both patients with peri-intubation cardiac arrest were induced with etomidate, achieved return of spontaneous circulation, and later expired in the hospital.

3.3 Multivariate Logistic Regression Modeling

In our multivariate logistic regression analysis (Table 4), we found that the strongest predictor of post-intubation hypotension was pre-intubation shock index ≥0.9 (aOR 3.34, 95% CI 1.58 to 7.07). There was no difference in the odds of post-intubation hypotension between patients who received ketamine or etomidate. Our analysis found pre-intubation antibiotic administration and sex to be insignificant as well.

4. Discussion

Ketamine and etomidate are two of the most commonly used induction agents for RSI in the ED due to their favorable hemodynamic profiles compared to other agents, such as propofol. Ketamine especially has seen a rise in popularity for RSI in patients with hemodynamic instability. We believe our study is the first to specifically evaluate the association between ketamine and etomidate in patients at risk of cardiovascular collapse in the ED at the time of intubation. In patients with a pre-intubation SI > 0.9, we found no difference in

post-intubation hypotension as a function of sedative selection. We also found that the most important factor associated with post-intubation cardiovascular collapse was an elevated pre-intubation SI, suggesting that appropriate resuscitation prior to intubation is more important than sedative selection. Additionally, we found that emergency physicians were more likely to select ketamine in patients at risk of hemodynamic instability in the post-intubation phase.

Prior studies evaluating cardiovascular collapse as a function of sedative choice have yielded conflicting results. A recent randomized clinical trial in ICU patients did not reveal any differences in heart rate, blood pressure, or 28-day survival, although interestingly the ketamine group had higher 7-day survival and was more likely to be treated with vasoactive medications. [38]. A randomized trial of ED and ICU patients in France did not report any differences in mortality between etomidate or ketamine, although this was published over a decade ago and included pre-hospital and ICU intubations [21]. Although our results are similar to these two randomized trials, key differences exist in the study populations: all intubations in one of the studies [38] were performed by a dedicated airway team consisting of anesthesiologists in the ICU and thus findings may not apply to the patient population in the emergency department. The other RCT was published in 2009 in Europe and included pre-hospital and ICU intubations [21], which again may not apply to what current emergency physicians experience. While Multiple additional observational studies have found no significant differences in outcomes between these two agents, none have stratified by SI [39-41].

Recently, the NEAR trial evaluated the use of ketamine and etomidate in sepsis patients [25]. The authors found that ketamine was associated with more post-intubation hypotension than etomidate in this patient population. Although the authors had an elegant study design including propensity matching, they did not specifically evaluate patients with pre-existing cardiovascular instability. Ketamine's mechanism of action includes a release of catecholamines which is likely impaired in critically ill patients who are deficient in catecholamines, which may explain their findings. However, the authors did not include peri-intubation vital signs in their analysis, which may explain the difference between their findings and that of others, including our own. A single-center study evaluating post-intubation hypotension in sepsis patients undergoing RSI with either ketamine or etomidate found conflicting results - patients who received ketamine had less post-intubation hypotension [19]. These conflicting results highlight the need for additional evaluation of sepsis patients undergoing RSI.

In patients with a pre-intubation SI > 0.9, we did not find a significant difference in the incidence of post-intubation hypotension and vasopressor use between etomidate and ketamine. However, in our overall cohort there was an increased risk of post-intubation hypotension and vasopressor use with ketamine, although this was not present in our multivariate model. This is consistent with our finding that the ketamine group was associated with higher pre-intubation SI. Our findings suggest that in patients undergoing RSI, etomidate and ketamine have similar hemodynamic consequences. While several studies have reported a higher incidence of post-intubation hypotension and vasopressor therapy in patients undergoing ketamine induction, they did not control for pre-RSI illness

severity using hemodynamic data, which we hypothesized could confound their results [19, 26]. Our findings support this hypothesis.

In our cohort, ketamine was more likely to be utilized in patients with higher illness severity. The ketamine group had a significantly higher average SI, a higher percentage of patients with an SI of > 0.9, lower average baseline blood pressure values, and a higher average SOFA score. The resultant differences in baseline characteristics demand that future studies comparing induction agents must consider patients' pre-intubation hemodynamics and illness severity to accurately compare outcome effects.

Given the deleterious effects of post-intubation hypotension on mortality and other patient-centered outcomes, our data suggest that the choice of induction agent is far less important than appropriate resuscitation prior to intubation. However, additional studies, ideally prospective randomized trials are required to validate this finding.

5. Limitations

We acknowledge that there are several limitations to our study. This is a retrospective study with potential confounding variables and selection bias. Vital sign recordings surrounding the intubation time were not standardized and occurred at variable time points. In some cases, vital sign data was missing leading to exclusion. The relatively small sample size impaired our ability to assess peri-intubation cardiac arrest. Furthermore, the peri-intubation cardiac arrest rate in our cohort was low relative to cited numbers in the literature [30, 34]. This may be secondary to our exclusion criteria which potentially excluded a higher proportion of peri-intubation cardiac arrest cases. It reasonably follows that patients with peri-intubation cardiac arrest may not have post-intubation data recorded and thus meet exclusion criteria. Additionally, while we analyzed data from two emergency departments over a three-year period, our results may not be generalizable to other centers.

6. Conclusions

In this single-center investigation, ED tracheal intubation with ketamine or etomidate had no difference in the incidence of post-intubation hypotension in multivariate logistic regression analysis or the subgroup with pre-intubation shock index > 0.9. An elevated pre-intubation shock index was the most significant risk factor for post-intubation hypotension. We found emergency physicians at our institution were more likely to use ketamine in hemodynamically unstable patients. A large prospective study is needed to determine the optimal induction agent, if any, for hemodynamically unstable patients undergoing tracheal intubation in the ED.

Conflicts of Interest

Dr. Wardi reports receiving funding from the Gordon and Betty Moore Foundation, the National Foundation of Emergency Medicine, and the National Institutes of Health (K23GM146092), although unrelated to this investigation. Dr. Foster, Mr. Gelber, Mr. Kennis, Dr. Self, Dr. Lasoff, and Dr. Hayden have no conflicts of interest to disclose.

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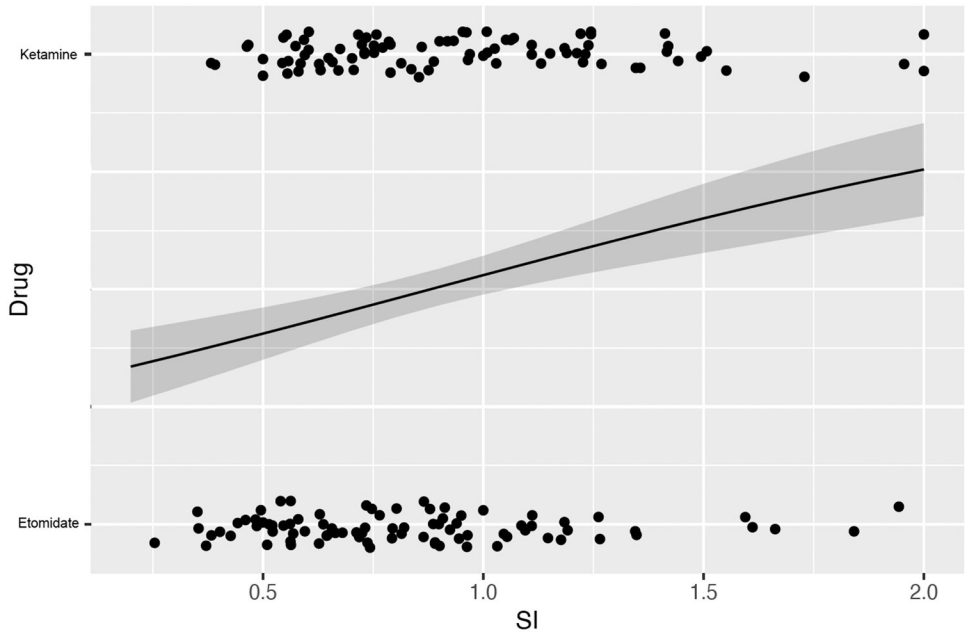

Figure 1. Logistic Regression Log Plot for Induction Agent by Shock Index

Note: Analysis includes full shock index range, but plot is clipped at 99th percentile shock index for size.

SI = shock index.

Table 1.

Demographic and Baseline Characteristics of Emergently Intubated Patients Grouped by Induction Agent.

Baseline Characteristics	Etomidate (n=272)	Ketamine (n=86)	Difference
Age, years, mean (SD)	56 (17.2)	55 (16.2)	1 (−3.1 to 5.1)
Female gender, n (%)	81 (30%)	29 (34%)	−4 (−15.4 to 7.4)
Male gender, n (%)	191 (70%)	57 (66%)	4 (−7.4 to 15.4)
Race/ethnicity, n (%)			
White	128 (47%)	42 (49%)	−2 (−14.1 to 10.1)
Black	40 (15%)	13 (15%)	0 (−8.6 to 8.6)
Hispanic	62 (23%)	16 (19%)	4 (−5.7 to 13.7)
Other/unknown	42 (15%)	15 (17%)	−2 (−11.0 to 7.0)
Weight, kg, mean (SD)	76.8 (20.6)	82.2 (25.4)	−5.4 (−10.7 to −0.1)
BMI, kg/m^2, mean (SD)	26.1 (6.2)	27.7 (7.6)	−1.6 (−3.2 to −0.1)
Means of arrival, n (%)			
EMS	215 (79%)	68 (79%)	0 (−9.9 to 9.9)
Private/walk-in	57 (21%)	18 (21%)	0 (−9.9 to 9.9)

*Categorical variables reported as difference in proportion (95% CI) and continuous variables reported as mean difference (95% CI).

Note: BMI = body mass index, Kg = kilogram, EMS = emergency medical services, CI = Confidence intervals.

Table 2.

Intubating Factors and Patient Characteristics Grouped by Induction Agent.

Baseline Characteristics	Etomidate (n=272)	Ketamine (n=86)	Difference
Neuromuscular Blocking Agent, n (%)			
Rocuronium	255 (94%)	62 (72%)	22 (12.9 to 32.5)
Succinylcholine	10 (4%)	5 (6%)	−2 (−9.4 to 2.5)
None recorded	7 (3%)	19 (22%)	−20 (−29.5 to −11.6)
Peri-intubation antibiotics, n (%)	54 (20%)	25 (29%)	−9 (−21.1 to −0.1)
Serum Lactate, mmol/L, mean (SD)	4.4 (4.3)	4.6 (4.5)	−0.2 (−1.3 to 0.9)
SOFA Score, mean (SD)	2.6 (3.2)	3.5 (2.8)	−0.9 (−1.7 to −0.1)
Baseline vital signs on arrival, mean (SD)			
Temperature, C°	36.6 (1.5)	36.6 (1.2)	0 (−0.3 to 0.3)
Oxygen saturation, %	95.7 (8)	95.3 (6)	0.4 (−1.4 to 2.2)
MAP, mmHg	98.3 (26.0)	89.9 (25.2)	8.4 (2.1 to 14.7)
SBP, mmHg	133.7 (33.1)	121.0 (34.6)	12.7 (4.6 to 20.8)
DBP, mmHg	80.5 (24.9)	73.8 (22.2)	6.7 (0.8 to 12.6)
Heart rate, beats per minute	104.2 (28.5)	103.9 (25.1)	0.3 (−6.4 to 7.0)
Pre-intubation Hypotension, n (%)	29 (11%)	20 (23%)	−12 (−21.6 to −2.4)
Pre-intubation SI, mean (SD)	0.83 (0.33)	0.97 (0.46)	−0.14 (−0.2 to −0.1)
Pre-intubation SI > 0.9, n (%)	90 (33%)	42 (59%)	−26 (−37.1 to −14.0)

*Data are reported as difference in proportion (95% CI) for categorical variables and mean difference (95% CI) using independent t-test for continuous variables.

**Pre-intubation Hypotension denotes cases with at least one pre-intubation blood pressure recording that meets our hypotension criteria.

Note: SOFA Score = sequential organ failure assessment score, MAP = mean arterial pressure, SBP = systolic blood pressure, DBP = diastolic blood pressure, SI = shock index, CI = confidence interval.

Table 3.

Outcome Measures Grouped by Induction Agent.

Outcomes	Etomidate (n=272)	Ketamine (n=86)	Difference in Proportion (95% CI)
Incidence of post-intubation hypotension, n (%)	49/272 (18%)	24/86 (28%)	−10 (−20.9 to −0.1)
Incidence of post-intubation hypotension in cases with pre-intubation SI >0.9, n (%)	27/90 (30%)	15/42 (33%)	−3 (−20.1 to 14.1)
Incidence of post-intubation hypotension in cases with pre-intubation, n (%)	15/29 (52%)	10/20 (50%)	2 (−26.5 to 30.5)
Incidence of post-intubation hypotension in those with normotension prior to intubation, n (%)	34/243 (14%)	14/66 (21%)	−7 (−17.8 to 3.8)
Vasopressor used, n (%)	40/272 (15%)	22/86 (26%)	−11 (−21.8 to −1.6)
Vasopressor used in cases with pre-intubation SI >0.9, n (%)	22/90 (25%)	13/42 (31%)	−6 (−22.6 to 10.6)
Mortality, n (%)	44/272 (16%)	15/86 (17%)	−1 (−7.1 to 11.1)
Mortality in cases with pre-intubation SI >0.9, n (%)	24/90 (27%)	12/42 (29%)	−2 (−19.1 to 13.2)

Note: SI = shock index, SBP = systolic blood pressure, CI = Confidence interval.

Table 4.

Multivariate Logistic Regression Results for Post-intubation Hypotension

Variable	Adjusted Odds Ratio (95% CI)
Age	1.03 (1.01 to 1.04)
Weight	1.01 (0.99 to 1.02)
Lactate	1.06 (0.99 to 1.12)
Induction agent (ketamine vs etomidate)	1.45 (0.79 to 2.65)
Pre-intubation shock index	3.34 (1.58 to 7.07)
C-statistic	0.72

Note: Data reported as adjusted odds ratio (95% confidence interval).