Sedative Dose for Rapid Sequence Intubation and Postintubation Hypotension: Is There an Association?

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Study objective: For patients with hemodynamic instability undergoing rapid sequence intubation, experts recommend reducing the sedative medication dose to minimize the risk of further hemodynamic deterioration. Scant data support this practice for etomidate and ketamine. We sought to determine if the dose of etomidate or ketamine was independently associated with postintubation hypotension.

Methods: We analyzed data from the National Emergency Airway Registry from January 2016 to December 2018. Patients aged 14 years or older were included if the first intubation attempt was facilitated with etomidate or ketamine. We used multivariable modeling to determine whether drug dose in milligrams per kilogram of patient weight was independently associated with postintubation hypotension (systolic blood pressure $<$ 100 mm Hg).

Results: We analyzed 12,175 intubation encounters facilitated by etomidate and 1,849 facilitated by ketamine. The median drug doses were 0.28 mg/kg (interquartile range [IQR] 0.22 mg/kg to 0.32 mg/kg) for etomidate and 1.33 mg/kg (IQR 1 mg/kg to 1.8 mg/kg) for ketamine. Postintubation hypotension occurred in 1,976 patients (16.2%) who received etomidate and in 537 patients (29.0%) who received ketamine. In multivariable models, neither the etomidate dose (adjusted odds ratio [aOR] 0.95, 95% confidence interval [CI] 0.90 to 1.01) nor ketamine dose (aOR 0.97, 95% CI 0.81 to 1.17) was associated with postintubation hypotension. Results were similar in sensitivity analyses excluding patients with preintubation hypotension and including only patients intubated for shock.

Conclusion: In this large registry of patients intubated after receiving either etomidate or ketamine, we observed no association between the weight-based sedative dose and postintubation hypotension. [Ann Emerg Med. 2023;■:1-8.]

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0196-0644/$-see front matter

https://doi.org/10.1016/j.annemergmed.2023.05.014

INTRODUCTION

Background

Hypotension after emergency tracheal intubation is common and associated with an increased risk of mortality.[1,2] Many experts advocate for reducing the dose of etomidate, which is typically dosed at 0.3 mg/kg intravenous push, or ketamine, typically dosed at 1.0 to 1.5 mg/kg intravenous push, for rapid sequence intubation to lessen the risk of hypotension in patients with or at risk of shock.[3-7] There is scant evidence to support this assertion, which may be extrapolated from other agents, such as propofol or midazolam, that are more likely to cause hypotension as the dose increases.[8,9]

Importance

Reducing the dose of the sedative administered during rapid sequence intubation could have unintended negative consequences. First, this may increase the risk of awareness during paralysis.[10,11] Second, the clinician might believe that dose reduction is effective in avoiding hypotension and thus will not fully optimize patient hemodynamics before intubation with intravenous fluids, vasopressors, and other therapies.

Goals of the Investigation

We sought to determine if the milligram per kilogram dose of etomidate or ketamine was associated with hypotension within 15 minutes of emergency intubation of adult emergency department (ED) patients. We hypothesized that higher doses of etomidate and ketamine would be associated with higher rates of postintubation hypotension.

MATERIALS AND METHODS

Study Design and Setting

We analyzed data from the National Emergency Airway Registry (NEAR), a prospective registry of ED intubations

Editor's Capsule Summary

What is already known on this topic

During rapid sequence intubation in patients at risk for shock, some recommend reduced sedative doses.

What question this study addressed

Is the selected rapid sequence intubation dose of etomidate or ketamine associated with postintubation hypotension?

What this study adds to our knowledge

In this multivariable analysis of 14,024 rapid sequence, intubation encounters in a prospective registry, there was no association of drug dose with postintubation hypotension in the main or sensitivity analyses.

How this is relevant to clinical practice

These data suggest that etomidate or ketamine dosing need not be reduced in rapid sequence intubation patients judged at risk for postintubation hypotension.

collected from an international network of academic hospitals. Each of these participating sites obtained approval from its local institutional review board. We report all data in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement.[12]

Methods of Measurement and Data Collection and Processing

Detailed methods outlining data collection methods have been published previously.[13,14] Briefly, intubating clinicians completed an online data collection instrument after each encounter to provide detailed information about the patient, intubation process, and outcomes (StudyTRAX; version 3.47.0011; ScienceTRAX, Macon, GA). The central coordinating center screens each entry for completeness and data consistency. Each study site must complete data collection forms for at least 90% of intubations performed. Relevant to the present study, the clinician records the patient's preintubation hemodynamic status as hypertensive, normotensive, hypotensive without treatment, hypotensive prompting treatment with intravenous fluids, or hypotensive prompting treatment with vasopressors in addition to fluid administration. Postintubation hypotension, defined as a systolic blood pressure <100 mm Hg in the 15 minutes following intubation, is recorded dichotomously; the postintubation systolic blood pressure value is not recorded. The clinician also documents whether the postintubation hypotension was treated with intravenous fluids or vasopressor agents.

Selection of Participants

We included patients 14 years and older who underwent orotracheal intubation and received etomidate or ketamine to facilitate tracheal intubation and whose data were entered into NEAR between January 1, 2016, and December 31, 2018 (the latest version of NEAR). We excluded patients who had intubation primarily facilitated by topical anesthesia; we also excluded those with missing data for the primary outcome, patient weight, etomidate or ketamine dose, or preintubation hemodynamic status. We did not exclude patients with other missing data unrelated to these variables.

We included patients who were recorded as having hypotension before intubation for 2 reasons. First, this is often corrected before intubation occurs.[15] Second, patients with hypotension (either corrected or not) are most likely to have a reduction in the dose of their rapid sequence intubation sedative agent and are at higher risk of postintubation hypotension.

Outcomes

The primary outcome was postintubation hypotension, defined as a systolic blood pressure <100 mm Hg in the 15 minutes following intubation. The secondary outcome was postintubation hypotension treated with intravenous fluids or vasopressor agents.

Primary Analysis

This analysis was not meant to compare etomidate with ketamine but rather sought to separately analyze the association between the dose (mg/kg of actual patient body weight) of each drug and the study outcomes.

We first tabulated all data stratified by receipt of etomidate or ketamine and generated descriptive statistics summarizing patient characteristics, intubation process measures, and the primary and secondary outcomes. We analyzed the association between sedative dose (mg/kg) and postintubation hypotension using generalized linear mixed effects models that allowed adjustment for clinically relevant potential confounders. Etomidate and ketamine were modeled separately. The independent variable of interest was the sedative drug dose (mg/kg of actual patient body weight). We selected the following model covariates a priori from prior literature[16,17] and clinical plausibility: age, female sex, need for immediate intubation, intubation

indication of shock for any reason, preintubation hemodynamic status, initial impression of difficult airway, and presence of one or more difficult airway characteristics. Specific characteristics were reduced neck mobility (including a cervical collar), reduced mouth opening, airway obstruction, facial trauma, and blood or vomit in airway. Study site was included as a random effect.

We created identical models for each drug for the secondary outcome.

We excluded cases with missing data for any variables from the models by listwise deletion. To prevent exclusion of significant proportions of cases, we did not consider variables with 1% or more data missing for inclusion in the model. We conducted all statistical analyses using Stata (version 15; StataCorp, College Station, TX).

Sensitivity Analyses

We performed 4 sensitivity analyses for the primary outcome by replicating the multivariable models used in the primary analysis but altering the study population to assess the robustness of our findings.

The first sensitivity analysis excluded patients in the etomidate and ketamine groups who had hypotension before intubation. The rationale for this sensitivity analysis is to exclude patients who had met the primary outcome at the time of sedative administration and therefore could not have been affected by sedative dose, acknowledging that patients who had hypotension that was corrected before intubation may likewise be excluded.

The second sensitivity analysis included patients intubated for the primary indication of shock. This was meant to capture the population at highest risk for postintubation hypotension and therefore potentially most susceptible to dose-dependent effects of etomidate or ketamine during rapid sequence intubation.

The third sensitivity analysis was performed in the etomidate group (excluding patients who received ketamine) and included patients from sites that did not appear to use weight-based etomidate dosing, defined as EDs in which more than 75% of etomidate administrations were at a dose of exactly 20 mg. Restricting the analysis to the sites that have adopted predominantly uniform-dosing of etomidate created a natural experiment where mg/kg dosing depended on patient weight and not the clinical judgment of the treating team. To explore this issue further, we also replicated the first two sensitivity analyses within the restricted study population of this third sensitivity analysis, sequentially excluding those with hypotension and then including only those intubated for shock.

Finally, we performed multiple imputation to conduct a sensitivity analysis to include all eligible patients who were excluded from multivariable modeling because of missing data for hemodynamic status, drug dose, weight, intubation indication of shock, need for immediate intubation, initial impression of difficult airway, and one or more difficult airway characteristics. We used fully conditional specification to create 10 imputed datasets using the imputed variables above as well as neuromuscular blocking agent use, intubator level of training, age, oxygen saturation before intubation, patient sex, and intubation reason of trauma vs medical. We then performed multivariable modeling identical to the main analysis with use of the *mi estimate* command in Stata, which uses Rubin's rules to combine imputed data to form valid statistical inferences.

RESULTS

Description of the Study Population

During the study period, 14,024 of 19,071 recorded intubation encounters met inclusion criteria for analysis, including 12,175 (86.8%) encounters using etomidate and 1,849 (13.2%) encounters using ketamine for rapid sequence intubation (Figure 1). Hypotension before intubation occurred in 1,727 (14.1%) encounters in the etomidate group and 610 (33.0%) encounters in the ketamine group. The median dose of etomidate was 0.28 mg/kg (interquartile range [IQR] 0.22 mg/kg to 0.32 mg/kg) and for ketamine was 1.33 mg/kg (IQR 1 mg/kg to 1.8 mg/kg). There were 1,885 patients (15.5%) who received <0.2 mg/kg of etomidate and 154 patients (8.3%) who received <0.75 mg/kg of ketamine. Other patient characteristics and intubation process measures are shown in Table 1.

Main Results

Postintubation hypotension occurred in 1,976 patients (16.2%) who received etomidate and in 537 patients (29.0%) who received ketamine. Postintubation hypotension treated with intravenous fluids or vasopressors occurred in 1,521 patients (12.5%) who received etomidate and 448 patients (24.2%) who received ketamine.

The multivariable models demonstrated that neither etomidate dose per 0.1 mg/kg (adjusted odds ratio [aOR] 0.95, 95% confidence interval [CI] 0.90 to 1.01) nor ketamine dose per 1 mg/kg (aOR 0.97, 95% CI 0.81 to 1.17) were associated with postintubation hypotension (Table 2 and Figure 2). Similarly, neither etomidate dose (aOR 0.94, 95% CI 0.89 to 1.00) nor ketamine dose (aOR 0.91, 95% CI 0.75 to 1.10) were significantly associated with postintubation hypotension treated with fluids or

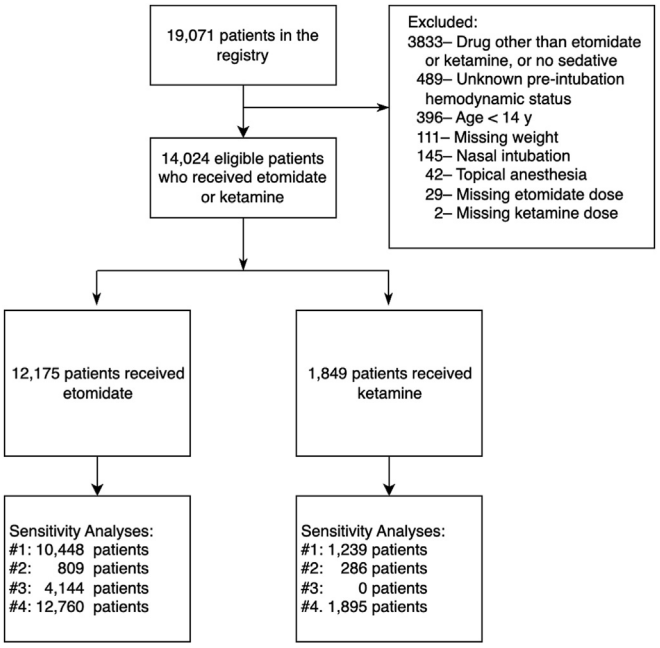

Figure 1. Selection of patients for analysis. Sensitivity analysis #1 excluded patients with hypotension before intubation. Sensitivity analysis #2 included only patients with shock as the primary indication for intubation. Sensitivity analysis #3 applied only to etomidate and included the 5 emergency departments where >75% of etomidate administrations were at a dose of exactly 20 mg (range 75.6% to 94.9% of etomidate administrations being 20 mg). Sensitivity analysis #4 used multiple imputation to include all adult patients who received etomidate or ketamine to facilitate orotracheal intubation for whom topical anesthesia was not the primary approach.

vasopressors (Table E1, available online at http://www.annemergmed.com).

Sensitivity Analyses

In the first sensitivity analysis that excluded 2,337 patients recorded as having hypotension before intubation, neither the etomidate dose (aOR 0.93, 95% CI 0.87 to 1.00) nor ketamine dose (aOR 1.11, 95 %CI 0.87 to 1.42) were associated with the outcome (Figure E1, available online at http://www.annemergmed.com).

In the second sensitivity analysis limiting the analytic sample to the 1,095 patients intubated for an indication of shock, neither etomidate dose (aOR 0.96, 95% CI 0.84 to 1.10) nor ketamine dose (aOR 0.75, 95% CI 0.50 to 1.11) was associated with postintubation hypotension.

The third sensitivity analysis identified 5 EDs where more than 75% of etomidate administrations were at a dose of 20 mg, totaling 4,144 patients (the percentage of patients in these EDs who received 20 mg of etomidate ranged from 75.6% to 94.9%). The median etomidate dose at these EDs was 0.25 mg/kg (IQR 0.21 to 0.29 mg/kg), with 673 patients (16.2%) receiving an etomidate dose less than 0.2 mg/kg. Postintubation hypotension occurred in 693 patients (15.6%). Etomidate dose was not associated with postintubation hypotension (aOR 0.89, 95% CI 0.77 to 1.02) in these 5 EDs. In the nested sensitivity analyses among these 5 EDs that 1) excluded patients with hypotension before intubation, and 2) included only patients intubated for shock, etomidate dose was not associated with postintubation hypotension.

The final sensitivity analysis of imputed data included data for 12,760 patients who received etomidate and 1,895 patients who received ketamine. In this analysis, neither the dose of etomidate (aOR 0.96, 95% CI 0.91 to 1.01) nor the dose of ketamine (aOR 0.99, 95% CI 0.83 to 1.19) was associated with postintubation hypotension. Repeating the other sensitivity analyses in this full imputed data set demonstrated nearly identical results to the sensitivity analyses without imputed data for both drugs.

LIMITATIONS

This study has important limitations. First, physicians may have selected a lower sedative dose for patients who they judged to be at higher risk of postintubation hypotension. This would make it difficult to discern the true effect of sedative drug dose and the risk of hypotension because of residual confounding. However, a sensitivity analysis that included sites with near-uniform use of 20 mg etomidate had results similar to the main analysis, as did a sensitivity analysis of those most at risk for postintubation hypotension. Second, some experts advocate sedative dosing based on lean body weight rather than actual body weight.[3] Only actual body weight is available in this data set, but we anticipate that many patients with actual weights of more than 100 kg (12% of patients in this study) would have a higher calculated mg/kg dose if the lean body weight had been used. Thus, if higher mg/kg doses caused more hypotension, an analysis including lean weights could better reveal this association. However, 964 patients (8% of etomidate administrations) received 0.4 mg or more etomidate in the present data, and there was no association between drug dose and the primary outcome, arguing against a large hypotensive effect with high drug doses. Third, postintubation hypotension was recorded as a dichotomous outcome. Ideally, the pre- and postintubation blood pressures would instead be used. This would quantify the absolute blood pressure change and its nadir. It is difficult to fully judge the clinical relevance of the present primary outcome without this information.

Table 1. Patient characteristics and intubation process measures.

Patient Characteristics or Intubation Process Measure	Etomidate Without Postintubation Hypotension N=10,199	Etomidate With Postintubation Hypotension N=1,976	Ketamine Without Postintubation Hypotension N=1,312	Ketamine With Postintubation Hypotension N=537
Age, median (IQR) (y)	52 (32 to 66)	60 (46 to 73)	50 (33 to 64)	61 (50 to 73)
Female sex	3,435 (33.7)	800 (40.5)	444 (33.8)	181 (33.7)
Weight, median (IQR) - kg	77 (67-90)	77 (65-92)	80 (69 to 95)	79 (64 to 91)
Patient habitus*				
Very thin or thin	1,852 (18.2)	444 (22.5)	271 (20.7)	153 (28.5)
Normal	5,212 (51.1)	827 (41.9)	594 (45.3)	194 (36.1)
Obese	2,665 (26.1)	574 (29.1)	335 (25.5)	146 (27.2)
Very obese	452 (4.4)	128 (6.5)	111 (8.5)	43 (8.0)
Medical indication*				
Altered mental status	3,795 (37.3)	547 (27.7)	283 (21.6)	126 (23.5)
Respiratory failure	1,089 (10.7)	313 (15.9)	294 (22.4)	104 (19.4)
Shock from medical cause	357 (3.5)	341 (17.3)	116 (8.9)	127 (23.7)
Cardiac arrest	300 (2.9)	109 (5.5)	13 (1.0)	15 (2.8)
Shock from trauma cause	61 (0.6)	50 (2.5)	26 (2.0)	17 (3.2)
Other	2,128 (20.9)	229 (11.6)	144 (11.0)	36 (6.7)
Trauma indication*	2,456 (24.1)	384 (19.4)	434 (33.1)	112 (20.9)
Need for immediate intubation (preoxygenation not possible)*	2,792 (27.4)	630 (31.9)	386 (29.4)	172 (32.0)
Hemodynamic status before intubation				
Hypertensive	4,101 (40.2)	290 (14.7)	407 (31.0)	44 (8.2)
Normotensive	5,287 (51.8)	770 (39.0)	631 (48.1)	157 (29.2)
Hypotensive without vasopressors used	577 (5.7)	637 (32.2)	169 (12.9)	201 (37.4)
Hypotensive with vasopressors used	234 (2.3)	279 (14.1)	105 (8.0)	135 (25.1)
Initial impression of difficult airway*	2,978 (29.2)	668 (33.8)	552 (42.1)	221 (41.2)
≥1 Difficult airway characteristics*†	5,162 (50.6)	968 (49.0)	719 (54.8)	280 (52.1)
Intubation Process Measures				
Sedative dose, median (IQR)—mg/kg	0.28 (0.22 to 0.32)	0.27 (0.20 to 0.31)	1.34 (1.0 to 1.82)	1.32 (1 to 1.75)
Reduced sedative dose‡	1,477 (14.5)	408 (20.6)	98 (7.5)	56 (10.4)
Increased sedative dose‡	804 (7.9)	160 (8.1)	32 (2.4)	11 (2.0)
Neuromuscular blocking agent, any	10,154 (99.6)	1,955 (98.9)	1,245 (94.9)	499 (92.9)
Video laryngoscope used	7,081 (69.4)	1,361 (68.9)	898 (68.4)	348 (64.8)

*These variables had missing data for etomidate and ketamine, respectively: 21 patients and 2 patients for body habitus; 16 patients and 2 patients for indication for intubation; 15 patients and 1 patient for need for immediate intubation; 105 patients and 5 patients missing data for initial impression of difficult airway; 8 patients and 0 patients for difficult airway characteristics.

†Difficult airway characteristics included cervical spine immobility, limited mouth opening, airway obstruction, facial trauma, or blood or vomit in the airway.

‡Reduced drug dose is defined as ≤0.2 mg/kg for etomidate and ≤0.75 mg/kg for ketamine. Increased drug dose is defined as ≥0.4 mg/kg for etomidate and ≥3 mg/kg for ketamine.

DISCUSSION

In this registry-based study of more than 14,000 patients who underwent ED rapid sequence intubation, we observed no association between the dose of either etomidate or ketamine, when indexed to actual body weight (mg/kg), and postintubation hypotension, including in multiple sensitivity analyses that included groups of patients most at risk for the primary outcome and in EDs that did not commonly use weight-based dosing. These findings do not support expert guidance to reduce the doses of these agents in critically ill patients with or at risk for hypotension.[3,4,15] These data suggest that other

Table 2. Multivariable modeling of the association between sedative drug dose and the primary outcome, postintubation hypotension.

	Adjusted Odds Ratio (95% CI)	
Variables	**Etomidate Model (N=12,061)**	**Ketamine Model (N=1,843)**
Sedative (etomidate or ketamine) dose - per 0.1 mg/kg for etomidate and per 1 mg/kg for ketamine	0.95 (0.90 to 1.01)	0.97 (0.81 to 1.17)
Age - y	1.02 (1.01 to 1.02)	1.03 (1.02 to 1.03)
Female vs male sex	1.18 (1.05 to 1.32)	0.85 (0.67 to 1.09)
Need for immediate intubation	1.18 (1.04 to 1.33)	1.21 (0.94 to 1.56)
Shock as an indication for intubation	2.10 (1.76 to 2.51)	1.37 (1.01 to 1.86)
Preintubation hemodynamic status		
Normal (reference value)	1	1
Hypertensive	0.43 (0.37 to 0.50)	0.41 (0.29 to 0.59)
Hypotensive *without* the use of vasopressors	6.26 (5.42 to 7.23)	4.67 (3.50 to 6.22)
Hypotensive *with* the use of vasopressors	5.51 (4.49 to 6.77)	4.11 (2.93 to 5.76)
≥1 Difficult airway characteristics	0.99 (0.88 to 1.11)	0.89 (0.69 to 1.14)
Initial impression of anticipated difficult intubation	1.38 (1.22 to 1.56)	1.23 (0.95 to 1.59)

resuscitative measures are more important in preventing postintubation hypotension.

There is little prior study of etomidate and ketamine dosing and postintubation hemodynamics. All prior work is observational and sedative dose is often not the primary exposure of interest. Heffner et al studied 278 ED patients, and Reich et al studied 415 operating room patients, both at single institutions, and both found in a univariate analysis that the etomidate dose was not associated with postintubation hypotension.[18,19] There are just 2 studies outside of the operating room that have specifically examined etomidate or ketamine dose and postintubation hypotension. Kim et al studied 324 patients who received etomidate, categorizing the dose into low (<0.23 mg, 70 patients), normal, or high categories, finding that a low etomidate dose was associated with lower odds of postintubation hypotension.[20] Ketamine dosing in 242 patients was also studied, and no dose association was observed. Krebs et al studied 130 patients tracheally intubated in the out-of-hospital environment, finding that

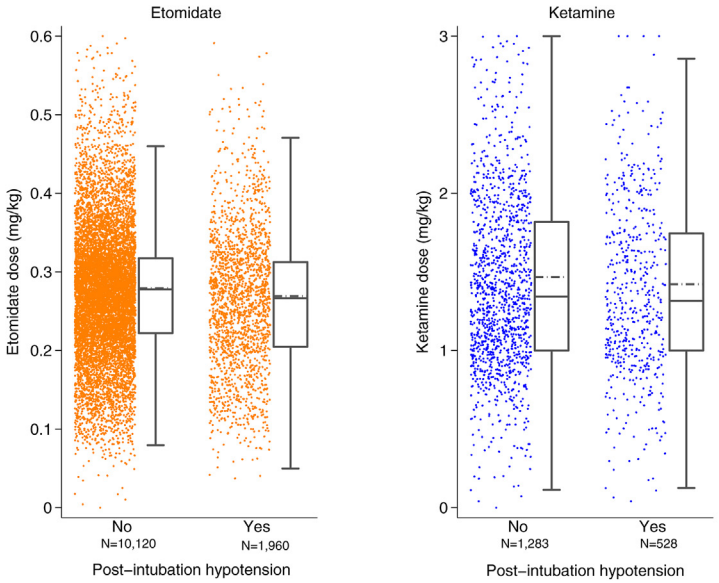

Figure 2. Postintubation hypotension by drug dose. Separately shown is the dose of etomidate and ketamine and whether postintubation occurred. All patients are shown except 95 patients who received >0.6 mg/kg of etomidate and 38 patients who received >3 mg/kg of ketamine. Jittering was used to improve visualization of individual patients.

ketamine doses categorized as high (>2 mg/kg, 50 patients) were associated with increased odds of postintubation hypotension.[21]

The present study, in contrast, included 14,024 patients from 25 EDs, including more than 2,000 patients with a low sedative dose, more than 1,000 patients with a high sedative dose, and more than 1,000 patients with shock as the primary indication for intubation. The primary analysis and multiple sensitivity analyses all demonstrated no association between sedative dose and postintubation hypotension. Although residual confounding remains possible, these data argue strongly for the absence of a large hypotension-preventing effect of the strategy of sedative dose reduction during rapid sequence intubation. Future randomized trials would certainly have equipoise for this clinical question.

Avoiding postintubation hypotension has obvious importance, and hypotension has been associated with an increased risk of mortality.[1,2] For patients with or at risk for hypotension, clinicians should focus efforts on resuscitative measures, including obtaining adequate intravenous access, volume resuscitation, and administration of vasopressor agents.[15] Another potential consequence of sedative dose reduction during rapid sequence intubation is awareness during paralysis, which has shown recently in 2 studies to occur in a substantial proportion of ED patients who have undergone rapid sequence intubation.[10,11] Future prospective research on sedative dosing strategy and hypotension is warranted and would ideally include patient-centered outcomes such as awareness of paralysis.

In conclusion, in this large registry of patients intubated with either etomidate or ketamine, we observed no association between the weight-based drug dose and subsequent postintubation hypotension.

Supervising editor: Steven M. Green, MD, MD, MPP. Specific detailed information about possible conflict of interest for individual editors is available at https://www.annemergmed.com/editors.

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Author contributions: BD, ST, MP, RR, and CAB conceived and designed the study. BD performed the data analysis. BD drafted the initial manuscript and made final editorial decisions; all authors contributed substantially to its revision. CAB is the registry's principal investigator. BD takes responsibility for the study as a whole.

Data sharing statement: The complete dataset and data dictionary are available for centers that participated in data collection, with a valid data use agreement, upon request to Andrea Fantegrossi at afantegrossi@partners.org.

All authors attest to meeting the four ICMJE.org authorship criteria: (1) Substantial contributions to the conception or design of the work; or the acquisition, analysis, or interpretation of data for the work; AND (2) Drafting the work or revising it critically for important intellectual content; AND (3) Final approval of the version to be published; AND (4) Agreement to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

Funding and support: By *Annals'* policy, all authors are required to disclose any and all commercial, financial, and other relationships in any way related to the subject of this article as per ICMJE conflict of interest guidelines (see www.icmje.org). The authors have stated that no such relationships exist.

Publication dates: Received for publication March 18, 2023. Revision received May 12, 2023. Accepted for publication May 24, 2023.

Previous presentation: Presented at the annual meeting for the Society of Academic Emergency Medicine, May 2023, Austin, TX.

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