



Predictors of the complication of postintubation hypotension during emergency airway management[☆]

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Keywords:

Intubation;
Complication;
Hypotension;
Post-intubation
hypotension;
Shock index

Abstract

Objective: Arterial hypotension is a recognized complication of emergency intubation that is independently associated with increased morbidity and mortality. Our aim was to identify factors associated with postintubation hypotension after emergency intubation.

Methods: Retrospective cohort study of tracheal intubations performed in a large, urban emergency department over a 1-year period. Patients were included if they were older than 17 years and had no systolic blood pressure measurements below 90 mm Hg for 30 consecutive minutes before intubation. Patients were analyzed in 2 groups, those with postintubation hypotension (PIH), defined as any recorded systolic blood pressure less than 90 mm Hg within 60 minutes of intubation, and those with no PIH. Multiple logistic regression modeling was used to define predictors of PIH.

Results: A total 465 patients underwent emergency intubation during the study period, and 300 met inclusion criteria for this study. Postintubation hypotension occurred in 66 (22%) of 300 patients, and these patients experienced significantly higher in-hospital mortality (35% vs 20%; odds ratio [OR] 2.1; 95% confidence interval [CI], 1.2-3.9). Multiple logistic regression analysis demonstrated that preintubation shock index (SI), chronic renal disease, intubation for acute respiratory failure, and age were independently associated with PIH. Of these, SI was the most strongly associated factor (OR, 55; 95% CI, 13-232). Receiver operating characteristic plot showed optimized SI 0.8 or higher predicting PIH with 67% sensitivity and 80% specificity. Rapid sequence intubation paralysis was associated with a lower incidence of PIH (OR, 0.04; 95% CI, 0.003-0.4).

Conclusions: Preintubation and peri-intubation factors predict the complication of PIH. Elevated SI strongly and independently forewarned of cardiovascular deterioration after emergency intubation with pre-RSI SI 0.8 or higher as the optimal threshold to identify patients at risk.

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[☆] Conflicts of interest: The authors have no conflicts of interest to report. There were no sponsors or funding sources for this research.

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1. Introduction

Emergency airway management is a core element of critical care support. The use of a rapid acting hypnotic and neuromuscular blocking agent, collectively referred to as rapid sequence intubation (RSI), has become the standard technique to facilitate emergency endotracheal intubation. Safety and efficacy of emergency RSI are well established [1-3], but emergency intubation remains a high-risk procedure [4,5]. Prior attention has focused on immediate airway related complications such as procedural failure, esophageal intubation, pulmonary aspiration, and hypoxemia, which are the most commonly reported complications of RSI [5,6]. Research interrogating hemodynamic consequences is limited [7].

Arterial hypotension is a late sign of cardiovascular insufficiency that is associated with adverse outcome regardless of the cause [8-10]. In contrast, postintubation hypotension (PIH) is frequently explained as a physiologic response to intubation due to multiple mechanisms including induction drug associated sympatholysis and effects of positive-pressure ventilation. This risks leading clinicians to assume that PIH is a benign, transient, or self-limited consequence of airway management [11]. In attempt to quantify the consequences of PIH, we recently demonstrated that PIH impacts nearly one-quarter of patients who are hemodynamically stable before intubation [12]. Furthermore, PIH is a high-risk sign that is independently associated with increased mortality and morbidity [12]. Warning markers for hemodynamic complications of emergency RSI are limited [11,13]. This study was performed to identify clinical factors predicting hemodynamic deterioration after emergency intubation.

2. Methods

2.1. Study design and setting

We conducted a retrospective cohort study of consecutive patients requiring emergency airway management from January 1, 2007, to December 31, 2007, in the emergency department (ED) at Carolinas Medical Center, a large, urban teaching hospital with more than 100,000 patient visits per year. This ED is staffed by emergency medicine residents supervised by board-certified emergency physicians. All attending physicians have privileges granted by the hospital credentialing committee for emergency airway management. Dedicated training in emergency airway management is included in the department annual core curriculum and follows the guidelines of The Difficult Airway Course with a written test and practical examination [14]. The Institutional Review Board and Privacy Board of Carolinas Healthcare System approved this study under waiver of informed consent.

2.2. Selection of patients and data collection

A mandatory audit tool for quality assurance is completed by the intubating physician for all ED intubations at our facility. All adult patients (>17 years old) undergoing emergency endotracheal intubation and who had no systolic blood pressure (SBP) measurements below 90 mm Hg for 30 consecutive minutes before intubation were eligible for this study. Patients were excluded if they were intubated before arrival, required vasopressor support, or had cardiac arrest within 30 minutes of intubation. We used a 2-step pathway to identify eligible study subjects. First, we queried the prospectively collected ED intubation quality assurance database for all patients meeting inclusion criteria during the study period. To ensure that no patients were missed, we then cross referenced all potential subjects with our ED billing database for the procedure code of endotracheal intubation.

A standardized data collection instrument and guidance tool was developed for data collection. Record review and data abstraction were performed by a single research assistant who underwent a standard training program for data abstraction. Regular meetings were conducted to address any problems encountered during the data collection phase. A random sample of 10% of charts was abstracted by a second reviewer who was blinded to the findings of the first observer to assess interobserver reliability as we have previously described [15]. Collected data elements included demographic information, medical history, outpatient and ED medications, ED clinical variables, and primary indication for emergency airway management.

For purposes of data analysis, patients were divided into 1 of 2 groups according to the presence or absence of PIH. *Postintubation hypotension* was defined as any recorded SBP less than 90 mm Hg within the 60 minute postintubation period. The primary outcome measure of the original study was in-hospital mortality. This is a preplanned secondary analysis focused to identify predictors of PIH.

2.3. Data analysis

Continuous data are presented as means \pm SD or medians and interquartile ranges and, when appropriate, were compared for statistical differences using unpaired *t* tests or Mann-Whitney *U* tests. Interobserver variability was examined with Cohen's κ . Categorical data are reported as counts and percentages and were tested for significance using χ^2 or Fisher exact tests when applicable. For all statistical tests, $P < .05$ was considered significant.

To account for potential confounders, a multivariable logistic regression model was created using PIH as the dependent variable. Candidate variables were selected based on known predictors of PIH and significant ($P < .05$) differences in PIH in the bivariate analysis. The model was refined using backwards stepwise elimination. Model fit was assessed using *C* statistic and the Hosmer-Lemeshow

goodness-of-fit test. Odds ratios (ORs) and 95% confidence intervals (CIs) are presented for the final model. The Cochran-Armitage test for trend was used to assess the relationship of pre-RSI shock index (SI) and PIH. All statistical analyses were done with SAS software package (SAS v9.2; SAS Institute, Cary, NC).

3. Results

We identified 542 patients who underwent ED endotracheal intubation during 2007, and 300 patients were analyzed in this study (Fig. 1). The demographic and clinical characteristics of the study subjects are shown in Table 1. Two independent reviewers had excellent agreement for the determination of the variable of PIH present or absent (κ , 0.85; 95% CI, 0.61-1.0). Most patients received etomidate and succinylcholine for RSI. Postintubation hypotension was observed in 66 of 300 (22%; 95% CI, 18%-27%) patients with 8 of 300 patients (3%; 95% CI, 1%-5%) experiencing cardiac arrest during the postintubation study interval. The median time to first PIH was 11 minutes (interquartile range, 4-27 minutes). Of 66 patients with PIH, 13 (20%; 95% CI, 12%-31%) received catecholamines for hemodynamic support. Patients with PIH were older and had more comorbid disease burden. They exhibited lower SBP and higher heart rate immediately before intubation, were more likely to require intubation for acute respiratory failure, and were less likely to receive neuromuscular blockade as part of the RSI drug regimen.

Of the 300 patients, 70 died in the hospital for an overall mortality rate of 23% (95% CI, 19%-29%). Patients in the PIH group had significantly higher in-hospital mortality (35% vs 20%; 95% CI for difference, 2%-27%), and PIH was associated

with a 2-fold increase in odds of in-hospital death (OR, 2.1; 95% CI, 1.2-3.9) compared with patients without PIH.

Factor associated with PIH included age, chronic obstructive pulmonary disease, diabetes mellitus, left ventricular dysfunction, chronic angiotensin-converting enzyme inhibitor therapy, primary reason for intubation, preintubation hemodynamic variables, and RSI paralysis. Shock index was calculated as the quotient of cardiac rate divided by SBP. These fields along with other clinically relevant variables were entered into the multivariable regression analysis, and the final model is reported in Table 2. The model demonstrated good fit by the Hosmer-Lemeshow goodness-of-fit test ($P = .35$). Pre-RSI SI was the variable most strongly associated with PIH (OR, 55; 95% CI, 13-232).

A receiver operating characteristic curve of pre-RSI SI as a predictor of PIH was produced to assess the sensitivity and specificity for PIH at various cut points of SI (Fig. 2). The aim was to identify an optimal SI threshold for predicting PIH. A SI threshold of 0.8 was most predictive, with SI of 0.8 or higher predicting PIH with 67% sensitivity and 80% specificity. Table 3 demonstrates an exposure-response relationship of pre-RSI SI and PIH.

Discussion

This study demonstrates several readily identifiable preintubation and peri-intubation factors that are independently associated with PIH. Within the context of a small body of literature focusing on this topic, our results provide several important insights.

Among hemodynamically stable patients undergoing emergency intubation, immediate pre-RSI SI was the strongest predictor of PIH. Although pre-RSI blood pressure

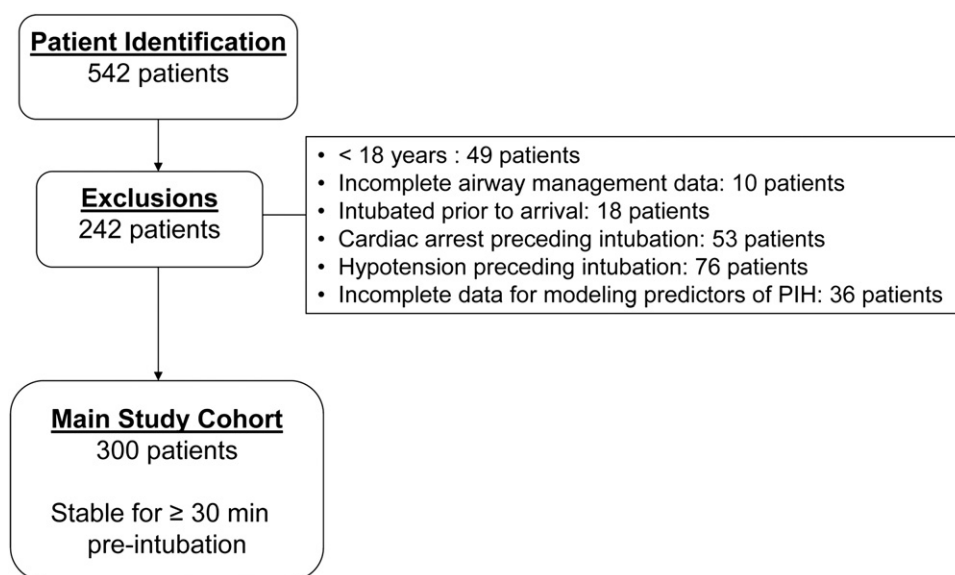


Fig. 1 Patient identification and exclusions for 542 patients intubated during the study period.

Table 1 Population characteristics grouped by outcome

	Total group (n = 300)	PIH absent (n = 234)	PIH present (n = 66)	P
Variable				
Age (y)	49 ± 19	48 ± 19	54 ± 20	.03
Sex (male)	196 (65)	156 (67)	40 (61)	.36
Race (white)	147 (49)	113 (48)	34 (52)	.64
Weight (kg)	81 ± 23	81 ± 23	83 ± 24	.60
Comorbidities				
Coronary artery disease	28 (9)	19 (8)	9 (14)	.17
Left heart failure	17 (6)	10 (4)	7 (11)	.07
Atrial fibrillation	27 (9)	18 (8)	9 (14)	.14
Hypertension	129 (43)	98 (42)	31 (47)	.46
COPD	37 (12)	24 (10)	13 (20)	.04
Asthma	20 (7)	14 (6)	6 (9)	.40
Chronic renal insufficiency	29 (10)	18 (8)	11 (17)	.03
End-stage liver disease	16 (5)	10 (4)	6 (9)	.13
End-stage renal disease	18 (6)	11 (5)	7 (11)	.08
Diabetes mellitus	67 (22)	45 (19)	22 (33)	.02
Outpatient medications				
ACE-inhibitor	53 (18)	46 (20)	7 (11)	.09
β-Blocker	53 (18)	37 (16)	16 (24)	.11
Calcium-channel blocker	47 (16)	34 (15)	13 (20)	.31
Case category				
Medical	171 (57)	132 (56)	39 (59)	.70
Trauma	129 (43)	102 (44)	27 (41)	
Reason for intubation				
Airway protection	236 (79)	194 (83)	42 (64)	<.01
Respiratory insufficiency	56 (19)	35 (15)	21 (32)	
Other	8 (3)	5 (2)	3 (5)	
Clinical variables				
Pre-RSI heart rate (BPM)	102 ± 25	100 ± 24	109 ± 24	<.01
Pre-RSI SBP (mm Hg)	153 ± 38	158 ± 36	134 ± 41	<.001
Pre-RSI SI	0.7 ± 0.3	0.7 ± 0.2	0.9 ± 0.3	<.001
Induction with etomidate	278 (93)	218 (93)	60 (91)	.59
Etomidate dose (mg/kg)	0.30 (0.24-0.37)	0.30 (0.24-0.37)	0.30 (0.24-0.36)	.98
RSI paralysis used	294 (98)	233 (100)	61 (92)	<.01
Paralysis with succinylcholine	255 (85)	199 (85)	56 (85)	.97
No. of intubation attempts				
1	236 (78.9)	182 (78.1)	54 (81.8)	.55
2	40 (13.4)	31 (13.3)	9 (13.6)	
3	23 (7.7)	20 (8.6)	3 (4.6)	

Abbreviations: ACE, angiotensin-converting enzyme; BPM, beats per minute.

and heart rate differed in the 2 study groups, neither was dramatically deranged from normal. One prior study demonstrated pre-RSI systolic pressure less than 140 as independently associated with PIH, but SI was not included in this analysis [14]. When controlling for blood pressure and heart rate in our group, only SI remained independently associated with PIH. Furthermore, an exposure-response relationship is demonstrated such that progressive deviation from normal SI is associated with increased risk of PIH. To our knowledge, this is the first study investigating SI during the peri-intubation period.

Conventional vital signs are poorly correlated with systemic hemodynamics and oxygen delivery [8]. Vital signs, therefore, provide limited insight into cardiovascular reserve and may remain deceptively normal despite evolving

shock [16,17]. As such, our finding of the superiority of SI compared with traditional vital signs is not surprising.

Shock index, calculated as the ratio of heart rate indexed to SBP, is an inverse surrogate of cardiac work such that elevated SI is associated with deteriorating cardiac performance [17]. Normal SI is 0.5 to 0.7, and elevated SI is an early sign of shock despite otherwise normal vital signs [18]. Elevated SI correlates with severity of illness and is associated with adverse outcome and need for therapeutic intervention in a number of diseases [16,19-22]. Our findings extend the utility of SI to serve as a marker of limited cardiovascular reserve during emergency airway management. Our receiver operating characteristic analysis demonstrates SI of 0.8 as the optimal threshold to predict PIH. This is consistent with previous studies showing SI greater than

Table 2 Results of logistic regression analysis for the end point of PIH

Variable	OR	95% CI
Preintubation SI	55.1	13-232
End-stage renal disease	3.7	1.1-13.1
Chronic renal insufficiency	3.4	1.2-9.6
Intubation for respiratory failure	2.1	1.0-4.5
Age	1.03	1.01-1.04
ACE inhibitor use	0.3	0.1-0.7
Intubation paralysis	0.04	0.003-0.4

Model fit: C statistic, 0.81; Hosmer-Lemeshow test, $P = .35$.

0.8 and 0.9 as a marker of serious and life-threatening disease [16,19,20,23]. The threshold of pre-RSI SI of 0.9 predicted PIH with 45% sensitivity and 89% specificity in our group.

Acute disease precipitating the need for intubation is also a useful marker for PIH. Lin et al [13] previously demonstrated that acute exacerbation of chronic obstructive pulmonary disease (COPD) and acute sepsis were both associated with PIH. Previous investigations identified that acute hypercapnic respiratory failure due to COPD and acute hypoxemic respiratory failure are also associated with PIH, although regression analysis was not performed to assess the strength of these associations [11,24]. Our study provides a corroborating signal. We categorized primary indication for intubation rather than precipitating disease and found that emergency intubation for acute respiratory insufficiency was independently associated with PIH. Exhaustion of physiologic reserve during respiratory distress before intubation may be one explanation for this finding.

Our analysis sheds new light on chronic comorbid diseases that contribute to PIH. Chronic renal disease including both chronic kidney and end-stage renal disease were both associated with PIH. Given the high rate of left ventricular diastolic dysfunction in these patients, we hypothesize that vulnerability to preload compromise stemming from peri-intubation events such as RSI medication associated sympatholysis and initiation of positive pressure ventilation may underlie this finding.

Hypnotic agent and dose have been associated with differential cardiovascular responses during RSI [24-27]. It is important to note that our high rate of PIH occurred with standard use of etomidate, which is generally regarded as an optimal and hemodynamically stable hypnotic agent [28]. Etomidate dose and RSI using an alternative hypnotic agent were not associated with PIH in our study group. Nonetheless, we recognize that standard RSI agent dosing recommendations assume hemodynamic stability and normal cardiovascular reserve. Although not specifically investigated in this study, we advocate 50% reduction in induction hypnotic dose (ie, etomidate 0.15 mg/kg IV) for at-risk patients consistent with that commonly used for RSI of patients in uncompensated shock.

In contrast, use of neuromuscular blockade as part of the intubation drug regimen was independently associated with

reduced risk of PIH. Prior observational trials demonstrate higher complication rates during emergency intubation undertaken without paralysis compared with true RSI including neuromuscular blockade, although hemodynamic deterioration was not specifically reported [29,30]. Complicated intubation as marked by the number of intubation attempts does not explain this finding in our group [31]. Routine use of paralysis is strongly advocated in our training program and is consistent with our historic practice [1]. As such, we speculate that another factor may have modified the airway management approach in patients at risk for PIH.

Patient age was the last factor associated with PIH in our study. Given the high rate of comorbid disease and limited cardiovascular reserve associated with aging, this finding is expected and consistent with prior reports [32,33]. However, practitioners should recognize that the aforementioned factors are much more strongly predictive of PIH compared with age.

The practical implications of our data are several-fold. Postintubation hypotension is an important complication of emergency airway management that is independently associated with in-hospital morbidity and mortality. It remains unclear if PIH causes injury or is simply a marker of worsened comorbidity [12]. Regardless of the mechanism, we have identified several clinically useful predictors for the complication of PIH. These warning markers warrant consideration to modify preintubation and peri-intubation management in attempt to avoid hemodynamic deterioration [28]. Timing of emergency intubation should balance respiratory and cardiovascular considerations. High-risk patients, especially those exhibiting pre-RSI SI of 0.8 or higher should undergo preintubation hemodynamic optimization as time permits.

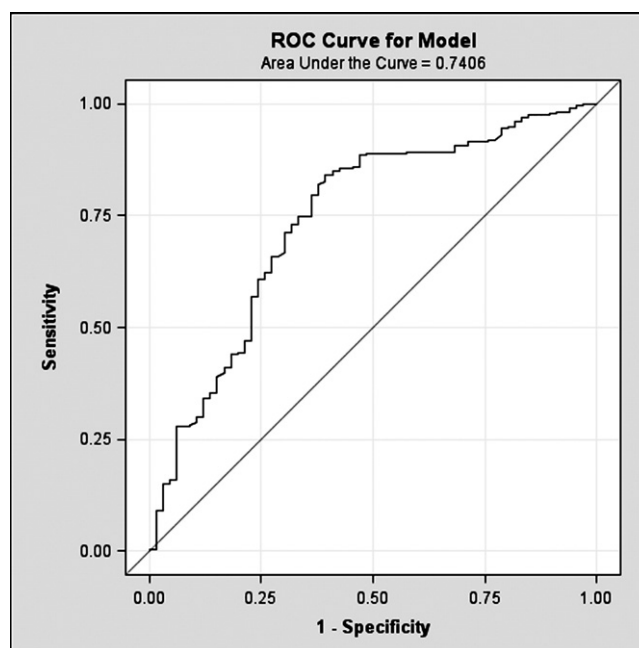


Fig. 2 Receiver operating characteristic plot for pre-RSI SI prediction of PIH.

Table 3 Postintubation PIH risk categorized by SI quintiles and ranges

Pre-RSI SI group	Total n = 300	PIH positive n = 66	PIH negative n = 234
<0.497	60 (20%)	4 (6.8%)	56 (93.3%)
0.497-0.627	60 (20%)	10 (16.7%)	50 (83.3%)
0.628-0.722	60 (20%)	6 (10.0%)	54 (90.0%)
0.723-0.873	60 (20%)	12 (20.0%)	48 (80.0%)
0.874-1.66	60 (20%)	34 (56.7%)	26 (43.3%)

Pre-RSI SI group	Total n = 300	PIH positive n = 66	PIH negative n = 234	OR	95% CI
<0.5	62	4 (6.5%)	58 (93.6%)	1.0	
0.5-0.69	101	13 (12.9%)	88 (87.1%)	2.1	0.7-6.9
0.7-0.89	84	21 (25.0%)	63 (75.0%)	4.8	1.6-14.9
0.9-1.09	30	16 (53.3%)	14 (46.7%)	16.6	4.8-57.3
>1.1	23	12 (52.2%)	11 (47.8%)	15.8	4.3-58.2

Cochrane-Armitage test for trend, $P < .001$.

Empiric volume loading, blood transfusion, and peri-RSI catecholamine support may be indicated depending on the clinical circumstances. The RSI regimen of these patients should also be individualized with selection of a hemodynamically stable induction agent such as etomidate or ketamine, at reduced dose, coupled with neuromuscular blockade, unless contraindicated. Lastly, attention to slow, low-tidal volume ventilation aims to limit intrathoracic pressure and its cardiovascular consequences. Potential next research steps include focus on a structured pre-RSI hemodynamic assessment tool or controlled trial of preintubation hemodynamic optimization or vasopressor support during intubation for at-risk patients in attempt to forestall hypotension and determine effect on mortality.

There are several limitations to our report that warrant discussion. First, this was a retrospective analysis and, as such, is associated with potential biases. To address these potential biases, we followed the steps recommended to minimize validity threats in chart review studies [34]. Although yearly airway education at our center seeks to provide consistent best practice, airway management is not rigidly standardized. Second, this study was performed at a single urban tertiary care hospital, and these results may not be generalizable to other centers treating a different patient population including those with a dissimilar level of acuity. Third, our study defined a discrete preprocedure period, and hemodynamic changes outside the study interval were not interrogated. Patients were monitored via noninvasive blood pressure assessment and preintubation and postintubation hemodynamic changes may have gone undetected due to the intermittent nature of this monitoring. In addition, we studied the presence of hypotension but did not attempt to document abnormal oxygen delivery, organ dysfunction, or other clinical signs suggestive of shock.

In conclusion, PIH is a common complication of emergency intubation that is associated with increased risk

of mortality. Patient variables independently associated with PIH serve as markers for this high-risk complication. Elevated pre-RSI SI strongly and independently forewarned of cardiovascular deterioration after intubation with pre-RSI SI of 0.8 or higher as the optimal threshold to identify patients at risk. Future studies should investigate therapeutic strategies aimed at patients with these markers in attempt to avoid PIH.

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