Risk factors of peri-intubation cardiovascular collapse in critically ill patients

Yoğun bakım hastalarında endotrakeal entübasyon esnasında gelişen kardiyovasküler kollapsa etki eden risk faktörleri

Omer Emgin[1], Bisar Ergun[2]

[1] Division of Internal Medicine and Intensive Care, Tepecik Training and Research Hospital

[2] Division of Internal Medicine and Intensive Care, Tekirdağ Dr. İsmail Fehmi Cumalıoğlu City Hospital

Abstract

Aim: Peri-intubation cardiovascular collapse is a common side effect of tracheal intubation and is associated with increased mortality in critically ill patients. We aimed to determine the potential risk factors associated with peri-intubation cardiovascular collapse.

Methods: This is a prospective observational study of patients who were critically ill and undergoing tracheal intubation from August 1, 2022 to February 28, 2023. The primary outcome was peri-intubation cardiovascular collapse. Peri-intubation cardiovascular collapse was defined as systolic blood pressure <65 mm Hg or new or increased need for vasopressors between induction and 2 minutes after tracheal intubation, or cardiac arrest or death between induction of anesthesia and 1 hour after tracheal intubation.

Results: A total of 87 eligible patients were included. Of them, 37 (42.5%) had peri-intubation cardiovascular collapse. Multivariate logistic regression analysis showed that a shock index ≥ 0.90 (odds ratio [OR] 9.87, 95% confidence interval [CI] 2.98–32.70, p<0.001) and older age (OR 1.07, 95%CI 1.01–1.13, p=0.013) were independent risk factors for peri-intubation cardiovascular collapse. The mortality rate was similar in patients with and without peri-intubation cardiovascular collapse (56.8% vs. 42.0%; p=0.198).

Conclusion: The incidence of peri-intubation cardiovascular collapse is high in critically ill patients. Precautions should be taken because of the increased risk of peri-intubation cardiovascular collapse in elderly patients and patients with a shock index ≥ 0.90.

Keywords: Airway management; cardiovascular collapse; intensive care unit; intubation; mortality; shock index.

Öz

Amaç: Kardiyovasküler kollaps, yoğun bakım hastalarında trakeal entübasyon esnasında sıklıkla meydana gelen ve mortaliteyi arttıran bir komplikasyondur. Biz bu çalışmada, trakeal entübasyon esnasında kardiyovasküler kollapsa neden olan risk faktörlerini araştırmayı amaçladık.

Yöntemler: Bu çalışma, 1 Ağustos 2022 ile 28 Şubat 2023 tarihleri arasında yoğun bakımda yatan ve trakeal entübasyon uygulanan hastalar üzerinde yapılan prospektif gözlemsel bir çalışmadır. Çalışmanın primer sonlanım noktası kardiyovasküler kollapstı. Kardiyovasküler kollaps, indüksiyonun başlangıcından trakeal entübasyondan sonraki 2. dakikaya kadar olan sürede sistolik kan basıncının 65 mm Hg altına düşmesi veya yeni vazopresör ihtiyacının ortaya çıkması veya zaten vazopresör alanlarda ihtiyacın artması veya indüksiyonun başlangıcından trakeal entübasyondan sonraki 1. saate kadar olan sürede kardiyak arrest veya ölüm saptanması olarak tanımlandı.

Bulgular: Toplam 87 hasta çalışmaya dahil edildi. Bu hastaların 37'sinde (%42,5) kardiyovasküler kollaps meydana geldi. Lojistik regresyon analizinde, şok indeksinin 0,90 ve/veya üzerinde olması (OR 9,87, %95CI 2,98–32,70, p<0,001) ve ileri yaş (OR 1,07, %95CI 1,01–1,13, p=0,013) kardiyovasküler kollaps için bağımsız risk faktörleri olarak bulundu. Mortalite oranı kardiyovasküler kollaps olan (%56,8) ve olmayan hastalarda (%42,0) benzer bulundu (p=0,198).

Sonuç: Yoğun bakım hastalarında trakeal entübasyon esnasında gelişen kardiyovasküler kollaps insidansı yüksektir. Yaşlı hastalarda ve şok indeksi ≥ 0.90 olan hastalarda kardiyovasküler kollaps riskinin artması nedeniyle uygun önlemler alınmalıdır.

Anahtar Sözcükler: Endotrakeal entübasyon; hava yolu yönetimi; kardiyovasküler kollaps; mortalite; şok indeks; yoğun bakım ünitesi.

Received/*Geliş* : 05.06.2023
Accepted/*Kabul*: 20.02.2024

DOI: 10.21673/anadoluklin.1310223

Corresponding author/*Yazışma yazarı*

Bişar Ergün
Tekirdağ Dr. İsmail Fehmi Cumalıoğlu Şehir Hastanesi, İç Hastalıkları ve Yoğun Bakım Ünitesi, Tekirdağ, Türkiye
E-mail: dr.bisarergun@hotmail.com

ORCID
Ömer Emgin: 0000-0001-5607-0858
Bişar Ergün: 0000-0003-4828-7576

INTRODUCTION

Tracheal intubation is a routine and life-saving procedure applied in the intensive care units (ICU) (1) complication rates may also differ. We undertook a prospective, observational study of tracheal intubation performed by critical care doctors in Scotland to identify practice, complications, and training. Methods. For 4 months, we collected data on any intubation performed by doctors working in critical care throughout Scotland except those in patients having elective surgery and those carried out before admission to hospital. We used a standardized data form to collect information on pre-induction physical state and organ support, the doctor carrying out the intubation, the techniques and drugs used, and complications noted. Results. Data from 794 intubations were analysed. Seventy per cent occurred in ICU and 18% occurred in emergency departments. The first-time intubation success rate was 91%, no patient required more than three attempts at intubation, and one patient required surgical tracheostomy. Severe hypoxaemia (<80%. Critically ill patients are more vulnerable to tracheal intubation procedures than non-critically ill patients due to limited physiologic reserve (2). Serious complications, including cardiovascular instability/collapse and cardiac arrest, can occur during the tracheal intubation procedure in this fragile population (3)as well as the association of ICU intubation–related cardiac arrest with 28-day mortality. Design: Retrospective analysis of prospectively collected data. Setting: Sixty-four French ICUs. Patients: Critically ill patients requiring intubation in the ICU. Interventions: None. Measurements and Main Results: During the 1,847 intubation procedures included, 49 cardiac arrests (2.7%. The most common cardiovascular instability during or immediately after tracheal intubation is hypotension (4). Cardiovascular instability/collapse/hypotension due to tracheal intubation has been studied with different definitions in various critically ill populations (5–9)2010, to December 31, 2014. We defined immunocompromised as patients with any solid organ or nonsolid organ malignancy or transplant, whether solid organ or not, requiring current chemotherapy. Postintubation hypotension was defined as a decrease in systolic blood pressure to less than 90 mm Hg or a decrease in mean arterial pressure to less than 65 mm Hg or the initiation of any vasopressor medication. Patients were then stratified based on development of postintubation hypotension. Potential risk factors and intensive care unit (ICU.

Cardiovascular collapse has been defined as systolic blood pressure (SBP) <65 mmHg or new or increased need for vasopressors between induction and two minutes after intubation, or cardiac arrest or death between induction and one hour after tracheal intubation (8,9)cardiac arrest, or death remains uncertain. Objective: To determine the effect of fluid bolus administration on the incidence of severe hypotension, cardiac arrest, and death. Design, Setting, and Participants: This randomized clinical trial enrolled 1067 critically ill adults undergoing tracheal intubation with sedation and positive pressure ventilation at 11 intensive care units in the US between February 1, 2019, and May 24, 2021. The date of final follow-up was June 21, 2021. Interventions: Patients were randomly assigned to receive either a 500-mL intravenous fluid bolus (n = 538. Peri-intubation cardiovascular collapse has been reported with an incidence of 18.0-43.4% in critically ill patients (7–9)systolic arterial pressure <65 mm Hg [once] or <90 mm Hg for >30 minutes; new/increased vasopressor requirement; fluid bolus >15 ml/kg, or cardiac arrest. Cardiovascular collapse increases mortality in critically ill patients (7)systolic arterial pressure <65 mm Hg [once] or <90 mm Hg for >30 minutes; new/increased vasopressor requirement; fluid bolus >15 ml/kg, or cardiac arrest. As nearly half of the critically ill patients who undergo tracheal intubation experience this complication, and it increases the risk of mortality, identifying the risk factors associated with cardiovascular collapse gains more importance.

Our primary outcome was peri-intubation cardiovascular collapse. The secondary outcome was the risk factors associated with the peri-intubation cardiovascular collapse in the critically ill population.

MATERIALS AND METHODS

Patients and Setting

We conducted a prospective observational study. This study was in line with the ethical principles of the Dec-

laration of Helsinki. This study was approved by the Tekirdağ Dr. Ismail Fehmi Cumalıoglu State Hospital Clinical Research Ethics Committee (date: 29.08.2022, decision number: 2022/726). All participants included in our study provided written informed consent. In cases where the patient was unable to give informed consent, informed consent was obtained by the representative. Between August 1, 2022 and February 28, 2023, all patients (18 years and older) who underwent tracheal intubation during ICU follow-up were assessed. All beds are monitored in the ICU. Invasive arterial monitoring is performed in most patients, especially when vasopressors are indicated. The exclusion criteria of the study were as follows: (1) tracheal intubation before admission to the ICU; (2) tracheal intubation for cardiac arrest; and (3) SBP <65 mmHg before induction of anesthesia regardless of vasopressor administration.

Variables

The following data were recorded for this study: (1) the demographic data (chronic underlying disease, Charlson Comorbidity Index [CCI], sex, age, and body mass index); (2) main critical events during the entire stay of ICU (sepsis, acute kidney injury [AKI], and vasopressor use); (3) mortality; (4) results of biochemical tests on the day of tracheal intubation and arterial blood gas measurement results immediately before or within six hours of intubation; (5) the main indication for intubation, tracheal intubation medications, hemodynamic and respiratory data immediately before induction of anesthesia and components of cardiovascular collapse.

Outcomes and Definitions

Our primary outcome was the peri-intubation cardiovascular collapse, defined as SBP <65 mm Hg or new or increased need for vasopressors between induction and two minutes after tracheal intubation, or death or cardiac arrest between induction of anesthesia and 1 hour after tracheal intubation (8,9)cardiac arrest, or death remains uncertain. Objective: To determine the effect of fluid bolus administration on the incidence of severe hypotension, cardiac arrest, and death. Design, Setting, and Participants: This randomized clinical trial enrolled 1067 critically ill adults undergoing tracheal intubation with sedation and positive pressure ventilation at 11 intensive care units in the US between February 1, 2019, and May 24, 2021. The date of final follow-up was June 21, 2021. Interventions: Patients were randomly assigned to receive either a 500-mL intravenous fluid bolus (n = 538. The secondary outcome was ICU mortality. The shock index was defined as a ratio of heart rate recorded just before induction divided by SBP recorded just before induction (10)conducted in eligible 140 adult intensive care unit (ICU. The choice of intubation drugs depended on the patient's history and clinical condition.

Statistical Analysis

All statistical analyses were performed using SPSS 25.0 (Statistical Package for the Social Sciences. IBM Corp. Armonk, NY). The categorical data were presented as count/percentage. Categorical variables between participants were compared with the Chi-Squared or Fisher's Exact Tests. The continuous data were presented as median and interquartile range. Continuous variables between the two groups were compared with Mann–Whitney U-test. Multivariate Logistic Regression Analysis was performed to assess the independent risk factors of peri-intubation cardiovascular collapse. A purposeful variable selection method was used to construct the model. A two-tailed p-value less than 0.05 was considered statistically significant.

RESULTS

Characteristics of Patients

Eighty-seven eligible patients who underwent tracheal intubation during ICU follow-up were included in the study. Of them, 37 (42.5%) had peri-intubation cardiovascular collapse. The median age of the study population was 72.0 (63.0-82.0) years, and 36 (41.4%) were women **(Table 1).**

Compared with the no cardiovascular collapse group, patients in the cardiovascular collapse group were significantly older (78.0 [67.5–86.0] vs. 67.0 [59.0–77.3] years; p = 0.001). Compared with the no cardiovascular collapse group, the percentage of patients with hypertension and congestive heart failure

Table 1. Demographic and clinical characteristics in patients with and without peri-intubation cardiovascular collapse.

Characteristics	All cases	Cardiovascular collapse	No cardiovascular collapse	p value
	(n=87)	(n=37)	(n=50)	
Age, years	72.0 (63.0-82.0)	78.0 (67.5-86.0)	67.0 (59.0-77.3)	**0.001**
Gender				
Female	36 (41.4)	16 (43.2)	20 (40.0)	0.827
Male	51 (58.6)	21 (56.8)	30 (60.0)	
Body mass index, kg/m²	24.7 (21.3-28.0)	22.4 (20.0-26.0)	26.2 (22.2-28.8)	**0.013**
Comorbidities				
Hypertension	50 (57.5)	27 (73.0)	23 (46.0)	**0.016**
Congestive heart failure	19 (21.8)	13 (35.1)	6 (10.0)	**0.017**
Diabetes Mellitus	27 (31.0)	12 (32.4)	15 (30.0)	0.819
Coronary artery disease	22 (25.3)	10 (27.0)	12 (24.0)	0.806
Chronic kidney disease	22 (25.3)	13 (35.1)	9 (18.0)	0.084
Malignancy	21 (24.1)	6 (16.2)	15 (30.0)	0.250
Dementia	17 (19.5)	10 (27.0)	7 (14.0)	0.173
COPD	14 (16.1)	7 (18.9)	7 (14.0)	0.567
Cirrhosis	3 (3.4)	1 (2.7)	2 (4.0)	1.000
Main reason for ICU admission				
Respiratory failure	40 (46.0)	19 (51.4)	21 (42.0)	0.514
Sepsis / septic shock	25 (28.7)	14 (37.8)	11 (22.0)	0.150
Neurological disease	7 (8.0)	1 (2.7)	6 (12.0)	0.231
Postoperative	6 (6.9)	1 (2.7)	5 (10.0)	0.234
Trauma	3 (3.4)	0 (0)	3 (6.0)	0.258
Other	6 (6.9)	1 (2.7)	5 (10.0)	0.234
APACHE II	24.0 (17.0-30.0)	28.0 (20.0-31.0)	20.5 (14.8-28.5)	**0.025**
SOFA[1]	7.0 (6.0-10.0)	8.0 (6.0-11.0)	7.0 (4.8-9.3)	0.131
CCI	6.0 (4.0-8.0)	7.0 (5.0-8.0)	5.0 (3.0-8.0)	0.059
Laboratory data within 24 h before tracheal intubation				
BUN, mg/dL	35.0 (23.5-59.0)	54.0 (30.3-78.3)	30.7 (16.3-54.1)	**0.001**
Creatinine, mg/dL	1.05 (0.71-2.02)	1.39 (0.88-2.25)	0.91 (0.59-1.80)	**0.011**
Albumin, g/dL	2.51 (2.31-2.97)	2.39 (2.19-2.59)	2.60 (2.39-3.23)	**0.002**
CRP, mg/L	153 (73-224)	161 (115-256)	115 (41-208)	**0.024**
ALT, U/L	25.0 (13.0-41.0)	20.0 (12.0-52.5)	25.5 (14.8-40.3)	0.747
Hemoglobin, g/dL	10.6 (9.7-12.5)	10.6 (9.6-12.7)	10.6 (9.7-12.4)	0.918
Arterial blood gas analysis just before tracheal intubation or within 6 h before tracheal intubation				
pH	7.38 (7.30-7.44)	7.33 (7.25-7.43)	7.41(7.34-7.46)	**0.006**
HCO_3, mmol/L	24.3 (21.0-28.0)	22.0 (18.0-28.0)	25.0 (22.8-28.2)	**0.032**
Lactate, mmol/L	2.10 (1.30-3.20)	2.60 (1.40-4.15)	1.70 (1.18-2.70)	**0.030**
PaO_2/FiO_2	124 (97-164)	124 (99-144)	122 (94-177)	0.293
Events/therapies during the entire ICU stay				
Sepsis	69 (79.3)	33 (89.2)	36 (72.0)	0.063
Vasopressor requirement	62 (71.3)	31 (83.8)	31 (62.0)	**0.032**
Acute kidney injury	35 (40.2)	16 (43.2)	19 (38.0)	0.663
Renal replacement therapy	18 (20.7)	8 (21.6)	10 (20.0)	1.000
Successful weaning	47 (54.0)	17 (45.9)	30 (60.0)	0.277
ICU length of stay (days)	10.0 (4.0-19.0)	9.0 (4.5-21.0)	10.5 (4.0-16.3)	0.850
ICU mortality	42 (48.3)	21 (56.8)	21 (42.0)	0.198

All values are expressed as numbers (percentages) or median (interquartile range).

APACHE II, Acute Physiology and Chronic Health Evaluation II; ALT, alanine transaminase; BUN, blood urea nitrogen; CCI, Charlson Comorbidity Index; COPD, chronic obstructive pulmonary disease; CRP, C-reactive protein; FiO_2, fraction of inspired oxygen; ICU, intensive care unit; LDH, lactate dehydrogenase; LVEF, left ventricular ejection fraction; PaO_2, partial pressure of arterial oxygen; $PaCO_2$, partial pressure of arterial carbon dioxide; SOFA Score, The Sequential Organ Failure Assessment Score.

1. Calculated on the day of tracheal intubation.

Table 2. Variables obtained before, during, and immediately after tracheal intubation.

Characteristics	All cases (n=87)	Cardiovascular collapse (n=37)	No cardiovascular collapse (n=50)	p value
Main indication for intubation				
Hypoxic respiratory failure	52 (59.8)	25 (67.6)	27 (54.0)	0.270
Hypercarbic respiratory failure	9 (10.3)	4 (10.8)	5 (10.0)	1.000
Altered mental status	13 (14.9)	2 (5.4)	11 (22.0)	**0.037**
Haemodynamic instability	8 (9.2)	5 (13.5)	3 (6.0)	0.277
Other	5 (5.7)	1 (2.7)	4 (8.0)	0.389
Tracheal intubation medications				
Midazolam, n (%)	64 (73.6)	27 (73.0)	37 (74.0)	1.000
Midazolam dose (mg/kg)	0.04 (0.03-0.06)	0.04 (0.03-0.06)	0.04 (0.03-0.06)	0.859
Propofol, n (%)	23 (26.4)	10 (27.0)	13 (26.0)	1.000
Propofol dose (mg/kg)	0.48 (0.37-0.64)	0.51 (0.34-0.64)	0.47 (0.35-0.60)	0.828
Fentanyl, n (%)	46 (52.9)	19 (51.4)	27 (54.0)	0.831
Fentanyl dose (mcg/kg)	1.60 (1.10-1.85)	1.60 (1.37-2.20)	1.50 (1.00-1.70)	0.077
Rocuronium, n (%)	83 (95.4)	35 (94.6)	48 (96.0)	1.000
Rocuronium dose (mg/kg)	0.83 (0.61-1.06)	0.90 (0.70-1.20)	0.80 (0.60-1.00)	0.116
Tracheal intubation events				
Difficult airway (=2 attempts)	8 (9.2)	3 (8.1)	5 (10.0)	1.000
Video laryngoscopy use	5 (5.7)	2 (5.4)	3 (6.0)	1.000
Medications administered within 24 hours before tracheal intubation, n (%)				
Diuretics	30 (34.5)	15 (40.5)	15 (30.0)	0.365
Calcium channel blockers	17 (19.5)	8 (21.6)	9 (18.0)	0.786
Beta-blockers	16 (18.4)	10 (27.0)	6 (12.0)	0.096
ACE-inhibitors and ARBs	6 (6.9)	3 (8.1)	3 (6.0)	0.696
Nitrates	2 (2.3)	2 (5.4)	0 (0)	0.178
Anti-arrhythmic agents	5 (5.7)	5 (13.5)	0 (0)	**0.012**
Alfa-blockers	2 (2.3)	1 (2.7)	1 (2.0)	1.000
Dexmedetomidine	18 (20.7)	8 (21.6)	10 (20.0)	1.000
Other sedative drugs	17 (19.5)	9 (24.2)	8 (16.0)	0.415
Fentanyl or other narcotics	8 (9.2)	2 (5.4)	2 (12.0)	0.458
Clinical data before tracheal intubation				
Use of vasopressors immediately before induction	39 (44.8)	22 (59.5)	17 (34.0)	**0.029**
Norepinephrine dose (mcg/kg/minute)	0.00 (0.00-0.10)	0.07 (0.00-0.16)	0.00 (0.00-0.08)	**0.034**
Receipt of NIV in 6 hours before intubation	58 (66.7)	25 (67.6)	33 (66.0)	1.000
Negative fluid balance in 6 hours before intubation	10 (11.5)	3 (8.1)	7 (14.0)	0.507
Presence of atrial fibrillation rhythm before intubation	19 (21.8)	15 (40.5)	4 (8.0)	**<0.001**
Hemodynamic and respiratory data immediately before induction of anesthesia				
SBP (mm Hg)	121 (105-143)	110 (104-121)	134 (116-151)	**<0.001**
DBP (mm Hg)	65 (57-78)	60 (51-69)	70 (60-84)	**0.001**
MAP (mm Hg)	83 (74-98)	76 (69-87)	94 (77-106)	**<0.001**
Heart rate (BPM)	109 (89-128)	116 (103-136)	101 (82-119)	**0.005**
Shock index	0.85 (0.72-1.07)	1.01 (0.82-1.18)	0.77 (0.63-0.87)	**<0.001**
Shock index ≥ 0.90, n (%)	36 (41.4)	26 (70.3)	10 (20.0)	**<0.001**
Respiratory rate (per minute)	29 (24-31)	30 (27-31)	27 (18-31)	0.070
Sp02, %	93 (86-98)	91 (85-95)	94 (88-100)	0.054
Body temperature, °C	36.8 (36.4-37.4)	37.0 (36.7-37.4)	36.8 (36.4-37.4)	0.351

All values are expressed as numbers (percentages) or median (interquartile range). ACE: Angiotensin converting enzymes, ARB: Angiotensin receptor blockers, BPM: Beats per minute, DBP: Diastolic blood pressure, MAP: Mean arterial blood pressure, NIV: Non-invasive ventilation, N/A: Not applicable, SBP: Systolic blood pressure, SpO2: Peripheral oxygen saturation.

Table 3. Data of cardiovascular collapse.

Characteristics	All cases	Cardiovascular collapse	No cardiovascular collapse	p value
	(n=87)	(n=37)	(n=50)	
Data of measurements between induction and 2 min after intubation				
Lowest SBP (mm Hg)	95 (68-120)	64 (60-76)	114 (100-133)	**<0.001**
Lowest DBP (mm Hg)	55 (41-65)	40 (36-46)	62 (57-72)	**<0.001**
Lowest MAP (mm Hg)	69 (52-82)	51 (43-54)	79 (73-93)	**<0.001**
Lowest Sp02, %	93 (86-98)	91 (85-95)	94 (88-100)	0.187
Components of the cardiovascular collapse				
New systolic blood pressure <65 mm Hg between induction and 2 min after intubation	19 (21.8)	19 (51.4)	N/A	N/A
New vasopressor between induction and 2 min after intubation	15 (17.2)	19 (40.5)	N/A	N/A
Increased vasopressor between induction and 2 min after intubation	22 (25.3)	22 (59.5)	N/A	N/A
Cardiac arrest within 1 h of intubation	2 (2.3)	2 (5.4)	N/A	N/A
Death within 1 h of intubation	1 (1.1)	1 (2.7)	N/A	N/A

All values are expressed as numbers (percentages) or median (interquartile range).

DBP: Ddiastolic blood pressure, MAP: Mean arterial blood pressure, N/A: Not applicable, SBP: Systolic blood pressure, SpO2: Pulse oxygen saturation.

Table 4. Logistic regression analysis for risk factors of peri-intubation cardiovascular collapse.

Risk Factors	OR (95% CI)	p value
Shock index ≥ 0.90	9.87 (2.98-32.70)	**<0.001**
Age, years	1.07 (1.01-1.13)	**0.013**
Gender	0.75 (0.22-2.60)	0.651
APACHE II	0.99 (0.92-1.07)	0.835
Congestive heart failure	1.68 (0.34-8.33)	0.529
Vasopressor use (Immediately before induction of anesthesia)	2.49 (0.76-8.12)	0.130
pH	0.07 (0.01-14.48)	0.333
Creatinine, mg/dL	0.95 (0.65-1.40)	0.801

APACHE II: Acute Physiology and Chronic Health Evaluation II, CI: confidence interval, OR: Odds ratio.

was higher in the cardiovascular collapse group (73.0% vs. 46.0%; p=0.016) and (35.1% vs. 10.0%; p=0.017) respectively. Patients with cardiovascular collapse had a higher Acute Physiology and Chronic Health Evaluation (APACHE) II score than patients without cardiovascular collapse (28.0 [20.0–31.0] vs. 20.5 [14.8–28.5]; p = 0.025). ICU mortality was 56.8% (n=21) in the cardiovascular collapse group and 42.0% (n=21) in the no cardiovascular collapse group (p = 0.198).

Laboratory Findings

Creatinine levels were higher in the cardiovascular collapse group than in the no cardiovascular collapse group (54.0 [30.3–78.3] vs. 30.7 [16.3–54.1] mg/dL; p = 0.001). Blood urea nitrogen levels were higher in the cardiovascular collapse group than in the no cardiovascular collapse group (1.39 [0.88–2.25] vs. 0.91 [0.59–1.80] mg/dL; p=0.011). C-reactive protein levels were higher in the cardiovascular collapse group than in the no cardiovascular collapse group (161 [115–256] vs. 115 [41–208] mg/dL; p = 0.024).

pH was lower in the cardiovascular collapse group when compared to the no cardiovascular collapse group (7.33 [7.25–7.43] vs. 7.41 [7.34–7.46]; p = 0.006). HCO3 was lower in the cardiovascular collapse group when compared to the no cardiovascular

collapse group (22.0 [18.0–28.0] vs. 25.0 [22.8–28.2] mmol/L; p = 0.032). Lactate levels were higher in the cardiovascular collapse group than in the no cardiovascular collapse group (2.60 [1.40–4.15] vs. 1.70 [1.18–2.70] mmol/L; p = 0.030).

Clinical characteristics before, during, and after tracheal intubation

The main indication for tracheal intubation was acute hypoxic respiratory failure with a rate of 59.8% (n=52; **Table 2**). Tracheal intubation was required in 13 patients (14.9%) due to altered mental status. Of these 13 patients, 2 (5.4%) patients had cardiovascular collapse, while 11 (22.0%) did not (p=0.037). The two most commonly used drugs for tracheal intubation were midazolam (73.6%) and rocuronium (95.4%). All medications and doses used for tracheal intubation were similar between groups.

The percentage of patients who received a vasopressor immediately before induction of anesthesia was higher in the cardiovascular collapse group compared to the no cardiovascular collapse group (59.5% vs. 34.0%; p = 0.029). The proportion of patients with atrial fibrillation rhythm immediately before induction of anesthesia was higher in the cardiovascular collapse group compared to the no cardiovascular collapse group (40.5% vs. 8.0%; p< 0.001).

SBP, DBP, and MAP measured immediately before induction of anesthesia was lower in the cardiovascular collapse group than in the no cardiovascular collapse group (110 [104–121] vs. 134 [116–151] mm Hg; p<0.001), (60 [51–69] vs. 70 [60–84] mm Hg; p=0.001), and (76 [69–87] vs. 94 [77–106] mm Hg; p<0.001) respectively.

SBP measured immediately before induction of anesthesia was lower in the cardiovascular collapse group than in the no cardiovascular collapse group (110 [104–121] vs. 134 [116–151] mm Hg; p<0.001). DBP measured immediately before induction of anesthesia was lower in the cardiovascular collapse group than in the no cardiovascular collapse group (60 [51–69] vs. 70 [60–84] mm Hg; p=0.001). MAP measured immediately before induction of anesthesia was lower in the cardiovascular collapse group than in the no cardiovascular collapse group (76 [69–87] vs. 94 [77–106] mm Hg; p<0.001). Heart rates recorded immediately before induction of anesthesia was higher in the cardiovascular collapse compared to the no cardiovascular collapse group (116 [103–136] vs. 101 [82–119] beats per minute; p=0.005).

The proportion of patients with a shock index ≥ 0.90 immediately before induction of anesthesia was higher in the cardiovascular collapse compared to the no cardiovascular collapse group (70.3% vs. 20.0%; p<0.001). Two patients had cardiac arrest during tracheal intubation and one of them died (**Table 3**).

Independent risk factors of cardiovascular collapse

In Multivariate Logistic Regression Analysis (**Table 4**), a shock index ≥ 0.90 (OR 9.87, 95%CI 2.98–32.70, p <0.001) and older age (OR 1.07, 95%CI 1.01–1.13, p=0.013) were significant factors that independently increased the risk of cardiovascular collapse.

DISCUSSION AND CONCLUSION

This prospective observational study addresses the possible risk factors for the peri-intubation cardiovascular collapse in critically ill patients and has three significant results. First, peri-intubation cardiovascular collapse incidence is 42.5% in this population. Second, advanced age and shock index ≥ 0.90 calculated immediately before induction of anesthesia independently increase the risk of peri-intubation cardiovascular collapse. Third, peri-intubation cardiovascular collapse does not increase the ICU mortality.

Peri-intubation cardiovascular collapse has been reported with an incidence of 18.0-43.4% in critically ill patients (7–9)systolic arterial pressure <65 mm Hg [once] or <90 mm Hg for >30 minutes; new/increased vasopressor requirement; fluid bolus >15 ml/kg, or cardiac arrest. The relatively higher incidence of cardiovascular collapse in this study may be attributed to the fact that our study population is older than other studies (8,9)cardiac arrest, or death remains uncertain. Objective: To determine the effect of fluid bolus administration on the incidence of severe hypotension, cardiac arrest, and death. Design, Setting, and Participants: This randomized clinical trial enrolled 1067 critically ill adults undergoing tracheal intubation with sedation and positive pressure ventilation at 11 intensive care units in the US between February 1, 2019, and May 24, 2021. The date of final follow-up was June 21,

2021. Interventions: Patients were randomly assigned to receive either a 500-mL intravenous fluid bolus (n = 538. Elderly patients have less organ reserves than younger ones (11). Additionally, hemodynamic compensatory mechanism becomes impaired with aging (12). Therefore, they are at higher risk for post-intubation hemodynamic instability (5)2010, to December 31, 2014. We defined immunocompromised as patients with any solid organ or nonsolid organ malignancy or transplant, whether solid organ or not, requiring current chemotherapy. Postintubation hypotension was defined as a decrease in systolic blood pressure to less than 90 mm Hg or a decrease in mean arterial pressure to less than 65 mm Hg or the initiation of any vasopressor medication. Patients were then stratified based on development of postintubation hypotension. Potential risk factors and intensive care unit (ICU. In this study, age was an independent predictor of peri-intubation cardiovascular collapse, similar to the literature (13). On the other hand, significant changes occur in the cardiovascular system and the incidence of hypertension and congestive heart failure increases with age (12). In a study conducted on patients with ST-elevation myocardial infarction, hypertension was a significant risk factor for post-intubation hypotension when Midazolam was used as an induction agent (14)136 patients (66 male and 70 females, mean age 72.25 ± 7.33 years. In this study, the comorbidities of hypertension and heart failure were risk factors for peri-intubation cardiovascular collapse. However, the independent effect of hypertension or congestive heart failure on peri-intubation cardiovascular collapse has to be further investigated as the aging process affects their incidence (12).

The relationship between sepsis and increased risk of hypotension during intubation has been shown in the literature (15,16)little is known about the relationship between pre-existing peripheral microvascular alteration and post-intubation hemodynamic instability (PIHI. In this study, sepsis-related parameters such as hemodynamic parameters (low SBP, DBP, and MAP), global perfusion parameters (increased arterial lactate levels), use of vasopressors, and higher dose of norepinephrine immediately before induction were risk factors for hypotension during intubation procedure. The shock index has been suggested as a bedside "easy-to-use" tool to assess the presence of hemodynamic compromise (10)conducted in eligible 140 adult intensive care unit (ICU. An elevated shock index may be an early sign of shock (17). The clinical significance of the shock index has been studied in critically ill patients, and it has been shown that a shock index ≥ 0.90 significantly increases the risk of post-intubation hypotension (6,10)conducted in eligible 140 adult intensive care unit (ICU. In this study of critically ill patients, we demonstrated that a shock index ≥ 0.90 independently predicts the risk of cardiovascular collapse during tracheal intubation.

The presence of atrial fibrillation may cause loss of atrial systole, tachycardia, and acute heart failure. As a result, cardiac output may decrease (18)but there is limited information regarding their temporal relations and the combined influence of these conditions on mortality. Methods and Results - We studied participants in the Framingham Study with new-onset AF or CHF. Multivariable Cox proportional hazards models with time-dependent variables were used to evaluate whether mortality after AF or CHF was affected by the occurrence and timing of the other condition. Hazard ratios (HRs. The higher incidence of cardiovascular collapse in patients with atrial fibrillation can be explained by these multifactorial effects of atrial fibrillation on the heart.

Albumin plays a crucial role in maintaining intravascular colloidal osmotic pressure. Hypoalbuminemia leads to the movement of fluids from blood vessels to tissues (19,20). Patients with septic shock may experience a further decline in their intravascular volume status due to fluid exchange (21)morphology, cell biology, biochemistry, immunology, and circulation. In our study, the correlation between low albumin levels and peri-intubation cardiovascular collapse may be explained by the lowered intravascular colloidal pressure.

Cardiovascular collapse during the intubation procedure is associated with increased mortality in ICU setting (7)systolic arterial pressure <65 mm Hg [once] or <90 mm Hg for >30 minutes; new/increased vasopressor requirement; fluid bolus >15 ml/kg, or cardiac arrest. In this study, there was no significant relationship between ICU mortality and peri-intubation cardiovascular collapse. It is possible that these findings were due to the limited size of our study population. Additionally,

mortality was higher in our entire population compared to the literature due to advanced age and multiple pre-existing complicated comorbid diseases (7,8)systolic arterial pressure <65 mm Hg [once] or <90 mm Hg for >30 minutes; new/increased vasopressor requirement; fluid bolus >15 ml/kg, or cardiac arrest. Due to high mortality rates, especially in the cardiovascular collapse group, patients had shorter ICU stays.

This research has several limitations. First, the echocardiographic evaluation was not performed during the intubation procedure to show volume status. Second, long-term outcomes and hospital mortality were not analyzed. Third, the results of this study cannot be generalized due to the relatively small sample size of our study. However, the study has some strengths. Tracheal intubation procedures were performed by expert physicians in the ICU setting. All patients were monitored, and most were under invasive arterial pressure monitoring which provides instant and accurate data on abrupt changes in blood pressure.

In conclusion, the frequency of peri-intubation cardiovascular collapse is high in the critically ill population. Intubation procedures should be performed with caution, especially in elderly patients. Shock index is a practical tool that can be calculated easily, quickly and inexpensively at the bedside before endotracheal intubation. A pre-intubation shock index ≥0.90 can be used to predict the risk of peri-intubation cardiovascular collapse.

Conflict-of-interest and financial disclosure

The authors declare that they have no conflict of interest to disclose. The authors also declare that they did not receive any financial support for the study.

REFERENCES

1. Simpson GD, Ross MJ, McKeown DW, Ray DC. Tracheal intubation in the critically ill: A multi-centre national study of practice and complications. Br J Anaesth. 2012;108(5):792–9.
2. Smischney NJ, Demirci O, Diedrich DA, et al. Incidence of and risk factors for post-intubation hypotension in the critically Ill. Med Sci Monit. 2016;22:346–55.
3. de Jong A, Rolle A, Molinari N, et al. Cardiac Arrest and Mortality Related to Intubation Procedure in Critically Ill Adult Patients: A Multicenter Cohort Study. Crit Care Med. 2018;46(4):532–9.
4. Shafi S, Gentilello L, Salomone JP,et al. Pre-hospital endotracheal intubation and positive pressure ventilation is associated with hypotension and decreased survival in hypovolemic trauma patients: An analysis of the national trauma data bank. J Trauma - Inj Infect Crit Care. 2005;59(5):1140–7.
5. Smischney NJ, Seisa MO, Cambest J, et al. The Incidence of and Risk Factors for Postintubation Hypotension in the Immunocompromised Critically Ill Adult. J Intensive Care Med. 2019;34(7):578–86.
6. Ergün B, Ergan B, Yakar MN, et al. Incidência e fatores de risco para hipotensão pós-intubação em pacientes críticos com COVID-19. Rev Bras Ter intensiva. 2022;34(1):131–40.
7. Russotto V, Tassistro E, Myatra SN, et al. Peri-intubation Cardiovascular Collapse in Patients Who Are Critically Ill Insights from the INTUBE Study. Am J Respir Crit Care Med. 2022;206(4):449–58.
8. Russell DW, Casey JD, Gibbs KW, et al. Effect of Fluid Bolus Administration on Cardiovascular Collapse Among Critically Ill Patients Undergoing Tracheal Intubation: A Randomized Clinical Trial. JAMA. 2022;328(3):270-9.
9. Janz DR, Casey JD, Semler MW, et al. Effect of a fluid bolus on cardiovascular collapse among critically ill adults undergoing tracheal intubation (PrePARE): a randomised controlled trial. Lancet Respir Med. 2019;7(12):1039–47.
10. Trivedi S, Demirci O, Arteaga G, Kashyap R, Smischney NJ. Evaluation of preintubation shock index and modified shock index as predictors of postintubation hypotension and other short-term outcomes. J Crit Care. 2015;30(4):861.e1-861.e8617.
11. Lally F, Crome P. Understanding frailty. Postgrad Med J. 2007;83(975):16–20.
12. Fleg JL, Strait J. Age-associated changes in cardiovascular structure and function: A fertile milieu for future disease. Heart Fail Rev. 2012;17(4–5):545–54.
13. Halliday SJ, Casey JD, Rice TW, et al. Risk Factors for Cardiovascular Collapse during Tracheal Intubation of Critically III Adults. Ann Am Thorac Soc. 2020;17(8):1021–4.
14. Zuin M, Rigatelli G, Dell'Avvocata F, et al. Ketamine and midazolam differently impact post-intubation hemodynamic profile when used as induction agents during emergency airway management in hemodynamically stable patients with ST elevation myocardial infarction. Heart Vessels. 2018;33(3):213–25.

15. Dubée V, Hariri G, Joffre J, et al. Peripheral tissue hypoperfusion predicts post intubation hemodynamic instability. Ann Intensive Care. 2022;12(1):68.
16. Smischney NJ, Kashyap R, Khanna AK, et al. Risk factors for and prediction of post-intubation hypotension in critically ill adults: A multicenter prospective cohort study. PLoS One. 2020;15(8):e0233852.
17. Allgöwer M, Burri C. "Schockindex" ["Shock index"]. Dtsch Med Wochenschr. 1967;92(43):1947-50.
18. Wang TJ, Larson MG, Levy D, et al. Temporal relations of atrial fibrillation and congestive heart failure and their joint influence on mortality: The Framingham heart study. Circulation. 2003;107(23):2920–5.
19. Fleck A, Hawker F, Wallace PI, et al. Increased Vascular Permeability: a Major Cause of Hypoalbuminaemia in Disease and Injury. Lancet. 1985;325(8432):781–4.
20. Starling EH. On the Absorption of Fluids from the Connective Tissue Spaces. J Physiol. 1896;19(4):312–26.
21. Singer M, Deutschman CS, Seymour C, et al. The third international consensus definitions for sepsis and septic shock (sepsis-3). JAMA - J Am Med Assoc. 2016;315(8):801–10.