

Predictors of prolonged use of mechanical ventilation in patients with acute respiratory failure and acute heart failure in the CVCU RSUD Dr. Saiful Anwar Malang

Puspa Lestari¹, Setyasih Anjarwani¹, Novi Kurnianingsih¹, Indra Prasetya¹,
Heny Martini¹

Abstract

Background: Acute respiratory failure (ARF) is a critical condition that often complicates hospitalization and commonly arises from cardiopulmonary dysfunctions such as acute heart failure. Prolonged mechanical ventilation (PMV) in these patients is associated with increased morbidity, mortality of about 30%, and greater healthcare resource utilization. Identifying predictors of PMV is essential to improve outcomes and optimize management strategies.

Methods: A retrospective cohort study was conducted on all patients who underwent endotracheal intubation in the Cardiovascular Care Unit (CVCU) of RSUD Dr. Saiful Anwar Malang from 2015 to 2021. Patients with incomplete medical records or who died within 14 days of mechanical ventilation were excluded. Univariate and multivariate logistic regression analyses identified independent predictors of PMV. Receiver operating characteristic (ROC) curves were generated to assess model discrimination using the area under the curve (AUC), with corresponding sensitivity and specificity. Data were analyzed using SPSS 22.0.

Results: Five independent predictors of PMV were identified: tachycardia ($p = 0.013$), metabolic acidosis ($p = 0.002$), impaired renal function ($p = 0.009$), shock ($p = 0.006$), and major bleeding ($p = 0.002$). Multivariate analysis showed the following odds ratios (OR, 95% CI): tachycardia 2.06 (1.09–5.99), metabolic acidosis 2.03 (1.09–6.33), impaired renal function 2.87 (1.28–6.46), shock 2.83 (1.13–7.06), and major bleeding 1.36 (1.18–2.15). The model demonstrated good discrimination with an AUC of 0.83 (95% CI 0.77–0.88), sensitivity 0.87, and specificity 0.73.

Conclusions: In patients with respiratory failure due to acute heart failure, tachycardia, metabolic acidosis, impaired renal function, shock, and major bleeding were independent predictors of prolonged mechanical ventilation. The predictive model showed high sensitivity and acceptable specificity, supporting its clinical usefulness for early identification of high-risk patients and targeted intervention.

(Indonesian J Cardiol, 2025;46:96-103)

¹Department Cardiology and Vascular Medicine, Faculty of Medicine, Universitas Brawijaya, Dr. Saiful Anwar Hospital, Malang, Indonesia.

Correspondence:
Puspa Lestari,

Department Cardiology and Vascular Medicine, Faculty of Medicine, Universitas Brawijaya, Dr. Saiful Anwar Hospital, Malang, Indonesia.

Email: pusparyath@gmail.com

Keywords: Predictor, Prolong Mechanical Ventilation, Acute Respiratory Failure, Acute Heart Failure

Introduction

Acute Respiratory Failure (ARF) can occur as a complication in hospitalized patients and is brought on by a variety of cardiopulmonary dysfunctions, such as Congestive Heart Failure (CHF), Chronic Obstructive Pulmonary Disease (COPD), pneumonia, and Acute Respiratory Distress Syndrome (ARDS). A patient's medical history and a clinical examination can be used to determine whether they have ARF, which is indicated by an arterial oxygen pressure of less than 60 mmHg. Intensive Care Unit (ICU) treatment is frequently prompted by ARF. Patients who need invasive mechanical ventilation have mortality rates in hospitals ranging from 33% to 37%. More than 365,000 patients in the Intensive Care Unit (ICU) in the United States are admitted with congestive heart failure and cardiogenic shock each year; 80,000 of these patients need mechanical breathing.¹

For critically ill patients, intubation and mechanical ventilation are necessary to maintain an open airway and guarantee appropriate gas exchange.² In order to prevent adverse effects, management should be informed about the benefits and drawbacks of invasive mechanical breathing.¹ The use of mechanical ventilation necessitates an understanding of the fundamental physiology of the respiratory system, work standards, main ventilation settings, and knowledge of potential problems. In cases of cardiopulmonary malfunction, the ventilation strategy should be tailored to particular situations, such as decompensated congestive heart failure, chronic obstructive pulmonary disease, right heart failure, and protective ventilation in patients with healthy lungs.^{1,3}

From a cardiac and respiratory standpoint, ventilator management for critically ill patients should be customized to meet the specific requirements of each patient in order to maximize benefits while minimizing risks. Depending on the clinical situation's pathophysiology and planned aims, there are different requirements for starting mechanical breathing. The cardiovascular system can be impacted positively or negatively by Positive-Pressure Ventilation (PPV), although no overall effect has been observed because of the body's capacity to adjust to variations in intrathoracic pressure. Cardiovascular performance can be significantly changed by variations in intrathoracic pressure that are conveyed to the heart and lungs. The physiological reaction of the right and left ventricles to variations in intrathoracic pressure differs significantly. A proficient cardiologist must be familiar with the evidence-based

uses of Invasive Mechanical Positive Pressure Ventilation (IM-PPV) and its interactions with the heart and lungs, as well as be able to modify ventilation techniques to the underlying cardiovascular status of each patient. All cardiologists who care for patients in the CVCU should be educated about these interventions as well.^{4,6}

Nearly 80% of all cardiogenic shock patients in everyday practice have Acute Myocardial Infarction (AMI), which is linked to significant mortality and morbidity. The usage of mechanical ventilation and respiratory failure have increased, according to prior research, in cardiogenic shock. Long-term use of mechanical ventilation in critically ill patients is linked to a mortality rate of about 30% and much higher resource demand.⁶ Identification of those who require long-term mechanical ventilation might change breathing techniques or possibly shorten the course of therapy and reduce associated problems. Therefore, it indicates that the length of time a patient uses mechanical ventilation is a substantial predictor of health issues. For medical objectives, with a focus on the most important factors, and to aid in clinical decisions about critical care patients, predictors of prolonged mechanical ventilation requirements among patients admitted to the CVCU are required.⁴

Methods

Study Design

This cohort retrospective study included patients intubated in CVCU room RSUD Dr. Saiful Anwar Malang from 2015 until 2021. There were 397 patients intubated in CVCU, 117 patients were excluded for the following reasons: incomplete medical record data and died before 14 days of mechanical ventilation. Data obtained from medical records include age, gender, vital signs (blood pressure, heart rate, respiratory rate), oxygen saturation, shock conditions, blood gas analysis (pH, PaO₂, PaCO₂, HCO₃, Base Excess), hemoglobin levels, leukocyte count, hematocrit level, platelet count, serum electrolytes (Sodium, Potassium, Chloride), random blood sugar, kidney function test (Urea, creatinine, estimated Glomerular Filtration Rate (eGFR)), pneumonia, emphysema lung, sepsis, major bleeding, Liver Function Test (AST/ALT), and Glasgow Coma Scale.

End Points and Operational Definitions

The primary endpoint of this study was the occurrence of Prolonged Mechanical Ventilation (PMV), defined as the requirement for invasive

mechanical ventilation for ≥ 14 consecutive days in patients with respiratory failure due to acute heart failure. Secondary endpoints included the identification of clinical and laboratory predictors associated with PMV, such as tachycardia, metabolic acidosis, impaired renal function, shock, and major bleeding, as well as the evaluation of the predictive performance of these variables. Predictive accuracy was assessed through sensitivity, specificity, Odds Ratios (ORs) with 95% confidence intervals, and the discriminative ability of the model, measured by the Area Under the Receiver Operating Characteristic (ROC) Curve (AUC).

For the purposes of this study, operational definitions were applied as follows: tachycardia was defined as a resting heart rate >100 beats per minute measured on admission or during CVCU stay; metabolic acidosis as arterial pH <7.25 on blood gas analysis; impaired renal function as an eGFR <30 mL/min/ 1.73m^2 using the CKD-EPI equation; shock as systolic blood pressure <90 mmHg or the requirement for vasopressor support to maintain adequate mean arterial pressure; and major bleeding as clinically overt bleeding resulting in a hemoglobin drop > 2 g/dL or requiring blood transfusion, hemodynamic support, or surgical intervention. Additional demographic, clinical, and laboratory variables—including age, sex, Glasgow Coma Scale, vital signs, oxygenation indices, complete blood count, serum electrolytes, renal and liver function tests, and co-

morbidities (e.g., pneumonia, sepsis) were also collected and analyzed.

Statistical Analysis

The mean and standard deviation for all data were displayed. The chi-square (χ^2) was used to compare categorical variables. Two-sample t-tests were used to analyze mean differences for continuous variables. To find potential PMV predictors, we conducted both univariate and multivariate analyses. A multivariate analysis was performed on all variables that had a p-value of 0.05 or above in the univariate analysis. Logistic regression was used for the multivariate analysis. For these variables, ROC curves were built. The area under the curve was used to compare receiver operating characteristic curves (AUC). The sensitivity and specificity calculated for each positive variable count. Using SPSS 22.0, the data were examined.

Results

In this study, there were 33 patients (13.36%) with prolonged use of mechanical ventilation (>14 days) and 247 patients (86.64%) patients with mechanical ventilation <14 days.

There was no significant difference in the number of acute heart failure patient with a wet-warm subset as well as a wet-cold subset between the PMV and non-PMV group.

Table 1. Characteristics of a group patient respiratory failure with acute failure using mechanical ventilation.

Variable	Non PMV (n=247)	PMV (n = 33)	p-value
Age (years), (mean \pm SD)	59.7 \pm 11.7	62.8 \pm 10.9	0.142
Gender			
Women (n, %)	103 (41.7%)	14 (42.4%)	0.93
Men (n, %)	144 (58.3%)	19 (57.6%)	
Glasgow Coma Scale (GCS)	11	11	0.762
Systolic Blood Pressure (mmHg)	115 \pm 27	116 \pm 29	0.843
Diastolic Blood Pressure (mmHg)	69 \pm 18	74 \pm 22	0.150
Heart Rate (beats per minute)	96 \pm 24	99 \pm 30	0.465
Temperature ($^{\circ}$ C)	36.5 \pm 0.2	36.6 \pm 0.2	0.403
Respiratory Rate (times per minute)	21 \pm 6	21 \pm 3	0.881
Oxygen saturation (%)	97.52 \pm 4.26	96.06 \pm 11.7	0.483
FiO ₂ (%)	82 \pm 23.2	79 \pm 23.2	0.473
PaO ₂ /FiO ₂	157 \pm 102	186 \pm 128	0.139
Hemoglobin (g/dL)	12.3 \pm 3.0	12.3 \pm 2.92	0.273
Hematocrit (%)	37.81 \pm 8.4	37.64 \pm 8.7	0.257
WBC (cells/ μ L)	15257 \pm 2174	24170 \pm 5601	0.186

Platelet count ($\times 10^3/\mu\text{L}$)	211 \pm 22	221 \pm 90	0.696
Serum Creatinine (mg/dL)	1.48 \pm 3.3	2.27 \pm 5.0	0.067
Ureum (mg/dL)	81.49 \pm 65.9	83.02 \pm 49.5	0.898
eGFR (mL/min/1.73m ²)	50.1 \pm 32.4	46.9 \pm 27.2	0.593
Aspartate Aminotransferase (AST, IU/L)	45.3 \pm 55.0	38.82 \pm 21.8	0.567
Alanine Aminotransferase (ALT, IU/L)	55.53 \pm 77.0	46.61 \pm 56.78	0.980
Random Blood Glucose (mg/dL)	182 \pm 122	163 \pm 101	0.986
Sodium (Na ⁺ , mEq/L)	134.2 \pm 5.16	134.2 \pm 4.6	0.517
Potassium (K ⁺ , mEq/L)	4.18 \pm 0.96	4.2 \pm 1.22	0.793
Chloride (Cl ⁻ , mEq/L)	105 \pm 14.7	105 \pm 6.2	0.852
Arterial pH	7.24 \pm 0.14	7.28 \pm 0.12	0.241
Bicarbonate (HCO ₃ ⁻ , mEq/L)	18.5 \pm 7.5	18.7 \pm 5.9	0.859
Partial Pressure of Oxygen (PaO ₂ , mmHg)	117.42 \pm 59.84	137.5 \pm 82.3	0.09
Partial Pressure of Carbon Dioxide (PaCO ₂ , mmHg)	41.74 \pm 23.87	40.19 \pm 13.96	0.719
Arterial Oxygen Saturation (SaO ₂ , %)	93.92 \pm 8.9	93.15 \pm 12.2	0.662
Tachycardia (HR > 100 beats per minute)	110 (44.9%)	23 (69.7%)	0.013
Metabolic acidosis (PH < 7.25)	115 (47.1%)	25 (75.8%)	0.002
Impaired renal function (eGFR < 30 mL/min/1.73m ²)	80 (32.7%)	19 (57.6%)	0.009
Shock Condition	127 (51.6%)	26 (78.8%)	0.006
Major Bleeding (n, %)	9 (3.6%)	6 (18.2%)	0.002
Lung Emphysema (n, %)	9 (3.6%)	0 (0%)	0.605
Acute Coronary Syndrome			
Anterior MI	20 (8.09%)	3 (9.09%)	0.989
Anterior Extensive MI	15 (6.07%)	2 (6.06%)	
Anteroseptal MI	6 (2.43%)	1 (3.03%)	
Inferior MI	16 (6.47%)	1 (3.03%)	
Inferoposterior MI	11 (4.45%)	4 (12.12%)	
Inferoposterior MI + RV Infarct	18 (7.28%)	4 (12.12%)	
Non ST Elevation	29 (11.74%)	1 (3.03%)	
Unstable Angina Pectoris	9 (3.64%)	7 (21.21%)	
Killip			
I	28 (11.33%)	7 (21.21%)	0.277
II	5 (2.02%)	0	
III	16 (6.47%)	0	
IV	74 (29.95%)	10 (30.30%)	
Pneumonia	165 (66.8%)	21 (63.6%)	0.86
Sepsis	57 (23.3%)	5 (15.2%)	0.414
Hypoxemia	34 (13.8%)	7 (21.2%)	0.38
Hypercapnia	60 (24.3%)	7 (21.2%)	0.86
Forrester			
Dry Warm (n, %)	0	0	0.86
Dry Cold (n, %)	0	0	
Wet Warm (n, %)	114 (46.2%)	16 (48.4%)	
Wet Cold (n, %)	133 (53.8%)	17 (51.6%)	

The PMV group had a significantly greater number of patients with tachycardia, acidosis, grade III chronic kidney disease, shock, and major bleeding compared to the non PMV group. Hence, from univariate analysis, these five variables were identified as the independent predictor

of PMV among the acute heart failure patients. Furthermore, multivariate analysis with logistic regression was performed on these variables.

Based on multivariate analysis, among the predictors, shock and impaired renal function had the highest odds of PMV. Furthermore, tachycardia

Table 2. Multivariate analysis with logistic regression.

Variable	Sig.	OR	95% CI	
			Lower	Upper
Tachycardia (HR > 100 beats per minute)	0.31	2.058	1.091	5.996
Metabolic acidosis (PH <7.25)	0.31	2.027	1.090	6.330
Impaired renal function (eGFR < 30 mL/min/1.73 m ²)	0.11	2.873	1.278	6.460
Shock Condition	0.26	2.828	1.133	7.062
Major Bleeding	0.28	1.359	1.177	2.150

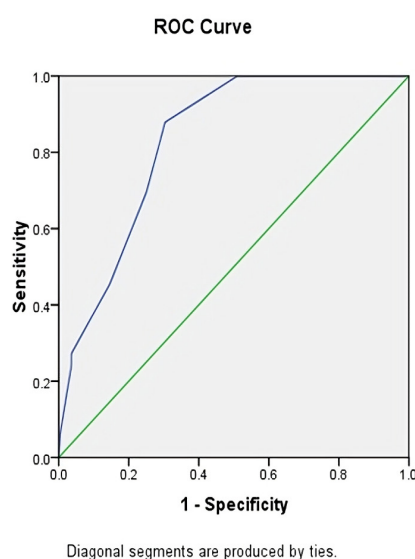


Figure 1. ROC curve of the predictor model for prolonged mechanical ventilation showing good discrimination (AUC = 0.83).

and metabolic acidosis had two times likelihood of PMV while major bleeding only had minor likelihood of PMV.

The predictor factors showed good discrimination with an area under the receiver operating curve (AUC) of 0.83 (95% CI 0.77–0.88) with a sensitivity of 0.87 (95% CI 0.82–0.94) and a specificity of 0.73 (95% CI 0.72–0.84).

Discussion

This study showed that tachycardia, metabolic acidosis, impaired renal function, shock, and major bleeding were important predictors of prolonged mechanical ventilation in patients with acute heart failure and respiratory failure. These findings highlight the clinical relevance of hemodynamic instability, acid base imbalance, renal dysfunction, and

bleeding complications as barriers to successful weaning. Recognizing these predictors early may enable clinicians to anticipate difficulties and adjust management strategies accordingly. Compared with the study by Clark et al., the average duration of mechanical ventilation in our cohort was shorter. This difference may partly be explained by our exclusion of patients who died before day 14 of ventilation, which was done to avoid misclassification of early death as successful weaning. However, this methodological choice may also have contributed to a shorter overall duration of ventilator use compared with other studies. Differences in patient characteristics, disease severity, and supportive care practices may also explain the observed variation.⁷

In this study, patients were intubated primarily to secure the airway and provide prolonged ventilatory support due to underlying cardiac complica-

tions, particularly acute heart failure. The classification of acute heart failure in the study was divided based on Forrester's criteria, including: Warm and dry (normal); Warm and wet (congestion); Cold and dry (hypoperfusion); and Cold and wet (Congestion and hypoperfusion). Patients with acute heart failure were predominantly classified into wet-warm and wet-cold subsets, with similar proportions observed between those who required prolonged ventilation and those who did not. Previous literature notes that acute respiratory failure in this setting is often related to cardiogenic pulmonary edema. Supportive management with oxygen therapy remains the mainstay to correct hypoxemia, while the presence of hypercapnia may necessitate escalation to mechanical ventilation.⁸ Mechanical ventilation provides cardiovascular system benefits in patients with left heart failure. In contrast, cardiovascular conditions are responsible for mechanical ventilation weaning failure in conditions of myocardial ischemia and cardiogenic pulmonary edema.⁹⁻¹⁰

Tachycardia conditions induce excessive catecholamine release in patients who have difficulty weaning mechanical ventilation and can shorten coronary perfusion time.¹¹⁻¹² This condition causes (1) increased oxygen demand due to increased respiratory effort, (2) decreased myocardial oxygen delivery. Because at the time of weaning on mechanical ventilation there is hypoxemia, the decrease in arterial diastolic pressure associated with a decrease in intrathoracic pressure during inspiration is a sign of significant respiratory effort. This mechanism can lead to prolongation of mechanical ventilation.¹²⁻¹⁵ Studies conducted by Hammash et al. in patients using mechanical ventilation who experience cardiac dysrhythmias, it is stated that tachycardia conditions contribute to the weaning process of mechanical ventilation, which can lead to prolonged use of mechanical ventilation. The negative effect of this tachycardia condition affects cardiac contractility and increases tissue oxygen demand, so that systematic heart rate evaluation in mechanically ventilated patients can help shorten weaning time and use mechanical ventilation.¹⁶

Metabolic acidosis also emerged as a relevant predictor of prolonged mechanical ventilation. Patients with lower pH values were more likely to remain ventilator-dependent compared to those with more stable acid-base balance. This finding emphasizes the importance of acid-base status in the weaning process, as persistent acidosis signals ongoing physiological stress that can delay recovery. In contrast, patients with adequate compensation

or closer to normal pH were more likely to tolerate weaning, since sufficient correction of acidosis is one of the key criteria for safe liberation from mechanical support.¹⁷

The effect of mechanical ventilation on renal function remains incompletely understood, although beyond changes in carbon dioxide levels, it is known to influence fluid and electrolyte balance through mechanisms involving vasopressin and the renin-angiotensin-aldosterone system. These alterations may contribute to fluid overload and disturbances in acid-base status, thereby complicating recovery. In this study, patients with impaired renal function were more likely to experience prolonged ventilator dependence compared to those with preserved kidney function. This suggests that renal dysfunction, by worsening systemic imbalance and delaying clearance of metabolic byproducts, plays a critical role in extending the duration of mechanical ventilation.¹⁸⁻¹⁹

Previous research by Kimura et al. demonstrated that shock was an important predictor of prolonged mechanical ventilation, and our findings are consistent with this observation. Patients who developed shock in our cohort were more likely to remain ventilator-dependent compared with those who maintained stable hemodynamics. This association highlights how inadequate tissue perfusion and the need for vasopressor support create conditions that delay recovery and complicate the weaning process.²⁰

Major bleeding was also associated with prolonged mechanical ventilation in this study. Patients who experienced significant bleeding were more likely to remain ventilator-dependent compared with those without bleeding complications. This can be explained by several mechanisms such as massive hemorrhage may precipitate hypovolemic shock and tissue hypoperfusion, transfusion can increase the risk of acute lung injury, and resuscitation efforts may lead to fluid overload that contributes to pulmonary edema. These combined effects create unfavorable respiratory conditions that make weaning from mechanical ventilation more difficult.²¹

Strengths and Limitations

The study benefited from a homogenous patient population and robust analytical methods, including multivariate logistic regression and ROC analysis, which strengthen confidence in the identified predictors. However, exclusion of patients who died before day 14 may have introduced survival bias, and outcome data after extubation were not available. As a single-center retrospective study, residual confounding cannot be excluded, and the relatively

small number of patients with prolonged ventilation may limit generalizability. Future prospective, multi-center studies with longer follow-up are warranted to validate these predictors and to assess whether targeted interventions can improve outcomes and reduce resource utilization.

Conclusion

Tachycardia, metabolic acidosis, impaired renal function, shock condition, and major bleeding were identified as the principal predictors of prolonged mechanical ventilation in patients with respiratory failure due to acute heart failure treated in the CVCU at RSUD Dr. Saiful Anwar Malang. These predictors demonstrated good discriminatory ability, with acceptable sensitivity and specificity for identifying patients at risk of prolonged mechanical ventilation. Their recognition may assist clinicians in anticipating high-risk cases, guiding preventive strategies, and promoting more cost-effective management of acute respiratory failure.

List of Abbreviations

AMI	Acute Myocardial Infarction
ARF	Acute Respiratory Failure
AUC	Area Under the Curve
CVCU	Cardiovascular Care Unit
COPD	Chronic Obstructive Pulmonary Disease
CHF	Congestive Heart Failure
eGFR	Estimated Glomerular Filtration Rate
ICU	Intensive Care Unit
IM-PPV	Invasive Mechanical Positive Pressure Ventilation
PPV	Positive Pressure Ventilation
PMV	Prolonged Mechanical Ventilation

Ethical Clearance

This study was reviewed and approved by the Ethics Commission of General Hospital Dr. Saiful Anwar, Malang, Indonesia (Approval No. 400/018/K.3/302/2022). All procedures were conducted in accordance with the ethical standards of the institutional and national research committee. The study adhered to the principles of the Declaration of Helsinki.

Publication Approval

All authors consent to the publication of this manuscript.

Authors Contributions

Idea/concept: PL. Design: PL, SA. Control/supervision: SA. Data collection/processing: PL. Analysis/interpretation: PL, SA, NK, IP, HM. Literature review: PL, SA, NK. Writing the article: PL. Critical review: All authors have critically reviewed and approved the final draft and are possible for the content and similarity index of the manuscript.

Acknowledgments

We thank the General Hospital Dr. Saiful Anwar, Malang, Indonesia.

Conflict of Interest

The authors declare that they have no conflicts of interest.

Availability of Data and Materials

De-identified data and analytic code are available from the corresponding author upon reasonable request and subject to institutional policies

Funding

This study was supported by institutional funding from RSUD Dr. Saiful Anwar Malang through the Regional Government Budget based on the Director's Decree of RSUD Dr. Saiful Anwar Malang No. SK 800/15689/302/2021.

Copyright/Permissions for Figures

Not applicable.

Generative AI and AI-Assisted Technologies in the Writing Process

The authors used PaperPal to assist in improving English language clarity and grammar during manuscript preparation. No AI tools were used to generate, analyze, or interpret data, figures, or scientific content. All text was reviewed, verified, and edited by the authors, who take full responsibility for the content.

References

1. Kuhn BT, Bradley LA, Dempsey TM, Puro AC, Adams JY. Management of mechanical ventilation in decompensated heart failure. *Journal*

- of cardiovascular development and disease. 2016;3(4):33.
2. Cherpanath T, Lagrand W, Schultz M, Groeneveld A. Cardiopulmonary interactions during mechanical ventilation in critically ill patients. *Netherlands Heart Journal*. 2013;21(4):166-72.
 3. Pham T, Brochard LJ, Slutsky AS, editors. *Mechanical ventilation: state of the art*. Mayo Clinic Proceedings; 2017: Elsevier.
 4. Alviar CL, Miller PE, McAreavey D, Katz JN, Lee B, Moriyama B, et al. Positive pressure ventilation in the cardiac intensive care unit. *Journal of the American College of Cardiology*. 2018;72(13):1532-53.
 5. Alviar CL, Rico-Mesa JS, Morrow DA, Thiele H, Miller PE, Maselli DJ, et al. Positive pressure ventilation in cardiogenic shock: review of the evidence and practical advice for patients with mechanical circulatory support. *Canadian Journal of Cardiology*. 2020;36(2):300-12.
 6. Bayram B, Şancı E. Invasive mechanical ventilation in the emergency department. *Turkish Journal of Emergency Medicine*. 2019;19(2):43-52.
 7. Clark PA, Lettieri CJ. Clinical model for predicting prolonged mechanical ventilation. *Journal of critical care*. 2013;28(5):880. e1-. e7.
 8. Gadre S.K. DA, Mireles C.E., Krishnan S., Wang X.F, Zell K. Acute Respiratory Failure Requiring Mechanical Ventilation. *Medicine (Baltimore)*. 2018(97 (17)):487.
 9. de Meirelles Almeida C, Nedel W, Morais V, Boniatti M, de Almeida-Filho O. Diastolic dysfunction as a predictor of weaning failure: a systematic review and meta-analysis. *Journal of Critical Care*. 2016;34:135-41.
 10. Teboul J-L. Weaning-induced cardiac dysfunction: where are we today? *Intensive care medicine*. 2014;40(8):1069-79.
 11. Routsis C, Stanopoulos I, Kokkoris S, Sideris A, Zakyntinos S. Weaning failure of cardiovascular origin: how to suspect, detect and treat—a review of the literature. *Annals of Intensive Care*. 2019;9(1):1-17.
 12. Boles J-M, Bion J, Connors A, Herridge M, Marsh B, Melot C, et al. Weaning from mechanical ventilation. *European Respiratory Journal*. 2007;29(5):1033-56.
 13. Ghiani A, Paderewska J, Sainis A, Crispin A, Walcher S, Neurohr C. Variables predicting weaning outcome in prolonged mechanically ventilated tracheotomized patients: a retrospective study. *Journal of intensive care*. 2020;8(1):1-10.
 14. Ghiani A, Paderewska J, Walcher S, Tsitouras K, Neurohr C, Kneidinger N. Mechanical power normalized to lung-thorax compliance indicates weaning readiness in prolonged ventilated patients. *Scientific reports*. 2022;12(1):1-9.
 15. Maggiore SM, Lellouche F, Pignataro C, Girou E, Maitre B, Richard J-CM, et al. Decreasing the adverse effects of endotracheal suctioning during mechanical ventilation by changing practice. *Respiratory care*. 2013;58(10):1588-97.
 16. Hammash MH. *Cardiac rhythm during mechanical ventilation and weaning from ventilation*: University of Kentucky; 2010.
 17. Cairo JM. *Pilbeam's mechanical ventilation: physiological and clinical applications*: Elsevier Health Sciences; 2015.
 18. Bellomo R, Kellum JA. CHAPTER 14 - Acid-Base Balance and Kidney-Lung Interaction. In: Papadakos PJ, Lachmann B, Visser-Isles L, editors. *Mechanical Ventilation*. Philadelphia: W.B. Saunders; 2008. p. 158-72.
 19. Imai Y, Parodo J, Kajikawa O, de Perrot M, Fischer S, Edwards V, et al. Injurious mechanical ventilation and end-organ epithelial cell apoptosis and organ dysfunction in an experimental model of acute respiratory distress syndrome. *Jama*. 2003;289(16):2104-12.
 20. Kimura N, Tanaka M, Kawahito K, Sanui M, Yamaguchi A, Ino T, et al. Risk factors for prolonged mechanical ventilation following surgery for acute type a aortic dissection. *Circulation Journal*. 2008;72(11):1751-7.
 21. Liu J, Shen F, Teboul J-L, Anguel N, Beurton A, Bezaz N, et al. Cardiac dysfunction induced by weaning from mechanical ventilation: incidence, risk factors, and effects of fluid removal. *Critical care*. 2016;20(1):1-14.