SCIENTIFIC JOURNAL
CEREM-GO

ISSN 2675-5009
SCIENTIFIC ARTICLE - ORIGINAL ARTICLE

ANALYSIS OF VENTILATORY PARAMETERS AND HOSPITAL OUTCOMES IN MECHANICALLY VENTILATED PATIENTS

ERIKA LETÍCIA GOMES NUNES[1,2]; ISADORA OLIVEIRA FREITAS BARBOSA[1]; MARISTELA LÚCIA SOARES CAMPOS[1]; BRUNA KELLY FERREIRA[1]; GIULLIANO GARDENGHI[3,4]

1. Hospital de Urgências de Goiás - Goiânia – GO.
2. Universidade Estadual de Goiás – UEG, Goiânia – GO.
3. Hospital ENCORE, Aparecida de Goiânia – GO.
4. Faculdade CEAFI, Goiânia – GO.

ABSTRACT

Introduction: Mechanical ventilation is essential for advanced life support, however, if conducted improperly, it can damage the lungs. Parameters such as driving pressure (DP) and mechanical power (MP) are important measurements that help to reduce ventilator-induced lung injuries (VILI).
Objective: To verify if the adjusted parameters in the intensive care units (ICU) in an emergency hospital, were within the recommended by the literature, and verify the patient's outcomes.
Methods: Cross-sectional study of individuals admitted to the ICUs of an emergency hospital. Data regarding hospitalization, vital signs and ventilatory parameters were collected.
Results: 99 individuals were studied, 71% male. Only 8.1% of the DP values were high (greater than 15 cmH2O). The MP, in 21.2% of the patients, was above 17J/min. The main outcome observed in the sample was the mortality rate of 63.8%. SpO2 values were above 95% in 64.6% of the evaluations.
Conclusion: In some situations, ventilatory parameters were adjusted in a non-protective way according to the scientific literature (8.1% increased DP and 21.2% increased MP). SpO2 values were frequently high, which is directly related to VILI. Such findings may be associated with the mortality in the studied population.

Keywords: Respiration Artificial; Ventilator-Induced Lung Injury; Intensive Care Units.

INTRODUCTION

Mechanical ventilation is an indispensable strategy for advanced life support; however, if conducted improperly, it can damage the lungs, leading to a process known as ventilator-induced lung injury (VILI). The physical mechanisms contributing to lung injury are increasingly well understood.[1]

These mechanisms include exposure to high transpulmonary inflation pressures, alveolar overdistension, and/or repetitive opening and closing of alveoli. In addition to direct lung damage, these mechanical forces can trigger a complex cascade of inflammatory mediators, resulting in local and systemic inflammatory responses (biotrauma), causing injuries to non-pulmonary organs[2]. This can lead to dysfunction of multiple organ systems and, potentially, patient mortality.[3]

The primary mechanical determinant of VILI is pulmonary overdistension due to high transpulmonary pressure, causing the lung to exceed its resting volume[3,4]. Lower tidal volumes, lower driving pressure (DP), lower plateau pressure (Pplat), and appropriate positive end-expiratory pressure (PEEP) are indicated to reduce mechanical stresses imposed on inflamed lung tissues, contributing to an effective lung protective strategy.[5]

Based on basic thermodynamic principles, lung injury is attributed to a rate of energy transfer (mechanical power) from the ventilator to the patient. This energy dissipation within the lungs can lead to the production of heat, inflammation, and disruptive deformation of cells and the extracellular matrix.[5,6] The measurement of these mechanical stresses can be performed using the predictive equations for driving pressure (DP) and mechanical power (MP).[7]

Driving pressure (DP) can be routinely calculated in patients who are not making inspiratory efforts by subtracting PEEP from Pplat,[8] recommending values ≤15 cmH2O. The easy bedside application also makes it possible to calculate mechanical power (MP), in which the formula basically involves tidal volume (TV), peak pressure, DP, and respiratory rate (RR). The result of the equation is given in joules per minute. Studies suggest values below 17 J/min, but the idea is to use as little energy as possible, as higher values are also strongly related to mortality.[7,9]

The main factor motivating the study is to raise awareness that the primary mission of the professional's approach should be: not to cause and/or exacerbate harm to the patient, as the intubation procedure itself already makes them vulnerable. The approach to mechanical ventilation (MV) should provide life support while minimizing unwanted toxicity, preventing sentinel events during intervention, and ensuring patient safety.[10] Therefore, the objective of this study was to verify whether the parameters adjusted in the intensive care units (ICUs) of an emergency hospital in the state of Goiás were within the recommendations of the literature, as well as the hospital outcomes of the sample.

METHODS

This is a cross-sectional observational study. With CAAE registration: 53496621.5.0000.0033, it was approved by the Research Ethics Committee of Hospital de Urgências de Goiás, Dr. Valdemiro Cruz (HUGO), under opinion number 5.409.088, in accordance with resolution 466/12 of the National Council of Research Ethics involving human beings.

Patients aged 18 years or older, admitted to the ICUs, receiving invasive ventilatory support in controlled mode, and whose guardians agreed to sign the informed consent form (ICF) were included. Asynchronous patients and those in spontaneous mode were excluded from the study.

After the ICF was signed by the guardian, personal data, medical history, diagnostic hypothesis, vital signs, peripheral oxygen saturation, and length of hospital stay of the patients were collected. The ventilatory parameters collected included tidal volume (TV), inspiratory pressure, PEEP, inspiratory time, peak pressure, Fraction of Inspired Oxygen (FiO2), spontaneous and set respiratory rate (RR), static lung compliance (CStat), driving pressure (DP), and mechanical power (MP). All data were recorded on an evaluation form prepared by the authors.

The data were categorized and tabulated in a spreadsheet using Microsoft Excel 2010 and then analyzed using the statistical software Statistical Package for Social Science, version 26.0. For the analysis of categorical variables, absolute frequency and relative frequency were used, and for continuous variables, mean and standard deviation were used. Tidal volume (TV) values were compared to predicted values using Student's t-test. The significance level adopted was 5% ($p < 0.05$).

RESULTS

Ninety-nine patients were included in the studies, with the sample being predominantly male, consisting of 74 men and 25 women. Table 1 describes the characterization of the sample.

Table 1. Characterization of the sample regarding age, height, predicted weight, and vital signs.
DP (Standard Deviation); HR (Heart Rate); RR (Respiratory Rate); SpO2 (Peripheral Oxygen Saturation); MV (Mechanical Ventilation); Source: Prepared by the authors.

	Mean +/- Standard deviation	Minimum	Maximum
Age	55.7 ± 18.7	18.00	91.00
Height	1.7 ± 0.08	1.58	1.88
Predicted weight	17.2 ± 6.2	3.00	21.00
HR	10.8 ± 4.0	2.00	14.00
RR	13.8 ± 0.5	12.00	14.00
SpO2	20.9 ± 0.4	18.00	21.00
Length of hospital stay	17.9 ± 13.8	1.00	62.00
Duration of Mechanical Ventilation (MV)	15.6 ± 11.5	1.00	55.00

Considering the ventilatory prosthesis, 55% used endotracheal tubes and 45% used tracheostomy. Regarding the assessment of oxygen saturation, we recorded 64.6% above 95%, which is associated with hyperoxia, and 3.0% below 88%, which is associated with hypoxia. The SpO2 values were around 95±5%. Table 2 describes the main ventilatory parameters adjusted and measured during mechanical ventilation.

Table 2. Ventilatory parameters
Oxygen); VC (Tidal Volume); DP (Driving Pressure); MP (Mechanical Power); Source: Prepared by the authors.

	Mean ± Standard deviation	Minimum	Maximum
PEEP	7.5 ± 1.5	5.00	13.00
FIO_2	35.9 ± 20.8	21.00	100.00
VC	400.7 ± 83.7	197.0	573.0
DP	10.47 ± 3.2	5.00	25.00
MP	13.77 ± 5.3	5.00	34.00

The predominant ventilatory mode used was Pressure Control (79%), followed by Volume Control (21%). Regarding variables related to lung injury, only 8.1% of the DP values were high (above 15 cmH2O). The DP found was 10.4±3.2 cmH2O. In 21.2% of the patients, the MP was above 17 J/min, which is associated with LPIV

The static lung compliance (Cstat) was 19.4±5.3 ml/cmH2O, which is considered very low. The respiratory rate (RR) found during mechanical ventilation was 21.2±3.6 breaths per minute. The tidal volume (TV) values did not show a significant difference when compared to the predicted tidal volume of 6 ml/kg (observed TV: 400.7±83.7 ml vs. predicted TV: 397.2±51.3 ml, p: 0.68).

Table 3. Hospital Outcomes
Legend: ICU (Intensive Care Unit); TQT (Tracheostomy); Source: Prepared by the authors.

Outcome	%
Extubation and ICU Discharge	14,90%
Death	63,80%
TQT	21,30%

Table 3 shows the hospital outcomes of the sample. It is noted that 63.8% of the sample progressed to death, 21.3% of the sample required a tracheostomy instead of direct extubation, and only 14.9% were discharged from the ICU.

DISCUSSION

The majority of the sample in this study was composed of men, accounting for 74.7%, and most had been on mechanical ventilation for more than seven days. This result can be explained by the fact that the research was conducted in an emergency and trauma hospital where the male population constitutes the majority of the patients served. These findings are consistent with data present in the literature[11].

It is known that tidal volume (TV) and driving pressure (DP) can impact the development of lung parenchymal injury by ventilation (LPIV) in patients with acute respiratory distress syndrome (ARDS)[7,8]. Other studies have sought to identify the relationship between driving pressure (DP) and in-hospital mortality in populations without acute respiratory distress syndrome (ARDS)[11]. In the study by Silveira Júnior, Cardoso e Rieder[11] the values of driving pressure (DP) and mechanical power (MP) were not associated with mortality in trauma patients without ARDS.

Other ventilatory parameters, such as respiratory rate, can also contribute to tissue damage. Measurements of tidal volume (TV) and driving pressure (DP) do not account for the effect of respiratory rate on the development of lung parenchymal injury by ventilation (LPIV). By calculating mechanical power (MP), it is possible to account for flow and respiratory rate, in addition to tidal volume and DP, representing the energy applied to the respiratory system[12].

Amato[8] and his research group demonstrated that the indiscriminate use of pressures causes deleterious effects and results in increased mortality. They showed that in the group where PEEP was maintained and DP values were increased (exceeding protective limits), there was a marked increase in mortality. In the group where both PEEP and DP were simultaneously increased, as long as DP values were controlled, mortality did not increase. Finally, in the group where PEEP was increased but DP was maintained and/or decreased, mortality decreased as long as PEEP remained above a certain value.[8]

In the current study, 8.1% of patients had a driving pressure (DP) higher than recommended by the literature (above 15 cmH2O), and 21.2% showed a mechanical power (MP) exceeding 17 J/min. Additionally, the tidal volume (TV) was in accordance with the predicted weight of the patients. However, despite most parameters being aligned with literature recommendations, 63.8% of the sample had death as the hospital outcome.

Factors such as advanced age, tracheostomy,[13] care by the healthcare team, and the structure and number of patients admitted to the ICU may be associated with mortality.[14] Additionally, individuals undergoing invasive mechanical ventilation (IMV) are believed to have a worse prognosis, including higher mortality rates and longer ICU stays.[15] All patients in this study were on

IMV, which may partly explain the high mortality rate found in the sample. However, investigating the risk factors associated with mortality was not the focus of this research. Severe patients who require IMV, such as those with acute neurological conditions, can have mortality rates around 33%[16-18]. Our study revealed a mortality rate significantly higher than this, which should prompt the hospital service to investigate the reasons for such an outcome. We hope that this finding encourages reflection among healthcare professionals in this regard.

Another important finding was that 64.6% of the sample exhibited hyperoxia. Although oxygen is essential for cellular respiration, excessive amounts lead to the production of reactive oxygen species, causing cellular damage and death. On the other hand, hyperoxia is not easily detected by pulse oximetry, which can result in significant discrepancies between SpO2 and arterial oxygen pressure (PaO2). Therefore, caution is needed when determining the ideal oxygen dosage using SpO2 records, and it is important to confirm with arterial blood gas analysis.[21]

Observational studies indicate that both high and low PaO2 levels are associated with mortality in critically ill patients.[21-23] In the review by Damiani et al[24] an association was observed between arterial hyperoxia and increased hospital mortality in critically ill patients with diagnoses such as stroke, traumatic brain injury, and post-cardiac arrest, clinical profiles similar to those in the present study. However, the authors caution that these data should be interpreted carefully due to the heterogeneity of criteria used to define exposure to hyperoxia.

Despite the findings contributing to future studies, this research had some limitations. The heterogeneity of the sample, as well as its size, were notable limitations. Additionally, the study's objective was to verify whether the parameters were in accordance with the literature, providing a more descriptive rather than inferential analysis.

CONCLUSION

In some situations, the ventilatory parameters were adjusted in a non-protective manner according to the scientific literature (8.1% with increased DP and 21.2% with increased MP). Mortality was observed in 63.8% of the sample. SpO2 values were above 95% in 64.6% of the assessments, which is directly related to LPIV. These findings may be associated with higher mortality in the studied population.

REFERENCES

1. Curley GF, Laffey JG, Zhang H, Slutsky AS. Biotrauma and ventilator-induced lung injury: clinical implications. Chest [Internet]. 2016 [cited 2024 Jun 19];150(5):1109-17. Available from: https://journal.chestnet.org/article/S0012-3692(16)52763-9/abstract. doi: 10.1016/j.chest.2016.07.019.

2. Tremblay LN, Slutsky AS. Ventilator-induced injury: from barotrauma to biotrauma. Proc Assoc Am Physicians [Internet]. 1998 [cited 2023 Jul 13];110(6):482-8. Available from: https://pubmed.ncbi.nlm.nih.gov/9824530/.

3. Fan E, Villar J, Slutsky AS. Novel approaches to minimize ventilator-induced lung injury. BMC Med. 2013 Mar 28;11(1).

4. Chiumello D, Carlesso E, Brioni M, Cressoni M. Airway driving pressure and lung stress in ARDS patients. Crit Care [Internet]. 2016 [cited 2024 Jun 19];20(1):1-10. Available from: https://ccforum.biomedcentral.com/articles/10.1186/s13054-016-1446-7. doi: 10.1186/s13054-016-1446-7 .

5. Costa ELV, Slutsky AS, Brochard LJ, Brower R, Serpa-Neto A, Cavalcanti AB, Mercat A, Meade M, Morais CAC, Goligher E, Carvalho CRR, Amato MBP. Ventilatory variables and mechanical power in patients with acute respiratory distress syndrome. Am J Respir Crit Care Med. 2021;204(3):303-11.

6. Cammaroto A, Brioni M, Montaruli C, Nikolla K, Guanziroli M, Dondossola D, Gatti S, Valerio V, Vergani GL, Pugni P, Cadringher P, Gagliano N, Gattinoni L. Mechanical power and development of ventilator-induced lung injury. Anesthesiology. 2016;124(5):1100-8.

7. Tonetti T, Vasques F, Rapetti F, Maiolo G, Collino F, Romitti F, Camporota L, Cressoni M, Cadringher P, Quintel M, Gattinoni L. Driving pressure and mechanical power: new targets for VILI prevention. Ann Transl Med. 2017;5(14):1-10.

8. Amato MBP, Meade MO, Slutsky AS, Brochard L, Costa ELV, Schoenfeld DA, Stewart TE, Briel M, Talmor D, Mercat A, Richard JC, Carvalho CR, Brower RG. Driving Pressure and Survival in the Acute Respiratory Distress Syndrome. N Engl J Med. 2015;372(8):747-55.

9. Toufen Junior C, De Santis Santiago RR, Hirota AS, Carvalho ARS, Gomes S, Amato MBP, Carvalho CRR. Driving pressure and long-term outcomes in moderate/severe acute respiratory distress syndrome. Ann Intensive Care [Internet]. 2018 [cited 2024 Jun 19];8(1). Available from: https://annalsofintensivecare.springeropen.com/articles/10.1186/s13613-018-0469-4. doi: 10.1186/s13613-018-0469-4.

10. Beitler JR, Malhotra A, Thompson BT. Ventilator-induced lung injury. Clin Chest Med. 2016 Dec 1;37(4):633-46.

11. Silveira Júnior JC, Cardoso EK, Rieder M de M. Driving pressure and mortality in trauma without acute respiratory distress syndrome: a prospective observational study. Rev Bras Ter Intensiva. 2021;33(2):261-5.

12. Dianti J, Matelski J, Tisminetzky M, Walkey AJ, Munshi L, Sorbo L Del, Fan E, Costa EL, Hodgson CL, Brochard L, Goligher EC. Comparing the effects of tidal volume, driving pressure, and mechanical power on mortality in trials of lung-protective mechanical ventilation. Respir Care. 2021;66(2):221-7.

13. Colpan A, Akinci E, Erbay A, Balaban N, Bodur H. Evaluation of risk factors for mortality in intensive care units: a prospective study from a referral hospital in Turkey. Am J Infect Control. 2005;33(1):42-7.

14. Weigl W, Adamski J, Goryński P, Kański A, Hultström M. ICU mortality and variables associated with ICU survival in Poland: a nationwide database study. Eur J Anaesthesiol. 2018;35(12):949-54.

15. Oliveira ABF, Dias OM, Mello MM, Araujo S, Dragosavac D, Nucci A, Falcão ALE. Fatores associados à maior mortalidade e tempo de internação prolongado em uma unidade de terapia intensiva de adultos. Rev Bras Ter Intensiva [Internet]. 2010 Sep [cited 2024 Jun 19];22(3):250–6. Available from: https://www.scielo.br/j/rbti/a/6qrwTw99v7yvyFSKH3b3VBH/abstract/?lang=pt#. doi: 10.1590/S0103-507X2010000300006.

16. Jeng JS, Huang SJ, Tang SC, Yip PK. Predictors of survival and functional outcome in acute stroke patients admitted to the stroke intensive care unit. J Neurol Sci. 2008;270(1–2):60-6.

17. Huang HY, Lee CS, Chiu TH, Chen HH, Chan LY, Chang CJ, Chang SC, Hu HC, Kao KC, Chen NH, Lin SM, Li LF. Clinical outcomes and prognostic factors for prolonged mechanical ventilation in patients with acute stroke and brain trauma. J Formos Med Assoc. 2022;121(1):162-9.

18. Pelosi P, Ferguson ND, Frutos-Vivar F, Anzueto A, Putensen C, Raymondos K, Apezteguia C, Desmery P, Hurtado J, Abroug F, Elizalde J, Tomicic V, Cakar N, Gonzalez M, Arabi Y, Moreno R, Esteban A, Ventila Study Group. Management and outcome of mechanically ventilated neurologic patients. Crit Care Med. 2011;39(6):1482-92.

19. Hochberg CH, Semler MW, Brower RG. Oxygen toxicity in critically ill adults. Am J Respir Crit Care Med. 2021;204(6):632-41.

20. Rogers LK, Cismowski MJ. Oxidative stress in the lung: the essential paradox. Curr Opin Toxicol. 2018;7:37-43.

21. Young PJ, Hodgson CL, Rasmussen BS. Oxygen targets. Intensive Care Med [Internet]. 2022 May 5[cited 2024 Jun 19];48(6):732–5. Available from: https://link.springer.com/article/10.1007/s00134-022-06714-0. doi: 10.1007/s00134-022-06714-0.

22. de Jonge E, Peelen L, Keijzers PJ, Joore H, de Lange D, van der Voort PHJ, Bosman RJ, de Waal RA, Wesselink R, de Keizer NF. Association between administered oxygen, arterial partial oxygen pressure and mortality in mechanically ventilated intensive care unit patients. Crit Care. 2008;12(6):1-8.

23. Palmer E, Post B, Klapaukh R, Marra G, MacCallum NS, Brealey D, Ercole A, Jones A, Ashworth S, Watkinson P, Beale R, Brett SJ, Young JD, Black C, Rashan A, Martin D, Singer M, Harris S. The association between supraphysiologic arterial oxygen levels and mortality in critically ill patients a multicenter observational cohort study. Am J Respir Crit Care Med. 2019;200(11):1373-80.

24. Damiani E, Adrario E, Girardis M, Romano R, Pelaia P, Singer M, Donati A. Arterial hyperoxia and mortality in critically ill patients: a systematic review and meta-analysis. Crit Care. 2014;18(6).

Erika Letícia Gomes Nunes
https://orcid.org/0000-0002-5754-7766 - http://lattes.cnpq.br/2912861835155118

Isadora Oliveira Freitas Barbosa
https://orcid.org/0000-0001-9585-6538 - http://lattes.cnpq.br/0930734495900171

Maristela Lúcia Soares Campos
https://orcid.org/0000-0002-7617-0538 - http://lattes.cnpq.br/3778037465953515

Bruna Kelly Ferreira
https://orcid.org/0000-0001-8808-471X - http://lattes.cnpq.br/7645606816676048

Giulliano Gardenghi
https://orcid.org/0000-0002-8763-561X - http://lattes.cnpq.br/1292197954351954

MAILING ADDRESS

ERIKA LETÍCIA GOMES NUNES
Hospital de Urgências de Goiás (HUGO) – COREMU;
Avenida 31 de Março, s/n, Av. Pedro Ludovico, Goiânia - GO, 74820-300
E-mail: erikalgn18@gmail.com

Spelling Check: Dario Alvares
Received: 18/06/24. Accepted: 15/07/24. Published on: 12/08/24.