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Original article

Inspiratory pauses of 0.5 and 2.0 seconds during respiratory mechanics assessment do not produce hemodynamic changes in mechanically ventilated patients: cross-sectional study

Pausas inspiratórias de 0,5 e 2,0 segundos durante avaliação de mecânica respiratória não produzem alterações hemodinâmicas em pacientes sob ventilação mecânica: estudo transversal

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ABSTRACT | INTRODUCTION: The heart-lung interaction is influenced by Mechanical Ventilation (MV), which directly impacts venous return and cardiac output through, but not limited to, adjustments in Positive End-Expiratory Pressure (PEEP) and mean airway pressure (Pmean). Additionally, inspiratory pauses for the assessment of pulmonary mechanics interrupt thoracic movement, potentially further impacting this interaction. **OBJECTIVE:** To compare hemodynamic changes during 0.5 and 2.0-second inspiratory pauses during respiratory mechanics measurements. **METHODS:** This is a cross-sectional study conducted in the intensive care units of a hospital in Salvador/BA. Patients on MV and over 18 years old were included. Exclusions were made for those with hemodynamic instability and sustained hypoxemia during the evaluation. For sample characterization, patients were divided into groups with and without pulmonary conditions. The main data collected and analyzed were PEEP, Pmean, Systolic Blood Pressure (SBP), Diastolic Blood Pressure (DBP), Mean Arterial Pressure (MAP), and Heart Rate (HR). For data comparison, Wilcoxon-Rank and Mann-Whitney tests were used for paired and unpaired data, respectively. **RESULTS:** Thirty-seven patients were included, with a median age of 63 years, 19 (51.4%) males, and 30 (81.1%) with an admission diagnosis of a clinical nature. No statistically significant hemodynamic changes were identified between the 0.5 and 2.0-second inspiratory pause times in the variables SBP (p=0.99), DBP (p=0.11), MAP (p=0.29), and HR (p=0.25). **CONCLUSION:** No hemodynamic variations were identified during respiratory mechanics measurements at 0.5 and 2.0-second inspiratory pauses.

KEYWORDS: Respiratory Mechanics. Physiotherapy. Hemodynamic Monitoring.

RESUMO | INTRODUÇÃO: A interação coração-pulmão influenciada pela Ventilação Mecânica (VM), que impacta diretamente no retorno venoso e débito cardíaco através, e não somente, de ajustes da Pressão Positiva Expiratória Final (PEEP) e Pressão média nas vias aéreas (Pmed). Além disso, as pausas inspiratórias para avaliação da mecânica pulmonar interrompem o movimento torácico, pode impactar mais nesta interação. **OBJETIVO:** Comparar as alterações hemodinâmicas durante os tempos de 0,5 e 2,0 segundos de pausa inspiratória durante as mensurações de mecânica respiratória. **MÉTODOS:** Trata-se de um estudo transversal, realizado nas unidades de terapia intensivas de um hospital público de Salvador/BA. Foram incluídos pacientes em uso de VM e acima de 18 anos. Os excluídos foram aqueles que apresentassem instabilidade hemodinâmica e hipoxemia sustentada durante a avaliação. Para caracterização amostral, os pacientes foram divididos em grupos daqueles com e sem afecções pulmonares. Os principais dados coletados e analisados foram PEEP, Pmed, Pressão Arterial Sistólica (PAS), Pressão Arterial Diastólica (PAD), Pressão Arterial Média (PAM), Frequência Cardíaca (FC). Para comparação de dados foram utilizados os testes *Wilcoxon-Rank* e *Mann-Whitney* para dados pareados e não pareados, respectivamente. **RESULTADOS:** Foram incluídos 37 pacientes, mediana de idade 63 anos, 19 (51,4%) do sexo masculino, 30 (81,1%) com diagnóstico admissional de natureza clínica. Não foram identificadas alterações hemodinâmicas estatisticamente significantes entre os tempos de pausa inspiratória de 0,5 e 2,0 segundos nas variáveis PAS (p=0,99), PAD (p=0,11), PAM (p=0,29) e FC (p=0,25). **CONCLUSÃO:** Não foram identificadas variações hemodinâmicas durante as mensurações da mecânica respiratória nas pausas de 0,5 e 2,0 segundos.

PALAVRAS-CHAVE: Mecânica Respiratória. Fisioterapia. Monitorização Hemodinâmica.

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1. Introduction

Ventilatory mechanics in patients undergoing mechanical ventilation (MV) are crucial during daily assessments in the intensive care unit (ICU). This involves measuring lung function through variables such as pressure, volume, flow, and their resulting effects on the respiratory system. Such evaluations can be conducted by implementing an inspiratory pause of 0.5 to 2.0 seconds, during which, in a near-zero flow situation, values such as quasi-static compliance (Cst), driving pressure (DP), respiratory system resistance (Rsr), and resistance pressure (Pres) can be calculated.[1-3]

Inspiratory pause values between 0.5 and 2.0 seconds are described as essential for assessing patients under MV, where the choice of duration may be linked to pulmonary characteristics, specifically the multicompartimentality related to different areas of ventilation and even pulmonary perfusion/circulation.[4-6]

However, in the context of heart-lung interaction, any non-physiological variation at the pulmonary level can directly impact venous return and cardiac output affecting this interaction.[7] The most commonly studied variable is positive end expiratory pressure (PEEP), where adjustments starting from 15 cmH_2O extrinsically increase transpulmonary pressure, directly impacting the right ventricular preload, and intrinsically compress pulmonary circulation blood vessels, increasing right ventricular afterload and decreasing left ventricular afterload.[8]

Another variable that is unfortunately under-studied but also has a close relationship with central venous pressure and cardiac output is the mean airway pressure (Pmean).[7,9] This type of evaluation addresses data related to both pulmonary filling pressures and stabilization, as well as the respiratory cycle itself (inspiration-expiration ratio and inspiratory time), closely linking it to cardiac function.[7,9]

In addition to cardiovascular changes caused by PEEP and reflected in Pmed, it is understood that, given the short duration of respiratory interruption, the inspiratory pause can also cause some hemodynamic variations, potentially altering volumes and pressures in the cardiac chambers.[8] However, few studies[10-12] have evaluated the effect of the inspiratory pause on the hemodynamics of patients under MV and, to date, no study has analyzed its effects during the assessment of pulmonary mechanics. Therefore, this study aims to investigate the possible existence of changes in hemodynamic variables during the evaluation of respiratory mechanics between inspiratory pause times of 0.5 and 2 seconds.

2. Method

This is a cross-sectional, prospective study, constructed according to the STROBE[13] (STrengthening the Reporting of OBservational studies in Epidemiology) guidelines, conducted in the ICUs of the Hospital do Subúrbio, Salvador – Bahia, from March 2022 to January 2024. This study was approved by the Research Ethics Committee under CAAE 57895516.8.0000.5028. The data were collected upon the signing of the Free and Informed Consent Term (physical or virtual) by the responsible parties of the respective patients.

Patients on mechanical ventilation of both sexes, aged 18 years or older, with no or low interaction with the ventilatory prosthesis visualized through graphic analysis on the ventilator, with stable hemodynamics characterized by the absence or low doses of vasoactive drugs (0.5 μg/ml/kg/min), absence of axial fractures and chest deformities and absence of unresolved intrathoracic impairments (pneumothorax and hemothorax) were included. Patients who presented severe hemodynamic instability during the ventilatory mechanics measurements, evaluated through a systolic blood pressure (SBP) of less than 90 mmHg and sustained hypoxemia analyzed through peripheral oxygen saturation (SpO_2) less than 90%, were excluded.

Primary data sources included the records of respiratory mechanics measurements (peak pressure [Ppeak], plateau pressure [Pplat], Cst and Rrs) with two different inspiratory pause times (0.5 and 2.0 seconds), as well as parameters of SpO_2 and hemodynamic function (SBP, diastolic blood pressure [DBP], mean arterial pressure [MAP], and Heart Rate [HR]). Secondary data such as age, sex, reason for hospitalization, intubation date, and presence of comorbidities were extracted from each patient's medical records.

The process of measuring the patients' ventilatory mechanics was performed using the Dräger Evita 4® and Dräger Savina® mechanical ventilators. Data collection was conducted by physiotherapists who were postgraduate degree students in the Intensive Care Physiotherapy Residency Program, previously trained to perform the evaluation. The ventilatory parameters for this measurement were: volume-controlled assist mode (VCV); tidal volume (Vt) calculated using the ARDSnet formula[14] (Equation 1), calculated as 6 ml/kg of predicted body weight; respiratory rate (RR) set at 15 bpm; flow of 40 L/min with a square wave; inspiratory pause initially set at 0.5 seconds for the first ventilatory mechanics measurement and then 2.0 seconds for the second. Only one measurement was taken for each pause. After adjustments, values for DP, Equation 2; Ppeak, Pplat, and Cest, as per Equation 3; Pres, as per Equation 4; and Pmean, as per Equation 5[2,15] were calculated.

$$\text{Predicted Body Weight}_{Men} = (\text{Height} - 152{,}4) \times 0{,}95 + 50$$
$$\text{Predicted Body Weight}_{Women} = (\text{Height} - 152{,}4) \times 0{,}95 + 45{,}5$$

Equation 1. Predicted Body Weight formula.

$$DP = Pplat - PEEP$$

Equation 2. Driving Pressure (DP) formula. PEEP: Positive End Expiratory Pressure

$$Cst = Vt / (Pplat - PEEP)$$

Equation 3. Quasi-Static Compliance (Cst) formula. Vt: Volume tidal; Pplat: Plateau Pressure; PEEP: Positive End Expiratory Pressure

$$Pres = Ppeak - Pplat$$

Equation 4. Resistive Pressure (Pres) formula. Ppeak: Peak Pressure; Pplato: Plateau Pressure

$$Pmean = (Ppeak - PEEP) / 2 \times (Tins/Ttot) - PEEP$$

Equation 5. Mean Airway Pressure (Pmean) formula. Ppeak: Peak Pressure; PEEP: Positive End Expiratory Pressure; Tins: Inspiratory Time; Ttot: Total respiratory cycle time

Upon obtaining all data, patients were divided into two groups: those with lung conditions, including those with pulmonary infections and/or comorbidities such as heart failure, smoking, obesity, chronic kidney disease; and those without lung conditions, with intubation determined by postoperative recovery that did not involve upper abdominal or chest surgery, decreased consciousness, stroke and seizure.

For statistical analysis, the Shapiro-Wilk test and histogram graphs[16] were used to assess data distribution. Since most data showed a non-normal distribution, the median (ME) was used as a measure of central tendency and the interquartile range (IQR) as a measure of dispersion. Categorical data were computed through absolute and relative frequencies. For group comparisons, non-parametric tests such as the Wilcoxon Rank test for paired data and the Mann-Whitney test for unpaired data were used. Correlations were assessed using the non-parametric Spearman's test.

The data were stored in Excel and analyzed using IBM SPSS (Statistical Package for the Social Sciences) software. P-values < 0.05 were considered statistically significant.

3. Results

A total of 37 patients were included in the study and none exhibited hemodynamic instability during the assessment. The sample data are presented in Table 1, with most participants being male (19, 51.4%), 30 (81.1%) hospitalized for clinical reasons, and the main comorbidity identified being systemic arterial hypertension in 24 (64.9%) of the participants (Table 1).

Table 1. Demographic and clinical variables of the research participants and changes in hemodynamic variables during the evaluation of respiratory mechanics between inspiratory pause times of 0.5 and 2 seconds

Variables	Sample (n=37)
Age in years, ME (IQR)	63 (47.5-75.5)
Male gender, n (%)	19 (51.4)
Height in cm, ME (IQR)	166.0 (158.5-171.5)
Predicted weight in kg, ME (IQR)	60.0 (51.0-66.5)
Clinical profile, n (%)	30 (81.1)
Surgical profile, n (%)	7 (18.9)
Presence of lung condition, n (%)	13 (35.1)
Comorbidities	
Hypertension, n (%)	24 (64.9)
Heart failure, n (%)	18 (48.6)
Diabetes mellitus, n (%)	14 (37.8)
Atrial fibrillation, n (%)	6 (16.2)

Subtitle: ME: Median; IQR: Interquartile Range; cm: Centimeters; Kg: Kilogram.
Source: the authors (2024).

Regarding the comparison of ventilatory and hemodynamic values during 0.5 and 2.0-second inspiratory pauses, Table 2 shows no statistically significant variations in SBP (p=0.99), DBP (p=0.11), MAP (p=0.29), and HR (p=0.25). However, the ventilatory values showed statistically significant differences between the two pause times, with Pmean during the 0.5-second pause showing values of 8.4 (6.9-12.7) cmH_2O versus 9.3 (7.6-12.2) cmH_2O during the 2.0-second pause (p<0.01) and Cst relative to predicted weight translating values during the 0.5-second pause of 0.60 (0.50-0.80) $ml/cmH_2O/kg$ versus 0.67 (0.60-0.76) $ml/cmH_2O/kg$ during the 2.0-second pause (p=0.01).

In comparisons between groups with and without lung conditions regarding values at 0.5-second versus 2.0-second pauses, there were statistically significant differences in Pmean (group without lung condition, 0.5-second pause resulting in 8.7 [6.9-13.1] cmH_2O versus 9.4 [7.8-12.2] cmH_2O during the 2.0-second pause, p<0.01) and Cst relative to predicted weight (group with lung condition, 0.5-second pause with values of 0.55 [0.47-0.61] $ml/cmH_2O/kg$ versus 0.63 [0.56-0.73] $ml/cmH_2O/kg$ during the 2.0-second pause). These data are found in Table 3.

Table 2. Ventilatory and hemodynamic variables during 0.5 and 2.0-second inspiratory pauses

Variables, ME (IQR)	0.5s Pause	2,0s Pause	p *value*[&]
Pmean (cmH_2O)	8.4 (6.9-12.7)	9.3 (7.6-12.2)	<0.01
Cst/PBW ($ml/cmH_2O/kg$)	0.67 (0.60-0.76)	0.60 (0.50-0.80)	0.01
SBP (mmhg)	125.0 (109.0-143.5)	129.0 (113.0-142.0)	0.99
DBP (mmhg)	66.0 (53.0-78.5)	65.0 (53.0-75.0)	0.11
MAP (mmhg)	81.0 (76.0-97.5)	85.0 (72.0-96.5)	0.29
HR (bpm)	86.0 (68.5-97.5)	87.0 (68.5-97.5)	0.25

Subtitle: ME: Median; IQR: Interquartile Range; s: seconds; cmH_2O: Centimeters of Water; Cst: Quasi-Static Compliance; PBW: Predicted Body Weight; ml: milliliters; SBP: Systolic Blood Pressure; DBP: Diastolic Blood Pressure; MAP: Mean Arterial Pressure; HR: Heart Rate; &: Wilcoxon Test.
Source: the authors (2024).

Upon identifying elevated PEEP values in the group with lung conditions, the impact of elevated PEEP parameters on patient hemodynamics was also evaluated. However, even when categorized into groups with a PEEP cutoff point of 8 cmH_2O and 10 cmH_2O, no statistically significant values were found in SBP, MAP, DBP and HR between the groups.

In addition to the descriptive and comparative data tests, correlations were also performed between hemodynamic variables and the ventilatory parameters most associated with their changes, such as PEEP and Pmean during a 2.0-second inspiratory pause. No statistically significant differences were found in hemodynamics versus PEEP values (SBP [p=0.26], MAP [p=0.46], DBP [p=0.91], and HR [p=0.68]) and versus Pmean values (SBP [p=0.62], DBP [p=0.35], MAP [p=0.78]).

Table 3. Ventilatory and hemodynamic variables of patients divided into groups with and without lung conditions and their comparisons between 0.5 and 2.0-second pauses

Variables, ME (IQR)	**Without Lung Condition (n=24)**	**With Lung Condition (n=13)**
Pmean 2.0 (cmH_2O)	9.4 (7.8-12.2)	9.0 (7.3-12.7)
Pmean 0.5 (cmH_2O)	8.7 (6.9-13.1)	8.5 (7.0-12.3)
p *value*&	<0.01	0.30
Cst/PBW 2.0*	0.67 (0.60-0.84)	0.63 (0.56-0.73)
Cst/PBW 0.5*	0.67 (0.50-0.85)	0.55 (0.47-0.61)
p *value*&	0.44	<0.01
SBP 2.0 (mmhg)	125.5 (107.2-142.0	129.0 (107.0-142.0)
SBP 0.5 (mmhg)	129.0 (106.5-147.5)	123.0 (113.5-143.5)
Δ	1 (-0.7-4.0)	-1.0 (-5.0-1.0)
p *value*&	0.25	0.13
DBP 2.0 (mmhg)	64.0 (55.2-81,0)	65.0 (49.0-74.5)
DBP 0.5 (mmhg)	66.0 (50.0-78,5)	69.0 (51.5-79.2)
Δ	1.0 (-1.5-3.0)	1.0 (-1.0-3.5)
p *value*&	0.24	0.23
MAP 2.0 (mmhg)	79.5 (72.0-99.5)	85.0 (79.5-93.5)
MAP 0.5 (mmhg)	80.5 (75.2-100.7)	82.0 (78.2-97.2)
Δ	0.0 (-2.0-3.0)	1.0 (-0.5-3.0)
p *value*&	0.17	0.95
HR 2.0 (bpm)	85.0 (67.0-106.0)	84.5 (67.2-94.5)
HR 0.5 (bpm)	84.0 (68.0-103.0)	82.0 (78.2-97.2)
Δ	-1.0 (-2.0-1.0)	0 (-3.0-2.0)
p *value*&	0.48	0.30

ME: Median; IQR: Interquartile Range; cmH_2O: Centimeters of Water; Cst: Quasi-Static Compliance; PBW: Predicted Body Weight in Kilograms; Kg: Kilogram; *: values in ml/cmH_2O/kg; Δ: comparison between 2.0 and 0.5 seconds; ml: milliliters; SBP: Systolic Blood Pressure; DBP: Diastolic Blood Pressure; MAP: Mean Arterial Pressure; HR: Heart Rate; &: Wilcoxon Test.
Source: the authors (2024).

4. Discussion

Based on the data analyzed in this study, no statistically significant differences were found in the hemodynamic values of SBP, DBP, MAP and HR during the assessment of ventilatory mechanics at inspiratory pauses of 0.5 and 2.0 seconds. Additionally, no significant correlations were identified between PEEP and Pmean values with hemodynamic variables. From these comparisons and the absence of hemodynamic adverse events during the respiratory mechanics evaluation, the present study suggests that variations in blood pressure and HR did not show any impact during inspiratory pause adjustments, regardless of pause duration.

Findings regarding inspiratory pause and clinically relevant hemodynamic changes for assessments are also corroborated by other studies[10-12] that performed such maneuvers for different strategies. For instance, the study by Galhardo et al.[12] demonstrated that the hyperinflation maneuver in MV with a 2.0-second inspiratory pause resulted in DBP alteration compared to the same maneuver without an inspiratory pause, without significant clinical changes. However, these alterations may be related to the incidence of AutoPEEP. Agreeing with the absence of clinical symptoms, the studies by Venezian et al.[11] and Chicayban[10] did not find significant differences in hemodynamics during inspiratory pauses of 2.5 seconds and 3.0 seconds, respectively, associated with pulmonary hyperinflation in MV. These data support the present study, which also did not identify instability or statistically significant hemodynamic differences between pauses of 0.5 and 2.0 seconds.

The reproducibility of the evaluated values, the study by Menezes Júnior et al.[4] demonstrated that using either 0.5 seconds or 2.0 seconds are reproducible parameters that yield similar results in patients with more homogeneous lungs. This study also emphasizes that using inadequate pause times or even those outside institutional protocols can generate incorrect measurements, and times longer than necessary can expose the patient to increased pulmonary and capillary stress. However, our study suggests that the measurement of ventilatory mechanics is hemodynamically safe even using a 2.0-second inspiratory pause.

Therefore, the determination of which pause time to use depends on pulmonary characteristics, especially acute and chronic pathological conditions. According to Henderson et al.[5], lungs affected by Acute Respiratory Distress Syndrome benefit from prolonged pauses (between 2 and 5 seconds) to eliminate heterogeneous issues of these parenchyma, responding to viscoelastic gaseous properties and alveolar recruitment due to gas redistribution. However, according to Menezes Júnior[4], patients with lungs close to homogeneity have reproducible values in both 0.5-second and 2.0-second pauses.

Concerning hemodynamic variations during MV, Long et al.[9] determined in their study that the relationship between ventilatory parameters and hemodynamics is closer to Pmean than to variations in Vt or inspiratory pauses alone. According to these authors, maintaining elevated Pmean (above 9.64 cmH_2O) significantly alters central venous pressure, which is associated with increased preload in the right ventricle, directly impacting systemic and pulmonary blood flow. In the present study, in addition to no macro hemodynamic alterations being identified, the median Pmean of the patients was 9.3 cmH_2O.

In this context, in addition to performing significant changes in central venous pressure, Pmean can be an important variable in determining the prognosis of critically ill patients under MV. According to the study by Sahetya et al.[15], the median Pmean was 13.0 cmH_2O in patients who died within 90 days of follow-up. Additionally, the same authors state that this parameter resembles Pplat and DP in terms of mortality prediction.

Besides Pmean, PEEP also directly interferes with the hemodynamics of patients under MV. If this parameter is set near or above 15 cmH_2O, transpulmonary pressure increases after expiration, hindering venous return and overloading the right ventricle due to imposed vascular resistance.[5] In this case, according to data reported by the ART trial[17], patients who received maximal alveolar recruitment maneuvers with PEEP up to 45 cmH_2O had a higher incidence of barotrauma, pneumothorax, and the need for vasopressors than the low PEEP group guided by the PEEP x fraction of inspired oxygen table. However, the group of patients with pulmonary involvement showed the highest median PEEP value at 9.1 cmH_2O, which seems to have no significant effect on hemodynamics during pulmonary mechanics evaluation.

It is important to highlight that, given the scarcity of other studies in the literature addressing comparisons in hemodynamic variability, this study also proposes to support new discussions on this topic. This study has some limitations: the absence of sample size calculation and a small sample size, which may limit the generalization of the data to a broader population; the absence of a severity score, meaning patients with higher clinical severity may present hemodynamic instability more easily than patients not using vasopressors, for example.

5. Conclusion

Based on the data obtained from this study, no adverse events or statistically significant differences were found in the values of PAS, PAD, PAM, and FC during the measurement of respiratory mechanics at inspiratory pauses of 0.5 and 2.0 seconds.

Author contributions

The authors declare that they made substantial contributions to the work, including the conception or design of the research; the acquisition, analysis, or interpretation of data for the work; and the drafting or critical revision of intellectually relevant content. All authors approved the final version to be published and agreed to assume public responsibility for all aspects of the work.

Conflicts of interest

No financial, legal, or political conflicts involving third parties (such as government, companies, and private foundations) have been declared for any aspect of the submitted work (including, but not limited to, grants and funding, advisory board participation, study design, manuscript preparation, statistical analysis, etc.).

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