

Mechanical power during mechanical ventilation

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ABSTRACT

Mechanical ventilation provides lifesaving support for patients with acute respiratory failure. However, the pressures and volumes required to maintain gas exchange can cause ventilator-induced lung injury. The current approach to mechanical ventilation involves attention to both tidal volume and airway pressures, in particular plateau pressures and driving pressures. The ventilator provides energy to overcome airway resistance and to inflate alveolar structures. This energy delivered to the respiratory system per unit time equals mechanical power. Calculation of mechanical power provides a composite number that integrates pressures, volumes, and respiratory rates. Increased levels of mechanical power have been associated with tissue injury in animal models. In patients, mechanical power can predict outcomes, such as ICU mortality, when used in multivariable analyses. Increases in mechanical power during the initial phase of ventilation have been associated with worse outcomes. Mechanical power calculations can be used in patients on noninvasive ventilation, and measurements of mechanical power have been used to compare ventilator modes. Calculation of mechanical power requires measurement of the area in a hysteresis loop. Alternatively, simplified formulas have been developed to provide this calculation. However, this information is not available on most ventilators. Therefore, clinicians will need to make this calculation. In summary, calculation of mechanical power provides an estimate of the energy requirements for mechanical ventilation based on a composite of factors, including airway resistance, lung elastance, respiratory rate, and tidal volume.

Keywords: mechanical ventilation, mechanical power, ventilator-induced lung injury, energy, work

INTRODUCTION

Mechanical ventilation provides lifesaving support for patients with acute respiratory failure. However, this support can also cause ventilator-induced lung injury.¹ The usual classification for ventilator-induced lung injury includes barotrauma, volutrauma, atelectrauma associated with the repeated opening and closing areas of the lung parenchyma, and biotrauma with the release of inflammatory markers into the lung and systemic circulation. Determining whether or not ventilator-induced lung disease develops in a patient on

mechanical ventilation is difficult since the initial disorder causing acute respiratory failure causes lung injury with edema formation, inflammation, and potentially fibrosis. Current ventilator standards concentrate on “safe ventilation” with smaller tidal volumes (6–8 ml/kg ideal body weight), reduced plateau pressures (<30 cm H₂O), and reduced driving pressures (plateau pressure – PEEP <15 cm H₂O). The respiratory rate and minute ventilation should be adjusted to maintain PaCO₂ levels at or above 40 mmHg. Patients usually require sedatives and narcotics for comfort and better interaction with ventilators. The FiO₂ and PEEP combination can be based on the low PEEP level or the high PEEP level tables. Patients with very poor gas exchange may benefit from short-term use of paralytic drugs and the use of prone positioning.¹ This approach to ventilator management focuses on static

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DOI: 10.12746/swrccc.v12i50.1275

intrapulmonary pressures with the expectation that lower pressures are associated with less lung injury. However, low pressure strategies generally require a higher minute ventilation to achieve equivalent gas exchange. The clinician can decrease the energy added per machine breath (tidal volume), but this may increase the energy added per unit of time (power). There are theoretical reasons with some support from experimental evidence that mechanical power may be a more important determinant of lung injury than the work of each delivered tidal volume.

Calculating mechanical power or the energy delivered to the lung per minute during mechanical ventilation provides an alternative approach to understanding the development of ventilator-induced lung injury. Gattinoni and colleagues developed the concept of mechanical power and its effect on the development of ventilator-induced lung injury.² Important parameters include pressures, volumes, flow, and respiratory rates. Abnormalities in the lung parenchyma during acute respiratory failure include differences in the disease process in various regions of the lung resulting in inhomogeneity, cyclic collapse and recruitment of the lung parenchyma, and the primary events associated with the development of lung injury, which include edema formation, inflammation, and fibrosis. The mechanical ventilator applies energy to the lung and chest wall during each ventilatory cycle; the energy per unit time is power. This energy is not distributed uniformly throughout the damaged lung during the respiratory cycle. Consequently, mechanical power calculations provide only an index of the overall mechanical events during the respiratory cycle.

Key equations used in the Gattinoni publication included:

$$W = \sum P dV$$

$$P = ELrs \times \Delta V + Raw \times F + PEEP,$$

Where $ELrs \times \Delta V = \Delta P$, i.e., pressure component due to elastic recoil

$Raw \times F = P_{peak} - P_{plat}$, i.e., pressure component due to air flow

PEEP = base line tension at end expiration

$$\text{Power} = RR (\Delta V^2 \times [0.5 \times ELrs + RR \times (1 + I: E) / (60 \times I: E) \times Raw] + \Delta V \times PEEP)$$

Where ΔV = tidal volume; EL = elastance of the system; $I: E$ = the inspiratory to expiratory time ratio; Raw = airway resistance.

METHODS TO MEASURE MECHANICAL POWER

Mechanical power can be calculated using graphs plotting changes in pressure versus changes in volume during a tidal breath. This requires software to measure this area. An ideal method is to directly measure the volume and pressure measurements using a high rate of sampling during 1 tidal breath. The energy is calculated by solving the integral of airway pressure with respect to change in volume, which represents the area of the pressure-volume loop. This requires high-quality data collection and software to make the calculations. Mechanical power can also be calculated by comprehensive formulas developed by Gattinoni, which requires multiple measurements to calculate the power needed to overcome resistance, elastic recoil, and PEEP. This approach is not practical for most clinicians for bedside management of ventilators. However, surrogate equations have been developed based on the comprehensive equation to provide simpler calculations at the bedside.^{2,3} These include:

1. Volume-controlled ventilation: $MP = [VE \times (Peak\ pressure + PEEP + inspiratory\ flow/6)]/20$
2. Pressure-controlled ventilation: $MP = 0.098 \times RR \times V_t [PEEP + pressure\ above\ PEEP]$

Pressure-volume curves plot changes in pressure against changes in volume during inspiration and expiration. Some of the work required to inflate the lung during inspiration is recovered by relaxation of elastic structures during exhalation. Work required to drive flow across resistance is lost, however, and the loss must be dissipated as heat. Hysteresis is another amount of energy required during inspiration that is not recovered during expiration (Figure 1). The lost energy is heat that may contribute to lung injury if the respiratory system cannot dissipate it. The main determinants of hysteresis are the air liquid surface forces in the alveoli, stress relaxation of lung tissue, and lung re-expansion and collapse during inflation

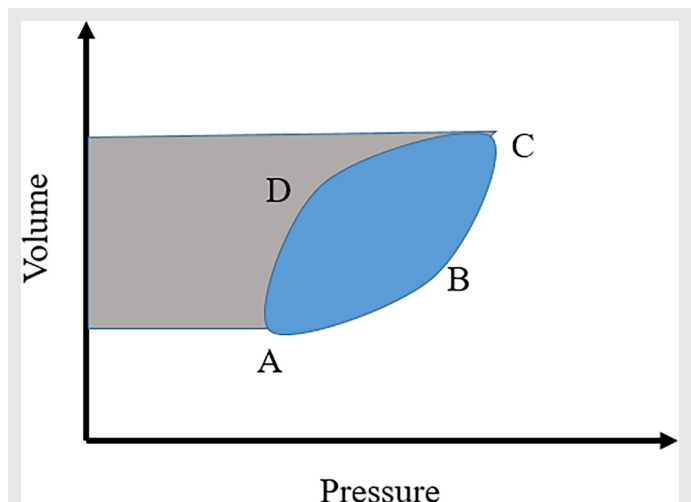


Figure 1. Graphical illustration of work or energy per breath. This is the traditional view of breath cycles in medicine and physiology. In this view, work or energy of movement in the plane is the area or integral to the LEFT of the curve. Physics and thermodynamics discussions display volume as the x-axis and pressure as the y-axis, so work is the integral under the curve. A breath cycle moves from A to B to C during inspiration and from C to D to A during exhalation. The gray area is the energy recovered as elastic tissues relax during exhalation. The gray + blue areas represent the work or energy required to inflate the lung during inspiration. The blue elliptical area represents the net work required to be added to the respiratory system for each breath. This is also the net energy per breath that must be dissipated as heat.

and deflation. These loops can be evaluated at different PEEP levels. If PEEP increases and lung volumes increase secondary to recruitment, there should be a change in the configuration of the loop. The mechanical energy calculated from a hysteresis loop should be compared to the energy calculated from various formulas that consider pressure, tidal volume, and resistance. The differences should represent energy lost to heat, associated with tissue injury, and stored in the lung parenchyma.

Gattinoni summarized recent studies on the utility of mechanical power in 2023.⁴ The mechanical power

formula multiplies each pressure component involved in mechanical ventilation by the tidal volume to calculate work or energy. It is then multiplied by the respiratory rate to determine power in joules per minute. The pressure components include elastic pressure, resistive pressure, and static pressure. The need for more mechanical power during mechanical ventilation is associated with mortality, but the boundaries for safe mechanical power levels are uncertain. In pigs, the safe threshold was between 4 to 7 J/min and 12 J/min. In animal studies, the experimental adjustment of respiratory rate, tidal volume, and PEEP cause the same level of lung damage provided mechanical power is similar, referred to as iso-power, at the various settings.⁵ It is likely that mechanical power needs to be normalized to other physical components of the lung, such as compliance or lung volume, or body weight. The distribution of mechanical power during the respiratory cycle may need to be considered, since it is unlikely to be uniform. In addition, the recovery of mechanical power during exhalation also depends on ventilator parameters.^{6,7} In most studies, the measured mechanical power is the energy needed to inflate both the lung and chest wall; to determine the mechanical power applied to the lung only would require placement of an esophageal balloon to measure transpulmonary pressures.

In summary, mechanical energy is used to create flow into the lungs, expand (inflate) the lungs, and maintain volume stability at a various pressure. It also creates heat and can cause tissue injury in some patients. Some energy is stored in the lung, and some is released during exhalation. Mechanical power can be calculated graphically using pressure volume curves. Alternatively, it can be calculated using a comprehensive formula developed by Gattinoni and coworkers. Finally, surrogate formulas have been developed to make it easier to calculate mechanical power at the bedside.³ Mechanical power has been studied in patients with acute respiratory failure to determine its association with outcomes and its changes during an episode of acute respiratory failure and to compare ventilator modes. The energy delivery has been studied in patients on noninvasive ventilation and in animal models. Some of these studies are discussed below.

MECHANICAL POWER AS PREDICTOR OF OUTCOMES

Serpa Neto et al. used two large databases to study the outcomes in patients with acute respiratory failure.⁸ The median mechanical power on the second day of ventilator care was 21.4 J/min in the first cohort and 16.0 J/min in the second cohort. Approximately 10% of the patients had ARDS, and the overall mortality was 29.9% and 31.0% in the 2 cohorts. Mechanical power was independently associated with in-hospital mortality. The odds ratio for each 5 J/min increase was 1.06 in the first cohort and 1.10 in the second cohort. Mechanical power was associated with ICU mortality, 30-day mortality, the number of ventilator-free days, and ICU and hospital length of stay. Higher mechanical power levels were associated with worse outcomes in patients who were on a low tidal volume ventilator strategy and had low driving pressures. Since mechanical power calculations include several ventilator parameters, it might be used as a method to determine optimal ventilator settings that potentially reduce lung injury. In this study the calculation for mechanical power was:

$$\text{MP (J/min)} = 0.098 \times V_t \times \text{RR} \times (\text{P}_{\text{peak}} - 1/2 \times \text{driving pressure}).$$

MECHANICAL POWER NORMALIZED TO BODY SIZE

Zhu used the data stored in a large critical care database.⁹ This study involved patients who were on invasive mechanical ventilation for at least 48 hours, and the mechanical power was normalized to the predicted body weight. This study eventually included 1301 patients; 365 patients died. Patients in the fourth quartile of normalized mechanical power had an increased ICU mortality rate, increased ICU length of stay, and a decreased number of ventilator-free days at 28 days of ventilation. The formula used in this study was:

$$\text{MP} = 0.098 \times V_t \times \text{RR} \times (\text{PIP} - \text{driving pressure} \times 0.5).$$

Serpa Neto combined the clinical and ventilator information from 2 large patient cohorts with acute respiratory failure.¹⁰ This study included 8191 patients requiring invasive ventilation. They calculated absolute mechanical power, mechanical power adjusted for predicted body weight, mechanical power normalized to body mass index, and mechanical power normalized to be a body surface area. All 4 values were increased in non-survivors in this cohort. However, these parameters were not significantly increased in the patients with ARDS. These results suggest that normalized mechanical power calculations can improve predictions of outcomes in patients with acute respiratory failure.

CHANGES IN MECHANICAL POWER DURING MECHANICAL VENTILATION

Chi et al. studied the outcomes of 602 patients who required mechanical ventilation for acute respiratory failure for more than 48 hours.¹¹ This study excluded patients with a mechanical power less than 10 J/min. Patients were classified as having a decrease in mechanical power at 24 hours or an increase or no change in mechanical power at 24 hours. The baseline mechanical power levels were 11.7 J/min in the group with increasing mechanical power and 12.2 J/min in the group with decreasing mechanical power at 24 hours. Patients who had decreased mechanical power had decreased mortality in comparison to the patients who did not. The mortality rates were 24% and 36%. The 24-hour mechanical power variation rate was associated with ICU mortality after adjusting for confounders. All mechanical power components improved in the group that had reduced levels at 24 hours. Minute ventilation and PEEP levels contributed to the increase in mechanical power in the group that had increases in mechanical power. The PaO₂ levels at 24 hours were identical in the 2 groups. Compliance improved or increased in the patients in the improved mechanical power levels. The formula used in this study was:

$$\text{MP} = 0.098 \times \text{RR} \times \text{TV} \times (\text{PIP} - 0.5 \text{ driving pressure}).$$

Pozzi et al. enrolled 69 patients with ARDS in a prospective study to determine outcomes and

mechanical ventilation variables, including PaO₂/FiO₂ ratios, mechanical power, and alveolar dead space fraction.¹² Thirty-six patients (52%) died during the study. The initial mechanical power in the entire cohort was 18.7 (14.7–22.2) J/min, and the mechanical power ratio was 7.0 (5.8–8.3). The PaO₂/FiO₂ was 139 (93–168) and the alveolar dead space fraction was 46 (30–62)%. The only difference between the 2 groups on admission was in the mechanical power ratio which was lower in survivors. Based on CT analysis, the total amount of nonaerated lung tissue was 47 (38–56)%. Patients who survived had higher PaO₂/FiO₂ ratios on the third day of mechanical ventilation in the ICU. These patients also had lower mechanical power ratios and lower alveolar dead space fractions. Based on the average values over 3 days of monitoring, the mechanical power ratio, the driving pressure, and the PaO₂/FiO₂ ratio were significantly associated with ICU mortality. In this study, the mechanical power ratio equals the measured mechanical power divided by a calculated ideal mechanical power based on equations that involved the ideal bodyweight, ideal respiratory rate, and ideal plateau pressure. This equation provides the expected mechanical power based on a healthy lung. In summary, monitoring these gas exchange variables and the energy requirement to deliver a tidal volume (i.e., mechanical power and mechanical power ratio) can predict outcomes in these patients during the initial phase of mechanical ventilation.

MECHANICAL POWER MEASUREMENTS TO COMPARE VENTILATOR MODES

Buiteman-Kruizinga measured mechanical power in 24 patients requiring mechanical ventilation for at least 1 day.¹³ Twelve patients were ventilated with adaptive support ventilation, and 12 patients were ventilated with pressure-controlled ventilation. Mechanical power was calculated 3 times per day. It was lower with adaptive support ventilation than pressure-controlled ventilation. The numbers were 15.1 J/min versus 22.9 J/min. The tidal volumes were similar, but the maximum pressure and respiratory rate were lower with adaptive support ventilation. They concluded that this mode of ventilation may have benefit since it requires lower levels of mechanical power.

MECHANICAL POWER IN COMPARISON TO RESPIRATORY RATE AND DRIVING PRESSURE TO PREDICT OUTCOMES

Costa et al. analyzed patient level data for 4549 patients with acute respiratory failure.¹⁴ The average mechanical power was 0.32 ± 0.14 J/min/kg of predicted body weight. The driving pressure was 15 ± 5.8 cm of water; the respiratory rate was 25.7 ± 7.4 breaths/min. The overall mortality was 38%. Univariable predictors of mortality included driving pressure, PEEP level, plateau pressure, respiratory rate, and mechanical power. Models were subsequently adjusted for a baseline risk factors in patients with ARDS. When all variables were entered into a multivariable model, only driving pressure and respiratory rate were significantly associated with mortality; the effect size of each 1 cm of water increase in driving pressure was approximately 4 times the effect size of a 1 breath/min increase in respiratory rate. The components of mechanical power were then introduced into the model. In this analysis the elastic dynamic component was associated with mortality and had a stronger effect than total power. A model that included a relationship between driving pressure and respiratory rate predicted mortality better than power.

Overall, this study suggested that driving pressure and respiratory rate were independently associated with survival. Mechanical power was independently associated with mortality, but this was attributed to the dynamic elastic component of this equation. Driving pressure had a greater effect on mortality than respiratory rate, which might suggest that adjusting the tidal volume to lower the driving pressure could have beneficial effects on overall mortality even if the respiratory rate is increased. The level of mechanical power needed for mechanical ventilation should reflect the disease severity. However, poorly adjusted ventilator settings with an unnecessarily high mechanical power may increase the potential for ventilator-induced lung injury. The stress and strain per breath applied to the lung is reflected in the driving pressure; the frequency of this stress/strain applied to the lung is reflected in the respiratory rate. These authors conclude that mechanical power is associated with mortality. However, driving pressure and the respiratory

rate are also predictors of mortality and are easier to measure at the bedside. Driving pressure potentially has more effects on mortality than respiratory rate and should be adjusted first.

MECHANICAL POWER DURING NONINVASIVE VENTILATION

Musso et al. measured mechanical power in patients with hypoxemic respiratory failure secondary to COVID-19.¹⁵ They analyzed the differences in mechanical power in the supine and prone position. This study included 216 patients who underwent noninvasive ventilation (NIV). They normalized the mechanical power to well aerated lung volumes determined by computed tomography scans. The prone position was associated with a 34% reduction in mechanical power. Patients with a high mechanical power during the first 24 hours of NIV had higher 28-day NIV failure and higher death rates. Mechanical power performed better than other ventilatory variables as a predictor of 28-day and NIV failure and death. It also predicted gas exchange, ultrasound changes in the lung, and inflammatory biomarker changes (CRP). In this study, mechanical power was calculated as: $MP = 0.098 \times RR \times V_t \times [PEEP + \Delta P_i]$ where ΔP_i = airway pressure above PEEP. The definition of high mechanical power in used this study was 9.1 J/min/ liter well aerated lung; low mechanical power was defined as less than 9.1 J/min/L well aerated lung. Throughout the first 7 days of patient management mechanical power decreased with every change from the supine to prone position. The median mechanical power on day 1 in patients who were ventilated in the prone position was 16.7 J/min. The mean mechanical power in patients in the supine position on day 1 was 16.9 J/min. This study demonstrates that mechanical power can be calculated in patients on noninvasive ventilation and has important associations with NIV failure and with death at 28 days of management.

MECHANICAL POWER STUDIES IN AN ANIMAL MODEL

Cressoni et al. used an animal model to try to determine the level of mechanical power which resulted in

ventilator-induced lung injury.¹⁶ These piglets were ventilated at a mechanical power level known to be lethal; the tidal volume was 38 mL per kilogram, the plateau pressure was 27 cm of water, and the respiratory rate was 15 breaths per minute. Other groups of piglets were ventilated with the same tidal volumes and plateau pressures but at lower respiratory rates. All animals were ventilated for 54 hours. Mechanical power levels greater than 12 J/min caused ventilator-induced lung injury. The animals at power levels greater than 12 J/min developed whole lung edema; animals ventilated below 12 J/min developed isolated densities in their lungs. These authors found a significant relationship between the mechanical power applied to the lung and increases in lung weight and lung elastance and decreases in PaO_2/FiO_2 ratios. There were significant changes in the configuration of the pressure volume loops in the animals receiving higher mechanical power levels at the end of the experiment.

Vassalli et al. used a porcine model to determine the effects of changes in mechanical power and the effect of the changes in tidal volume, respiratory rate, and PEEP in a model using adjustments to maintain a similar or iso-mechanical power in the animals with different respiratory parameters.⁵ In the iso-mechanical power studies, the tidal volume was twice the functional residual capacity, the respiratory rate was 40 breaths/min, and the PEEP level was 25 cm of water. The mechanical power levels were 15 and 30 J/min, and the treatment protocol was 48 hours. They found that the lung weight, wet to dry ratio, and histologic scores were similar regardless of the ventilatory strategies and the power levels. The high PEEP level group had larger changes in hemodynamics and required increased fluid administration. The authors suggest that understanding ventilator-induced lung injury requires an assessment of all relevant lung parameters, including tidal volume, respiratory rate, and PEEP level. There were no differences in lung histology in animals in the 2 power groups, but it is possible that 15 J/min is high enough to cause a significant lung injury.

CRITIQUE OF MECHANICAL POWER AS A GAUGE FOR LUNG INJURY

The work of breathing, whether expressed as energy per breath or expressed as energy per time,

i.e., mechanical power, is a cause of lung injury, an indicator of lung injury, or both. As lungs become stiffer, more energy is required to inflate them. At one point, it became fashionable to adjust ventilator knobs in such a way as to minimize the work of breathing. However, that strategy ignored some simple truths. If the goal of ventilator strategy were to minimize the work of breathing, then every patient should be on neuromuscular blockade 24/7. If the goal of ventilation were to minimize the work of breathing, the clinician should stop the ventilator completely – a ridiculous suggestion. The goal of ventilation is gas exchange, not some exercise in minimum heat transfer. Decreases in tidal volume require increases in rate to maintain equal levels of gas exchange. The clinician can reduce the work required for each breath at the expense of increasing mechanical power. Is this good or bad? The lack of knowledge of underlying mechanisms leading to lung injury limit any conclusions about the correct answer to this question. More basic observational evidence is needed to define the power thresholds which cause lung tissue temperatures to rise, which would seem necessary for any mechanism of injury based on mechanical power. Associations of increased mechanical power with poor outcomes must somehow correct for the difficulty that worse disease requires greater mechanical power to inflate the lungs and achieve adequate gas exchange. Consideration of mechanical power is useful with these limitations in mind. Delivering mechanical insults that cause harm more frequently over time will lead to greater injury than the same insult delivered less frequently. Once thresholds for harm have been exceeded, increasing either rate or energy per breath will be harmful, so practitioners should be careful not to decrease one parameter while increasing the other parameter without limit.

Marini and co-authors have written a succinct review of the mechanical factors potentially causing ventilator-induced lung injury.¹⁷ They focus on both static and dynamic contributors to injury. Important considerations include the reduced size of the injured lung, the heterogeneous distribution of the injury, the heterogeneous pathologic processes involved in the injury, the energy applied to the lung per cycle and per time unit, and the fact that overtime the distribution of absorbed energy in the lung likely opens up new

areas in the damaged lung which are then at risk for mechanical trauma. Their conclusions provide eight statements and questions which illustrate the complexity of any analysis trying to understand ventilator-induced lung injury and suggest that more studies are needed to understand the utility of mechanical power measurements in patients with acute respiratory failure.

CONCLUSIONS

Mechanical ventilation provides lifesaving support for patients with acute respiratory failure. However, the pressures and volumes required to maintain gas exchange can cause ventilator-induced lung injury. The current approach to mechanical ventilation involves attention to both tidal volume and airway pressures, in particular plateau pressures and driving pressures. The ventilator provides energy to overcome airway resistance and to inflate alveolar structures and the chest wall. This energy delivered to the respiratory system per unit time equals mechanical power, and calculation of mechanical provides a composite number which integrates pressures, volumes, and respiratory rates. Increased levels of mechanical power have been associated with tissue injury in animal models. In patients, mechanical power can predict outcomes, such as ICU mortality, when used in multivariable analyses. Increases in mechanical power during the first day of ventilation have been associated with worse outcomes. Mechanical power calculations can be used in patients on noninvasive ventilation, and measurements of mechanical power have been used to compare ventilator modes. This calculation requires measurement of the area in a hysteresis loop. Alternatively, simplified formulas have been developed to provide this calculation. However, this information is not available on most ventilators, and clinicians will need to make this calculation. In summary, calculation of mechanical power provides an estimate of the energy requirements for mechanical ventilation based on a composite of factors, including airway resistance, lung elastance, respiratory rate, and tidal volume. Manufacturers should add mechanical power calculations to the software of new ventilators.

Article citation: Nugent K, Berdine G. Mechanical power during mechanical ventilation. *The Southwest Respiratory and Critical Care Chronicles* 2024; 12(50):16–23

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Submitted: 12/1/2023

Accepted: 12/30/2023

Conflicts of interest: none

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