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Effects of passive mobilization on acute hemodynamic responses in mechanically ventilated patients

Efeitos da mobilização passiva nas respostas hemodinâmicas agudas em pacientes sob ventilação mecânica

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Conflicts of interest: None.

Submitted on January 11, 2012
Accepted on March 13, 2012

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ABSTRACT

Objective: To assess the effects of passive mobilization on acute hemodynamic responses in mechanically ventilated patients.

Methods: This cross-sectional, quantitative, observational study enrolled patients who were admitted to the intensive care unit, sedated and mechanically ventilated. The infusion of sedative and analgesic drugs aimed to maintain a Ramsay scale sedation level of 4 to 6. Passive mobilization consisted of hip and knee flexion-extension movements for five minutes. After 10 minutes of rest, an additional five minutes of flexion-extension passive movements was performed for the shoulders. Hemodynamic assessments (heart rate and systolic, diastolic and mean blood pressure) were performed one minute before the mobilization protocol and one minute after each phase. The double product and myocardial oxygen consumption were calculated using appropriate formulas.

Results: A total of 13 patients (69.2% male, with a mean age of 69.1 ± 15.8 years) were admitted from June to December, 2011. Passive mobilization led to statistically significant increases in heart rate, double product and myocardial oxygen consumption. However, mean blood pressure was not significantly altered.

Conclusions: Our results suggest that passive mobilization of mechanically ventilated and sedated patients is safe and provides beneficial effects on acute hemodynamic parameters, particularly heart rate, although mean blood pressure is not significantly altered.

Keywords: Hemodynamics; Intensive care unit; Artificial respiration

INTRODUCTION

Critically ill patients who require sedation and mechanical ventilation are restricted to bed for long periods. This confinement is a risk factor for dysfunction in various organ systems, often becoming more severe than the underlying condition itself.[1] In the intensive care unit (ICU), patients' limbs are routinely mobilized by the unit's physiotherapists, aiming to preserve the range of motion, improve or preserve soft tissue length, maintain muscle condition and reduce the risk of thromboembolism.[2] Mechanical stress from limb mobilization may alter hemodynamic responses such as heart rate (HR), blood pressure (BP) and myocardial oxygen consumption (mVO_2).[3] Cardiac output depends on the interaction between two main functions: 1) heart function, which is determined by HR, contractility and pre- and afterload, and 2) return function, which is determined by the volume of

venous return, venous drainage resistance and right atrial pressure.[4,5] It has also been demonstrated that the distension and shortening of muscle fibers may activate mechanoreceptors, leading to cardiovascular adjustments via parasympathetic inhibition and sympathetic activation.[4-8]

The potential benefits of exercise for inactive ICU patients have been reported previously.[9,10] Passive lower limb mobilization in critically ill patients prevents muscle fibers atrophy,[11] increases oxygen consumption (VO_2) and reduces venous blood oxygen saturation (SVO_2), most likely due to an increased oxygen extraction rate (O_2ER) and cardiac index.[12] However, the physiological mechanisms behind hemodynamic responses to passive mobilization in mechanically ventilated patients are not fully known.

This study aimed to assess the effects of passive mobilization on acute hemodynamic responses in mechanically ventilated patients.

METHODS

The study protocol was approved by the ethics committee of Irmandade Santa Casa de Londrina (ISCAL) (project #380/11), and written informed consent forms were signed by designated family members for each patient. The study design was a cross-sectional, quantitative, observational format following the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) criteria.[13]

The patients included were aged over 18 years who were maintained on pressure-controlled-mode mechanical ventilation (MV) (Newport Wave E200, Newmed, CA, USA) for more than 48 hours with the following parameters: a positive end-expiratory pressure (PEEP) between 5 and 8 cmH_2O, a tidal volume between 6 and 8 mL/kg and an inspired oxygen fraction (FiO_2) of 21-50%. The infusion of sedative and analgesic drugs aimed at a sedation level between 4 (brisk response to stimulus) and 6 (no response to painful stimulus) on the Ramsay scale.[14] All patients were administered vasoactive drugs, and mean blood pressure (MBP) was maintained above 60 mmHg. The following subjects were excluded from the study: hemodynamically unstable patients (MBP < 60 mmHg), patients showing agitation during the maneuvers, and those with resistance to movement, dropping oxygen saturation (< 90%), an intra-aortic balloon, complex arrhythmias, neurologic and/or motor deficits or musculoskeletal limitations preventing the protocol-determined movements.

Passive mobilization (PM) protocol

The patients were maintained in a supine position, with the bed head raised at 30%. PM consisted of hip and knee flexion-extension movements for five minutes (90º flexion). After 10 minutes of rest, an additional five minutes of flexion-extension passive movements was performed for the shoulders (90º). PM was simultaneously performed by two physiotherapists alternating flexion and extension of the right and left limbs at a frequency of 30 movements/minute. To maintain a steady frequency, a metronome was used (KORG MA-30, Japan). Hemodynamic assessments (measurements of HR and systolic (SBP), diastolic (DBP) and mean (MBP) blood pressures) were performed one minute before the mobilization protocol and one minute after the end of each phase.

Clinical signs were continuously monitored using a multi-parameter monitor (Dixtal DX 2010 - Dixtal, Manaus, Brazil) to provide electrocardiogram (ECG), HR, SBP, DBP and MBP measurements. The variable double product (DP) was calculated as the product of SBP and HR (DP = SBP × HR), and myocardial oxygen consumption (mVO_2) was calculated using the formula $mVO_2 = (DP \times 0.0014) - 6.3$.[15] Demographics and Acute Physiology Chronic Health Evaluation II (APACHE II) scores[16] were recorded for all patients. Drug infusions and mechanical ventilator settings were constant during the protocol.

Statistical analysis

The normality of the data was confirmed using the Kolmogorov-Smirnov test. Categorical variables are presented as absolute values and proportions, and continuous variables are reported as the means with standard deviations (±SD). The assessments before and after PM were compared using the pairwise samples parametric Student's t-test. A one-way ANOVA was used to compare pre- and post-PM assessments for the upper and lower limb mobilizations. Differences between categorical variables were compared using the Chi-squared test. The Statistical Package for Social Sciences (SPSS 17.0) software was used for the analyses, and a value of $p < 0.05$ was considered to be significant.

RESULTS

A total of 13 patients (69.2% male, with a mean age 69.1 ± 15.8 years) were enrolled from June to December, 2011. These patients had been admitted to the ICU of Hospital Santa Casa de Londrina - Paraná,

Brazil. Table 1 lists the patients' baseline characteristics. The Ramsay scale was adopted to assess the level of consciousness. One patient was categorized as Ramsay 4, three as Ramsay 5 and nine as Ramsay 6.

Table 2 presents the patients' hemodynamic parameters according to the protocol applied to the upper and lower limbs. Statistically significant differences were found for HR (lower limbs, p = 0.015; upper limbs, p = 0.034), DP (lower limbs, p = 0.012; upper limbs, p = 0.025) and mVO_2 (lower limbs, p = 0.011; upper limbs, p = 0.024). Immediately after PM, MBP showed a mild but not statistically significant increase.

When the responses to the lower and upper limb mobilizations were compared, no difference was found for the pre- and post-PM assessed variables (Table 3).

No adverse events, such as desaturation or agitation, were recorded during the PM maneuvers.

Table 1 - Baseline characteristics of the study patients

Characteristics	Result	p value
Age (years)	69.1 ± 15.8 (41 - 88)	0.4979
Gender		
Male	9 (69.2)	0.9992
Female	4 (30.8)	
APACHE II score	25 ± 4.1 (18 - 33)	1.000
Risk of death	52.2 ± 13.4 (29.1 - 78.6)	0.9098
Glasgow score	5.5 – 1.0 (4 - 8)	0.8971
Ramsay score	5.0 – 0.58 (4 - 6)	1.0000
Mechanical ventilation time	10.5 ± 8.6 (2 - 30)	0.9216
Diagnosis		
Septic shock	03 (23.1)	0.0861
Pneumonia	06 (46.1)	
Chronic obstructive pulmonary disease	03 (23.1)	
Postoperative (abdominal)	01 (7.7)	

APACHE II - Acute Physiology and Chronic Health Evaluation. Results are expressed as the mean ± standard deviation or number (%) standard deviation.

Table 2 - Hemodynamic parameters following passive lower and upper limb mobilizations

Parameters			Mean ± SD	p value
HR (bpm)	Lower limbs	Rest	84 ± 21	0.015*
		Following PM	95 ± 28	
	Upper limbs	Rest	85 ± 15	0.034*
		Following PM	98 ± 30	
MBP (mmHg)	Lower limbs	Rest	99 ± 16	0.816
		Following PM	100 ± 14	
	Upper limbs	Rest	96 ± 10	0.320
		Following PM	99 ± 12	
Double product (mmHg × bpm)	Lower limbs	Rest	10477 ± 2949	0.012*
		Following PM	11670 ± 3197	
	Upper limbs	Rest	10114 ± 2240	0.025*
		Following PM	11502 ± 2686	
mVO_2 (mL O_2/100 g VE/min)	Lower limbs	Rest	8.4 ± 4.1	0.011*
		Following PM	10.0 ± 4.5	
	Upper limbs	Rest	7.9 ± 3.1	0.024*
		Following PM	9.8 ± 3.8	

HR - heart rate; MBP - mean blood pressure; mVO_2 - myocardial oxygen consumption; PM - passive mobilization; SD - standard deviation.

Table 3 - Comparison of hemodynamic parameters following passive lower and upper limb mobilization

Parameters		Lower limbs Mean ± SD	Upper limbs Mean ± SD	p value
HR (bpm)	Rest	84 ± 21	85 ± 15	0.3445
	Following PM	95 ± 28	98 ± 30	
MBP (mmHg)	Rest	99 ± 16	96 ± 10	0.9025
	Following PM	100 ± 14	99 ± 12	
Double product (mmHg × bpm)	Rest	10477 ± 2949	10114 ± 2240	0.5840
	Following PM	11670 ± 3197	11502 ± 2686	
mVO_2 (mL O_2/100 g VE/min)	Rest	8.4 ± 4.1	7.9 ± 3.1	0.5895
	Following PM	10.0 ± 4.5	9.8 ± 3.8	

HR - heart rate; MBP - mean blood pressure; mVO_2 - myocardial oxygen consumption; PM - passive mobilization; SD - standard deviation.

DISCUSSION

In this study, passive lower and upper limb mobilizations promoted significant increases in HR, DP and mVO_2, but not in MBP. The physiological mechanisms of hemodynamic responses during passive mobilization are not fully known. Although HR increases with cardiac output (CO) during exercise, CO increase should not be interpreted as resulting only from increased HR but rather from the interaction of two main factors: 1) heart function, which is determined by HR, contractility and pre- and afterload, and 2) return function, which is determined by the volume of venous blood return, resistance to venous return and right atrial pressure.[4,5] Although passive mobilization produces no muscle contraction, Doppler tests conducted for the assessment of active and passive kinesiotherapy have shown an increased venous blood flow from the sural pump during passive kinesiotherapy that was well above the baseline.[17]

Muscle tension caused by passive movements can also lead to increased HR due to the activation of tendinous mechanoreceptors.[8,18] Prior studies have shown that simultaneous muscle stretching and shortening, as in typical passive mobilization, causes the activation of mechanoreceptors and type III fibers, which can induce vagal activation and stimulate baroreceptors, thereby contributing to the overall cardiovascular response.[19,20] The mobilization of large muscle groups (such as the hip, knee or shoulder) is another factor likely contributing to the increased HR observed in our study. A similar observation was recently reported by Farinatti et al.,[21] who assessed cardiovascular responses to static flexibility with passive movements in healthy subjects. Their results showed that HR was consistently higher with hip flexion (involving the ischiotibial muscles, a large group) than with ankle dorsal flexion (involving the gastrocnemius, a small group). Additionally supporting our findings, these authors[21] also reported that DP increased with the passive mobilization of both small and large muscle groups, concluding that passive stretching of muscle groups may affect cardiovascular responses. Gladwell and Coote[22] also assessed HR and SBP during one-minute passive and sustained sural triceps stretching, reporting significantly increased HR but not SBP, which are findings similar to our own.

Recently Magder (2012)[23] reviewed the physiological mechanisms regulating HR and the relevance of mobilizing critically ill patients, concluding that HR may be interpreted within the overall hemodynamic condition of the patient and that HR and CO regulation during exertion is reflected in a range of physiological responses, but that HR is the major cardiovascular system component responsible for adjusting CO.[24] Additionally, when the HR response is limited by underlying disease or pharmacological interventions, changes in the systolic volume may act to compensate for such limitations. However, this ability may in turn be limited by the passive filling of the left ventricle.

In our study, MBP was not observed to significantly increase in response to passive mobilization. This observation may be explained by noting that increased peripheral vascular resistance is also influenced by sustained muscle contraction, consequently increasing BP.[25] However, our protocol used a low cycle frequency, maintaining 30 movement cycles/minute for five minutes without active contraction.

Based on our findings, the increased DP may have been caused by increased HR, as no significant BP

increase was observed,[26] shown by the MBP results. The DP is usually used to estimate cardiac workload during aerobic and strength exercises.[24] However, several authors have reported that during static flexibility exercises, DP may reach levels that are similar to the levels found during high-intensity, low-repetition resistance exercise.[27,28]

Comparative studies between upper and lower limb exertion tests have shown that at the same level of workload, the CO may be similar that for lower loads.[29-31] This difference may explain our results when comparing the responses for the mobilizations of the upper and lower limbs, where no differences was found for any of the variables, as the loads were the same for both the upper and lower limbs (30 movements/minute).

Cardiac metabolism is influenced both by chrono- and inotropisms, and both have an influence on myocardial overload and oxygen demand.[32] Myocardial oxygen demand may be measured as myocardial oxygen consumption (mVO_2), which is determined by the interactions among myocardial tension, heart muscle contractility and HR. All of these factors change during physical exertion, increasing the myocardial requirements for nutrients and oxygen and causing increased coronary flow.[33] A linear correlation between mVO_2 and coronary blood flow has been demonstrated, thus providing information on cardiac overload, i.e., the work performed by the heart to meet the body's demand. The product of HR and SBP, the DP, is highly correlated with mVO_2 (r^2=0.88).[32] Hellerstein & Wenger[15] described a mathematical function to convert DP into mVO_2 (mVO_2 = (DP × 0.00014) - 6.3), allowing the estimation of cardiac effort. This calculation was applied in our study; the results suggest that due to increased HR, which in turn influenced the DP, heart muscle overload occurred, with a consequently significant increase in mVO_2. The hemodynamic and metabolic effects of cyclic passive lower limb mobilization in mechanically ventilated patients was previously assessed by Savi et al.,[12] who reported that all five assessed patients displayed increased oxygen consumption (VO_2) concomitant with a drop in venous blood oxygen saturation (SVO_2), most likely due to an increased oxygen extraction rate (O_2ER) and cardiac index (CI). The authors additionally concluded that cyclic passive lower limb mobilization may influence the hemodynamic and metabolic conditions of sedated patients who are dependent on mechanical ventilation.[12] Additionally, aiming to assess exertion induced hemodynamic changes, Bittencourt et al. (2008) evaluated HR, DP, BP and mVO_2 in 11 male subjects. In agreement with our findings, their results demonstrated that physical activity significantly changes HR, DP and mVO_2.[34]

Our study had several limitations: 1) the small sample size may have influenced the lack of statistical significance for MBP and 2) the study included patients with Ramsay levels of 4 to 6. Ramsay 4 patients may have had a certain degree of active/assisted muscle activity; however, no comparative intergroup analysis was conducted due to the small sample size.

CONCLUSION

Our results suggest that passive mobilization of the lower and upper limbs produces acute hemodynamic effects in mechanically ventilated and sedated patients, particularly increasing HR; however, MBP was not significantly affected. It should be noted that considering the assessed variables, no changes were observed that may be considered dangerous according to the available literature. Our procedure resulted in beneficial heart muscle overload in critically ill mechanically ventilated patients.

This work was an initial study, and further studies will be necessary to explore this subject more deeply.

RESUMO

Objetivo: Avaliar as respostas hemodinâmicas agudas da mobilização passiva de pacientes sob ventilação mecânica.

Métodos: Estudo de investigação clínica do tipo transversal, quantitativa e observacional. Incluindo pacientes internados na unidade de terapia intensiva, sedados e sob ventilação mecânica. A infusão de drogas sedativas e analgésicas visava o grau de sedação de 4 a 6 de acordo com a escala de Ramsay. A mobilizaçao passiva consistiu em movimentos de flexo-extensão de quadril e joelho durante cinco minutos. Após 10 minutos de repouso, foram realizados mais cinco minutos de mobilização passiva com flexo-extensão de ombro. As mensurações hemodinâmicas (freqüência cardíaca, pressão arterial sistólica e diastólica e pressão arterial média) foram realizadas 1 minuto antes da realização do protocolo e no primeiro minuto após o término. O duplo produto e a medida do consumo ou captação de oxigênio pelo miocárdio foram obtidas por meio de fórmulas.

Resultados: Entre junho a dezembro de 2011 foram incluídos 13 pacientes (69,2% homens) com idade média de 69,1 ± 15,8 anos. A mobilização passiva de membros inferiores e de membros superiores provocou aumentos da frequência cardíaca,

do duplo produto e do consumo ou captação de oxigênio pelo miocárdio com diferença estatisticamente significante. Entretanto a pressão arterial média não apresentou diferença significativa.

Conclusões: Os resultados sugerem que a mobilização passiva de membros inferiores e superiores em pacientes sedados sob ventilação mecânica influencia de forma segura nos efeitos hemodinâmicos agudos, particularmente na frequência cardíaca, porém sem alterar significativamente a pressão arterial média.

Descritores: Hemodinâmica; Unidades de terapia intensiva; Respiração artificial

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