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Hemodynamic and metabolic effects of passive leg movement in mechanically ventilated patients

Efeitos hemodinâmicos e metabólicos da movimentação passiva dos membros inferiores em pacientes sob ventilação mecânica

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Conflict of Interest
The authors declare no conflict of interest

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ABSTRACT

Objective: Limb movements, passively performed by a physiotherapist, have been shown to result in significant increases in critically ill patients' metabolic and hemodynamic variables. This study objective was to determine whether passive cycling leg movement increases hemodynamic and metabolic variables in sedated mechanical ventilation dependent patients.

Methods: Five sedated mechanical ventilation dependent patients in a 18-bed intensive care unit of a university hospital were evaluated. Passive cycling leg movements were performed for 10min at a 30 movements/min rate. Complete hemodynamical data were recorded and arterial and mixed venous blood sample were collected 5 minutes before and after 5 minutes after the maneuver completion.

Results: All patients had increased oxygen consumption (VO_2). The VO_2 increase occurred with a concomitant drop in mixed venous blood saturation (SvO_2), likely from both oxygen extraction ratio (O_2ER) and cardiac index (CI) increase.

Conclusion: passive cycling leg movements may influence hemodynamical and metabolic status in sedated mechanical ventilation-dependent patients.

Keywords: Range of motion, articular; Hemodynamics; Respiration, artificial

INTRODUCTION

The cardiovascular system constantly copes with the body's oxygen requirements to provide appropriate oxygen delivery (DO_2) to the tissues in relation to their consumption (VO_2).(1) In critically ill patients, the VO_2 can be influenced not only by the acute illness severity and inflammatory response degree, but also by a number of other factors, such as the stress and anxiety levels. In addition, intensive care unit (ICU) standard procedures such as nursing care and respiratory physiotherapy also can increase VO_2.(2,3)

The hemodynamical and metabolic effects of respiratory physiotherapy in mechanically ventilated (MV) patients have been extensively investigated.(4) Authors evaluating the effects of chest physiotherapy on hemodynamics and VO_2 (measured by indirect calorimetry) in ten MV-dependent patients showed that no significant increase occurred in VO_2 and hemodynamic status.(5) However, other authors have documented significant detrimental hemodynamic and metabolic responses to multimodality respiratory physiotherapy.[6-8]

Limb exercises are performed by critical care patients with the goal of maintaining joint range of motion, improving soft-tissue length, muscle strength and function, and decreasing the thromboembolism risk.[9] Also Griffiths et al. described the effect of continuous passive motion of one leg in critically ill patients with respiratory failure during neuromuscular blockade, with the contralateral leg serving as a control.[9] This intervention prevented muscle fiber atrophy in those with highly severe illness. Other studies described the harms of immobility in MV-dependent patients and many have speculated on the potential benefits of physical activity in inactive ICU patients.[10, 11]

This technique hemodynamic effects were not appropriately evaluated, and only one small study, using passive limb movements, has been shown to result in significant increases in metabolic and hemodynamic variables (increase 15% VO_2).[12]

This pilot study evaluated if passive cycling leg movement (PCLM) can increase VO_2 in sedated MV-dependent patients and if the degree of cardiac dysfunction changes this hemodynamic response.

METHODS

Patients

We included consecutive patients under MV for more than 48h continuously monitored with pulmonary-artery catheter (Swan-Ganz catheter, Abbott Laboratories, North Chicago, IL, USA). Infusions of sedative and analgesic drugs were compatible with maximal sedation level as evaluated with the Ramsay scale.[13] All patients were under pressure-controlled mode ventilation, with positive end-expiratory pressure level between 6-8cmH_2O, tidal volume 6-8mL/kg and inspired oxygen fraction between 35-50% (Servo 900, Siemes-Elema, Sweden). The patients received continuous noradrenaline infusions to maintain mean arterial pressure (MAP) >60mmHg.

Exclusion criteria were age <16 years, hemodynamic instability (MAP <60mmHg or change in the vasopressor dose in the last 12h, agitation during the maneuver and drop in oxygen saturation.

PCLM protocol

All patients laid in semi-recumbent 30º position during the physical therapy assistance. PCLM consisted of passive flexion-extension movements of the hip and knee during 10 minutes at a 30movements/min rate. Two physiotherapists were required for the maneuver. When a physiotherapist promoted flexion movement in the patient's leg, the other promoted extension movement in the other leg. The movement achieved 90° of flexion of both hip and knee. The rate was kept with a metronome (KORG MA-30, Japan). Hemodynamic measurements (blood pressure, pulmonary artery pressure, pulmonary artery occluded pressure, heart rate, cardiac index [CI]) and arterial and mixed venous blood analysis were obtained 5 minutes before, and 5 minutes after the PCLM maneuver end. All pressures were measured by end-expiration. The CI was determined by thermodilution technique (HELLIGE, SMU-612, Germany) using 10mL aliquots of a cold saline solution and a closed system. Five measurements, within a 10% range, were averaged to measure the actual cardiac output. Immediately after this, arterial and mixed venous blood gases were determined in an autoanalyzer (ABL 520, Radiometer, Copenhagen, Denmark). Hemoglobin concentration and oxygen saturation were measured and DO_2 and VO_2 were calculated as follows:

DO_2 (mL/min/m^2) = CI x CaO_2 x 10
VO_2 (mL/min/m^2) = CI x (CaO_2 – CvO_2) x 10
were CaO_2 and CvO_2 representing the arterial and mixed oxygen contents respectively, calculated by:
CaO_2 = (1.39 x Hb x SaO_2) + (0.0031 x PaO_2)
and
CvO_2 = (1.39 x Hb x SvO_2) + (0.0031 x PvO_2)

where Hb is the hemoglobin concentration and SaO_2 and SvO_2 the arterial and mixed oxygen saturations, respectively.[14,15] During the protocol no infusion drugs or ventilator settings were changed.

Statistical analysis

All statistical analysis was performed with Statistical Package for Social Science (SPSS 17.0, Chicago, IL). Continuous variables were expressed as medians (interquartile range). Differences between before and after PCLM times were evaluated using Wilcoxon, Mann-Whitney U tests comparing not normally distributed variables. For all statistical tests a type I error was adopted.

RESULTS

The patients' baseline characteristics, reason for ICU admission, and drugs use are presented in Table 1. All

Table 1 - Patient's characteristics

Patient number	Age (years)	Gender	APACHE II score	MV days	Condition	Noradrenaline (μg/kg/min)	Sedative (mg/kg/h)	Analgesic	Ramsay
1	55	M	14	3	Post-operative	0.5	Midazolam (0.08)	Fentanil	6
2	56	M	20	5	Post-operative	0.35	Midazolam (0,08)	Morphine	6
3	60	M	19	4	ARDS	0.4	Propofol	Fentanil	6
4	53	F	16	6	Septic shock	0.25	Midazolam (0.1)	Fentanil	6
5	62	F	21	3	ARDS	0.45	Midazolam (0.06)	Fentanil	6

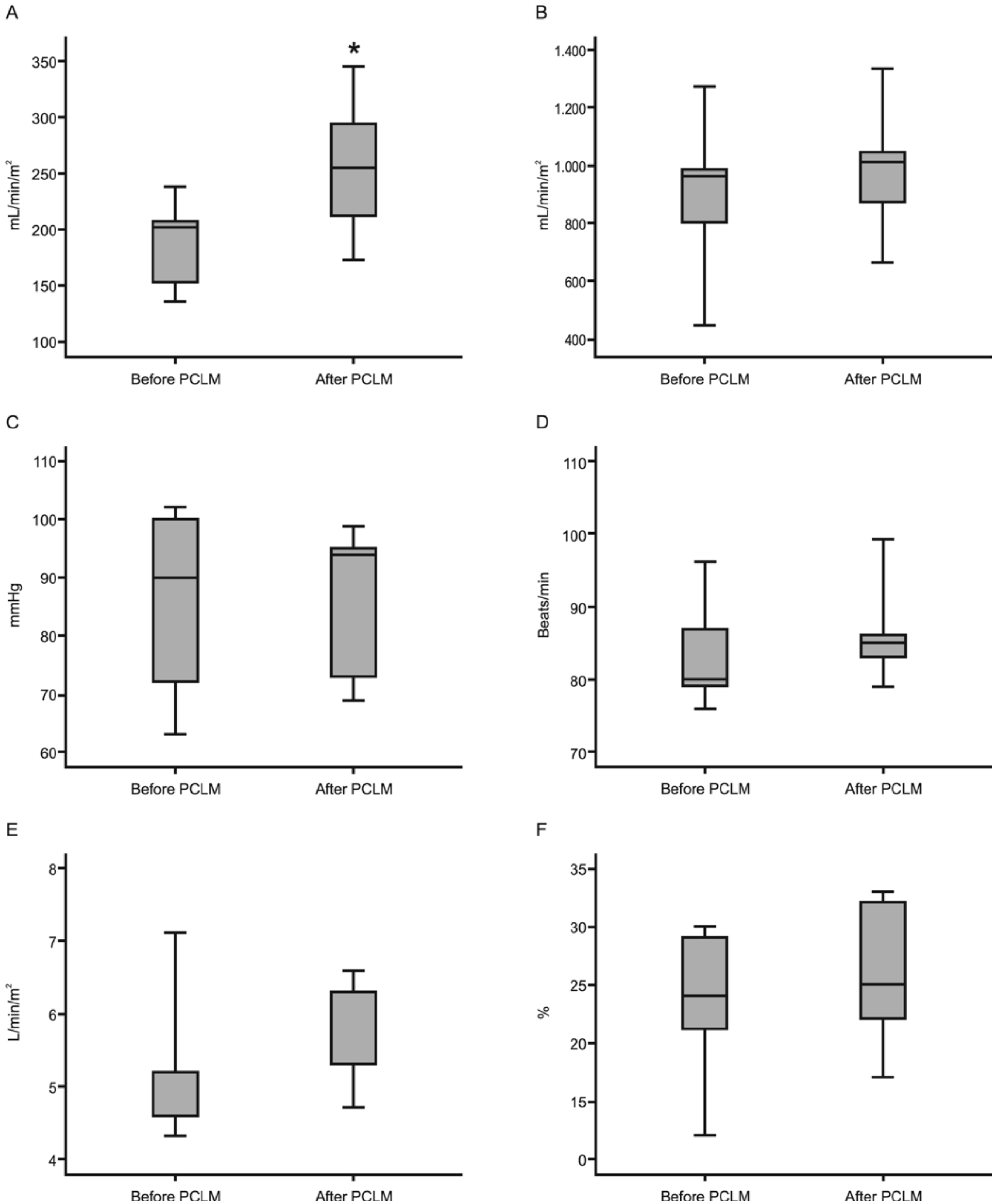
APACHE - Acute Physiologic and Chronic Health Evaluation; MV - mechanical ventilation; ARDS – acute respiratory distress syndrome.

Table 2 - Hemodynamic and oxygen derived parameters before and after PCLM

		Patients						
		1	2	3	4	5	Median (interquartile range)	p value*
PAOP (mmHg)	Rest	15	16	8	13	14	14 (11 – 16)	0.27
	After PCLM	12	13	8	13	16	13 (10 – 15)	
MAP (mmHg)	Rest	100	102	71	90	63	90 (68 – 101)	0.78
	After PCLM	99	94	73	95	69	94 (71 – 97)	
HR (beats/min)	Rest	76	80	87	96	79	80 (78 – 92)	0.078
	After PCLM	79	83	86	100	85	85 (81 – 93)	
DO_2 (mL/min/m²)	Rest	987	960	1276	450	804	960 (627 – 1131)	0.34
	After PCLM	1331	1046	1011	662	875	1011 (769 – 1189)	
VO_2 (mL/min/m²)	Rest	238	207	153	135	201	201 (144 – 223)	0.04
	After PCLM	294	345	172	212	254	254 (192 – 320)	
SvO_2 (%)	Rest	74	69	88	65	72	72 (67 – 81)	0.04
	After PCLM	66	63	70	62	68	66 (63 – 69)	
O_2ER (%)	Rest	24	21	12	30	25	24 (16 – 30)	0.41
	After PCLM	22	33	17	32	29	25 (20 – 33)	
CI (L/min/m²)	Rest	5.2	5.2	7.2	4.3	4.6	5,2 (4,5 – 6,2)	0.68
	After PCLM	6.3	5.3	6.6	4.7	5.3	5,3 (5 – 6,5)	

PAOP - pulmonary artery occlusion pressure; MAP - mean arterial pressure; HR - heart rate; DO_2 - oxygen delivery; VO_2 - oxygen consumption; SvO2 - mixed oxygen saturation; O_2ER - oxygen extraction ratio; CI – cardiac index. *compared with rest.

patients showed a VO_2 increase, from 201 (144 - 223) to 254 (192 - 320) mL/min/m² ($p = 0.043$). Immediately after PCLM, heart rate and mean arterial pressure increases were seen, however not statistically significant. Mixed venous saturation decreased from 72 (67 – 81) to 66 (63 – 69) % ($p = 0.043$), although no significant change in oxygen extraction ratio and cardiac index was found (Figure 1 and Table 2). No adverse event, such as hemodynamic instability, desaturation or agitation occurred during the PCLM.



PCLM - passive cycling leg movement; VO_2 - oxygen consumption; DO_2 - oxygen delivery; MAP - mean arterial pressure; HR - heart rate; CI - cardiac index; O_2ER - oxygen extraction ratio. * $p < 0.05$ compared with rest.

Figure 1- (A) Boxplot of VO_2; (B) DO_2; (C) MAP; (D) HR; (E) CI and (F) O_2ER.

DISCUSSION

This study showed that passive cycling leg movement is able to increase the oxygen consumption in sedated MV-dependent patients.

The hemodynamic and metabolic effects of respiratory physiotherapy in MV-patients have been extensively investigated. Horiuchi et al.[16] investigated the cause for the increased metabolic and hemodynamic responses during chest physiotherapy during MV-support after major vascular or abdominal surgery. All patients underwent two standardized physiotherapy treatments (first treatment preceded by midazolam and the second treatment preceded by vecuronium). These authors found that the administration of vecuronium suppressed the metabolic demands increase seen during the physiotherapy treatment preceded by midazolam, while hemodynamic responses were not changed by vecuronium administration. Thus, they hypothesized that the increased of metabolic demand during multimodality physiotherapy was similar to exercise-like response resulting from increased muscular activity, while the increased hemodynamic responses are most likely due to a stress like response associated with increased sympathetic output.

Differently from chest physiotherapy, passive leg movements metabolic and hemodynamic effects in critical ill patients were so far poorly assessed. Norrenberg et al.[15] studied the effects of passive leg movement in 16 critical ill MV or spontaneously breathing patients without sedation, and observed that simple maneuvers like passive leg movement were able to increase the VO_2 (123 ± 23 to 143 ± 34mL/min/m^2, $p < 0.05$). This increase was variable according to the patient's cardiovascular status. In patients with cardiac dysfunction, the VO_2 increase was met by O_2ER increase without a significant CI increase. In patients without cardiac dysfunction, the VO_2 increase was met by an CI increase without a significant O_2ER increase. No electromyography (EMG) was performed to confirm if the no sedated patients had muscular contractions, which could markedly increase VO_2. In our study all patients received continuous sedatives infusions and were deeply sedated, so most likely had no voluntary muscle contractions during PCLM. According to the Fick principle, VO_2 depends on DO_2 and O_2ER. DO_2 is defined by the product of cardiac output and the arterial oxygen content. O_2ER represents the tissues O_2 pickup in relation of oxygen offer. In our sample we identified no DO_2 and O_2ER changes, but did find a SvO_2 drop. SvO_2 is a clinical marker of systemic oxygen use, and its measurement is part of the of critically ill patient monitoring routine.[17] The O_2 demand increase during PCLM could directly contribute to a decrease in SvO_2, as no change in O_2 offer or transport occurs during the PCLM. Nóbrega et al. studied the effects of passive cycling movements in healthy subjects on a tandem bicycle with second riders performing the active movement.[18] They observed increased VO_2 but the EMG did not show any actual muscle contraction. The hemodynamic variation likely occurred due to an increased venous return from lower limbs or by a muscle mechanoreceptor-evoked myocardial contractility increase. Morikawa et al. reported that passive flexion-extension of knee significantly increased the VO_2 in healthy subjects but not in traumatic spinal section patients, implicating the activation of the mechanoreceptors in a reflex muscle tone increase.[19] Similarly to a subgroup of patients studied by Weissman during chest physiotherapy[20] our patients had VO_2 increase both by CI and O_2ER increase (Table 2).

Strengths and limitations: our study had a small number of patients. Therefore we could not statistically estimate the PCLM impact. Our study results suggest that in sedated mechanically ventilated patients with no history of cardiovascular disease, PCLM can increase $VO_{2,}$ and these results confirm the reported by other study groups. We suggest that these patients' nutritional offer maybe have been adjusted to equilibrate the caloric waste. Other studies are required to approach PCLM effects on this.

RESUMO

Objetivo: A movimentação passiva dos membros executada por um fisioterapeuta demonstrou resultar em aumentos significantes nas variáveis metabólicas e hemodinâmicas em pacientes criticamente enfermos. O objetivo deste estudo foi determinar se o movimento cíclico passivo das pernas aumenta as variáveis hemodinâmicas e metabólicas em pacientes sedados dependentes de ventilação mecânica.

Métodos: Foram estudados cinco pacientes sedados, dependentes de ventilação mecânica, internados em uma unidade de terapia intensiva com 18 leitos de um hospital universitário. A movimentação cíclica passiva das pernas foi realizada por 10 minutos em uma freqüência de 30 movimentos por minuto. Foram registrados dados hemodinâmicos completos, e colhidas amostras de sangue arterial e venoso 5 minutos antes e 5 minutos após o término das manobras.

Resultados: Todos os pacientes apresentaram aumento do consumo de oxigênio (VO_2). O aumento do VO_2 ocorreu concomitantemente a uma queda na saturação de oxigênio no sangue venoso (SvO_2), provavelmente ocorrendo por um aumento na taxa de extração de oxigênio (O_2ER) e índice cardíaco (IC).

Conclusões: Os movimentos cíclicos passivos das pernas podem influenciar a condição hemodinâmica e metabólica de pacientes sedados dependentes de ventilação mecânica.

Descritores: Amplitude de movimento articular; Hemodinâmica; Respiração artificial

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