The effects of orthostatism in adult intensive care unit patients

Efeito imediato do ortostatismo em pacientes internados na unidade de terapia intensiva de adultos

INTRODUCTION

The musculoskeletal system is designed for movement. Just seven days at rest can result in a 30% drop in muscle strength, with an additional 20% loss of remaining strength each additional week.\(^1\),\(^2\) Long stays in the intensive care unit (ICU) and mechanical ventilation (MV) are associated with functional decline, morbidity, mortality, high care costs and long-term hospitalization.\(^2\),\(^3\) Sepsis, hydroelectrolytic disorders and immobility can cause polyneuromyopathy in critically ill patients. Excessive use of sedating drugs, neuromuscular blockers, corticosteroids and MV can aggravate ICU-acquired weakness.\(^1\),\(^4\)
Prolonged bed rest results in changes to muscle fibers. Myosin isoforms change slow-contracting fibers to fast-contracting fibers, and protein synthesis is reduced; disuse atrophies skeletal muscles. Under inflammatory disease conditions, muscle proteolysis is increased, and the muscles may suffer “functional denervation,” related to reduced nervous impulses reaching the muscle membranes. This type of weakness affects both the limbs and respiratory muscles, and this process delays extubation and prolongs MV. Curiously, bed-assisted exercises are not sufficient for preventing the adverse effects of rest. This fact is related to changes in the intravascular fluid of the extremities, due to the lack of gravitational stress. However, assuming a vertical position helps in maintaining appropriate fluid distribution and in moving the abdominal viscera down. For this process, orthostatism is recommended to be included in early mobilization programs, with the goal of minimizing the adverse effects of immobility.

Orthostatism, as a therapeutic resource, may be provided either passive or actively for motor stimulation, cardiovascular function improvement and alertness. The use of an orthostatic board is indicated for the readaptation of patients to the vertical position, when they are unable to stand alone safely or even with considerable assistance.

Orthostatism has been encouraged in critically ill patients based on its supposed benefits, which include improved autonomic cardiovascular control, oxygenation, ventilation, alertness, vestibular stimulation, antigravity postural response and prevention of articular contractures and pressure ulcers.

The increased ventilation provided by this therapy is believed to prevent pulmonary complications. Chang et al. reported in their article that pulmonary volume optimization might be associated with pulmonary secretion redistribution and the easing of coughs and of secretion suctioning. This technique could also be beneficial for patients not able to participate actively in pulmonary expansion exercises.

There is no consensus on this technique’s indications and contraindications. However, the III Brazilian Consensus on Mechanical Ventilation recommends that an orthostatic board be used only in chronic and clinically stable patients. Recent studies have shown the need to monitor continuously systemic blood pressure, heart rate, oxygen saturation, the presence/absence of fatigue, discomfort and changed respiratory patterns during inclination.

Considering that the effects of this technique have not been sufficiently studied, this study aimed to assess the consciousness level, pulmonary and hemodynamic effects in intensive care patients during orthostatic positioning.

**METHODS**

**Subjects**

This was a prospective, interventional clinical trial conducted from April 2008 to July 2009 in the Adult ICU of Hospital das Clínicas (HC), Universidade Estadual de Campinas (UNICAMP), Campinas, São Paulo, Brazil. This study protocol was approved by the UNICAMP Medical College’s ethics committee under number 225/2008. For all of the included patients, a written informed consent form was signed by a legally responsible relative.

Male and female patients admitted to the ICU older than 18 years old and younger than 65 years old who were intubated and under mechanical ventilation for more than seven days were included. The subjects were required to have a tracheotomy, to be under intermittent nebulization for more than three days; to have maximal inspiratory pressure (PImax) less than -25 cm H2O; to have a Tobin score (TS) less than 105, collaborative (Glasgow coma scale [GCS] ≥ 8, motor response = 6, verbal response = 1; eye opening = 1) to allow for vital capacity (VC) and thoracoabdominal cirtometry; to have preserved respiratory drive, partial arterial oxygen pressure (PaO2) greater than 70 mm Hg and oxygen saturation (SatO2) greater than 90%; to be hemodynamically stable; and to use no vasoactive, inotropic and/or sedative drugs. Patients were excluded if they had proven cardiac changes on electrocardiogram (ECG); a bronchopleural fistula; deep vein thrombosis; thrombocytopenia (platelet count less than 50,000); a body temperature greater than 37.8ºC; bone fractures in the lower limbs; osteomuscular changes preventing orthostatism; spinal cord injuries; significant calcaneal pressure ulcers; an intra-aortic balloon (IAB); an intracranial pressure (ICP)-monitoring catheter; and/or external ventricular derivation (EVD).

**Description of the interventions**

During the procedure, the following measurements were assessed: consciousness level and degree of alertness; thoracoabdominal cirtometry; vital capacity (VC); tidal volume (TV); minute volume (V̇E); respiratory muscle strength; respiratory rate (RR); mean blood pressure (MBP); and heart rate (HR). The consciousness level...
and degree of alertness were assessed using the Glasgow coma scale (GCS) and the blinking reflex.

Thoracoabdominal cirtometry was performed using a plain metric tape; the chest was measured at the 4th costal arch level, and the abdominal measurement was taken over the navel while the patient breathed in and out deeply.

The VC, Ve and TV measurements were performed using a Ferrares Mark 8 Wright Respirometer®. To measure VC, the patient was asked to perform maximal inspiration and then maximal expiration, close to the residual volume, with the tracheotomy adapted to the ventilometer. Ve was measured based on regular respiratory cycles with effortless inspirations and expirations for one minute. TV was measured using the following equation: TV = Ve/RR. The RR was measured during the Ve measurement, by direct observation of chest movements.

Respiratory muscle strength was assessed by measuring the maximal inspiratory pressure (PImax) and maximal expiratory pressure (PEmax), both performed with a Comercial Médica® manovacuometer. PImax was measured by adapting the manovacuometer to the patient's tracheotomy by means of a unidirectional valve. The patient was asked to perform maximal expiration followed by a maximal inspiratory effort against the closed airway for 20 seconds. Three maximal inspiration measurements were measured with a one-minute interval between them, and the highest value was recorded. PEmax was measured with the manovacuometer adapted to the patient's tracheotomy. Later, the patient inspired until reaching maximal pulmonary capacity and then made a maximal expiratory effort. Three maximal expiration measurements were measured with a one-minute interval between them, and the highest value was recorded.

Statistical analysis
ANOVA for repeated measures with rank-transformation was used to compare the continued variables (MBP, HR, RR, chest cirtometry, abdominal cirtometry, PImax, PEmax, VC, MV and TV) among the 0º, 30º and 50º inclination angles, considering the non-normal data, thereby reducing the asymmetry of the recorded values. Where significant differences were found, multiple comparison tests (contrast) were performed to identify the differences. A 5% level was adopted for statistical significance, i.e., a p value ≤ 0.05.

RESULTS
Eight of the 15 patients were female (53.3%), and seven (46.6%) were male; the mean age was 42.5 ±16.2 years old. The mean Acute Physiology Health Evaluation II (APACHE II) score was 14.5 ±4.5.

Only two patients failed to complete the study protocol. One had a relevant MBP and HR increase in the 30º inclination and was immediately returned back to 0º and improved without any other interventions. However, no variables could be recorded, so this subject was withdrawn from the study. The other patient had significant clonus at 50º, preventing the assessment of thoracoabdominal cirtometry and respiratory muscle strength (PImax and PEmax) at this inclination level. The patient was repositioned back into bed, and the parameters normalized. The other 13 patients completed the full protocol without reporting any discomfort or showing instability. The admission diagnoses and comorbidities are shown in table 1.

Considering neurological level and alertness (GCS and blinking reflex), the patients underwent no statistically significant changes during the procedures, as shown in table 2.

Regarding the respiratory variables (Table 2), RR decreased at 30º and underwent a mild increase at 50º; however, these differences were not statistically significant.

Chest cirtometry increased in the 30º inclination and then was decreased at 50º; however, neither change was statistically significant. Abdominal cirtometry showed gradually increasing abdominal circumference, with no statistical significance.

PImax showed a statistically significant and gradual decrease at 30º and 50º, and PEmax showed a statistically significant increase at 50º.

The patient was monitored the entire time, and in cases of discomfort or clinical instability, the procedure was immediately discontinued.
immediate effects of orthostatism in ICU patients

The effects of orthostatism, particularly the influence of angle inclination on various physiological parameters, were investigated in this study. As the angle inclination increased, the inspiratory strength showed a statistically significant increase (p = 0.0218). When the inclinations were compared, a statistically significant increase was identified only for the comparison between 50º and 0º (p = 0.025). PE_max's behavior was similar to PI_max's; however, the observed increase was not statistically significant. VC underwent a statistically significant increase during the maneuver (p = 0.024) regarding the comparison of the values at 50º versus 0º (p = 0.003). The analysis of V_E showed an initial drop and then an increase; however, the changes were not statistically significant for the different angle inclinations. Regarding TV, a statistically significant and gradual increase was observed during the maneuver (p = 0.012). Comparing the different angle inclinations, significant differences were found for 30º versus 0º (p = 0.040) and 50º versus 0º (p = 0.017).

Regarding the hemodynamic variables (table 2), MBP trended to increase (p = 0.051); the comparison of 50º versus 0º was statistically significant (p = 0.016). The analysis of HR showed a gradual increase (p = 0.001) for the comparisons between 30º and 0º (p = 0.025), between 50º and 0º (p = 0.001) and between 50º and 30º (p = 0.002).

### Table 1 - Demographic information

<table>
<thead>
<tr>
<th>Subject</th>
<th>Age</th>
<th>Diagnosis and comorbidities</th>
<th>APACHE II</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>18</td>
<td>Post-operatively of renal transplantation; pleural effusion; bronchopneumonia; and sepsis</td>
<td>16</td>
</tr>
<tr>
<td>2</td>
<td>31</td>
<td>Post-operatively of excision of retroperitoneal tumor; hypovolemic shock; sepsis; testis tumor; and injury to the vena cava</td>
<td>12</td>
</tr>
<tr>
<td>3</td>
<td>24</td>
<td>Head trauma and polytrauma</td>
<td>5</td>
</tr>
<tr>
<td>4</td>
<td>59</td>
<td>Reduced consciousness level and sepsis from pulmonary focus</td>
<td>12</td>
</tr>
<tr>
<td>5</td>
<td>18</td>
<td>Head trauma and drainage of epidural hematoma</td>
<td>17</td>
</tr>
<tr>
<td>6</td>
<td>43</td>
<td>Post-operatively of cerebral aneurysm clipping</td>
<td>11</td>
</tr>
<tr>
<td>7</td>
<td>56</td>
<td>Postoperative maxillectomy; bronchopneumonia; and sepsis</td>
<td>19</td>
</tr>
<tr>
<td>8</td>
<td>38</td>
<td>Thrombosis of sagittal sinus</td>
<td>9</td>
</tr>
<tr>
<td>9</td>
<td>65</td>
<td>Post-operatively of carotid aneurysm clipping</td>
<td>17</td>
</tr>
<tr>
<td>10</td>
<td>37</td>
<td>Postoperatively of hemotoma drainage and aneurysm clipping</td>
<td>15</td>
</tr>
<tr>
<td>11</td>
<td>60</td>
<td>Head trauma; polytrauma; pulmonary contusion; and costal arch fracture</td>
<td>12</td>
</tr>
<tr>
<td>12</td>
<td>57</td>
<td>Septic shock; systemic hypertension; and diabetes mellitus</td>
<td>21</td>
</tr>
<tr>
<td>13</td>
<td>54</td>
<td>Correction of thoracic aorta aneurysm; systemic hypertension; and sepsis</td>
<td>14</td>
</tr>
<tr>
<td>14</td>
<td>34</td>
<td>Acute pancreatitis; sepsis; systemic hypertension; and diabetes mellitus</td>
<td>21</td>
</tr>
<tr>
<td>15</td>
<td>45</td>
<td>Post-operatively of carotid aneurysm clipping</td>
<td>13</td>
</tr>
</tbody>
</table>

### Table 2 - Neurological, respiratory and hemodynamic variables in the 0º, 30º and 50º positions

<table>
<thead>
<tr>
<th>Variables</th>
<th>0º</th>
<th>30º</th>
<th>50º</th>
<th>p value for the sample</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glasgow coma scale</td>
<td>11</td>
<td>11</td>
<td>11</td>
<td>1</td>
</tr>
<tr>
<td>Blinking reflex (presence)</td>
<td>14</td>
<td>14</td>
<td>14</td>
<td>1</td>
</tr>
<tr>
<td>Respiratory rate (ipm)</td>
<td>30.8 ± 9.2</td>
<td>26.6 ± 7.1</td>
<td>27.1 ± 6.5</td>
<td>0.203</td>
</tr>
<tr>
<td>Chest cirtometry (cm)</td>
<td>1.2 ± 0.5</td>
<td>1.3 ± 0.6</td>
<td>1.1 ± 0.5</td>
<td>0.631</td>
</tr>
<tr>
<td>Abdomen cirtometry (cm)</td>
<td>1.6 ± 1.0</td>
<td>1.8 ± 1</td>
<td>2.0 ± 1</td>
<td>0.110</td>
</tr>
<tr>
<td>Maximal inspiratory pressure (cm H2O)</td>
<td>-59.7 ± 18.0</td>
<td>-62.2 ± 20.9</td>
<td>-67.1 ± 23.6</td>
<td>0.021</td>
</tr>
<tr>
<td>Maximal expiratory pressure (cm H2O)</td>
<td>44.7 ± 30.2</td>
<td>47.2 ± 32.5</td>
<td>49.6 ± 32.5</td>
<td>0.244</td>
</tr>
<tr>
<td>Vital capacity (mL)</td>
<td>1612.1 ± 753.4</td>
<td>1802.1 ± 992.3</td>
<td>1932.8 ± 883.6</td>
<td>0.024</td>
</tr>
<tr>
<td>Minute volume (mL)</td>
<td>11167.1 ± 3300.4</td>
<td>10886.4 ± 3301.6</td>
<td>11288.5 ± 2583.3</td>
<td>0.883</td>
</tr>
<tr>
<td>Tidal volume (mL)</td>
<td>381.4 ± 124.3</td>
<td>420.1 ± 137.0</td>
<td>443.3 ± 144.8</td>
<td>0.012</td>
</tr>
<tr>
<td>Mean blood pressure (mm Hg)</td>
<td>98.7 ± 17.5</td>
<td>102.6 ± 18.3</td>
<td>102.7 ± 18.3</td>
<td>0.051</td>
</tr>
<tr>
<td>Heart rate (bpm)</td>
<td>93.1 ± 14.9</td>
<td>99.0 ± 16.6</td>
<td>104.5 ± 18.2</td>
<td>0.001</td>
</tr>
</tbody>
</table>

* p< 0.05 versus 0º; ** p< 0.05 versus 30º.
DISCUSSION

No changes were observed at the neurological level. The observed changes in \( f \), \( V_E \), PEmax, chest cirtometry and abdomen cirtometry were not statistically significant. Regarding PImax and VC, a statistically significant increase was seen in the comparison between the 50º and 0º inclinations. However, TV increased for the comparisons between 30º and 0º and between 50º and 0º. Mean blood pressure increased only for the comparison of 50º versus 0º. Heart rate increased for all angle inclinations.

This study showed that during the procedure, there were no changes in neurological variables as assessed with the GCS and blinking reflex; however, the study by Velar and Júnior reported significant improvement in consciousness level and alertness in patients undergoing 70º orthostatism for approximately 20 seconds. According to these authors, this result occurred because the alert systems were less stimulated than during orthostatism.\(^8\)

Our HR findings diverged from those in the literature. In this study, no differences were found for this variable, in agreement with previous studies;\(^{10-12}\) however, all of the previous studies were conducted in healthy subjects. However, Gisolf studied orthostatism with the support of nine mathematical models simulating the cardiorespiratory system and found reduced HR after five minutes in an orthostatic position.\(^\) In contrast, in the study by Chang et al., in 15 intensive care patients, HR increased with 70º inclination maintained for five minutes.\(^9\) Bourdin et al. assessed the ICU’s most frequently acquired physiotherapy measurements, as well as orthostatism's effects, in 20 critically ill patients and reported a statistically significant HR increase during passive orthostatism.\(^{14}\)

Regarding cirtometry, no changes in chest or abdominal circumference were detected. We could not find any articles in the literature reporting on this variable during passive orthostatism. In a study in which respiratory mechanics were assessed in 28 mechanically ventilated patients, it was found that the more verticalized the patient was, higher pulmonary compliance and expansibility were. This finding was ascribed to a smaller dependent zone in this position, assuming that free zones are more compliant and thus favoring respiratory mechanics.\(^{15}\) Another study reported on thoracic and abdominal expansibility, both in supine and active orthostatic positions, in healthy subjects. Thoracic expansion was greater during orthostatism, and abdominal expansion was greater in the supine position. This change was likely due to downward movement of the viscera, allowing for improved thoracic expansion.\(^{10}\)

This study showed statistically significant differences only for PImax and not for PEmax. In a study by Fiz et al. assessing PImax and PEmax in supine, seated and standing positions in 10 obese patients and 10 normal body mass index subjects, it was observed that the values were higher for the standing position in comparison to other positions; therefore, they were in agreement with our PImax increase.\(^{16}\) In another study by Roquejani et al., the influence of body position was assessed on the respiratory muscle strength of healthy subjects. Orthostatic position was not assessed; however, more verticalized positions showed no differences for PImax and PEmax in comparison with other positions.\(^{17}\)

The analysis of VC showed statistically significant increases when the 50º and 0º inclinations were compared, in agreement with other authors’ findings. Already in 1927, Wilson suggested that VC decreased due to a pulmonary congestion in the supine position and the diaphragm elevation decreased due to the pressure of abdominal viscera, consequently reducing the volume of the chest.\(^{18}\) Blair and Hickman described, in their study of 11 healthy subjects, that residual volume (RV), residual functional capacity (RFC) and total pulmonary capacity (TPC) were significantly reduced when changing from an orthostatic to a supine position.\(^{19}\) In the same article, the authors stated that these changes apparently resulted from progressive diaphragm elevation, presumed to occur due to the pressure of the abdominal viscera and decreased rest diaphragm tonus in the supine position.\(^{19}\) During the first studies of human neural drive to the diaphragm, it was identified that muscle activity was increased by an average of four- to five-fold when the subjects changed from supine to orthostatic positions.\(^{20}\) This finding means that the neural drive to the diaphragm likely increases considerably to compensate for different loads on the diaphragm in different positions, perhaps due to increased diaphragm proprioceptive reflexes stimulating the subjects to breathe more deeply, increasing the VC.\(^{10,21}\)

In our study, \( V_E \) showed an initial decrease, followed by an increase, with orthostatism; however, this difference was not significant. In agreement with this study, Butler et al.\(^{16}\) found no difference for \( V_E \) in the comparison between the supine and orthostatic positions. In the same study, respiratory muscle motor
units were studied separately, aiming to assess the neural activity of the diaphragm and the intercostal muscles in the supine position; no change was found for the frequency of discharge in either muscle of the motor units.\textsuperscript{(10)} However, in other studies in which $V_E$ was compared between different positions, increased values were found during orthostatism.\textsuperscript{(9,11-13)}

Regarding TV this study showed a statistically significant increase with time between 30° and 0° and between 50° and 0°. In a case reported by Dean and Ross, a mechanically ventilated patient started training on an orthostatic board during the fifth day after ICU admission. The patient was initially positioned at 15° for five minutes and then at 45° for 10 minutes. The study identified improved ventilation parameters and increased pulmonary volumes using radiographic analysis; however, the lack of quantitative data prevented the determination of the effects of the position on ventilation.\textsuperscript{(22)} Other authors have described similar results.\textsuperscript{(9,11-13)} However, the mechanics behind the ventilation change are not fully understood.

According to Yoshizaki,\textsuperscript{(11)} the afferent signs that originate from the lower limbs and that are related to maintaining posture are activated during orthostatism. This information is projected to the respiratory center in the brain, resulting in increased ventilation. In his study, contraction of the gastrocnemius muscle, detected by electroneuromyography, supported this possibility.\textsuperscript{(11)}

The effort necessary to support the position on the orthostatic board can also increase energy expenditure, thereby increasing ventilation.\textsuperscript{(23)} However, some studies have shown that this increased $O_2$ consumption occurs when the subject is not firmly fixed to the board, resulting in increased muscle activity.\textsuperscript{(21)}

Another possible mechanism for the TV increase is that changing from a supine to an orthostatic position may increase RFC and diaphragm mobility, lowering the abdominal contents. This RFC change modifies the point at which each tidal volume occurs on the pressure-volume curve, resulting in increased respiratory system compliance and implying higher inspired volumes in an orthostatic position.\textsuperscript{(9,12)}

Orthostatic positioning may promote increased ventilation due to vestibular stimulation. Vestibular nerve stimulation in anesthetized cats was reported to increase phrenic nerve and respiratory muscle activity, indicating that there is some vestibular stimulation in breathing.\textsuperscript{(24)} However, in human models, vestibular stimulation failed to show ventilation effects.\textsuperscript{(25)}

In addition, several types of chest wall receptors have been reported to be responsible for ventilation increases. In the supine position, C pulmonary fibers are activated as a result of the increased volume of pulmonary blood, leading to tachypnea and reduced alveolar ventilation. However, intercostal muscles are activated during inclination and might induce hyperventilation (increased RR and TV).\textsuperscript{(26)} The patient’s alertness during orthostatism might also support the theory of increased ventilation;\textsuperscript{(9)} however, in our study, the alertness level did not change with orthostatism.

Finally, the inclination procedure increases the patient’s anxiety, thereby stimulating sympathetic activity. This process may have effects on breathing. Sympathetic nerve stimulation is known to cause bronchodilation, increase RR and reduce TV.\textsuperscript{(27)} In this study, the respiratory response to orthostatism was characterized by increased TV and unchanged RR during the intervention, suggesting that other factors might influence ventilation, as sympathetic stimulation alone would have reduced TV, as previously reported in healthy young subjects. Similarly, the increased MBP should have been associated with reduced RR and TV in response to increased stimulation of arterial baroreceptors;\textsuperscript{(28)} therefore, it was not in agreement with this study’s findings. There is evidence that increased afferent signs from baroreceptors inhibit breathing via an integrated central mechanism.\textsuperscript{(28)} However, in disagreement with this study, the trial by Buttler found no TV or neural drive changes in the respiratory muscles during active orthostatism.\textsuperscript{(28)}

In agreement with the literature, HR and MBP showed statistically significant increases with inclination.\textsuperscript{(29,30)} When the posture is changed, carotid baroreceptors equally change in relation to the heart and can induce blood pressure changes. Baroreceptors are located approximately 25 cm above the heart, and they detect blood pressure at a mean 18 mm Hg less than the heart level. Therefore, the blood pressure at the heart level should change from 15 to 20 mm Hg upon changing from a supine to standing position. However, aortic baroreceptors detect higher pressures due to their proximity to the heart and might potentially neutralize this MBP increase. Conversely, the filling of the cardiac chambers is affected by postural change, as arteries and veins situated below the heart level are filled more greatly during orthostatism. This substantial reduction of central filling pressure would cause a significant drop in ejection fraction and cardiac output, even though baroreflexive mechanisms induce an HR increase, which is not sufficient to normalize cardiac output. Consequently,
there is peripheral vasoconstriction when the body is changed from a supine to orthostatic position.

Gravitational stress is also responsible for increasing the secretion of hormones such as noradrenalin, adrenalin and aldosterone, contributing to increases in HR and MBP. Orthostatism can also cause a significant reduction of cardiac vagal activity in comparison with the supine position. In contrast, sympathetic activity can significantly increase during elevation. These findings support the provisional hypothesis regarding the influence of body posture on autonomic nervous system regulation. It could be said that orthostatism induces increased sympathetic tonus and decreased parasympathetic tonus.

This study has limitations that include its small sample size, which was due to difficulty in screening subjects who fit the inclusion criteria. This limitation impaired the observation of more subtle changes in the assessed parameters. The exclusion of two subjects from some variables’ analyses as a result of instability might also have contributed to the lack of significant changes in respiratory parameters, which failed to show relevant changes. Additionally, the patients were not assessed after the intervention; therefore, it is not possible to say for how long the changes were maintained.

CONCLUSION

It is concluded that orthostatism does not change consciousness level or alertness; however, it provides improved TV, VC, and PImax, and it increases HR and MBP in critically ill patients who are restricted to bed but who have clinical conditions that allow the maneuver.

REFERENCES

10. Butler JE, McKenzie DK, Gandevia SC. Discharge

RESUMO

Objetivo: Analisar o nível de consciência, efeitos pulmonares e hemodinâmicos em pacientes intensivos durante a posição ortostática.

Métodos: Estudo realizado de abril de 2008 a julho de 2009 na unidade de terapia intensiva adulto do HC-UNICAMP. Foram incluídos quinze pacientes que estiveram mecanicamente ventilados por mais de sete dias; traqueostomizados; em nebulização intermitente; pressão inspiratória máxima inferior a -25 cmH2O; índice de Tobin inferior a 10; drive ventilatório preservado, ausência de sedativos; pressão parcial de oxigênio arterial maior que 70 mmHg; saturação de oxigênio maior que 90% e estabilidade hemodinâmica. Os parâmetros avaliados, nas inclinações de 0º, 30º e 50º, foram o nível de consciência; reflexo de blinking; cirtometria tórcico-abdominal; capacidade vital; volume corrente; volume minuto; força da musculatura respiratória e sinais vitais.

Resultados: Não houve alteração do nível neurológico. A frequência respiratória (f) e V E reduziram-se em 30º com posterior aumento em 50º, no entanto, essas alterações não foram estatisticamente significativas. A cirtometria abdominal e a pressão expiratória máxima apresentaram aumento, novamente sem significância estatística. Em relação à pressão inspiratória máxima e a capacidade vital observou-se aumento estatisticamente significante na comparação entre as angulações 50º e 0º. Já o volume corrente aumentou ao longo do tempo, na comparação entre as angulações 30º e 0º, e entre 50º e 0º. A pressão arterial média sofreu incremento somente na comparação entre 50º e 0º. A frequência cardíaca elevou-se ao longo do tempo e quando comparada entre 30º e 0º, 50º e 0º, e 50º e 30º.

Conclusão: O ortostatismo passivo proporcionou melhora do volume corrente, capacidade vital, pressão inspiratória máxima, e aumento da frequência cardíaca e pressão arterial média em pacientes críticos.

Descritores: Serviço hospitalar de fisioterapia; Reabilitação; Unidades de terapia intensiva


