Analysis of Ventilation and Hemodynamic Changes Resulting from Noninvasive Bilevel Pressure Mechanical Ventilation Applied to Patients with Congestive Heart Failure

Ariadne Fernandes de Barros, Luciana Costa de Barros, Márcia Cristina Sangean, Joaquim Minuzo Vega
Hospital Estadual Mario Covas - Santo André, SP, Brazil

Objective: To observe in practice how noninvasive bilevel positive pressure, applied at different levels, may interfere with systolic arterial pressure (SAP), diastolic arterial pressure (DAP), mean arterial pressure (MAP), heart rate (HR), respiratory rate (RR), and peripheral oxygen saturation (SpO2), in patients with congestive heart failure (CHF).

Methods: Fourteen CHF patients (mean age 62.85 years) were treated with noninvasive bilevel mechanical ventilation. Patients were consecutively treated with an expiratory positive airway pressure (EPAP) of 5 cmH2O, 10 cmH2O, 15 cmH2O, 10 cmH2O, and 5 cmH2O, maintaining a variation in pressure (ΔP) of 5 cmH2O between the inspiratory pressure (IPAP) and the expiratory pressure (EPAP). Ventilation and hemodynamic data were collected at these time points, as well as 5 minutes before the beginning of the protocol and 5 minutes after its completion.

Results: A statistically significant difference was observed in the respiratory rate between the moment just before the beginning of the protocol and 5 minutes after positive pressure mask placement (p=0.022), and in oxygen saturation, between the final minute of EPAP at 5 cmH2O and after the removal of the mask (p=0.05).

Conclusion: Noninvasive bilevel mechanical ventilation improves oxygenation and reduces respiratory work, thus being beneficial for patients with congestive heart failure. It was not possible to observe statistically significant changes in the hemodynamic data due to the small number of patients and to other associated heart diseases.

Key words: Heart failure, congestive heart failure, artificial respiration, positive pressure respiration

The combination of mechanical ventilation and positive pressure (PEEP) aims to increase arterial oxygenation and maintain life support for patients who have lost the capacity for spontaneous ventilation[1].

The goal of PEEP application is to keep positive pressure in the airways throughout the expiratory phase, including its end stage. However, the technique consists of applying a resistance to the expiratory flow of the ventilation cycle, keeping in mind that the effectiveness of the technique lies in adjusting the PEEP level to the pathophysiological process that is impairing lung function[2].

Lenique et al[3] have shown that the use of PEEP significantly improves gas exchange due to the recruitment of collapsed alveoli, and, according to Barbas et al.[4], this results in increased arterial oxygen pressure (PaO_2) and decreased arterial carbon dioxide pressure ($PaCO_2$).

The application of positive end-expiratory pressure increases air pressure in the mediastinum. With this mechanism, and with the lung overdistended, the boost in external juxtacardiac pressure results in increased intracavity pressures, due to the decline in myocardial compliance and venous return (VR)[5].

PEEP also affects the hemodynamic function. As intrathoracic pressure is raised, there is a reduction in the systemic vascular resistance (SVR), compression of the superior and inferior vena cavas, a drop in ventricular filling, as well as in left atrium (LA) filling and, finally, a reduction in cardiac output (CO). As a result of the increased intrathoracic pressure and reduced cardiac output, a significant fall in mean arterial blood pressure (MAP) takes place[6-8].

The clinical benefits of positive pressure ventilation in treating congestive heart failure patients are clearly observed**:** improvement in oxygenation, reduction in respiratory work, as well as improvement in left ventricular function and cardiac output[9].

There is a difference between the proper application of PEEP to cardiopathic patients and to patients with normal LV function. While these are minimally affected by changes in the postload, cardiopathic patients with LV dilation are more sensitive to changes in postload[10].

Therefore, one of the main factors for the development of acute respiratory failure in cardiopathic patients is the reduction of lung compliance, which increases the respiratory work. This reduction results from the circulatory failure caused by LV dysfunction due to preload and postload changes. Consequently,

Mailing Address: Ariadne Fernandes de Barros •
Rua Dona Carlota , 242/62 – 09040-250 – Santo André, SP, Brazil
E-mail: dinny_fernandes@yahoo.com.br
Manuscript received August 8, 2005; Revised manuscript received May 31, 2006; Accepted July 3, 2006.

the application of positive pressure reduces the respiratory work, resulting in beneficial changes in transpulmonary pressure and reduced $PaCO_2$. Moreover, there is also a significant increase in lung compliance associated with the improvement in LV due to increased intrathoracic pressure[11].

Hence, application of PEEP to patients with CHF significantly improves arterial oxygenation by reducing the respiratory work. According to Hoyos et al, higher intrathoracic pressure raises the CO by means of an increase in LV ejection fraction, since the main factors that determine myocardial reversion are represented by preload, postload, myocardial contractility, and heart rate (HR)[3,9,12].

The objective of this study was to show how positive end-expiratory pressure (PEEP), applied at different levels, interferes with systolic arterial pressure (SAP), diastolic arterial pressure (DAP), mean arterial pressure (MAP), heart rate (HR), respiratory rate (RR), and peripheral oxygen saturation (SpO_2) in cardiopathic patients receiving noninvasive bilevel ventilation.

Methods

Patients included in the protocol were those admitted to the adult intensive care unit of *Hospital Estadual Mário Covas*, in Santo André, state of São Paulo, between May and August 2003, who met the inclusion criteria (cardiac dysfunction and acute pulmonary edema) and did not have any of the exclusion criteria. Exclusion criteria in this protocol were**:** any pulmonary condition that would contraindicate changes in PEEP values (chronic obstructive pulmonary disease, particularly those with pulmonary bubbles, bronchial fistula, and pulmonary abscesses); hemodynamically unstable patients not receiving vasoactive drugs; patients with acute circulatory failure; patients with arterial hypotension (MAP ≤ 70 mmHg); patients with intracranial hypertension; cardiopathies requiring corrective surgery (valve alterations, myocardial ischemia, drug-resistant arrhythmias); cranial-encephalic trauma; recent facial surgery or anatomic nasopharyngeal anomalies; patients with hypersecretion who are unable to cough; upper digestive hemorrhage and upper digestive tract surgery; non-drained pneumothorax; hypersecretion; severe encephalopathy (Glasgow < 10); noncollaborative patient; extreme anxiety.

Patient's personal data (age and gender), data about the disease, reasons for the decompensation, hemodynamic data (HR, SAP, DAP, MAP) collected by non-invasive Dixtal 1020 monitor, ventilatory data (RR, SpO_2), respiratory rate measured by the physiotherapist who applied the protocol, and SpO_2 (also registered by the Dixtal 1020 monitor) were all recorded in the Case Report Form – CRF. Drugs administered or any other relevant observation that could directly interfere with the results were also recorded.

Next, the noninvasive mechanical ventilation mask was applied (BREAS PV101 bilevel device), with EPAP and IPAP values set at 5 cmH_2O and 10 cmH_2O, respectively, and maintaining a pressure variation of 5 cmH_2O (ΔP) throughout all EPAP levels during 15 minutes. Hemodynamic and ventilatory data were recorded at each five-minute interval with this EPAP level. After 15 minutes, the EPAP level was increased to 10 cmH_2O and maintained for 15 minutes; hemodynamic and ventilatory data were recorded again for each five-minute interval. The same procedures was followed when the EPAP was increased to 15 cmH_2O. From this level on, the EPAP value was reduced to 10 cmH_2O, and then to 5 cmH_2O, with a 15-minute interval between each change; hemodynamic and ventilatory data were recorded again at each five-minute interval. Finally, the noninvasive mechanical ventilation mask was removed and data were recorded once more after 5 minutes. If the patient needed support ventilation, the mask was applied again.

NOTE: If at any point of the protocol the patient developed any of the exclusion criteria, he/she would be dismissed from the study and the necessary measures would be taken. However, these patients would be included in the statistical analysis, and the complication, as well as the reason for the interruption of the protocol, would be discussed. Subsequently, a statistical analysis of the hemodynamic and respiratory variations resulting from the changes in EPAP values was performed.

Results

Fourteen patients with acute pulmonary edema due to cardiac decompensation participated in the study. Table 1 shows age, diagnosis and drugs used for each patient enrolled in the trial.

The mean heart rate (beats per minute) before the study was 82 ± 22; with an initial EPAP of 5 cmH_2O, 82 ± 21; with an initial EPAP of 10 cmH_2O, 81 ± 22; with an EPAP of 15 cmH_2O, 83 ± 21; with a final EPAP of 10 cmH_2O, 80 ± 19; with a final EPAP of 5 cmH_2O, 79 ± 21; and after the mask had been removed, 80 ± 22. No statistically significant difference was observed at any time point in this parameter (table 2).

The respiratory rate (breaths per minute) before the start of the protocol was 24 ± 11; with an initial EPAP of 5 cmH_2O, 20 ± 6; with an initial EPAP of 10 cmH_2O, 20 ± 5; with an EPAP of 15 cmH_2O, 20 ± 3; with a final EPAP of 10 cmH_2O, 20 ± 3; with a final EPAP of 5 cmH_2O, 20 ± 4; and after the mask had been removed, 20 ± 8. A statistically significant difference was observed between the initial time point, before the noninvasive ventilation mask was placed, and the initial EPAP of 5 cmH_2O (p=0.0222) (Table 2).

Table 2 shows the mean values, and their respective standard deviations, of the parameters evaluated before and during the application of the technique using different EPAP levels, and at the end of the application without the utilization of EPAP.

The mean systolic arterial pressure (mmHg) before the start of the protocol was 129 ± 22; with an initial EPAP of 5 cmH_2O, 131 ± 21; with an initial EPAP of 10 cmH_2O, 132 ± 21; with an EPAP of 15 cmH_2O, 130 ± 23; with a final EPAP of 10 cmH_2O, 129 ± 22; with a final EPAP of 5 cmH_2O, 128 ± 22; and after the mask had been removed, 124 ± 23. No statistically significant difference was observed at any time point in this parameter (Table 2).

The mean diastolic arterial pressure (mmHg) before the start of the protocol was 79 ± 15; with an initial EPAP of 5 cmH2O, 79 ± 14; with an initial EPAP of 10 cmH_2O, 80 ±

P	Age (years)	Diagnosis	Drugs
1	67	DM; reversed VF (2 episodes); acute arterial embolism in lower left limb; ARF; APE	Amiodarone; Furosemide; Heparin; Dobutrex; Deslanoside; Spironolactone
2	33	MS; RF; CHF; ischemic CVA	Capoten; Furosemide; Acetylsalicylic acid
3	70	Consumptive syndrome; DM; APE	Capoten
4	86	DCHF; APE	Dobutrex; Noradrenaline; Furosemide
5	86	DCHF; APE; BF	Capoten; Enoxoparin; Furosemide; Vallium; Dipyrone
6	36	DCHF; APE; coronary insufficiency; acute respiratory failure	Furosemide; Noradrenaline
7	70	CRF; pyelonephritis; DCHF; APE; acute AF (high response); IM	Enoxoparin; Amiodarone; Capoten; Furosemide
8	68	CHF; APE; BF; chronic AF; acute CRF; AMI	Enoxoparin; Furosemide; Acetylsalicylic acid
9	54	DCHF; chronic AF; APE	Amiodarone
10	45	DCHF; APE; chronic AF; PE (minimum)	Furosemide;Enoxaparin;Deslanoside
11	60	DCHF; APE	Sodium nitroprussiate; Capoten; Spironolactone; Furosemide; Enoxaparin; Deslanoside
12	70	CHF; APE; IM; diabetes mellitus; systemic arterial hypertension	Heparin; Deslanoside; Capoten; Spironolactone
13	67	DM; reversed VF (2 episodes); acute arterial embolism in lower left limb; ARF; APE	Amiodarone; Furosemide; Heparin; Dobutrex; Deslanoside
14	68	DCHF; APE	Noradrenaline; Capoten; Acetylsalicylic acid; Furosemide

DCHF = decompensated congestive heart failure; DM = dilated myocardiopathy; IM = ischemic myocardiopathy; APE = acute pulmonary edema; AMI = acute myocardial infarction; MS = mitral stenosis; RF = rheumatic fever; PE = pericardial effusion; CVA = cerebrovascular accident; AF = atrial fibrillation; VF = ventricular fibrillation; ARF = acute renal failure; CRF = chronic renal failure; BF = bronchopneumonia

Table 1 - Patients' age, diagnosis and drug therapy

14; with an EPAP of 15 cmH_2O, 82 ± 18; with a final EPAP of 10 cmH_2O, 83 ± 18; with a final EPAP of 5 cmH_2O, 77 ± 17; and after the mask had been removed, 76 ± 18. No statistically significant difference was observed at any time point in this parameter (Table 2).

The mean arterial pressure (mmHg) before the start of the protocol was 94 ± 12; with an initial EPAP of 5 cmH_2O, 98 ± 13; with an initial EPAP of 10 cmH_2O, 96 ± 13; with an EPAP of 15 cmH_2O, 96 ± 13; with a final EPAP of 10 cmH_2O, 96 ± 15; with a final EPAP of 5 cmH_2O, 93 ± 14; and after the mask had been removed, 89 ± 14. No statistically significant difference was observed at any time point with this parameter (Table 2).

The mean peripheral oxygen saturation (SpO_2) before the start of the protocol was 95 ± 2; with an initial EPAP of 5 cmH_2O, 95 ± 1; with an initial EPAP of 10 cmH_2O, 95 ± 1; with an EPAP of 15 cmH_2O, 95 ± 1; with a final EPAP of 10 cmH_2O, 95 ± 2, with a final EPAP of 5 cmH_2O, 96 ± 1; and after the mask had been removed, 95 ± 1. A statistically significant difference was observed between the moment the mask was being used at an EPAP of 5 cmH_2O, and after the mask had been removed (p=0.051) (Table 2).

Discussion

Our results indicate that patients with congestive heart failure (CHF) benefit from noninvasive bilevel positive airway pressure ventilation. This benefit was observed not only by evaluating data recorded, but also by the patients' treatment acceptance and willingness to collaborate, as well as the reported improvement in dyspnea. Patients with congestive heart failure have to deal with increased respiratory rates caused by the decrease in lung compliance and volume, as well as the increase in intrapulmonary shunt. This is due to increased pulmonary fluids caused by changes in hydrostatic relative to oncotic pressures, both intravascular and interstitial.[3, 13, 14]

The presence of cardiogenic pulmonary edema leads to airflow obstruction through the increase in pulmonary vascular pressure, resulting in reflex bronchoconstriction. Due to the decrease in pulmonary volume caused by reduced lung compliance, the airways are narrowed, i.e. the bronchial lumen is obstructed by edema fluid, and the bronchial mucosa is dilated, resulting in increased respiratory elastic work and dyspnea. [15-18]

Reduction in lung compliance leads to an increase in respiratory work and oxygen consumption in order to maintain adequate ventilation. As a result, greater negative intrathoracic pressures are needed to maintain an adequate pulmonary ventilation.[11, 19]

The application of positive pressure increases the inspiratory flow rate, which, in turn, generates a significant increase in pulmonary volume and lung compliance. This redistributes fluid within the alveoli (by alveolar recruitment), improving gas exchange and alveolar ventilation, i.e., reducing the intrapulmonary shunt.[3, 14, 19, 20]

Changes in PEEP levels promote directly proportional alterations in RFC and in pulmonary compliance. That is,

		BEFORE	EPAP 5	EPAP 10	EPAP 15	EPAP 10	EPAP 5	AFTER
HR	MEAN	129.0	131.0	132.0	130.0	129.0	128,0	124,0
	SD	22.9	21.8	21.6	23.3	22.5	22,7	23,1
f	MEAN	24.3	20.6	20.0	20.7	20.3	20,8	20,9
	SD	11.8	6.9	5.2	3.5	3.6	4,8	8,5
SAP	MEAN	82.4	82.9	81.4	83.1	80.1	79,4	80,5
	SD	22.6	21.6	22.7	21.2	19.8	21,7	22,9
DAP	MEAN	79.5	79.7	80.5	82.3	83.1	77,7	76,9
	SD	15.1	14.9	14.9	18.1	18.1	17,5	18,5
MAP	MEAN	94.4	98.5	97.0	96.6	97.0	93,8	89,7
	SD	12.6	13.7	13.9	13.7	15.2	14,6	14,7
SatO2	MEAN	95.4	95.4	95.6	95.6	95.8	96,1	95,2
	SD	2.7	1.8	1.4	1.4	2.2	1,5	1,5

HR = heart rate (bpm – beats per minute); f = respiratory rate (bpm – breaths per minute); SAP = systolic arterial pressure (mmHg); DAP = diastolic arterial pressure (mmHg); MAP = mean arterial pressure (mmHg); SatO2 = peripheral oxygen saturation (%)

Table 2 - Mean values and standard deviations of the parameters before, after, and during the application of positive pressure with the values described, according to the treatment given.

the greater the level of PEEP applied, the greater the alveolar volume, thus increasing transpulmonary pressure at the base of the lungs, and also greater the intra-alveolar pressure, which will take on, at the end of the expiration, the level of the PEEP used.[2]

Therefore, the application of PEEP at levels lower than 10 cmH_20 shows a linear increase in alveolar diameter, which improves gas exchanges due to the increase in RFC and in the surface where gas exchange takes place - between the alveolar epithelium and the capillary endothelium.[21]

Furthermore, the application of positive pressure reduces inspiratory resistance and increases the mean airway pressure. For this reason, there is a significant reduction in respiratory muscle overload and inhibition of the sympathetic response to these muscles.[14, 18, 22, 23]

Moreover, CHF patients have to deal with increased respiratory rates due to stimulation of the atrial chamber, as well as an increase of the afferent impulse of the receptors in pulmonary vessels and stimulation of the J mechanoreceptors, which result in rapid and shallow breathing. For this reason, the sympathetic nervous system is activated leading to an increase in HR in order to maintain an adequate CO. [13, 22, 24]

The application of noninvasive mechanical ventilation with positive pressure in patients with cardiac abnormalities increases pleural pressure, thus increasing intrathoracic pressure, and resulting in inhibition of the autonomous sympathetic nervous system.[10, 20, 22, 25]

In our study, patients who received noninvasive mechanical ventilation (NIMV) showed a statistically significant reduction in the respiratory rate (p=0.022, T test) at the moment immediately before the application of the NIMV mask, and after it had been applied, with an initial EPAP of 5 cmH_2O (Table 2). No statistically significant difference was observed at any other time point in this parameter, which indicates that there was a reduction in the respiratory work after 5 minutes of mask use, and that this reduction was sustained throughout the protocol (Figure 1).

According to the findings above, there was a contribution towards the increase in peripheral oxygen saturation.

In this study, a statistically significant increase was observed in peripheral oxygen saturation (p=0.05, T test) between the final EPAP of 5 cmH_2O and 5 minutes after the NIMV mask had been removed.

The application of positive pressure to patients with CHF improved peripheral oxygen saturation due to the increased pulmonary volume and compliance, which redistributed alveolar fluid, improved gas exchange and increased the arterial oxygen reserve[3, 14, 26, 27].

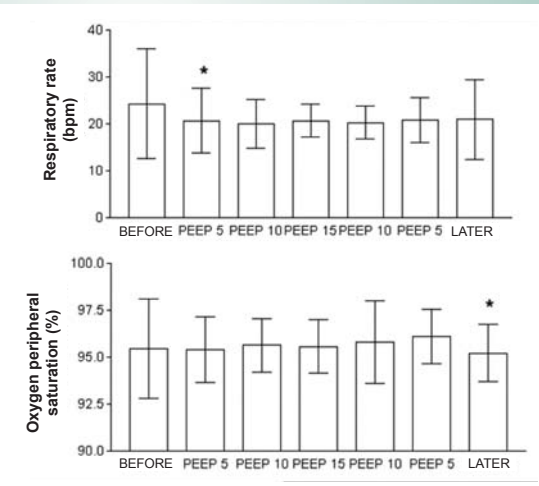

Fig. 1 - *Valores médios de freqüência respiratória (rpm) e saturação periférica de oxigênio (%) (médias e desvios-padrão) dos pacientes em cada momento do atendimento: antes, EPAP5, EPAP 10, EPAP 15, EPAP 10, EPAP 5 e depois. (* p = 0,022 e * p = 0,05, respectivamente).*

According to Barbas et al[4], increased arterial oxygen values lead to vasodilation, a decrease in pulmonary vascular resistance (PVR) and a considerable increase in CO.

According to Bradley et al, the increase in CO and systolic volume results in reduced LV postload due to the reduction in its transmural pressure. All these associated factors significantly improve the LV ejection fraction[22,28].

Cardiac ouput in patients with no cardiac dysfunction is significantly dependent on the preload, since during PEEP application CO is diminished due to the reduction in VR and LV filling. In patients with CHF and LV dysfunction, on the other hand, CO is dependent on postload changes. Thus, a considerable preload increase in the patient with LV dysfunction impairs the cardiac function due to the changes that take place in myocardial tension and length properties, mainly at the left heart chamber[28].

With PEEP application, intrathoracic pressure increases and cardiac output falls significantly, since the compression of the pulmonary artery reduces pulmonary flow and increases PVR. This leads to a reduced systemic venous return, decreasing the ventricular filling due to a significant reduction of CO to the left atrium[6].

According to SANDUR and STOLLER[29], with the decrease in VR, the pressure within the right atrium (RA) raises leading to a fall in the LV systolic transmural pressure, thus reducing the pressure gradient for systemic venous return, with a consequent decline in RV filling and systolic volume[27]. As a consequence, there is an increase in RV postload, reducing the VR to the right side of the heart. However, the reduced venous return to the left side of the heart is a direct consequence of increased intrathoracic pressure, leading to a compression of inferior and superior vena cavas and increasing the PVR, which results in increased pressure of the pulmonary veins and LA preload[5].

The systolic arterial pressure (SAP) may vary due to the direct transmission of the increased intrathoracic pressure to the aorta[30].

During the inspiratory phase, the increase in SAP occurs because of a reduction in LV ejection volume, which, associated with the reduced VR, leads to increased pulmonary blood blow volume, causing a reduction in LV preload. Therefore, the systolic arterial pressure remains high because of the increased intrathoracic pressure[31]. On the other hand, SAP falls during the expiratory phase because of the reduction in aortic blood volume[30].

With PEEP application during mechanical ventilation, there is a reduction in VR due to the increased intrathoracic pressure and the atrial chambers do not distend. Therefore, through the neurohumoral reflex response, renal flow is reduced, leading to antidiuresis and fluid retention[32]. This results in reduced urinary output and sodium excretion, and these effects are greater in individuals with normal or reduced circulatory volume, in an attempt to regulate volemy[33].

The reduction in diuresis associated with reduced atrial distensibility is caused by the atrial myocites, which, in response to increased atrial tension and distension, secrete the atrial natriuretic factor (ANF) that acts on the kidneys to increase urinary flow, sodium excretion, and renal blood flow, thus increasing the glomerular filtration rate. The limited atrial distension causes a negative feedback to this mechanism, reducing the urinary flow in an attempt to increase the VR[34].

The application of positive pressure, however, significantly reduces plasma ANF concentration because of the increased intrathoracic pressure over the pericardium, consequently reducing the pressure of final LV diastolic filling. As a result, the function of the papillary muscles becomes effective and improves mitral valve insufficiency by reducing LV transmural pressure. Additionally, by reducing the activity of the sympathetic autonomous nervous system, associated with coronary vasoconstriction, it also reduces myocardial ischemia[34].

When the atrium is overdistended, it sends out signals to the central nervous system through mechanoreceptors in order to inhibit the release of the antidiuretic hormone (ADH)[35].

With an extreme loss of body volume, volume receptors in the kidneys are activated. These receptors are located in the smooth muscle cells of the afferent and efferent arterioles in the renal glomerule, and are innervated by sympathetic nerve fibers, where they synthesize, store and release the rennin hormone, a large part of which travels and circulates throughout the bloodstream[36].

When the rennin synthesis process is over, the hormone produces angiotensin I, which, through the angiotensin-converting enzyme (ACE), is converted into angiotensin II. Its functions are systemic vasoconstriction and arterial pressure increase, since it constricts the systemic veins, promoting greater venous return to the heart and increasing systemic vascular resistance. As a consequence, there is an increase in the intensity of myocardial contraction, that is, an increase in blood volume ejected by the LV[36].

However, when the arterial pressure falls to abnormally low levels, the renin-angiotensin-aldosterone system is stimulated by intrinsic kidney reactions, thus regulating the blood volume in the body[37].

Therefore, the primary factor responsible for the changes in systemic arterial pressure (SAP) is associated with increased intrathoracic pressure or ventricular interdependence. However, the secondary factor is associated with changes in LV ejection, as well as LV systolic and end-diastolic volumes[30,38].

The changes in LV ejection and filling volumes are caused mainly by increased intrathoracic pressure on the myocardium, leading to changes in LV shape and size, particularly because of the changes in the right cardiac chamber that result in an important displacement of the intraventricular septum. The increased intrathoracic pressure from positive pressure ventilation causes a reduction in LV transmural pressure and VR, and an increase in the alveolar volume, thus elevating PVR. These factors result in an increased postload and overburden of the RV. The overload on the RV causes the intraventricular septum to shift to the left, changing the shape of the LV[8].

According to Pinsky et al, the elevation in CO in patients with CHF is basically caused by increased diastolic pressure and LV systolic volume during application of PEEP[39].

CO in patients with CHF undergoes a relative modification, with changes and reduction in the cardiac function. However, VR remains stable, consequently worsening the insufficiency of the left heart chamber[40].

The positive pressure applied to patients with CHF causes a significant reduction in LV transmural pressure (myocardial systolic force generation index) and in HR (oxygen consumption), associated with a drop in respiratory rate (f), resulting in reduced muscle and myocardial energy consumption. This translates into a significant improvement in the redistribution of blood flow throughout the organs, subendocardial perfusion, and LV diastolic filling. Therefore, due to increased intrathoracic pressure, LV postload decreases and results in an inspiratory muscle overload reduction[22].

In this study, no statistically significant difference was observed regarding noninvasive hemodynamic alterations (heart rate, systolic arterial pressure, diastolic arterial pressure, and mean arterial pressure).

This may be because patients in this study had other cardiac abnormalities besides CHF which may have interfered with the hemodynamic response triggered by the use of noninvasive positive pressure mechanical ventilation, when compared to reports of changes observed in other studies.

Kiely et al[41], who studied patients with CHF and sinus rhythm comparing them with those who had atrial fibrillation, observed that during the application of a CPAP of 0 (ZEEP), 5 cmH_2O, and 10 cmH_2O, patients with sinus rhythm had a reduction in their left ventricular transmural pressure followed by a reduction in postload. This resulted in an increase of the systolic volume and, consequently, an increased cardiac rate. On the other hand, patients with atrial fibrillation (patients 7, 8, 9, and 10 in our study) experienced a reduction in their heart rate with the application of CPAP, resulting in changes in systolic volume index and no alterations in the heart rate. Moreover, ventricular diastolic filling in patients with sinus rhythm is significantly increased by atrial contraction. However, patients with atrial fibrillation do not show the same response since CPAP had a deleterious effect on ventricular preload[40].

Moreover, the study conducted by Mehta et al[42]., which compared the application of CPAP to patients with ischemic myocardiopathy and patients with dilated cardiomyopathy, reported that the latter (in our study, patients 1, 12, and 13) benefited more from the utilization of CPAP than patients with ischemic myocardiopathy (in our study, patients 3 and 7), because the use of positive pressure reduces left ventricular preload, increases left ventricular end-diastolic volume, and reduces postload in this chamber. This last factor results in reduced sympathetic autonomous nerve system activity and systemic arterial pressure, since in the long run they increase LV ejection fraction. Increase in the ejection fraction is associated with the utilization of CPAP, since this leads to increased intrathoracic pressure which reduces the curve radius of the right ventricle and changes the geometric configuration of the left ventricle by shifting the intraventricular septum. The greater the cardiac dilation and the more compliant the cardiac chambers, the more pronounced this response is. Thus, a pronounced reduction in the end left ventricular systolic volume takes place, as well as an increase in the ejection fraction in this cardiac chamber. This suggests that the application of CPAP to patients with dilated cardioyopathy and elevated left ventricular volume, in the short run, counterbalances the trend towards dilation of the left ventricle. This results in a consistent reduction of its postload, improving, in the long run, the ejection fraction from this heart chamber .

Another factor of utmost importance for our findings was the extremely small number of patients. Further studies with more patients need to be conducted in order to identify the best EPAP value that can yield greater benefits to this group of patients.

Conclusion

Noninvasive bilevel positive pressure mechanical ventilation in the treatment of patients with congestive heart failure is commonly used in clinical practice. It has been widely demonstrated that the use of positive pressure, by increasing pulmonary volume and lung compliance, promotes increased residual functional capacity, lower inspiratory resistance, and increased mean airway pressure, resulting in a significant reduction in respiratory work. Patients with CHF associated with other diseases, such as atrial fibrillation, dilated cardiomyopathy, and ischemic cardiomyopathy who received noninvasive positive pressure mechanical ventilation, experienced considerable hemodynamic changes. In our study, however, these were not statistically significant, since there was an increase in transmural pressure directly reflected in the myocardium, and changes of pleural pressure due to the increased intrathoracic pressure. Therefore, further studies with larger numbers of patients should be conducted in order to define an EPAP level that can yield greater benefits to this group of patients.

References

1. Nasi L, Torres G, Manfroi W. Interação coração-pulmão em pacientes críticos: aplicação da ventilação mecânica como terapêutica não farmacológica na disfunção ventricular. Revista HCPA 1999; 19: 382-7.
2. Azeredo CAC. Fisioterapia respiratória no Hospital Geral. São Paulo: Manole; 2000.
3. Lenique F, Habis M, Lofaso F, Dubois-Randé JL, Harf A, Brochard L. Ventilatory and hemodynamic effects of continuous positive airway pressure in left heart failure. Am J Respir Crit Care Med. 1997; 155: 500-5.
4. Barbas CSV, Bueno MAS, Amato MBP, Hoelz C, Rodrigues Jr M. Interação cardiopulmonar durante a ventilação mecânica. Rev Soc Cardiol Estado de São Paulo. 1998; 8: 406-19.
5. Culver BH, Marini JJ, Butler J. Lung volume and pleural pressure effects on ventricular function. J Appl Physiol. 1981; 50: 630-5.
6. Bersten AD, Holt AW, Vedig AE, Skowronski GA, Baggoley CJ. Treatment of severe cardiogenic pulmonary edema with continuous positive pressure delivered by face mask. N Engl J Med. 1991; 325: 1825-30.

7. Bowie RA, O´Connor PJ, Hardman JG, Mahajan RP. The effect of continuous positive on cerebral blood flow velocity in awake volunteers. Anesth Analg. 2001; 92: 415-7.

8. Iliceto S, Dambrosio M, Sorino M, Dambrosio G, Amico A, Fiore T, et al. Effects of acute intrathoracic pressure changes on left ventricular geometry and filling. Am Heart J. 1988; 116: 455-64.

9. Acosta B, DiBenedetto R, Rahimi A, Acosta MF, Cuadra O, Nguyen AV, et al. Hemodynamic effects of noninvasive bilevel positive airway pressure on patients with chronic congestive heart failure with systolic dysfunction. Chest. 2000; 118: 1004-9.

10. Fellahi JL, Valtier B, Beauchet A, Bourdarias JP, Jardin F. Does positive end-expiratory pressure ventilation improve left ventricular function? Chest. 1998; 114: 556-62.

11. Räsänen J, Väisänen IT, Heikkila J, Nikki P. Acute myocardial infarction complicated by left ventricular dysfunction and respiratory failure. Chest. 1985; 82: 158-62.

12. De Hoyos A, Liu PP, Benard DC, Bradley TD. Haemodynamic effects of continuous positive airway pressure in humans with normal and impaired left ventricular function. Clin Sci (Lond).1995; 88: 173-8.

13. Naughton MT, Floras JS, Rahman MA, Jamal M, Bradley TD. Respiratory correlates of muscles sympathetic nerve activity in heart failure. Clin Sci (Lond). 1998; 95: 277-85.

14. Arena R, Myers J, Aslam SS, Varughese Eb, Peberdy MA. Technical considerations related to the minute ventilation/carbon dioxide output slope in patients with heart failure. Chest. 2003; 124: 720-7.

15. Gehlbach BK, Geppert E. The pulmonary manifestations of left heart failure. Chest. 2004; 125: 669-82.

16. Manning HL, Schwartzstein RM. Pathophysiology of dyspnea. N Engl J Med. 1995; 333: 1547-53.

17. Yan AT, Bradley TD, Liu PP. The roles of continuous positive airway pressure in the treatment of congestive heart failure. Chest. 2001; 120: 1675-85.

18. Khayat RN, Xic A, Patel AK, Kaminski A, Skatrud JB. Cardiorespiratory effects of added dead space in patients with heart failure and central sleep apnea. Chest. 2003; 123: 1551-60.

19. Meyer EC, Lorenzi Filho G, Schettino GPP, Carvalho RR. Ventilação não-invasiva no cardiopata grave. Rev Soc Cardiol Estado de São Paulo 1998; 3: 420-7.

20. Perel A, Williamson DC, Modell JH. Effectiveness of CPAP by mask for pulmonary edema associated with hypercarbia. Intensive Care Med. 1983; 9: 17-9.

21. Auler Junior JOC, Amaral G. Assistência ventilatória mecânica. São Paulo: Atheneu; 1998.

22. Naughton MT, Rahman A, Hara K, Flora JS, Bradley D. Effect of continuous positive airway pressure on intrathoracic and left ventricular transmural pressure in patients with congestive heart failure. Circulation. 1995; 91: 725-31.

23. Shuster S, Erbel R, Weilemann LS, Lu W, Henkel B, Wellek S, et al. Hemodynamics during PEEP ventilation in patients with severe left ventricular failure studied by transesophageal echocardiography. Chest. 1990; 97: 1181-9.

24. Krachman SL, D´Alonzo Gilbert, Berger TJ, Eisen HJ. Comparison of oxygen therapy with nasal continuous positive airway pressure on cheyne-stokes respiration during sleep in congestive heart failure. Chest. 1999; 116: 1550-7.

25. Mehta S, Jay GD, Woolard RH, Hipona RA, Connolly EM, Cimini DM, et al. Randomized, prospective trial of bilevel versus continuous positive airway pressure in acute pulmonary edema. Crit Care Med. 1997; 25: 620-8.

26. Krachman SL, Crocetti J, Berger TJ, Chatila W, Eisen HJ, D´Alonzo GE. Effects of nasal continuous positive airway pressure on oxygen body stores in patients with cheyne-stokes respiration and congestive heart failure. Chest. 2003; 123: 59-66.

27. Park M, Lorenzi Filho G, Feltrim MI, Viecili PRN, Sangean MC, Volpe M, et al. Oxigenoterapia, pressão positiva contínua em vias aéreas ou ventilação não invasiva em dois níveis de pressão no tratamento de edema agudo de pulmão cardiogênico. Arq Bras Cardiol. 2001; 76: 221-5.

28. Bradley TD, Holloway RM, McLaughlin PR, Ross BL, Walters J, Liu PP. Cardiac output response to continuous positive airway pressure in congestive heart failure. Am Rev Respir Dis. 1992; 145: 377-82.

29. Sandur S, Stoller JK. Pulmonary complications of mechanical ventilation. Clin Chest Med. 1999; 20: 223-47.

30. Denault AY, Gasior TA, Gorcsan J, Mandarino WA, Deneault LG, Pinsky RM. Determinants of aortic pressure variation during positive-pressure venlilation in man. Chest. 1999; 116: 176-86.

31. Morélot-Panzini C, Lefort Y, Derenne JP, Similowski T. Simplified method to measure respiratory-related changes in arterial pulse pressure in patients receiving mechanical ventilation. Chest. 2003; 124: 665-70.

32. Farge D, Coussaye JEDL, Beloucif S, Fratacci MD, Payen DM. Interactions between hemodynamic and hormonal modifications during PEE-induced antidiuresis and antinatriuresis. Chest. 1995; 107: 1095-100.

33. Beuret P, Feihl F, Nussberger J, Brunner HR, Perret C. Volemic: status influences the response of plasma atrial natriuretic factor to positive airway pressure. Chest. 1996; 109: 1313-9.

34. Tkacova R, Liu PP, Naughton MT, Bradley TD. Effect of continuous positive airway pressure on mitral regurgitant fraction and atrial natriuretic peptide in patients with heart failure. J Am Coll Cardiol. 1997; 30: 739-45.

35. Auler Junior JOC, Carmona MJC, Barbas CV, Saldiva PHN, Malbouisson LMS. The effects of positive end-expiratory pressure on respiratory system mechanics and hemodynamics in postoperative cardiac surgery patients. Braz J Med Res. 2000; 33: 31-42.

36. Berne RM, Levy MN, Koeppen MB, Stanton BA. Fisiologia. 45a ed. Rio de Janeiro: Guanabara Koogan; 2000.

37. Guyton A. Tratado de fisiologia médica. 8a ed. Rio de Janeiro: Guanabara Koogan; 1992.

38. Vieillard-Baron A, Chergui K, Augarde R, Prin S, Page B, Beauchet A, et al. Cyclic changes in arterial pulse during respiratory support revisited by doppler echocardiography. Am J Respir Crit Care Med. 2003; 168: 671-6.

39. Pinsky MR, Matuschak GM, Klain M. Determinants of cardiac augmentation by elevations in intrathoracic pressure. J Appl Physiol. 1985; 58: 1189-98.

40. Baratz DM, Westbrook PR, Shah PK, Mohsenifar Z. Effect of nasal continuous positive airway pressure on cardiac output and oxigen delivery in patients with congestive heart failure. Chest. 1992; 102: 1397-401.

41. Kiely JL, Deegan P, Buckley A, Shiels P, Maurer B, McNicholas W T. Efficacy of nasal continuous positive airway pressure therapy in chronic heart failure: importance of underlying cardiac rhythm. Thorax. 1998; 53: 957-62.

42. Mehta S, Liu PP, Fitzgerald FS, Allidina YK, Bradley D. Effects of continuous positive airway pressure on cardiac volumes in patients with ischemic and dilated cardiomyopathy. Am J Respir Crit Care Med. 2000; 161: 128-34.