

Acute Effects of Continuous Positive Airway Pressure on Pulse Pressure in CHF

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Today's reality on non-invasive ventilation (NIV) use has nothing but four key A level indications supported by evidence-based medicine. These, which would be chronic obstructive pulmonary disease (COPD) exacerbation, cardiogenic pulmonary edema, pulmonary infiltrates in immunocompromised patients, and the weaning of already extubated COPD patients, are the so called "the fabulous four"¹. But is this the maximum therapeutic potential of NIV? Probably not. If so, which would be the next one on this selected "fabulous four" group? Maybe it is stable chronic heart failure (CHF). If so, we would be facing a new frontier, yet unexplored, of those chronically stable not respiratory but cardiac patients, opening new applications, none existent up to today.

Quintão et al.² move on into the next step to conquer this new frontier, the NIV application on stable CHF. They do so, analyzing the NIV (Continuous Positive Airway Pressure - CPAP) effects on pulse pressure (PP), as a risk factor with independent negative predictive value for adverse cardiovascular events, followed by left ventricular dysfunction, especially type II, caused by acute myocardial ischemia. They prove not only to affect PP reduction, but also heart rate (HR), mean arterial pressure (MAP), systolic blood pressure (SBP) and respiratory rate (RR).

The results will be explained through the relationship between positive pressure ventilation effects³ on the cardiorespiratory system. In the left heart, pulmonary vein compression followed to translung pressure increases, improving venous return and so the preload. In addition, this translung pressure increase contributes to squeeze the heart chambers and discharge them, this "dUp"⁴ effect increases

the stroke volume (SV) and improves left systolic output. The afterload reduction is secondary to the systemic vasodilatation effect as a response to intrathoracic pressure elevation. As a final result, HR, MAP, SAP and PP decrease, protecting myocardial oxygenation and reducing the myocardial infarction risk. In the right heart, translung pressure reduces preload secondary to the vena cava squeeze and elevates afterload³ by the increase in pulmonary vascular resistances. As a result a "dDown"⁴ effect and right SV reduction occur, reducing vascular congestion and lung edema, and once again improving oxygenation and ventilation. Regarding respiratory effects, there will be direct oxygenation by O₂ administration and also the alveolar recruitment effect. As a final result, PaO₂ and mixed venous oxygen (SVO₂) raise, and RR and HR decrease.

In a study by Quintão et al.², hemodynamic monitoring was not continuous, but manually measured (sphygmomanometer); thus, a continuous monitoring might offer more accurate and precise data. Actually additional monitoring with echocardiography will allow to expand data, calculate ejection fraction and SV, which will allow to establish the relationship between PP reduction and ventricular output improvement. The trial lasted 30 minutes, enough to confirm the hypothesis, but a longer time will allow maximum effect assessment to possibly define the best CPAP potential on this matter. Finally, although a CPAP pressure of 6 cm H₂O is in fact the usual level used in those studies, a bigger pressure of 8 cm H₂O will probably have a bigger effect, as we usually see in everyday work.

Keywords

Heart Failure; Continuous Positive Airway Pressure; Pulse.

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Reply Letter to Editor

I am very thankful to Dr. Blanco for his comments regarding our study. Non-invasive ventilation (NIV) has been our focus of study, especially with chronic heart failure (CHF), thus, an opportunity to discuss it is always welcome. In our experience, we have observed benefits with the use of NIV with lower CPAP levels to exercise tolerance in CHF patients¹. Other authors^{2,3} have published studies before showing hemodynamic effects with lower CPAP levels as adequate and safer. This gave us an incentive to use that form, since our patients were stable and, therefore, no upper levels were necessary, which could be uncomfortable, and, consequently, promote increase of some parameters. Thus, we observed in a scale of 3-6 cm H₂O the greatest CPAP pressure, which showed a decrease in hemodynamics parameters with the least discomfort sensation, as in other studies⁴. The hemodynamics parameters were measured in periods, but not continuously. In fact, in previous studies they were measured continuously in many forms, by use of catheterization or echocardiography, and others, in a

similar form to ours^{5,6}. Our group has studied hemodynamic parameters beat to beat and will publish the results soon.

In our CPAP experience in CHF, we also observed that the main hemodynamic changes occur between 10 and 20 minutes, and, after that, there is very few or no significant difference from baseline. Furthermore, 30-minute protocols for CPAP have proven to be enough to provide satisfactory results, even in patients with CHF exacerbation⁷. Patients with CHF undergo many phases of both functional and respiratory worsening in the course of their illness. Non-invasive ventilation with CPAP may be a method available to improve quality of life. Our group has also used CPAP as a non-pharmacological resource for the relief of dyspnea to reduce any minimum hemodynamic load caused by the mechanism of that syndrome. In HF outpatient context, we used that device as a complementary treatment for HF. Our future results will demonstrate the magnitude of the use of this non-pharmacological resource in different hemodynamic variables and the benefits to the quality of life of patients with HF.

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