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Short Communication

Advances in Physiological Research: Consideration on Arterial Hypercapnia in Acute Cardiogenic Pulmonary Edema (ACPE)

Andrea Bellone*

Department of Emergency, ASST Grand Metropolitan Hospital Niguarda, Milano, Italy

Abstract

The causes of hypercapnia in Acute Cardiogenic Pulmonary Edema are still unknown. Our hypothesis recognizes an alteration of the ventilation-perfusion ratio in the apical areas of the lung as the major cause. The redistribution of blood to the apical lung zone in hypertensive pulmonary circulation determines an excess of perfusion compared to ventilation and consequently hypercapnia. Our suggestion might be in favor of Bilevel-PAP over Continuous Positive Airway Pressure (CPAP) as the first line of therapy.

More Information

***Address for correspondence:** Andrea Bellone, Department of Emergency, ASST Grand Metropolitan Hospital Niguarda, Milano, Italy,
Email: andreabellone@libero.it

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The majority of patients who enter the emergency room with acute respiratory failure secondary to Acute Cardiogenic Pulmonary Edema (ACPE) show a blood gas analysis characterized by the presence of hypoxemia (always) and hypercapnia (almost always) [1-5].

Patients with ACPE complain of dyspnea, and tachypnea, and are diaphoretic with pulmonary rales. A percentage greater than 50% - 60% are patients with preserved systolic function and usually secondary to a sudden increase in systemic blood pressure values in concentric hypertrophy of the left ventricle, in other cases the patients are affected by severe-moderate systolic dysfunction associated with the classic signs of venous congestion, jugular turgor, hepato-jugular reflux, and sloping edema [6].

The cause of hypoxemia is known to be secondary to the shunt effect. The interstitial-alveolar exudate occupies almost the entire alveolar compartment, thus creating an obstacle to gas exchange. In this case, a significant portion of non-arterialized blood causes hypoxemia, poorly responsive to oxygen (0_2)-therapy. Even by increasing the inspiratory fraction by 0_2, an optimal arterial oxygen tension ($Pa0_2$) value is not obtained.

Conversely, it is necessary to introduce positive pressure into the airways, both continuously throughout the respiratory cycle (CPAP) and in Bilevel mode (Bilevel- Positive Airway Pressure (BiPAP) [7].

The most important effects of positive pressure can be summarized in:

a) **Hemodynamics:** Reduction of right ventricular pre-load with relative improvement in the performance of the left ventricle, and reduction of transmural pressure of the left ventricle with an increase in systolic volume,

b) **Mechanical:** Increase in Functional Residual Capacity (FRC), reduction in Respiratory Rate (RR), reduction in physiological dead space,

c) **Neuromediated:** Reset of the respiratory centers

d) **Gas exchange:** Alveolar recruitment with reduction of the shunt rate.

Conversely, the cause of hypercapnia during EPAC has never been widely studied.

In the literature [8], the most widespread theory was that hypercapnia could be secondary to an overload imposed on the inspiratory muscles linked to the shallow breathing type ventilatory pattern adopted by patients with ACPE. The persistence of tachypnea would have led to progressive exhaustion of the inspiratory muscles, in particular the diaphragm, with consequent alveolar hypoventilation and relative hypercapnia.

But this theory does not hold up, it could be so in the presence of a picture of alveolar hyperinflation and relative

shortening and lowering of the muscle fibers of the diaphragm typical of pulmonary emphysema. Only in this case is it possible that the inspiratory muscles, already mechanically disadvantaged, may undergo muscle exhaustion as when in the course of an exacerbated COPD the persistence of tachypnea is a cause of hypercapnia due to a reduction in alveolar ventilation. This is why Bilevel Positive Airway Pressure (PAP) is mandatory in these patients, especially for supporting pressure support on the inspiratory muscles. Therefore, in the presence of ACPE and comorbidities such as COPD or obesity-related hypoventilation, the physiological conditions and also the therapeutical approach could be different.

In a previous study, Bellone and colleagues [9] randomized 36 patients with ACPE and hypercapnia to NIV (15/5 cm H_2O) or CPAP (10 cm H_2O). All two modalities of ventilation were shown to be as safe as effective, suggesting that the mechanism responsible for the improvement in hypercapnic patients with ACPE is more likely related to the application of positive airway pressure and not pressure support.

In my opinion, the cause of hypercapnia in ACPE has another explanation:

1) We are in the presence (ACPE) of a serious picture of diffuse alveolar congestion with an imbibed lung, a lung with low functional compliance of a "restrictive" type,

2) Ventilatory pattern is characterized by rapid and shallow breaths,

3) The tidal volume of these patients occurs below the FRC, therefore in areas of the lung where the intrapleural pressure exceeds the alveolar pressure, therefore ventilation is distributed mainly in the apical portions of the lung (Figure 1) [10],

4) Since we have a significant increase in left atrial pressure and consequent significant redistribution of pulmonary blood flow towards the apices of the lung,

5) In addition, we must also remember the effects of gravity in patients who are forced into a semi-supine position and take rapid, shallow breaths,

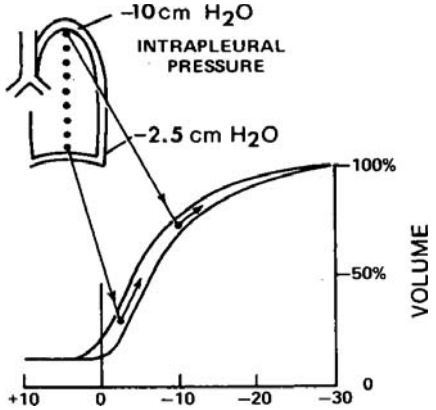

Figure 1: Effect of regional differences of intrapleural pressure on the distribution of ventilation (from West) [10].

6) The lung apexes become the site of gas exchange where an excess of perfusion in a hypertensive pulmonary circulation occurs in relation to ventilation. The result is the reduction of the ventilation-perfusion ratio due to excess perfusion and hypercapnia.

Conclusion

The causes of hypoxemia during ACPE are widely explained but not so for hypercapnia. Our effort was to understand the mechanisms responsible for hypercapnia during ACPE. In summary, hypercapnia during EPAC is secondary to a ventilation-perfusion mismatch while hypoxemia has a dual cause: primarily a shunt effect as well as a ventilation-perfusion mismatch. This suggestion might be in favor of Bilevel-PAP over CPAP as the first line of therapy to accelerate the resolution of pulmonary edema.

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