Why I Ventilate a Patient Non Invasively

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We will start by answering a simple question. What characterizes the acute respiratory failure that best responds to non invasive ventilation (NIV)? Certainly, as shown in Fig. 1.1, one characteristic is the presence of hypercapnia and, therefore, an impairment in the respiratory pump, which comprises the central nervous system, peripheral nerves, and the respiratory muscles.

A pump deficit always leads to hypercapnia and, when not compensated, to acidosis through the mechanism of alveolar hypoventilation. This is described as the condition in which the tidal volume of gas that enters and leaves the lungs (minute volume) is no longer sufficient to meet the metabolic requirements of the body. In contrast to patients with compensated or so-called chronic respiratory acidosis, hypercapnic individuals cannot achieve a balance between the metabolic production of CO_2 and its elimination.

In simple words, for the same minute ventilation (respiratory rate $\times$ tidal volume), alveolar ventilation could be completely different. Let's consider the case of patient A, who breathes at a rate of 10 breaths/min with a tidal volume of 500 mL, and that of patient B, who breathes at a rate of 20 breaths/min with a tidal volume of 250 mL. Both have the same minute ventilation (5 L/min), but they have completely different values of alveolar ventilation. If the dead space in the two patients is the same, for example 150 mL, the alveolar ventilation in the former patient is 3.5 L/min (500−150 mL = 350 mL $\times$ 10 breaths/min) while that in the latter is 2 L/min (250−150 mL = 100 mL $\times$ 20 breaths/min). It is this respiratory pattern, characterized by breathing that is rapid (high respiratory rates) and shallow (low tidal volumes), which leads to the development of hypercapnia.

The diagnosis of acute, hypercapnic respiratory failure is based above all on values of PaO_2 (otherwise what respiratory failure would it be?) <60 mmHg in room air and $PaCO_2 > 45$–50 mmHg with a pH < 7.35.

These formal limits do not, however, take into consideration numerous factors, the foremost being the time variable, as well as the way the episode developed and the age of the patient. For example, a $PaCO_2$ of 70 mmHg that develops over several weeks has a different significance from the same value reached in a few hours, so our diagnosis must be based on the pH (or degree of compensation).

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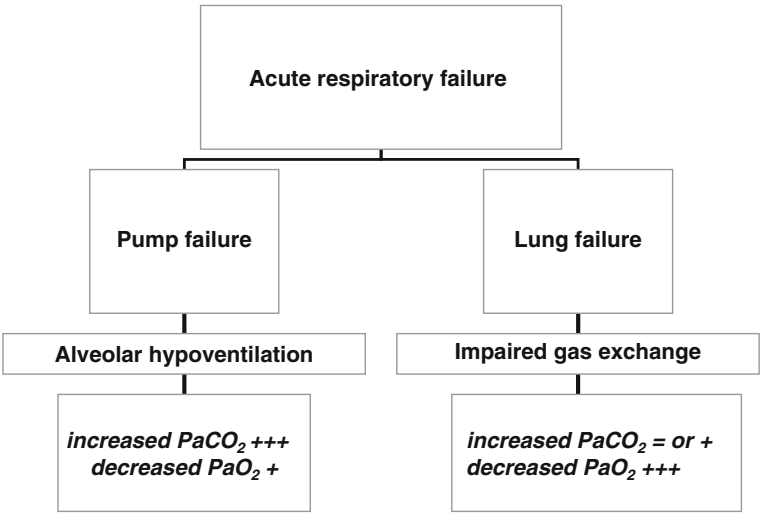

Fig. 1.1 Types of respiratory failure

In most cases, NIV is able to return our patients' blood-gas values to more appropriate, even if not completely normal, levels by correcting the respiratory pattern, that is, by increasing the tidal volume with the help of the ventilator and at the same time reducing the respiratory rate. In patients with chronic obstructive pulmonary disease (COPD), this latter effect also has the result of giving the individuals more time to exhale and, thereby, reduces the degree of dynamic hyperinflation.

The pathophysiology of purely hypoxic respiratory failure is more complex and depends of various factors, not all of which involve the lungs, such as cardiocirculatory failure. However, the most common changes in the so-called respiratory failure of parenchymal origin are those in the ventilation/perfusion ratio, shunt, and diffusion.

The classical definition of acute respiratory failure is based on a $PaO_2/FiO_2 < 300$, with increasing severity as the value of this ratio decreases. In these forms of respiratory failure, NIV is often not as effective as invasive ventilation which is, therefore, preferred as the first-line treatment, at least in some cases, for questions of safety. The reasons why intubation is often used are well-known:

- to protect the airways;
- because of the need for continuous ventilation and, therefore, sedation and sometimes even neuromuscular blockade;
- severe hemodynamic instability;
- use of high fractions of inspired oxygen, which is sometimes not possible with non invasive ventilators.

There are, however, conditions characterized by hypoxic respiratory failure which respond very well to NIV, particularly acute pulmonary edema and pneumonia in immunocompromised subjects (these conditions are discussed later in specific chapters).

Suggested Reading

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