pO₂ – Do We Treat the Right Thing?

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Oxygen is essential for human life. Following the technical advances in the last century, pO₂ was among the first parameters that were relatively easy to measure, which might explain its popularity in critical care and pulmonary medicine to assess oxygen supply [1]. pO₂ appears to be a sensitive and rapidly changing parameter reflecting alterations of the respiratory status. However, according to physiologists, for ATP production and oxygen delivery DO₂ only the oxygen content CaO₂ and perfusion (CO) are relevant: $DO_2 = CaO_2 \times CO$. The oxygen content is only influenced if pO₂ is markedly reduced (below 55 mm Hg), which is explained by the oxygen haemoglobin (Hb) dissociation curve. CaO₂ is proportional to Hb concentration and oxygen saturation (SaO₂), a relationship that is sometimes not given sufficient attention in pulmonary and critical care medicine: $CaO_2 = SaO_2 \times Hb \times 1.34$.

In a recent issue of *Respiration*, Gorecka et al. [2] reported on the use of almitrine in COPD patients with a PAO₂ ranging between 55 and 60 mm Hg. While almitrine improved the pO₂, it remained without effect on the patients' subjective well-being, even after a treatment period of 12 months. This is in line with older studies which failed to show a survival benefit of almitrine therapy [3]. On the other hand, long-term oxygen therapy (LTOT) greatly benefits COPD patients with a pO₂ < 55 mm Hg [4, 5]. One would thus expect that a minor difference in survival would also be visible in patients with less reduced pO₂ (55–60 mm Hg), but this was not the case in a recently

published controlled study [6]. How can this dilemma be solved? In earlier studies on LTOT [4, 5], most patients not only showed a reduction in pO₂, but also an increase in pCO₂ (44–45 mm Hg [4] and 54 mm Hg [5]). Recently published studies on the outcome of patients with respiratory insufficiency and LTOT have shown that the significance of pCO₂ was underestimated [7–9].

If patients were constantly hypercapnic, pCO₂ correlated with a prolonged life expectancy [7, 8]. This effect was even more marked in hypercapnic patients and more isolated restrictive disorders such as sequelae of tuberculosis with emphysema [9].

Oxygen treatment improves pO₂, but reduces minute ventilation, as reflected in an increase in pCO₂. This reduction in minute ventilation equals the reduction in the work of breathing, leading to an unloading of the respiratory muscles. Further unloading by non-invasive ventilation additionally increases the patient's endurance [10]. This may prevent the exacerbation of COPD [11], and may thus explain the prolonged life expectancy under LTOT. The combined effect of increased pO₂ and unloading of the respiratory muscles as an explanation for the improved survival is underestimated in the literature. Almitrine only elevates pO₂ by increasing ventilation. This challenge to the respiratory muscles might be deleterious in the long term. The results of older and newer studies on almitrine do not support its current use in COPD.

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