Use and misuse of oxygen administration

In this issue of the Journal of Postgraduate Medicine, the prospective study carried out by Abhilash et al. investigates the impact of an algorithm on oxygen use in the emergency department.[1] Abhilash et al. should be commended for insightful research that, thanks to the type of inquiry they chose, offers an opportunity for a thorough reappraisal of understanding of oxygen administration in today’s clinical practice.

The protocol and the algorithm they studied before and after implementation highlights how oxygen therapy is commonly administered in hospitals with poor adherence to treatment recommendations, exposing patients to a severe risk of untoward effects.[2] Moreover, this study underscores the possibility of treatment cost reductions without exposing patients to further mortality risks.

The benefits accruing from the use of oxygen have long been known but, at the same time, it has also been recognized to be among the most inappropriately administered drugs. Oxygen (O₂), like any other drug, should only be administered when strictly indicated, and at the appropriate dose. Unmonitored and unrestricted administration can be potentially harmful to patients.[3]

Severe hypoxemia should be immediately treated by achieving high FiO₂ values, but oxygen concentration must be decreased as soon as possible to avoid the risk of hyperoxia. However, despite increasing reports of injury from the inappropriate and protracted use of oxygen, the unrestricted use of oxygen therapy remains entrenched in many clinical settings.[4]

In 2017, the British Thoracic Society (BTS) formulated specific guidelines recommending oxygen use when there is a specific indication and when peripheral saturation (SpO₂) falls below 94%, or below 88% in patients with chronic obstructive pulmonary disease (COPD).[5]

The side-effects of high oxygen doses have been described in COPD, obesity hyperventilation syndrome, and myocardial infarction.[6] Hypercapnic patients with COPD administered oxygen are exposed to the risk of hypoventilation which can lead to apnea because of the depression of the respiratory nerve centers.

A precise risk threshold for oxygen toxicity is unknown because it is impossible to carry out studies among healthy subjects for ethical reasons, but the evidence is clear about the harmfulness of high oxygen concentrations which play a decisive role in the retinopathy of prematurity,[7] respiratory distress syndrome, and bronchopulmonary dysplasia of the premature infant.[8]

Complications of inappropriate oxygen use may be direct as well as indirect.

A high oxygen concentration causes a direct injury to lung tissue with characteristic oxidative stress, oxygen free radical production, and exposes all body organs to risks of cytotoxicity and functional impairment. Oxygen can also be a powerful irritant and potentially exacerbate patients’ respiratory disease.[9]

The damage induced in type I and II pneumocytes (the latter being responsible for the production of surfactant) with the inhibition and inactivation of surfactant has been underestimated and is often ignored. Surfactant deficiency increases alveolar capillary permeability, leading to alveolar collapse. Alveolar surface instability favors atelectasis development. Atelectasis and pneumonia may be considered concomitant because the changes associated with atelectasis may predispose to pneumonia.[10]

Indirect complications are the result of dry and cold oxygen administration in spontaneous breathing. Unwarmed and inappropriately humidified oxygen, delivered in concentrations above 4–5 L/min, damages the mucous membrane of the airways, exacerbating airway dryness and provoking a sensation of pain behind the sternum by inducing the dehydration and hyper-viscosity of bronchial secretions. The consolidation of these secretions makes their elimination difficult with spontaneous coughing as well as with bronchoaspiration in intubated patients (with consequent trauma of the airways and alveolar collapse due to the excessive negative pressure applied during aspiration). Furthermore, it reduces mucociliary clearance, promotes terminal bronchiole occlusion and, thus, the formation of atelectasis supporting a superimposed infection process. We should also bear in mind that oxygen concentrations above 90% is the cause of resorption atelectasis.

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The more important studies of lung injuries induced by oxygen have been carried out in the setting of anesthesia where it has been demonstrated that the administration of oxygen at 100%—even for a brief period of time—is at the origin of atelectasis which rapidly forms when anesthesia is induced and when the patient is extubated.[11]

The existing literature suggests an appropriate use to improve saturation by accepting SpO\textsubscript{2} limits of 92% to 94% to reduce the risks connected with oxygen concentration. Unfortunately, everything is left to personal interpretation of the existing data and to the preference of healthcare providers who tend to rely on the use of higher concentrations for supposed safety reasons and thus underestimate the risk to which patients are exposed.[12]

The administration of oxygen, and both invasive and noninvasive ventilation must be implemented in logical and consequent progression by carefully evaluating the pros and cons of the treatment. The approach must assess whether the patient requires supplemental oxygen or artificial ventilation because the work of breathing and the correlated fatigue have become excessive and unsustainable.

In summary, a cautious reappraisal of the use of oxygen ventilation in clinical settings across medical specialties is needed to secure the formulation of appropriate protocols and monitoring models to use whenever this gas is to be administered. Such a reassessment should also—besides reducing the unwanted side-effects of administration which can impact morbidity and worsen respiratory pathology—help in bringing about a notable reduction in healthcare treatment costs.

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