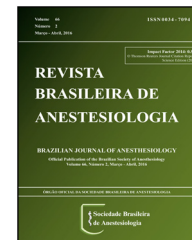




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LETTER TO THE EDITOR

Severe hypoxemia follows hypoxic pulmonary vasoconstriction and/or hypoxic pulmonary vasoconstriction inhibition by inhaled anesthetics: prognostic potential of 100% shunt fractions

Hipoxemia grave após VPH e/ou inibição da VPH por anestésicos inalatórios: potencial prognóstico de frações de shunt de 100%

Dear Editor:

Hypoxic Pulmonary Vasoconstriction (HPV) was first reported by Bindsley et al. in adult patients and was managed using double lumen catheters to ventilate one lung with 100% oxygen and the other with 95% N₂ and 5% oxygen during intravenous barbiturate and fentanyl anesthesia.¹ However, "HPV inhibition" has predominantly been attributed to the use of inhalation agents and is considered as a cause of hypoxia during anesthesia. Moreover, both in vitro and in vivo studies have demonstrated that inhalation agents inhibit HPV under a range of conditions,²⁻⁵ and sevoflurane has been shown to decrease HPV in a dose-dependent manner.^{4,6,7}

Case description

Here we report a case of severe hypoxemia during sevoflurane induction in a 6 year-old boy who was scheduled for adenotonsillectomy. The patient was treated according to American Society of Anesthesiology (ASA) class I and weighed 22 kg. Pre-operative assessments, physical examinations, and laboratory investigations were unremarkable, and the patient had a hemoglobin level of 12 mg/dL and a hematocrit of 36%. Following transfer to the operating room with no pre-medication, routine Electrocardiography (ECG), non-invasive blood pressure, and SpO₂ levels were monitored. Anesthesia was introduced via a facemask and a pediatric circle system providing 8% sevoflurane in 100% oxygen at a

flow rate of 6 L/min. Subsequently, sevoflurane concentrations were reduced to 5% within the first minute and to 2% on loss of eyelash reflexes. Following intravenous cannulation, rocuronium (0.6 mg/kg) was administered, and SpO₂ levels rapidly and progressively decreased from 98% to 38% at 10 and 15 min of induction, respectively, with no clinical explanation. As a consequence, the patients' Heart Rate (HR) suddenly decreased from 109 to 90 bpm in response to hypoxia, and sevoflurane concentrations were reduced to 2% and subsequently discontinued prior to tracheal intubation and ventilation with 100% oxygen. Efficient lung ventilation was possible throughout the period. A clinical improvement was observed within seconds of intubation and ventilation, and SpO₂ levels and HR returned to 100% and 118 bpm, respectively. No blood pressure abnormalities were observed during the procedure, and the rapid restoration of SpO₂ and HR with 100% oxygen was considered symptomatic of an adverse drug reaction. Thus, anesthesia was maintained with 1%–1.5% sevoflurane and oxygen in 50% N₂O.

Discussion

HPV is considered as a protective mechanism that optimizes systemic oxygen delivery. Thus, the inhibition of HPV by inhalational agents is believed to cause hypoxia during anesthesia. However, the protective effects of HPV against hypoxia may depend on the size of affected lung regions. Lung tissues are globally affected by all pulmonary anesthetics, and sevoflurane has been shown to induce pulmonary vessel dilatation in normoxia.⁷ Therefore, HPV should be considered as a multifactorial response to local or global pulmonary hypoxia during acute hypoxia. Thus, the present observations suggest that the degree of acute local shunting can be used to determine whether HPV mechanism is useful or not. The type of anesthetics can be significant with regard to this reaction.

It is widely accepted that inhalation agents have global effects on pulmonary vessels during either induction or maintenance of anesthesia. Thus, hypoxemia because of global HPV is likely in the presence of 100% intrapulmonary shunt fractions, and the protective effects of HPV-influenced perfusion to the better ventilated lung regions to improve oxygenation may be abolished under these conditions. In contrast, inhibition of HPV by inhalation agents in commonly

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affected lungs leads to rapid onset of hypoxemia because of normal effects on perfusion and alveolar hypoxia and direct effects of anesthesia. Thus, the effect of HPV inhibition by inhalation agents or hypoxia may depend on global effects on lungs and the ensuing shunt fractions. Therefore, we suggest that both HPV and HPV inhibition may cause hypoxemia under certain conditions. However, the relationship between pulmonary anesthetic administration and HPV remains controversial.

In conclusion, global effects of inhalation induction with sevoflurane at high concentrations may cause oxygen desaturation in lungs that are normally ventilated with 100% oxygen, potentially resulting in global inhibition of HPV. However, these conditions may be a consequence of global HPV and associated 100% shunt fractions. Thus, despite the high level evidence of no differences in outcomes following pulmonary and intravenous anesthesia, the hypoxic consequences of inhalation anesthesia require further clarification.

Consent

Written informed consent was obtained from the patient for publication of this case report. A copy of the written consent is available for review by the Editor of this journal.

Conflicts interest

The authors declare no conflicts of interest.

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