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Hyperoxia and cardiopulmonary resuscitation outcome: where is the data?

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Association between arterial hyperoxia following resuscitation from cardiac arrest and in-hospital mortality

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Aims of this study

The purpose of this multicenter study was to examine whether delivery of excessive oxygen concentrations (hyperoxia) in the post-resuscitative period to victims of in- and out-of-hospital sudden cardiac death (SCD) with return of spontaneous circulation (ROSC) increased mortality.

Methods

This study was of data derived from the Project IMPACT administrative database. This database was developed by the Society of Critical Care Medicine. Data is collected from more than 130 intensive care units (ICU) in the Unites States. Adult patients who had undergone non-traumatic SCD and were admitted within 24 hours of the event to the ICU in 2001-2005 were included; provided they had undergone arterial blood gas testing (ABG) within the first 24 hours post ICU arrival. The patient variables that were extracted from the database included demographic data, clinical co-morbidities, vital signs, functional status (pre-admission and at hospital discharge where relevant), life support interventions (including delivered oxygen concentrations) and ABGs. Sensitivity analysis utilizing propensity scores was performed in order to examine whether hyperoxia was an independent predictor of in-hospital death.

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Results

Seventy two percent of the patients who met inclusion criteria also had a full dataset available for the analysis (6326/8736). Sixty-six percent of these were living independently prior to the SCD. Patients were classified into three groups based on ABG results; 1156 patients were in the hyperoxia group (PaO2 of 300 mmHg or higher), 1171 were in the normoxia group and 3999 were in the hypoxia group (PaO2 of 60 mmHg or lower). Sixty percent of the patients required treatment with vasopressors during their

first 24 hours in the ICU. The median length of ICU stay was 4 days. The authors reported a mortality rate of 69% in the hyperoxia groups (95% CI, 65%-72%), compared to 57% in the hypoxia group (95% CI, 56%-59%) and 50% in the normoxia group (95% CI, 48%-52%).

Conclusions

The authors concluded that in patients admitted to the ICU after SCD, hyperoxia was associated with a higher mortality than normoxia or hypoxia.

Commentary

This work by Kilgannon and coworkers is the first multicenter study to question the effects of hyperoxia in adults after resuscitation and, as such, is of particular interest. (1) However, inferring that post-resuscitation hyperoxia is detrimental based on this study alone is premature. Retrospective analysis of existing data is always limited by the structure of the database which was not necessarily constructed to address the particular question at hand and this study clearly presents some of the limitations of this method.

One of the inclusion criteria was cardiopulmonary resuscitation within the 24 hours prior to ICU admission; some of the patients may have been admitted a significant time after the resuscitation. The detrimental effect of delayed ICU admission is well documented. (2) Furthermore, patients with delayed ICU admission may have received high oxygen concentrations for several hours before admission; "the first blood gas within 24 hours of ICU admission" provides no information on the duration of hyperoxia. This leads to concern that these patients may have been hyperoxic for almost 24 hours prior to ICU admission. This study could therefore have included patients that were placed on an FiO2 of 100% after intubation, were admitted to the ICU only the next day and were tested for ABG only two days after intubation. The association of free oxygen radical damage with long-term pulmonary problems in the ICU is well known, yet in the present study the element of time was virtually ignored. (3,4)

In addition, arterial blood gas values were not obtained within the first 24 ICU hours in a fourth of the study patients. This raises questions regarding the quality of ICU care. The presence of hyperoxia may reflect a lag in treatment rather than patient response. Needless to say, ICU level of care is a major outcome predictor for critically ill patients. (5)

More patients in the hyperoxia and hypoxia groups were receiving vasopressors. We would like to put forward that hyperoxia may have been provided to "compensate" for hypotension and vice versa. This practice does not represent either standard care or a good medical approach but is, unfortunately, widespread.

Finally, although data regarding both acute and chronic comorbidities is given, no information is provided regarding the actual cause of arrest. The hyperoxia group may have included more cases with primary pulmonary rather than primary cardiac arrest; the former tend to require higher levels of PEEP and have higher peak airway pressures which are damaging unto themselves.

The history of supra-normal oxygen delivery in the ICU suggests that caution should be used when singling out a single variable in a multivariate environment for a cause-effect model. We therefore urge clinicians to avoid immediate extrapolation of the study findings to their clinical practice but rather follow the literature on this subject in the future; we are likely looking at the makings of a new and fascinating debate. In neonatal resuscitation this dispute has been ongoing for more than a decade.

Acknowledgement

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