Pulse Oximetry in Acute Respiratory Failure: What Should Be Expected?

To the Editor:

In the April issue of RESPIRATORY CARE, Amalakanti and Pentakota[1] present a comparison of arterial oxygen saturation values between a 2-wavelength pulse oximeter and blood gas multi-wavelength hemoximetry in COPD subjects with acute respiratory failure. The authors conclude that the 2-wavelength pulse oximeter significantly overestimates arterial oxygen saturation and that COPD and its phenotypes have a causal role. However, this study has a number of shortcomings, which may limit the applicability of the authors' conclusions.

By comparing functional and fractional oxygen saturations, the authors are really comparing green apples with red apples. Two-wavelength pulse oximeters report functional oxygen saturation. Functional oxygen saturation (S_{pO_2} via pulse oximetry) only accounts for oxyhemoglobin and reduced hemoglobin: oxyhemoglobin/(oxyhemoglobin + reduced hemoglobin) × 100. Functional oxygen saturation can also be calculated by blood gases (SaO_2). Multi-wavelength oximeters measure fractional oxygen saturation, which includes carboxyhemoglobin and methemoglobin: oxyhemoglobin/(oxyhemoglobin + reduced hemoglobin + carboxyhemoglobin + methemoglobin) × 100. It is well documented that 2-wavelength pulse oximeters are not reliable in the presence of increased carboxyhemoglobin and methemoglobin.[2,3] The authors concede that not accounting for the effect of carboxyhemoglobin on their results is a shortcoming; however, the methods section states that carboxyhemoglobin was measured, but it was not reported in the study. Had the authors compared functional saturation by the 2 methods, they would have probably seen closer agreement, as shown in Figure 1.[4] To compare oxyhemoglobin between blood hemoximetry and pulse oximetry, a multi-wavelength pulse oximeter is required.[5] It is notable that the mean P_{O_2} the authors reported is what would be expected from the mean S_{pO_2} via pulse oximetry (a slight rightward shift of the oxyhemoglobin dissociation curve due to hypercapnia and presumed acidosis). For the purposes of oxygen titration and the avoidance of hyperoxemia, the data seem to indicate that pulse oximetry performed quite well.

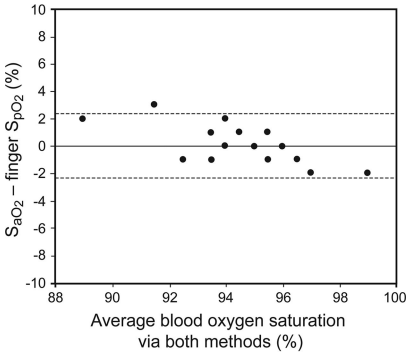

Fig. 1. Bland-Altman analysis of the agreement between functional oxygen saturation values from blood gas analysis and pulse oximetry. From Reference 4.

The authors correctly used Bland-Altman analysis to assess agreement between the 2 methods and conclude that "the pulse oximeter consistently overestimates oxygen saturation." However, the agreement plots show that in many instances, pulse oximetry markedly underestimated oxyhemoglobin ($S_{aO_2} - S_{pO_2} > 0$). The agreement plots also indicate that in some comparisons, the disparity between the 2 methods is quite large ($> -30\%$ and $> +20\%$). These findings are unusual, yet there is no recognition or explanation for these observations in the paper.

The authors propose a causal relationship between COPD (and 2 of its phenotypes) and pulse oximetry inaccuracy. A control group is necessary to make the case for a causal relationship. In this study, a control group (ie, subjects with acute respiratory failure without COPD) is missing, precluded by design a priori. There is a subgroup called "hypoxia" in a table but there is no description of these subjects in the paper. In addition, the use of an $FEV_1/FVC < 0.7$ to define COPD instead of a statistically valid lower limit of normal (eg, Z score −1.64) can be expected to include subjects *without* COPD.[6] Using a binary approach to COPD phenotyping (ie, chronic bronchitis or emphysema) is inappropriate because COPD has several phenotypes.[7]

In our opinion, this study fails to test what can be expected from a 2-wavelength pulse oximeter and by design cannot validate a causal relationship between COPD phenotypes and pulse oximetry inaccuracy.

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The authors have disclosed no conflicts of interest.

DOI: 10.4187/respcare.04965

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Pulse Oximetry in Acute Respiratory Failure: What Should Be Expected?—Reply

In reply:

We thank Haynes and Ruppel for their detailed comments on our work.[1] We also applaud the RESPIRATORY CARE team for maintaining this erudite platform for scholarly discussion.

Regarding the various points raised by Haynes and Ruppel, we were trying to show that pulse oximetry, which is used as a surrogate for arterial blood gas analysis, has limitations. As described in our article, various other researchers have compared the utility of pulse oximetry with arterial blood gas analysis. We were only trying to assess whether green apples are as palatable as red apples.

Given our small sample size, outliers appear important in the Bland-Altman plot (see Fig. 1).[1] We tried to offer a possible explanation for our results in COPD subjects. A study with a control group would, of course, be more conclusive. The hypoxemic group was extracted from the same subjects who had low oxygen by arterial blood gas analysis. This was done to show the performance of pulse oximetry in these cases.

We used this simplistic approach to COPD to find the differences in extremes of the spectrum of the disease. A more detailed study is needed to consider all of the phenotypes. Our study still shows the pitfalls of pulse oximetry in COPD.

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The authors have disclosed no conflicts of interest.

DOI: 10.4187/respcare.05087

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