EDITORIAL

Cerebral oximetry and cardiac arrest: a window still open

Giovanni Landoni[1,2], Antonio Pisano[3]

1 Vita-Salute San Raffaele University, Milan, Italy
2 Department of Anesthesia and Intensive Care, IRCCS San Raffaele Scientific Institute, Milan, Italy
3 Department of Critical Care, Cardiac Anaesthesia and Intensive Care Unit, AORN "Dei Colli," Monaldi Hospital, Naples, Italy

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Correspondence to:
Giovanni Landoni, MD, Department of Anaesthesia and Intensive Care, IRCCS San Raffaele Scientific Institute, 60 Via Olgettina, 20132 Milan, Italy, phone: +39 02 26436154, email: landoni.giovanni@hsr.it

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In Europe, 1.5 to 2.8 per 1000 hospitalized patients per year suffer from in-hospital cardiac arrest (IHCA). Survival or hospital discharge rates at 30 days post-IHCA range from 15% to 34%, with survivors often having poor neurological outcome, especially in the countries not routinely practicing withdrawal of life-sustaining treatment.[1]

Although intensivists, anesthesiologists, and emergency medicine physicians frequently face IHCA in their clinical practice, cardiopulmonary resuscitation (CPR) protocols have remained substantially unchanged for decades, and mainly rely on a very low level of evidence or are not evidence-based. For example, only 1% of the American Heart Association advanced life support recommendations are based on the level of evidence (LOE) A, 17% on LOE B (randomized), and 25% on LOE B (nonrandomized).[2] In particular, apart from some recommendations mostly based on common sense, it is often difficult to predict the chances of achieving return of spontaneous circulation (ROSC) or the neurological outcome in survivors, especially after a prolonged CPR. The decision when to stop CPR, or even when to initiate it, is often arbitrary and with the looming specter of creating severe neurological disability (with all related issues and often only delayed death) rather than saving a life.

In this issue of *Polish Archives of Internal Medicine*, Putowski and colleagues[3] report the findings of an interesting prospective observational study comparing near-infrared spectroscopy (NIRS)-based regional cerebral oxygen saturation (rSO_2) and end-tidal CO_2 ($ETCO_2$) as predictors of both ROSC and neurological outcome in 104 patients with IHCA.

NIRS cerebral oximetry uses NIR light, to which the brain is mostly transparent (except for a few molecules such as hemoglobins, which absorb it), to measure mixed oxygen saturation in a small area of the brain (more or less a square centimeter). As in a pulse oximeter, the measurement of rSO_2 is performed according to the Beer–Lambert law, but in combination with complex technology, such as spatially resolved spectroscopy or others, to estimate the amount of light backscattered toward the receiving optode, that is, the actual pathlength traveled by the NIR light in the brain.[4,5]

In essence, rSO_2 represents a balance between oxygen supply and consumption in a small sample area of the brain. Accordingly, it can be used to monitor the adequacy of global cerebral perfusion / oxygenation and, more extensively, as a hemodynamic monitoring tool. Cerebral oximetry is often used in cardiac surgery (although with a large group of cardiac anesthesiologists who do not use it)[4,6] and in neonatal intensive care units,[4] and its possible role during cardiac arrest is gaining increasing interest.[4]

Several studies already showed outcomes similar to those reported by Putowski et al.[3] In a multicenter investigation of 183 patients with IHCA, mean rSO_2 during the last 5 minutes of CPR was the best predictor of ROSC (area under the curve [AUC], 0.76; 95% CI, 0.69–0.83), with values of 25% or higher showing 100% negative predictive value, and values of 65% or higher showing 93% positive predictive value. Moreover, time with rSO_2 above 50% during CPR was the best predictor of cerebral performance category 1–2 (AUC, 0.79; 95% CI, 0.7–0.88).[7] A similar relationship between rSO_2 and ROSC was found in 329 patients with out-of-hospital cardiac arrest (OHCA), in whom an increase above 15% in rSO_2 was significantly associated with ROSC (odds ratio [OR], 4.5; 95% CI, 2.75–7.41; $P < 0.001$).[8]

More recently, such an association between rSO_2 and both ROSC and neurological outcome after CA was confirmed in the setting of pediatric IHCA[9] and in patients undergoing extracorporeal CPR (eCPR) after either IHCA or OHCA.[10] In a multicenter investigation of 93 patients aged

0.1–1.4 years, higher median rSO_2 values during the whole CPR procedure and both the first and last 5 minutes of CPR increased the likelihood of ROSC, survival to hospital discharge, and more favorable neurological outcome (pediatric cerebral performance category 1–2 or no changes as compared with before arrest).[9] A meta-analysis of 3 observational studies and 245 adult patients showed that precannulation rSO_2 values below 15%–16% in IHCA/OHCA patients undergoing eCPR were associated with increased mortality and poor neurological outcome.[10]

In addition to the association between rSO_2 values during CPR and both ROSC and neurological outcome, Putowski et al[3] also compared rSO_2 with $ETCO_2$, a tool recommended by CPR guidelines, although mostly to confirm tracheal intubation, assess the adequacy of external cardiac massage, and suggest ROSC when it increases abruptly.[1] They also investigated the role of rSO_2 values in the first 24 hours after ROSC in predicting 30-day mortality and poor neurological outcome. While the results of their investigation seem to suggest that $rScO_2$ could be a more sensitive predictor of ROSC after CA than $ETCO_2$, a prospective study of 100 patients with OHCA found a similar AUC for rSO_2 and $ETCO_2$, with better sensitivity for $ETCO_2$ (100%; 95% CI, 87–100 vs 48%; 95% CI, 31–66) and better specificity for rSO_2 (85%; 95% CI, 74–92 vs 45%; 95% CI, 33–57) at the optimal cutoffs identified (20 mm Hg and 50%, respectively).[11] Finally, regarding the association between rSO_2 values in the first hours after ROSC and either neurological outcome or survival to hospital discharge, a multicenter study of 87 IHCA patients[12] yielded similar findings to those by Putowski and colleagues.[3]

An association between rSO_2 values (or changes) during CPR and both ROSC and neurological outcome certainly makes sense (or is probably even intuitive). Moreover, cerebral oximetry has the advantage of not requiring a pulsatile flow, making it particularly suitable during CA or eCPR.[4,13] As compared with $ETCO_2$, rSO_2 has another advantage of not requiring advanced airway management in order to be monitored.[13] Moreover, it could be argued that, while $ETCO_2$ mainly reflects the effectiveness of external cardiac massage, rSO_2 is more widely (and more comprehensively) influenced by the general clinical status of a patient (including the conditions that led to CA) and, accordingly, it could be more informative about the chances of ROSC and the neurological outcome. However, this also makes investigating this relationship, as well as translating the findings of such studies into knowledge useful in clinical practice, very challenging. The main reason for this probably lies in the very nature of rSO_2 which, simply, does not exist (unless you measure it)![14] In other words, rSO_2 is not a physiological parameter, but a measurement invented by the technology used to measure it, and whose values strictly depend even on where it is measured.

Studies such as the one by Putowski et al[3] and the others mentioned above investigate a very complex clinical scenario, with various (and often not reported) causes, with often unknown times between the onset of CA and CPR initiation, and with a lack of information on the maneuvers actually performed and the drugs administered (all of which can heavily influence the outcomes, including ROSC). Moreover, the authors choose rather arbitrary time points at which to record rSO_2 values. Most importantly, different studies use regional oximeters from different manufacturers, whose readings are not comparable with each other.[14,15] For example, a small investigation including 10 cardiac surgery patients found that not only absolute rSO_2 values, but also their variations over time, were significantly different when simultaneously recorded using 2 different oximeters.[15] Those may be the reasons why these investigations found different cutoffs, and even different variations of rSO_2 values during CPR (eg, from 4% in Putowski et al[3] to 15% in Genbrugge et al[8]), which are associated with ROSC or other outcomes.

All these findings are highly intriguing and important, especially in the field where evidence is scant, but we seem to be far from a wide and useful clinical application.

ARTICLE INFORMATION

DISCLAIMER The opinions expressed by the author(s) are not necessarily those of the journal editors, Polish Society of Internal Medicine, or publisher.

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