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Pulse oximetry plethysmography: a new approach for physiology-directed CPR?

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The overarching aim of cardiopulmonary resuscitation (CPR) is to generate sufficient myocardial and cerebral blood flow to allow for survival with favorable neurologic outcome.[1] The adequacy of myocardial and cerebral blood flow during CPR depends, in part, on force of chest compressions, rate of compressions, chest compression fraction, and allowing full chest recoil for sufficient venous return.[2–6] Therefore, the core tenets of CPR are to push hard and push fast, minimize interruptions, and allow full chest recoil.[7] But how hard and fast should we push?

One approach to determine CPR quality is to measure the depth and rate of compressions (i.e., CPR mechanics) in hopes that achieving mechanical targets will provide adequate myocardial and cerebral perfusion. However, variability in chest wall size and compliance and heterogeneity in cardiac arrest etiology and underlying physiology suggest that a "one-size-fits-all" approach may not be optimal.[8] Not surprisingly, translational animal studies show that physiology-directed CPR titrated to coronary perfusion pressure (CoPP), blood pressure, or end-tidal carbon dioxide (ETCO2) can improve myocardial and cerebral blood flow and result in superior survival rates and neurologic outcomes.[9–13] Clinical studies confirm that higher CoPP, arterial diastolic blood pressures (DBP), and ETCO2 during CPR are associated with survival outcomes, thus supporting the premise of physiology-directed CPR.[1, 14–16] However, measurement of CoPP or DBP requires invasive hemodynamic monitoring, and ETCO2 measurement is most reliable in patients who are tracheally intubated. These limitations of established physiologic indicators of CPR quality provide the impetus for evaluating additional, non-invasive tools that can be used when CoPP, DBP, or ETCO2 are unavailable.

Pulse oximeters are widely used clinical monitors that are commonly applied to patients both in and out of the hospital to measure the oxygenation of peripheral blood. Independent of pulse oximetry's utility for determining oxygen saturation, the pulse oximetry plethysmography (POP) waveform generated by measuring pulsatile blood flow

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has clinical utility. For example, the POP waveform may be useful to assess vasomotor tone,[17] volume status[18, 19] and blood pressure.[20] Case reports and small series suggest that POP during CPR may provide clinically relevant information.[21–23] Promising animal studies indicate that POP can be used both for monitoring CPR quality[24] and for identification of return of spontaneous circulation (ROSC) during active CPR.[25, 26] In a porcine model, Xu et al. demonstrated correlations of POP amplitude (Amp) and POP area under the curve (AUCp) with higher-quality CPR, CoPP, and ETCO2.[27]

In this issue of *Resuscitation*, Xu and colleagues build upon their prior translational laboratory work with a large clinical study in which POP was evaluated during CPR to assess its utility in discriminating between patients with and without ROSC.[28] They measured POP Amp, AUCp, and ETCO2 in 150 out-of-hospital cardiac arrest (OHCA) patients and 291 in-hospital cardiac arrest (IHCA) patients across 14 teaching hospitals in China. In the 299 patients with evaluable POP and ETCO2 data, they found that those with ROSC had higher Amp, AUCp, and ETCO2 values during CPR than those without ROSC. During the early stage of CPR, POP and ETCO2 had similar abilities in discriminating between patients with and without ROSC. However, ETCO2 performed better than POP in the final two minutes of CPR and over the course of the entire resuscitation event. Additionally, the authors proposed cutoff values for POP Amp and AUCp to predict ROSC.

There are important limitations to the study that must be considered in interpreting and applying its results. Most notably, the investigators excluded 585 patients with conditions anticipated to be associated with extremely poor perfusion or low hemoglobin concentrations. This included patients with rib fractures, hemorrhagic shock, pulmonary embolism, pericardial tamponade, severe anemia, and tension pneumothorax without drainage. It was reasonable to exclude these patients in an initial clinical study to enrich the study cohort into one in which POP's potential benefit could be detected. However, the sum of these conditions was common in the patient population. Importantly, these conditions are not always evident to the rescuers providing CPR, and their association with POP efficacy is unclear. Thus, these exclusion criteria limit the generalizability of the data at present. Future prospective studies should attempt to assess POP's utility in broader OHCA and IHCA populations. Additionally, calculating POP Amp and AUCp requires post-event processing algorithms and analyses that are not immediately available at the bedside, thus limiting its current clinical applicability.

Regardless of these limitations, we commend the authors for this exciting exploratory contribution to the literature. The authors first established the physiologic premise of POP use in animal studies, and they now provide initial clinical data to support its role in assessing CPR quality and predicting outcome. Notably, this technology is ubiquitous, cheap, non-invasive, and easily applied, and therefore has the potential to impact nearly every cardiac arrest victim, including when invasive monitoring data is not available. Use of POP in conjunction with other physiologic indicators of CPR quality may better inform providers about multiple intertwined components of cardiac arrest physiology. Although further study is needed to understand how to interpret and apply this technology in real-time, the use of the pulse oximeter to inform physiology-directed CPR carries great promise.

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