

Is There Too Much “Pulmonary” in Cardiopulmonary Resuscitation?

Cardiopulmonary resuscitation (CPR) can be an effective life-saving technique. Basic life support with chest compressions and rescue breathing is taught in a standard, rigid fashion, as if all cardiac arrests were the same, in part because of practical educational considerations.¹ It is quite likely that optimal CPR varies, depending on the specific underlying and ongoing pathophysiology and pathoanatomy. For example, chest compressions without rescue breathing seem to be the preferred telephone-directed CPR technique for sudden collapse *ventricular fibrillation* (VF) cardiac arrests.^{1,2} In contrast, rescue breathing is critical for resuscitation from a cardiac arrest secondary to *acute asphyxia*.³ Importantly, cardiopulmonary interactions certainly raise substantive issues during the low-flow circulatory state of CPR, as noted by Yannopoulos et al, in this issue of RESPIRATORY CARE.⁴

Readers of this Journal are quite familiar with the potential profound effects of cardiopulmonary interactions. These issues are especially pertinent in certain extreme pathophysiological circumstances, such as severe circulatory shock or ventilation with high mean intrathoracic pressure. High levels of positive end-expiratory pressure can impede venous return and decrease cardiac output and blood pressure. Rapid positive-pressure rescue breathing for severe life-threatening respiratory failure due to asthma can also impede venous return, and can even result in profound shock and death.⁵ During CPR for cardiac arrest in animal models, the cardiac output is quite low, typically 10–20% of the baseline cardiac output (even with excellent continuous, uninterrupted, forceful chest compressions—an ideal circumstance rarely applicable to real-life CPR).^{1,6–9}

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Over the last few years, several investigators have observed that lay basic-life-support students, medical students, and emergency medical services providers cannot provide 2 rescue breaths in 4 seconds during single-rescuer manikin CPR, as recommended by the American Heart Association Guidelines.^{10–13} Instead, each interruption for 2 breaths generally requires 14–16 seconds for the rescuer to move from chest to head, attain a good mouth-to-mouth seal, provide the breaths, and move back from the head to the side of the manikin. As specialists in respiratory care,

we are not surprised by these observations that assisted ventilation is indeed a complex psychomotor task.

Over the last decade there has been increasing interest in chest-compression-only CPR for sudden collapse VF.^{6,7,14–19} During chest-compression-only CPR for VF, the lungs initially serve as a reservoir for exchange of oxygen and CO₂, as blood flows slowly through the pulmonary circulation. In addition, chest-compression-induced gas exchange can occur (ie, air forced out of the chest during the compression phase and allowed into the chest because of elastic recoil during the relaxation phase). Finally, animals and humans often gasp during CPR if it is provided soon after the onset of VF.^{6,7,16,18,20–22} In swine models of VF, approximately 2–5 L/min of mean expiratory minute ventilation has been documented during chest-compression-only CPR (typically 100 compressions/min with 10–40 mL/breath), mostly from chest-compression-induced gas exchange.^{7,16,18,19} This process bears some similarity to high-frequency oscillator ventilation at approximately 2 Hz. Not surprisingly, blood gases after 7 min of chest-compression-only CPR in one swine VF experiment revealed arterial oxygen saturation of 76 ± 6 mm Hg, P_{aCO₂} of 37 ± 5 mm Hg, and arterial pH of 7.41 ± 0.03, compared with arterial oxygen saturation of 92 ± 1 mm Hg, P_{aCO₂} of 25 ± 2 mm Hg, and arterial pH of 7.49 ± 0.02 in the control group treated with chest compressions and assisted ventilation at a ratio of 15:2.⁷ Because the chest-compression-only group had higher microsphere-determined myocardial blood flow, the 2 groups in that study had similar myocardial oxygen delivery. Consequently, the outcomes of both groups of animals were nearly identical, as in many other such studies by our group and others.^{7,19} In contrast to these excellent outcomes with chest-compression-only CPR for VF, physiological variables and outcomes from asphyxia-induced cardiac arrests are far superior with chest compressions plus rescue breathing.³

How do these animal studies relate to the real world of VF cardiac arrests? In Kouwenhoven et al's original 1960 description of closed-chest cardiac massage, 7 of the 20 patients received chest compressions alone, without rescue breathing.²³ Fourteen of the 20 patients treated with this new closed-chest cardiac massage survived to hospital discharge, some of whom had no assisted ventilation. There are also 3 published retrospective observational studies of adult out-of-hospital cardiac arrest victims documenting

similar outcomes if the bystander provided chest-compression-only CPR or chest compressions plus rescue breathing. Bossaert et al observed long-term survival in 71/443 (16%) adults treated with good chest compressions and good rescue breathing, 17/116 (15%) treated with good chest compressions alone, and 123/2055 (6%) who received no bystander CPR.^{24,25} Holmberg et al demonstrated long-term survival in 19/178 (7%) adults treated with chest-compression-only, 176/1,812 (10%) treated with chest compressions and rescue breathing, and 27/620 (4%) of those not receiving bystander CPR.²⁶ Finally, Waalewijn et al reported long-term survival in 6/41 (15%) adults treated with chest-compression-only, 61/437 (14%) treated with chest compressions and rescue breathing, and 26/429 (6%) not treated with any bystander CPR.²⁷ In all 3 observational studies, both bystander treatment groups differed from the no-bystander group, but not from each other.

Furthermore, Hallstrom et al performed a randomized clinical trial comparing telephone-dispatcher-directed CPR with chest-compression-only versus chest compressions and rescue breathing in 520 adult cardiac arrest victims.² Survival to hospital discharge occurred in 35/240 (15%) after chest-compression-only and 29/278 (10%) after chest compressions and rescue breathing ($p = 0.18$). In summary, even when combining all types of adult out-of-hospital arrests together, chest-compression-only resulted in similar outcomes to standard CPR, and was far superior to no-bystander CPR.

Recent observations by Aufderheide et al suggest that "over-ventilation" (ie, assisted ventilation rates of 30–40 breaths/min) is common during CPR for out-of-hospital cardiac arrests.²⁸ Presuming that these high rates could cause circulatory embarrassment, they studied this phenomenon in a swine VF cardiac arrest model and demonstrated that these high ventilation rates were uniformly lethal, whereas standard chest compression and assisted ventilation rates were routinely successful. "Over-ventilation" during CPR is not restricted to the out-of-hospital setting. Abella et al confirmed a previous study by my colleagues at the University of Arizona indicating that assisted ventilation rates were typically about 35 breaths/min for in-hospital arrests.^{8,29} Are we often harming our patients by impeding venous return and cardiac output because of inadvertent "over-ventilation" emanating from resuscitation-induced excitement and anxiety? Unfortunately, the discouraging answer is becoming increasingly clear.

In this issue of the Journal, Yannopoulos et al⁴ further investigate the interesting and important issue of cardiopulmonary interactions during CPR. Specifically, they have established that CPR with a 15:2 compression:ventilation ratio can adversely effect coronary and cerebral perfusion, compared with a 15:1 ratio in a swine VF model. The authors offer the intriguing argument that modest change

in intratracheal pressure (approximately 1 mm Hg) can result in substantial downstream effects on systemic blood pressure during the low-flow state of CPR. They assert that the increase in intrathoracic pressure interferes with venous return, thereby decreasing thoracic pump output and increasing intracranial pressure. Their data are consistent with their assertions.

In conclusion, cardiopulmonary interactions during the low-flow circulatory state of CPR can have profound clinically important implications. This is a fruitful subject for further investigations that may change our present understanding of CPR physiology and may ultimately save lives through changes in our approach to CPR.

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