Hands-Only Cardiopulmonary Resuscitation: Is It Really Dangerous?

In this issue of RESPIRATORY CARE, Lurie and colleagues report a laboratory investigation in which they compared ventilation rates of 10 breaths/min and 2 breaths/min during cardiopulmonary resuscitation (CPR) of swine.[1] This is an important hypothesis-generating study regarding cardiopulmonary interactions during CPR. In their model, their data establish that carotid-artery blood flow and brain tissue oxygen tension were lower with the 2-breaths/min strategy than with the 10-breaths/min strategy. However, they make an unsupportable claim based on that physiological observation, that "it is clear that low or no ventilation during CPR can be dangerous and should not be recommended except in circumstances where untrained CPR providers are not willing to perform mouth-to-mouth."

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Lurie and colleagues speculate that the worse cerebral hemodynamic and tissue oxygenation effects with 2 breaths/min were due to greater atelectasis, resulting in increased pulmonary vascular resistance and decreased transpulmonary flow.[1] These speculations are plausible, but they did not measure lung volumes or pulmonary vascular resistance, so their proposed mechanism for these findings could not be confirmed. Nevertheless, the lower mixed venous oxygen saturation values in the 2-breaths/min group support their contention that cardiac output may have been lower in that group, and that decreased blood flow through the lungs may have resulted in decreased cardiac output and cerebral blood flow. However, the right atrial compression pressures ("systolic" pressures) were substantially lower in the 2-breaths/min groups than in the 10-breaths/min groups (eg, 35.8 ± 12.9 mm Hg vs 54.6 ± 18.9 mm Hg, respectively, in the groups in which they did not use the impedance threshold device [$p = 0.007$]), which suggests that the force of compressions was substantially lower in these 2-breaths/min groups. If the force of compressions was indeed lower, the decreased cardiac output and decreased carotid-artery blood flow would be expected without any need to invoke the other cardiopulmonary interactions postulated by Lurie et al.

Although we applaud Lurie et al for this excellent hypothesis-generating physiological investigation, we are uncomfortable with their assertion that, "When combined with recent data that showed the harmful, if not deadly, effects of hyperventilation during CPR, these results support the conclusion that there is an ideal range of ventilation rate during CPR, and both too many and too few breaths per minute are dangerous. Markedly higher and lower rates result in physiologically detrimental cardiopulmonary and thoraco-cerebral interactions that substantially reduce the effectiveness of CPR." Even if the different blood flows in the 2-breaths/min groups were due to the mechanism postulated by Lurie et al, rather than inadvertent decreases in the force of compressions, differences in physiology do not necessarily indicate that any harm has occurred. Clearly, there is no blood flow to the brain during the "no-flow" state of untreated cardiac arrest, yet patients with many minutes of untreated cardiac arrest can be successfully resuscitated without demonstrable untoward effects. Moreover, though many elegant physiological studies in animals and humans in the 1980s and 1990s demonstrated that high-dose epinephrine (compared to standard-dose epinephrine) improved blood flow during CPR,[2-6] numerous outcome studies established that standard-dose epinephrine was at least as effective as high-dose epinephrine.[7-11] In fact, 2 human studies found better outcomes with standard-dose epinephrine than with high-dose epinephrine.[12,13] Interventions that cause adverse physiologic differences during CPR do not consistently result in harmful effects or differences in outcomes. The swine CPR investigation by Lurie and colleagues[1] provides interesting physiological data, but no outcome data.

What do we know about the lowest CPR ventilation rate, which is no assisted ventilation (also known as "chest-compression-only CPR" and "hands-only CPR")? For more than 2 decades there has been great interest in chest-compression-only CPR, based on exciting animal investigations and limited human experience.[14,15] Physiologic animal data indicated that hands-only CPR resulted in lower arterial oxygen saturation and higher myocardial blood flow than standard CPR, and the resultant mean myocardial oxygen delivery was very similar with either technique.[16,17] However, those animal data assumed that a single rescuer could provide 2 rescue breaths over 4 seconds, as recommended by the American Heart Association at that time. Manikin studies showed that chest compressions are typically interrupted for 14–16 seconds while a lay rescuer or medical student moves from the chest to the head, repositions the head, provides 2 rescue breaths, and returns to the chest to provide compressions.[18,19] Animal

models that incorporated this more realistic time interval of interrupted chest compressions for rescue breaths have established that the physiological and outcome data are substantially better with single-rescuer hands-only CPR for ventricular fibrillation, than with either the 15:2 compression:ventilation standard CPR or the newer 2005 guidelines 30:2 compression:ventilation ratio.[20-22] More importantly, recent additional human outcome data persuaded the American Heart Association's Basic Life Support committee to recommend hands-only bystander CPR.[23-27]

Hallstrom and colleagues found, in a randomized controlled trial, that hands-only CPR was at least as effective as standard chest compressions with rescue breathing in patients with sudden cardiac arrest.[24] When people without CPR training called 911 regarding an apparent cardiac arrest, the telephone dispatcher taught either hands-only CPR or chest compressions plus rescue breathing. Survival to hospital discharge was attained in 35 of 240 (15%) treated with hands-only CPR, versus 29 of 278 (10%) treated with standard CPR. Based on those data and the difficulty of teaching rescue breathing via telephone, the National Academies of Emergency Dispatch and the American Heart Association recommend hands-only CPR rather than standard CPR as the treatment of choice for telephone-directed CPR.[15,28]

Over the last year, 3 large observational human studies showed that patients who received hands-only bystander CPR were at least as likely to survive an out-of-hospital cardiac arrest as were patients who received standard bystander CPR with chest compressions and rescue breathing. Among 4,068 patients with witnessed out-of-hospital cardiac arrests in the SOS-KANTO (Survey of Survivors after out-of-hospital cardiac arrest in the Kanto area) study from Tokyo, 439 (11%) received hands-only bystander CPR, 712 (18%) received conventional CPR with chest compressions and rescue breathing, and 2,917 (72%) received no bystander CPR.[25] The etiology of the arrest was presumed cardiac in 70%, but was not cardiac in 30%. Favorable neurological status at 30 days post-arrest was much more common with any bystander CPR technique than with no bystander CPR (5.0% vs 2.2%, $p < 0.001$), and tended to be more common after hands-only bystander CPR than after conventional CPR (6% vs 4%, unadjusted odds ratio 1.5, 95% confidence interval 0.9–2.5). The adjusted odds ratios among patients with known resuscitation-related time intervals indicated that 30-day favorable neurological outcomes were more common after hands-only bystander CPR than after conventional CPR (odds ratio 2.22, 95% confidence interval 1.17–4.21). Importantly, the group with the greatest opportunity to survive with good outcomes (patients with ventricular fibrillation or ventricular tachycardia as the initial cardiac rhythm) were much more likely to have 30-day favorable neurological outcomes after hands-only CPR than after conventional CPR (19% vs 11%, adjusted odds ratio 8.00, 95% confidence interval 3.48–18.41). Interestingly, only 38% of the bystanders who provided hands-only CPR had previous CPR training, whereas 67% of the bystanders who provided conventional CPR had previous CPR training (ie, less-trained bystanders providing hands-only CPR were more likely to save a life than better-trained bystanders providing conventional CPR).

Two other large series were published in December 2007, which showed comparable outcomes with either hands-only bystander CPR or conventional bystander CPR. Among 4,902 witnessed out-of-hospital cardiac arrests of presumed cardiac etiology in Osaka, Japan, 1-year favorable neurological outcomes were attained in 2.1% with no bystander CPR, 3.5% with hands-only bystander CPR, and 3.6% with conventional bystander CPR.[26] Similarly, among 9,354 patients treated with either hands-only bystander CPR ($n = 1,145$) or conventional bystander CPR ($n = 8,209$) for their out-of-hospital cardiac arrests in the Swedish Cardiac Arrest Registry, the 1-month survival rate was 7% in each group. The patients in the Swedish registry included all cardiac arrests; 70% were presumably due to cardiac etiologies and 30% not cardiac.[27]

Despite the strong statements by Lurie et al[1] about the dangers of CPR with a ventilation rate of 2 breaths/min, there are presently no human or animal outcome data to support that concern. In contrast, there is abundant information that most bystanders do not provide any CPR. Furthermore, the published human outcome data indicate that hands-only bystander CPR and conventional bystander CPR have similar effectiveness in saving lives with favorable neurological outcomes. The American Heart Association now recommends that: (1) untrained bystanders should provide hands-only CPR, (2) trained bystanders who are confident in their ability to provide CPR should provide either conventional CPR or hands-only CPR, and (3) trained bystanders who are not confident in their ability to provide conventional CPR should provide hands-only CPR.[23]

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