Death by hyperventilation: A common and life-threatening problem during cardiopulmonary resuscitation

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Context: **This translational research initiative focused on the physiology of cardiopulmonary resuscitation (CPR) initiated by a clinical observation of consistent hyperventilation by professional rescuers in out-of-hospital cardiac arrest. This observation generated scientific hypotheses that could only ethically be tested in the animal laboratory.**

Objective: **To examine the hypothesis that excessive ventilation rates during performance of CPR by overzealous but well-trained rescue personnel causes a significant decrease in coronary perfusion pressure and an increased likelihood of death.**

Design and Setting: **In the *in vivo* human aspect of the study, we set out to objectively and electronically record rate and duration of ventilation during performance of CPR by trained professional rescue personnel in a prospective clinical trial in intubated, adult patients with out-of-hospital cardiac arrest. In the *in vivo* animal aspect of the study, to simulate the clinically observed hyperventilation, nine pigs in cardiac arrest were ventilated in a random order with 12, 20, or 30 breaths/min, and physiologic variables were assessed. Next, three groups of seven pigs in cardiac arrest were ventilated at 12 breaths/min with 100% oxygen, 30 breaths/min with 100% oxygen, or 30 breaths/min with 5% Co_2/95% oxygen, and survival was assessed.**

Main Outcome Measures: **Ventilation rate and duration in humans; mean intratracheal pressure, coronary perfusion pressure, and survival rates in animals.**

Results: **In 13 consecutive adults (average age, 63 ± 5.8 yrs) receiving CPR (seven men) the average ventilation rate was 30 ± 3.2 breaths/min (range, 15 to 49 breaths/min) and the average duration of each breath was 1.0 ± 0.07 sec. The average percentage of time in which a positive pressure was recorded in the lungs was 47.3 ± 4.3%. No patient survived. In animals treated with 12, 20, and 30 breaths/min, the mean intratracheal pressures and coronary perfusion pressures were 7.1 ± 0.7, 11.6 ± 0.7, 17.5 ± 1.0 mm Hg/min ($p < .0001$) and 23.4 ± 1.0, 19.5 ± 1.8, 16.9 ± 1.8 mm Hg ($p = .03$) with each of the different ventilation rates, respectively ($p =$ comparison of 12 breaths/min vs. 30 breaths/min for mean intratracheal pressure and coronary perfusion pressure). Survival rates were six of seven, one of seven, and one of seven with 12, 30, and 30 + Co_2 breaths/min, respectively ($p = .006$).**

Conclusions: **Despite seemingly adequate training, professional rescuers consistently hyperventilated patients during out-of-hospital CPR. Subsequent hemodynamic and survival studies in pigs demonstrated that excessive ventilation rates significantly decreased coronary perfusion pressures and survival rates, despite supplemental Co_2 to prevent hypocapnia. This translational research initiative demonstrates an inversely proportional relationship between mean intratracheal pressure and coronary perfusion pressure during CPR. Additional education of CPR providers is urgently needed to reduce these newly identified and deadly consequences of hyperventilation during CPR. These findings also have significant implications for interpretation and design of resuscitation research, CPR guidelines, education, the development of biomedical devices, emergency medical services quality assurance, and clinical practice. (Crit Care Med 2004; 32[Suppl.]:S345–S351)**

KEY WORDS: cardiopulmonary resuscitation; sudden death; heart arrest; ventilation; hypotension; hyperventilation; cardiac arrest; emergency medical services; out-of-hospital cardiopulmonary resuscitation; paramedic

With >1,000 victims daily, cardiac arrest remains one of the nation's most significant healthcare issues (1, 2). Despite widespread cardiopulmonary resuscitation (CPR) training, survival rates after cardiac arrest remain dismal for most patients (3–5).

Recent experimental animal data suggest that there may be deleterious effects of excessive rescue breathing, in part because ventilations interrupt chest compression and thereby reduce vital organ perfusion (6–8). We theorized that positive pressure ventilation may also be deleterious because it prohibits the development of negative intrathoracic pressure during chest wall recoil, inhibiting venous blood return to the right heart and thereby decreasing the hemodynamic effectiveness of CPR (9–13).

Paramedics and emergency medical technicians are required to maintain certification and deliver CPR to victims of out-of-hospital cardiac arrest according to national guidelines. At present, the American Heart Association recommends 12–15 breaths/min in patients with secured airways during the performance of CPR by professional rescuers (14).

This investigation consists of two studies: a clinical trial and a porcine animal study. The purpose of the clinical trial (a prospective, observational, case

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series) was to objectively and electronically record actual ventilation frequency and duration and the percentage of time in which a positive pressure was present in the lungs during CPR performed by trained and certified paramedics or emergency medical technicians in humans at the scene of out-of-hospital cardiac arrests. This trial led to the clinical observation that professional rescuers consistently hyperventilated patients during CPR (14a).

Based on these clinical observations, the following hypotheses were generated: 1) excessive ventilation rates inhibit venous blood return to the right heart during CPR, 2) ventilation rates during CPR of >12 breaths/min are inversely proportional to coronary perfusion pressure, and 3) excessive ventilation rates during CPR are associated with decreased survival rates (14a). Due to ethical concerns, these hypotheses were tested in a porcine model of ventricular fibrillation.

OBSERVATIONAL STUDY IN HUMANS

Methods

This study was performed on patients with an out-of-hospital cardiac arrest with an exception from informed consent requirements for emergency research (21 CFR Part 50.24) after community consultation and public notification. The Human Research Review Committee at the Medical College of Wisconsin and seven additional institutional research committees representing 13 receiving hospitals in the Milwaukee area approved the study (14a).

The clinical study was conducted in the two-tiered, urban, City of Milwaukee emergency medical services (EMS) system, described elsewhere (14a, 15). For the present study, noninvasive monitoring of intratracheal pressures in patients undergoing CPR was performed as part of a prospective, randomized, clinical trial designed to evaluate the effects of a new CPR-assist device. Entry criteria for the study were: 1) adult patients (presumed or known to be ≥21 yrs old) believed to be in cardiac arrest of presumed cardiac etiology and 2) patients who were successfully intubated with an endotracheal tube who were undergoing CPR at the time of scene arrival of the research team. The research physician and paramedic carried a portable Propaq (Welch Allyn Protocol, Beaverton, OR) monitoring system for electronic measurement of airway pressures, a surrogate for intrathoracic pressures. At arrival, the research team connected the noninvasive sensor between the endotracheal tube and the bag-valve resuscitator. Ventilations were then continuously recorded until resuscitation attempts were discontinued or the patient was resuscitated. There are a variety of factors that may affect ventilation rate throughout the resuscitation efforts, including the practice of hyperventilating immediately before and after intubation. For this reason, we sought to determine the maximum ventilation rate, defined as the highest ventilation rate recorded during CPR over a 16-sec period occurring at least 2 mins after intubation. The ventilation frequency and duration and percentage of time in which a positive pressure was recorded in the lungs were then calculated using a digital caliper (14a).

In the City of Milwaukee EMS system, both paramedics and emergency medical technicians manage the victim's airway and provide rescue breathing during out-of-hospital treatment for cardiac arrest. All professional rescuers must continuously remain certified in basic life support and demonstrate acceptable performance of CPR to maintain their licensure, and they must also attend CPR refresher courses approximately every 2 yrs. In addition, paramedics and emergency medical technicians were rigorously retrained and demonstrated CPR performance according to American Heart Association guidelines before the onset of the study.

The first seven consecutive cases constitute group 1. After recognizing that rescuers were consistently hyperventilating patients in cardiac arrest, investigators immediately retrained all EMS personnel to provide ventilations at a rate of 12 breaths/min during CPR after establishment of a secured airway. The duration of each ventilation was not addressed during retraining. The subsequent six consecutive cases (after retraining) constitute group 2. Data were also analyzed combining groups 1 and 2 (group 3). Differences between the mean values of groups 1 and 2 were statistically analyzed by analysis of variance. A p value of <.05 was considered statistically significant. All data are expressed as the mean ± SEM (SEM) (14a).

Results

The average age of the 13 consecutive patients (six women, seven men) was 63 ± 5.8 yrs (range, 34–96 yrs). Three patients had ventricular fibrillation, five had pulseless electrical activity, and five had asystole. Overall, the maximum ventilation rate was observed an average of 18.8 ± 11.9 mins after intubation (range, 2–39 mins). No patient survived. Table 1 documents the ventilation rate, ventilation duration, and percentage of time in which a positive pressure was recorded in the lungs in groups 1, 2, and 3 (14a).

Six of seven group 1 patients had ventilation rates of ≥32 breaths/min. The average ventilation rate for group 1 patients was 37 ± 4 breaths/min (range, 19–49 breaths/min). The average ventilation duration was 0.85 ± 0.07 secs/breath, and the average percentage of time in which a positive pressure was recorded in the lungs was 50 ± 4% (14a).

After the retraining of EMS personnel, three of six group 2 patients had ventilation rates of ≥26 breaths/min. The average ventilation rate for these six patients was signifi-

Table 1. Clinical observational study

Consecutive Case	Ventilation Rate (Breaths/Min)	Ventilation Duration (Secs/Breath)	% Positive Pressure
Group 1			
1	32	1.15	61
2	45	0.85	64
3	34	0.91	51
4	49	0.64	52
5	19	0.99	31
6	39	0.60	40
7	38	0.78	49
Mean ± SEM	37 ± 4[a]	0.85 ± 0.07[b]	50 ± 4
Group 2			
8	15	1.10	27
9	31	1.38	71
10	15	1.12	28
11	15	1.00	25
12	26	1.30	57
13	30	1.17	59
Mean ± SEM	22 ± 3[a]	1.18 ± 0.06[b]	44.5 ± 8.2
Group 3	30 ± 3	1.0 ± 0.7	47.3 ± 4.3

Group 1, first seven consecutive cases; group 2, subsequent six consecutive cases (after retraining); group 3, groups 1 and 2 combined.

[a]$p < .05$; [b]$p < .05$. Maximum ventilation rate, duration, and percentage of time in which a positive pressure was recorded in the lungs (mean ± SEM) (14a). Reprinted with permission.

cantly slower than group 1 patients at 22 ± 3 breaths/min (range, 15 to 31 breaths/min; $p < .05$). Although ventilation rates were slower after retraining, average ventilation duration was significantly longer than in group 1 patients (1.18 ± 0.06 vs. 0.85 ± 0.07 secs/breath, respectively; $p < .05$). As a result, the average percentage of time in which a positive pressure was recorded in the lungs was similar in group 2 and group 1 patients (44.5 ± 8.2% vs. 50 ± 4%, respectively; p = not significant). Combining groups 1 and 2 (group 3), the average ventilation rate for all 13 patients was 30 breaths/min (twice that recommended by the American Heart Association) (14a).

Individual recordings provide insight into the rate and duration of ventilations provided by professional rescuers. Figure 1*A*, represents delivery of CPR relatively close to the American Heart Association guidelines. Only one such case was observed. Figures 1, *B* and *C*, illustrates representative examples of hyperventilation observed in the majority of cases before retraining. After retraining, slower ventilation rates were seen in group 2 patients, but ventilation duration was more prolonged (Fig. 1*D*). As a result, the percentage of time in which a positive pressure was recorded in the airway was not significantly different between groups 2 and 1 (14a).

PORCINE ANIMAL STUDY

Based on these clinical observations, we tested the hypothesis that rapid ventilation rates during CPR increase intrathoracic pressures, impede venous return to the heart, and thus decrease coronary perfusion and survival rates.

Methods

The porcine study was approved by the Committee of Animal Experimentation at the University of Minnesota. The animal preparation and surgical techniques have been previously described in detail (13).

Two separate protocols were performed. Protocol 1 involved nine pigs. The purpose of this protocol was to evaluate the hemodynamic consequences of three different ventilation rates in a controlled model of CPR. Protocol 2 involved three groups with seven pigs per group. The purpose of protocol 2 was to determine whether excessive ventilation rates were associated with increased mortality rates. One group of animals in protocol 2 received supplemental Co_2 to assess whether correction of hypocarbia would influence survival in the hyperventilation group.

Protocol 1. After the initial 2 mins of CPR, each animal received three different ventilation rates (12, 20, and 30 breaths/min) in a computer-generated random order, with each phase lasting for 2 mins (Fig. 2). These three different ventilation-rate interventions were delivered in an asynchronous manner, either every 5 secs (12 breaths/min), every 3 secs (20 breaths/min), or every other second (30 breaths/min), with each breath delivered during a period of 1 sec (14a).

Closed-chest standard CPR was performed continuously with a pneumatically-driven automatic piston device (CPR Controller, AMBU International, Glostrup, Denmark) (13, 14a). The compression rate was 100 compressions/min with a 50% duty cycle, and the compression depth was 25% of the anteroposterior diameter of the chest wall. After each compression, the chest wall was allowed to recoil completely and without any impedance from the compression device. During CPR, aortic, right atrial, and intratracheal pressures were continuously recorded. End-tidal Co_2 and oxygen saturation were also measured continuously and recorded every minute. Arterial blood gases were collected before induction of ventricular fibrillation and at the end of each

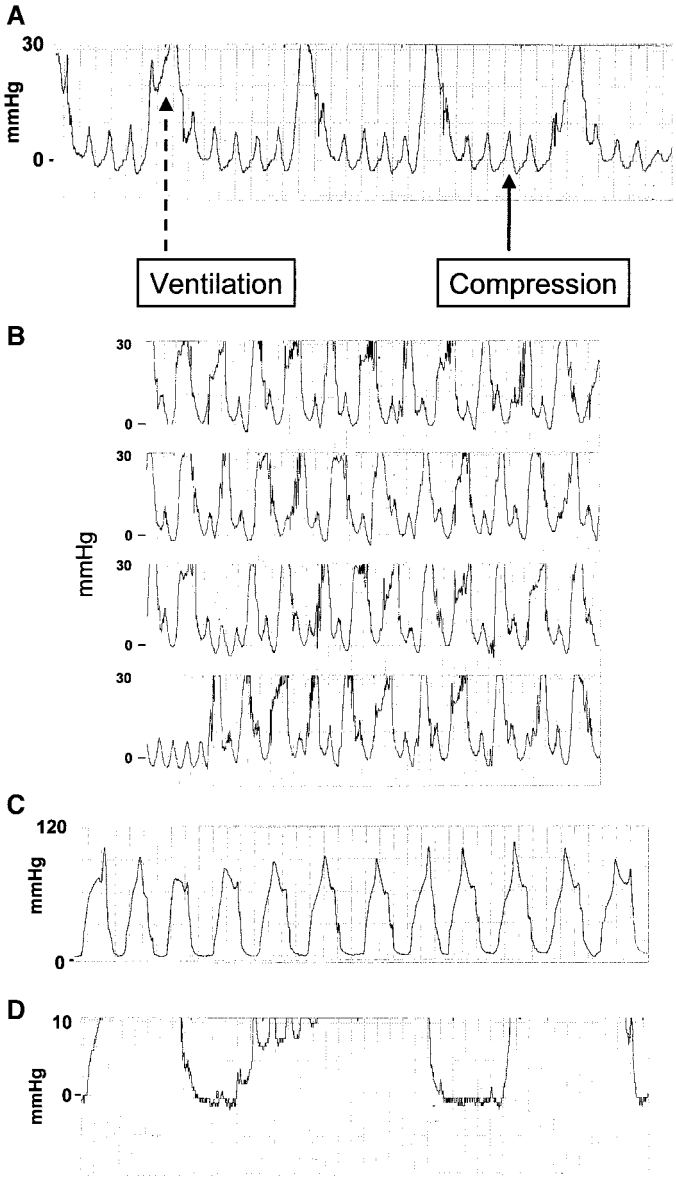

Figure 1. *A*, 16-sec intrathoracic pressure recording depicting cardiopulmonary resuscitation performed relatively close to American Heart Association guidelines. Large-amplitude waves represent ventilations (16 breaths/min); small-amplitude waves represent chest compressions (100 compressions/min). *B*, 64-sec intrathoracic pressure recording (from group 1) demonstrating a ventilation rate of 47 breaths/min. *C*, 16-sec intrathoracic pressure recording (from group 1) representing a ventilation rate of 46 breaths/min. *D*, after retraining, this 16-sec recording from a group 2 patient demonstrates a slower ventilation rate (11 breaths/min) but increased ventilation duration (>4 secs/breath), leaving little time (20%) during cardiopulmonary resuscitation for the development of low or negative intrathoracic pressure (14a). Reprinted with permission.

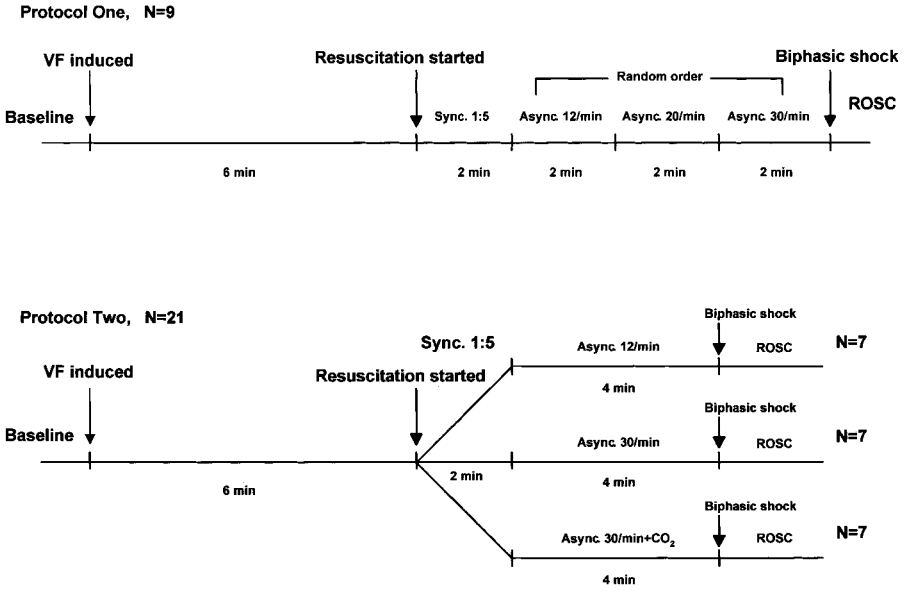

Figure 2. Sequence of interventions for protocol 1 (n = 9), designed to evaluate the hemodynamic consequences of three different ventilation rates, and for protocol 2 (n = 21), designed to determine whether excessive ventilation rates were associated with increased mortality rates. *VF*, ventricular fibrillation; *Sync*, synchronous ventilation rate intervention; *Async*, asynchronous ventilation rate intervention; *ROSC*, return of spontaneous circulation.

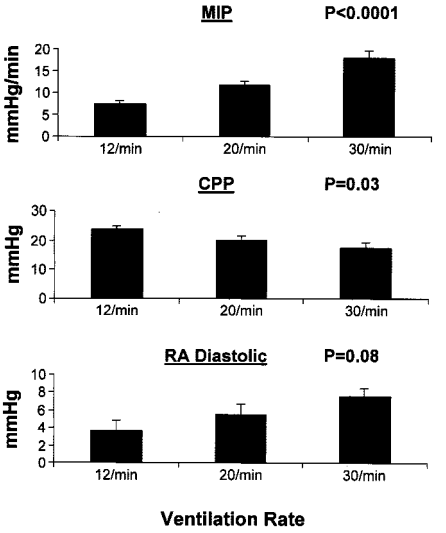

Figure 3. Hemodynamic study (n = 9); changes in mean intrathoracic pressure (*MIP*), coronary perfusion pressure (*CPP*), and right atrial diastolic pressure (*RA diastolic*) with different ventilation rates during resuscitation in a porcine model of cardiac arrest. A p value of $<.05$ was considered statistically significant based on analysis of variance of the three groups (14a). Reprinted with permission.

ventilation rate phase (after minutes 8, 10, 12, and 14 of cardiac arrest) (14a).

Protocol 2. Ventilation during the first 2 mins of CPR was delivered synchronously with a 5:1 compression/ventilation ratio (Fig. 2). After the initial 2 mins of CPR, each animal was randomized to receive 4 mins of CPR with one of the three different ventilation modes: 1) 12 breaths/min with 100% oxygen, 2) 30 breaths/min with 100% oxygen, and 3) 30 breaths/min with 5% Co_2 and 95% oxygen. Five-percent Co_2 was added to inspiratory gases in the third group to evaluate the effect of hyperventilation on survival in the absence of hypocarbia. During these interventions, ventilations were delivered in an asynchronous manner every 5 secs (12 breaths/min) or every other second (30 breaths/min), with each ventilation delivered over 1 sec (14a).

During CPR, aortic, right atrial, and intratracheal pressures and end-tidal Co_2 and oxygen saturation were continuously recorded. Arterial blood gas samples were assessed before induction of ventricular fibrillation and at the end of each ventilation phase (14a).

At the end of each protocol (Fig. 2), the animals were shocked with a biphasic defibrillator (Zoll M Series, Zoll Medical, Burlington, MA) using 150 J, up to three times, as needed (16). If resuscitation was successful, animals were ventilated by the positive pressure ventilator with supplemental oxygen. Return of spontaneous circulation was defined as a stable blood-perfusing rhythm generating a measurable blood pressure during the first hour of observation after resuscitation. No other therapeutic interventions were performed after return of spontaneous circulation. At the end of each study protocol described above, the animals were killed using an intravenous bolus of propofol, 60 mg, and then 10M KCl (14a).

All values are expressed as mean ± SEM. Coronary perfusion pressure was calculated as the difference between aortic diastolic pressure and right atrial diastolic pressure. For each animal, ten measurements were performed for both aortic diastolic pressure and right atrial diastolic pressure, and the average difference was used as the representative value for each animal. Mean intratracheal pressure was measured as the time-averaged value from continuous measurements acquired over a 10-sec period. Comparison between groups was done by analysis of variance and paired Student's t-test. Survival was calculated with chi-square tests and Fisher's exact tests. A p value of $<.05$ was considered statistically significant (14a).

Results

Protocol 1. Increased ventilation rate was associated with significantly higher mean intratracheal pressures ($p < .0001$) and significantly lower coronary perfusion pressures ($p = .03$, Fig. 3) and arterial pH but no change in Pao_2. There was also an increase in right atrial diastolic pressure with increased ventilation rate (Fig. 3). This was only significantly lower in the 12 breaths/min vs. 30 breaths/min groups (3.5 ± 1.1 vs. 7.3 ± 1.0, respectively; $p = .02$). The return of spontaneous circulation rate in protocol 1 was three of nine pigs. Two of three pigs that survived received 12 breaths/min as the terminal ventilation rate sequence (14a).

Protocol 2. The survival rate in pigs ventilated at 12 breaths/min with 100% oxygen was six of seven (86%) compared with a survival rate of one of seven (17%) at a rate of 30 breaths/min with 100% oxygen and one of seven (17%) at a ventilation rate of 30 breaths/min with 5% Co_2 and 95% oxygen ($p = .006$) (Fig. 4). Mean intratracheal pressures were significantly higher with the higher ventilation rates ($p < .0001$) and coronary perfusion pressures were lower (Fig. 4). Pigs ventilated at 30 breaths/min (100% oxygen) had lower levels of $Paco_2$. Supplemental Co_2 resulted in correction of hypocapnia, maintaining Co_2 at an average level of 35.4 ± 0.6 mm Hg (Fig. 4) (14a).

DISCUSSION

These results demonstrate that ventilation rates during the field-application of CPR in a city with well-trained EMS personnel were far in excess of those recommended by the American Heart Association. To our knowledge, this represents the first time that ventilation frequency, duration, and percentage of time with a positive pulmonary pressure have been objectively and electronically recorded during CPR performed by professional rescuers at the scene of out-of-hospital cardiac arrests. Based on these observations, animal studies were performed demonstrating that excessive ventilation

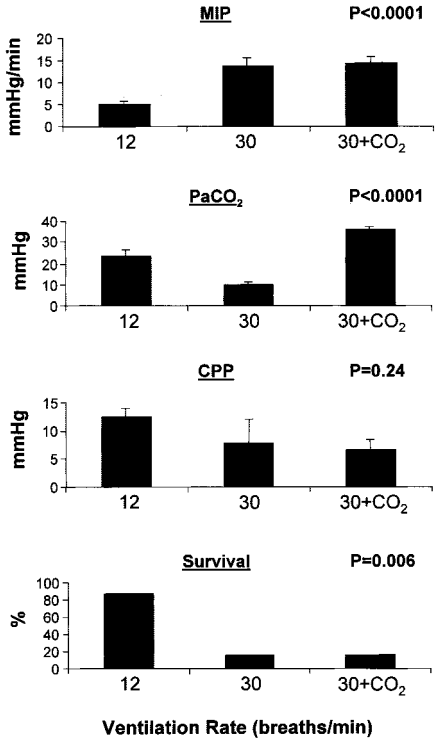

Figure 4. Survival study (n = 7 pigs/group); changes in mean intrathoracic pressure (*MIP*), $Paco_2$, coronary perfusion pressure (*CPP*), and survival rate, with hyperventilation and correction of hypocapnia ($+Co_2$). A p value of $<.05$ was considered statistically significant based on an analysis of variance of the three groups (14a). Reprinted with permission.

rates during CPR are associated with a marked decrease in coronary perfusion pressure and decreased survival rates in a well-accepted animal model of cardiac arrest (14a).

Continuous recording of ventilations in this study also allowed further characterization of ventilation patterns and of how those patterns contributed to a substantial percentage of time that positive pressure was recorded in the lungs. Both rapid-rate, short-duration ventilations and slow-rate, long-duration ventilations contributed to an excessively high percentage of time that pressure in the chest was increased. As confirmed by the animal studies, the increased positive intrathoracic pressure was associated with decreased coronary perfusion and decreased survival rates.

The physiologic consequences of hyperventilation and of prolonged ventilation intervals result in well-recognized decreases in cardiac preload and cardiac output (17–20) and impede right ventricular function (21). Increased tidal volume is also known to adversely affect cardiac output (22). In the present study, the mean intrathoracic pressure was significantly elevated in patients and animals treated with higher ventilation rates. We speculate that the elevated mean intrathoracic pressures inhibited venous blood flow back to the right heart, as there was never enough time to allow for the development of negative intrathoracic pressure between compressions. It is important to note that the intrathoracic pressure was always >0 mm Hg when ventilation rates were 30 breaths/min. The current results also support the contention that hypocapnea is not the cause of decreased coronary perfusion pressure and death in animals ventilated at 30 breaths/min. When supplemental Co_2 at 5% was delivered, a concentration identical to the amount found in expiratory gases, in one group of pigs to prevent hypocapnia without causing hypercarbia, the arterial Co_2 level did not fall to $<35.4 \pm 0.6$ mm Hg and survival rates (one of seven pigs) were identical to pigs hyperventilated with 100% oxygen (14a).

These observations have significant implications for CPR guidelines, education, and clinical practice. Despite being trained more thoroughly than the average rescuer to provide correct ventilation rates, professional rescuers in this study consistently and inadvertently hyperventilated patients during actual resuscitations. Before this investigation, the potential harmful effects of excessive ventilation rates during CPR remained unknown and have therefore not been a major focus during CPR education. We speculate that the hyperventilation observed in this study is also occurring undetected in many other EMS systems nationally and internationally. Excessive ventilation rates have been previously reported in patients with an in-hospital arrest undergoing CPR (23). Nonetheless, the extent to which this clinical observation applies to other EMS systems needs to be determined through additional study. The authors encourage medical directors to assess whether hyperventilation during CPR is inadvertently occurring in the care systems under their medical direction. Irrespective, these data demonstrate that any incident of hyperventilation is likely to have detrimental hemodynamic and survival consequences during low-flow states such as CPR. Unrecognized and inadvertent hyperventilation may contribute to the currently dismal survival rates of cardiac arrest. Similar detrimental effects of hyperventilation have recently been described in the setting of hemorrhagic shock (14a, 24).

The optimal ventilation rate for patients in cardiac arrest has yet to be defined and may well be significantly lower than 12 breaths/min (14a). As observed clinically, it is also important to limit ventilation duration, as both ventilation rate and ventilation duration contribute to mean intrathoracic pressure during CPR. In addition to data presented in this report, studies by others demonstrate that the hemodynamic consequences of interrupting compressions to deliver ventilations are also grave (25, 26). This has lead to a reappraisal of the importance of ventilation, in general, during the initial phases of CPR (25–28).

Clinical Implications. The current American Heart Association guidelines (14) are unfortunately confusing when it comes to guidance regarding ventilation during CPR. They recommend: 1) 12–15 breaths/min; 2) a compression-to-ventilation ratio of 5:1 (which calculates to a ventilation rate of 20 breaths/min at a compression rate of 100 compressions/min); or 3) a compression/ventilation ratio of 15:2, or about 16 breaths/min at a compression rate of 100 compressions/min for professional rescuers treating adult victims of cardiac arrest (with or without a secured airway). Based on the results of the current study, we believe there is an urgent need for the American Heart Association and other resuscitation councils to reevaluate current recommendations and to place new emphasis on the importance of slower ventilation rates of limited duration during CPR. It is possible that by consistently delivering rescue breaths at 12 breaths/min with limited duration, professional rescuers could considerably improve survival of cardiac arrest. Additional education of all professional rescuers (e.g., physicians, nurses, respiratory therapists, nursing assistants, first responders, emergency medical technicians, and paramedics) is urgently needed to reduce any incidents of these newly identified and deadly consequences of hyperventilation during CPR (14a).

The consistent and inadvertent hyperventilation observed in this study by trained, retrained, and certified professional rescuers demonstrates a significant difference between CPR performance in the classroom and CPR performance during an actual cardiac arrest. As such, this study indicates that there are significant patient safety issues

Excessive ventilation rates significantly decreased coronary perfusion pressures and survival rates, despite supplemental Co_2 to prevent hypocapnia.

when it comes to the delivery of CPR. It also highlights a potential direct relationship between the quality of CPR delivered and victim survival (14a). Developers of CPR training programs should recognize these important issues and develop, test, and implement educational strategies that maximize quality in learning the psychomotor skills of CPR and improve consistency in performance from the classroom to actual emergencies. This is particularly appropriate for ventilation rate and duration but may apply to other aspects of CPR performance as well. For example, the investigators of this study have identified frequent incomplete chest wall decompression during the performance of CPR by professional rescuers, resulting in similar hemodynamic consequences as seen with excessive ventilation rates (the subject of a separate study) (Fig. 5).

Significant opportunities are now emerging for EMS to improve the patient–care delivery system (29–31). These include devices to guide rescuers on when to deliver ventilations, more advanced and automatic data collection, the communication of real-time, waveform-based physiologic information for off-site assessment, wireless video, wireless transmission of field data to other locations via handheld devices, and use of remotely controlled medical devices (29–31). These technological advances provide the opportunity for significantly improved medical control of the delivery of EMS services. Pilot EMS programs using some of these advanced technologies, targeting rapid and accurate triage for victims of stroke and trauma, have already been implemented (29–31). The potential benefits of using such advanced technology to monitor the EMS delivery of CPR also deserve investigation.

There are several important limitations to this study. First, the clinical observations were only made in a single city and with a limited number of patients. We did not believe it was ethical to continue to collect data once we recognized the potential lethal nature of the observed hyperventilation. Moreover, in discussions with EMS directors across the country, it became clear that the observations of excessive ventilation rates in the current study were not limited to this test site alone. Nonetheless, we do not know how widespread this problem is, and further study is warranted to characterize its prevalence nationally and internationally. Second, the animal hemodynamic studies focused on coronary perfusion pressures and intrathoracic pressures. The physiologic effects of excessive ventilation rates may be underestimated by not measuring actual blood flow. Finally, animal survival studies were not performed at ventilation rates of <12 breaths/min. The optimal ventilation rate and duration for patients in cardiac arrest has yet to be defined and may well be <12 breaths/min (14a).

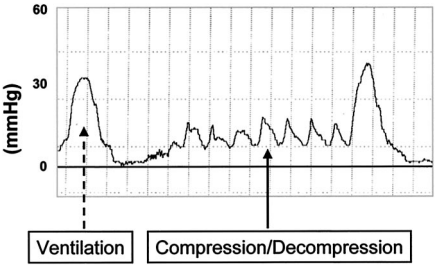

Figure 5. Sixteen-second intrathoracic pressure recording illustrating incomplete chest wall decompression performed by a rescuer. Hemodynamic consequences, similar to those observed with excessive ventilation rates, were identified by investigators in a separate study, indicating that there may be several aspects of cardiopulmonary resuscitation performance critical to optimizing forward flow.

CONCLUSIONS

Despite seemingly adequate training, professional rescuers consistently hyperventilated patients during out-of-hospital CPR. Subsequent hemodynamic and survival studies in pigs demonstrated that excessive ventilation rates significantly decreased coronary perfusion pressures and survival rates, despite supplemental Co_2 to prevent hypocapnia. This translational research initiative demonstrates an inversely proportional relationship between mean intratracheal pressure and coronary perfusion pressure during CPR. Additional education of CPR providers is urgently needed to reduce these newly identified and deadly consequences of hyperventilation during CPR. These findings also have significant implications for interpretation and design of resuscitation research, CPR guidelines, education, the development of biomedical devices, EMS quality assurance, and clinical practice (14a).

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