

# Clinical and hemodynamic comparison of 15:2 and 30:2 compression-to-ventilation ratios for cardiopulmonary resuscitation\*

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**Objective:** To compare cardiopulmonary resuscitation (CPR) with a compression to ventilation (C:V) ratio of 15:2 vs. 30:2, with and without use of an impedance threshold device (ITD).

**Design:** Prospective randomized animal and manikin study.

**Setting:** Animal laboratory and emergency medical technician training facilities.

**Subjects:** Twenty female pigs and 20 Basic Life Support (BLS)-certified rescuers.

**Interventions, Measurements, and Main Results: Animals:** Acid-base status, cerebral, and cardiovascular hemodynamics were evaluated in 18 pigs in cardiac arrest randomized to a C:V ratio of 15:2 or 30:2. After 6 mins of cardiac arrest and 6 mins of CPR, an ITD was added. Compared to 15:2, 30:2 significantly increased diastolic blood pressure ( $20 \pm 1$  to  $26 \pm 1$ ;  $p < .01$ ); coronary perfusion pressure ( $18 \pm 1$  to  $25 \pm 2$ ;  $p = .04$ ); cerebral perfusion pressure ( $16 \pm 3$  to  $18 \pm 3$ ;  $p = .07$ ); common carotid blood flow ( $48 \pm 5$  to  $82 \pm 5$  mL/min;  $p < .001$ ); end-tidal CO<sub>2</sub>

( $7.7 \pm 0.9$  to  $15.7 \pm 2.4$ ;  $p < .0001$ ); and mixed venous oxygen saturation ( $26 \pm 5$  to  $36 \pm 5$ ,  $p < .05$ ). Hemodynamics improved further with the ITD. Oxygenation and arterial pH were similar. Only one of nine pigs had return of spontaneous circulation with 15:2, vs. six of nine with 30:2 ( $p < 0.03$ ). **Humans:** Fatigue and quality of CPR performance were evaluated in 20 BLS-certified rescuers randomized to perform CPR for 5 mins at 15:2 or 30:2 on a recording CPR manikin. There were no significant differences in the quality of CPR performance or measurement of fatigue. Significantly more compressions per minute were delivered with 30:2 in both the animal and human studies.

**Conclusions:** These data strongly support the contention that a ratio of 30:2 is superior to 15:2 during manual CPR and that the ITD further enhances circulation with both C:V ratios. (Crit Care Med 2006; 34:1444–1449)

**KEY WORDS:** cardiopulmonary resuscitation; hemodynamics; perfusion; ventilation; impedance threshold device

Compression-to-ventilation (C:V) ratios greater than the 15:2 ratio currently recommended by the American Heart Association (AHA) have been suggested by some investigators for standard cardiopulmonary resuscitation (CPR) (1–8). Higher ratios may improve hemodynamics by decreasing interruptions for ventilation and delivering more compressions per minute. Computer modeling suggests that vital organ blood flow may be superior with a 30:2 ratio and with the use of an

impedance threshold device (ITD) (4, 5). Moreover, the optimal balance between circulation (compressions) and ventilation during CPR has not been established. Higher C:V ratios may be associated with increased rescuer fatigue and poorer performance of CPR (9, 10). Fewer ventilations may compromise acid-base and oxygenation status (11, 12).

This translational research was designed to evaluate the effect of a C:V ratio of 30:2 vs. 15:2 on hemodynamics in a porcine of ventricular fibrillation

(VF) cardiac arrest, with and without use of an inspiratory ITD (13–18), and to evaluate the effect of a CV ratio of 30:2 vs. 15:2 on fatigue and quality of CPR delivered as performed by Basic Life Support (BLS)-certified rescuers.

## METHODS

### Animal Study

The study was approved by the Institutional Animal Care Committee of the involved

#### \*See also p. 1563.

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Dr. Aufderheide has consulted for MedtronicPhysio and has received grants from NHLBI and SBIR. He also volunteers on AHA Basic Life Support (BLS) subcommittees and participated in funded research with the ITD in a previous study. Dr. Pirrallo owns stock in Boll Medical Inc. and is receiving grants from NIH. Dr. Becker has received research report (to the University of Chicago) from Philips Medical Systems, Laerdal Medical, Alsius Corporation, and the NIH. Dr. Becker has also consulted for Abbott Labs, Philips Medical Systems, and the NIH Data Safety Monitoring Board and Protocol Review Committee. Dr. Becker has been a special government employee, FDA, and CPR Device Panel member. Dr. Vanden Hoek has received re-

search support (to the University of Chicago) from the NIH and DOD/Office of Naval Research. Dr. Idris has received NIH and Defense Department Grants to study the ITD. He owns stock in ACSL, Inc. and has grants pending from the US Army.

Keith G. Lurie is the inventor of the Inspiratory Impedance Threshold Device used in this study and founded Advanced Circulatory Systems, Incorporated (ACSI) to develop this technology.

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institutions. The animals received treatment and care in compliance with the 1996 Guide for the Care and Use of Laboratory Animals by the National Research Council, in accord with the United States Department of Agriculture (USDA) Animal Welfare Act, Public Health Service (PHS) Policy, and the American Association for Accreditation of Laboratory Animal Care. Anesthesia was used in all surgical interventions to avoid all unnecessary suffering. The animal portion of this study was performed on female farm pigs ( $29 \pm 2.5$  kg).

**Preparatory Phase.** The preparatory phase has been described in detail previously (17). In brief, initial sedation was achieved with 700 mg of intramuscular ketamine. Propofol anesthesia (PropoFlo, Abbott Laboratories, North Chicago, IL; 2.3 mg/kg) was also delivered as an intravenous (iv) bolus via the lateral ear vein. After intubation with a 7.5-mm cuffed endotracheal tube, a propofol infusion of 160  $\mu$ g/kg/min was started.

Subsequently, under aseptic conditions, a burr hole was made at the middle of the distance between the left eyebrow and the posterior bony prominence of the skull. An intracranial pressure transducer (Camino, Intra Life Sciences) was used to record real-time intracranial pressure. Systolic, diastolic, and mean intracranial pressures were recorded every minute. The left common carotid artery was surgically exposed, and an ultrasonic flow probe (Transonic 400-Series Multi-Channel, Transonic Systems, Ithaca, NY) was placed around it to quantify blood flow.

Unilateral femoral artery cannulation was performed at the supine position. Central aortic and right external jugular vein pressures were recorded continuously, with two micromanometer-tipped catheters (Mikro-Tip Transducer, Millar Instruments, Houston, TX). All animals were treated with a heparin bolus (100 units/kg iv) once catheters were in place. During the preparatory phase, animals were ventilated with room air via a volume-control ventilator (Harvard Apparatus Co., Dover, MA), with the tidal volume of 12 mL/kg and rate adjusted to continually maintain an arterial  $P_{aCO_2}$  of 40 mm Hg and  $P_{aO_2}$  of >80 mm Hg (oxygen saturation, >95%), based on analysis of arterial blood gases (IL Synthesis, Instrumentation Laboratory, Lexington, MA). There was continuous electrocardiographic monitoring. Endotracheal pressure (ETP) was measured continuously with a micromanometer-tipped catheter positioned 2 cm above the carina. All data were recorded by a digital recording system as previously described (17). End-tidal  $CO_2$  ( $ETCO_2$ ), tidal volumes, and oxygen saturation were continuously measured ( $CO_2$ SMO Plus, Novamatrix Medical Systems, Wallingford, CT).

All the parameters (aortic, right atrial, endotracheal, intracranial, common carotid blood flow, coronary perfusion, and cerebral perfusion pressures) were analyzed with data from the fourth, fifth, ninth, and tenth mins of

CPR. Coronary perfusion pressure (CPP) during CPR was calculated with two different methods. The first method used the calculated area between the aortic pressure (AoP) and right atrial pressure (RAP) curves when  $AoP > RAP$ . This estimated the total positive CPP area over the whole minute (mm Hg  $\times$  secs). The second method used discrete CPP during the decompression phase of CPR, based on the nadir of the right atrial pressure and the coincident aortic pressure. Three consecutive decompression measurements before the delivery of ventilation were averaged. These measurements were repeated three times within each minute studied, and the average of the three mean values was reported as the mean discrete CPP in mm Hg during each minute. Cerebral perfusion pressure was calculated as the difference between the mean values of aortic pressure and intracranial pressure, with use of the mean value of the digitalized aortic and intracranial pressure tracings. Common carotid blood flow was reported as the mean blood flow recorded over 1 min (mL/min). Arterial and mixed venous blood gases were collected at baseline and at minutes 5 and 9 of CPR. Cardiac output was calculated by the Fick method (19).

**Experimental Protocol.** After VF was induced by delivering direct electrical current via a temporary pacing wire (Daig Division, St. Jude Medical, Minnetonka, MN) positioned in the right ventricle, a computer-generated randomization list was used to determine treatment assignment. After induction, the ventilator was disconnected from the endotracheal tube, and the dose of propofol was reduced to 100  $\mu$ g/kg/min. After 6 mins of untreated VF, closed-chest standard CPR was performed with a pneumatically driven automatic piston device (Pneumatic Compression Controller, AMBU International, Glostrup, Denmark), as previously described (17). Positive pressure ventilations were delivered with a resuscitator bag (Smart Bag, O2 Systems, Toronto, Canada), with controlled peak airway pressure of 40 mm Hg. The resuscitator bag was filled with oxygen at a rate of 10 L/min. Ventilations were initiated 2 secs after compressions were discontinued, and each ventilation was delivered over 2 secs. Tidal volumes of approximately 400 mL were delivered with each breath.

After 6 mins of untreated VF, animals were prospectively randomized to 6 mins of a C:V ratio of either 30:2 or 15:2. After 6 mins of CPR with the prespecified ratio, an inspiratory impedance threshold device (ITD) (Advanced Circulatory Systems, Eden Prairie, MN) was added, and another 4 mins of CPR was performed with use of the same ratio. At the end of 10 mins of CPR, animals were defibrillated with a Zoll M Series biphasic defibrillator (Zoll Medical, Chelmsford, MA), starting at a setting of  $120 \times 3$ . If VF persisted, epinephrine was administered at a dose of 45  $\mu$ g/kg, and then three more shocks (at 120) were delivered. If VF persisted, all resuscitation efforts were terminated. Fifteen minutes later, surviving ani-

mals were euthanized by a 10-mL intracardiac injection of 10 M potassium chloride.

**Statistical Analysis.** Values are expressed as mean  $\pm$  SEM. The primary end points were coronary and cerebral perfusion pressure and common carotid blood flow. Repeated-measurements analysis of variance (ANOVA) and unpaired Student's *t*-tests were used to determine statistical significance between different ratios, and a paired Student's *t*-test was used to evaluate significance within the same ratio, with and without the ITD. A *p* value of <.05 was considered statistically significant. Fisher's exact test was used to compare return of spontaneous circulation rates between groups.

**Findings.** An increase of the C:V ratio from 15:2 to 30:2 resulted in a significant increase in compressions delivered over 1 min ( $66 \pm 2$  to  $84 \pm 2$ ; *p* < .0001) and in a significant reduction in the amount of time when no compressions were performed each minute, from  $20.33 \pm 1.44$  to  $9.6 \pm 0.96$  secs/min (*p* < .0001) (Table 1).

The C:V ratio of 30:2 significantly improved diastolic aortic pressure from  $20 \pm 1$  to  $26 \pm 1$  mm Hg (*p* < .001). There was a significant improvement in coronary and cerebral perfusion pressures and a 70% increase in common carotid blood flow (Table 1 and Fig. 1).

Significant incremental hemodynamic improvement was observed with the addition of the ITD with use of 15:2 and 30:2 ratios. There was a 135% increase in common carotid blood flow in the 30:2 + ITD group vs. the 15:2–no ITD group. Cardiac output significantly and incrementally improved from 15:2 to the 30:2 ratio and further improved with the addition of an ITD (Table 1 and Fig. 1). CPP area over 1 min demonstrated similar incremental improvement. The return of spontaneous circulation rate was one of nine with the 15:2 ratio and six of nine with the 30:2 ratio (*p* < .03).

Arterial and venous blood gas results are shown in Table 2. There was no difference between oxygenation and acid-base balance between the 15:2–no ITD and 30:2–no ITD groups.  $ETCO_2$  significantly increased with the 30:2 ratio ( $7.7 \pm 0.9$  to  $15.7 \pm 2.4$  mm Hg; *p* = .001) (Fig. 1). Mixed venous oxygen saturation was significantly higher in the 30:2 group (Table 2). Addition of the ITD resulted in higher arterial  $P_{aO_2}$  and  $P_{aCO_2}$  and lower pH but no change in base excess (Table 2).  $P_{aCO_2}$  minus  $ETCO_2$  values significantly decreased with a ratio of 30:2 and ITD use (Table 2, Fig. 1).

## Human Study

The primary objective of the human study was to compare CPR performance and fatigue with use of 15:2 and 30:2 ratios in 20 volunteer subjects certified in BLS (10 emergency medical technicians [EMTs] and 10 laypersons). A secondary purpose was to compare CPR performance and fatigue between the 10 EMTs and 10 laypersons.

Table 1. Mean ± SEM values for hemodynamic parameters

Hemodynamic Parameters	15:2	15:2 + ITD	30:2	30:2 + ITD
SBP, mm Hg	53 ± 5 <sup>§</sup>	68 ± 5	55 ± 5	62 ± 4 <sup>§</sup>
DBP, mm Hg	20 ± 1 <sup>†§</sup>	21 ± 2 <sup>‡*</sup>	26 ± 1 <sup>†‡¶</sup>	30 ± 1 <sup>*¶§</sup>
MAP, mm Hg	36 ± 3 <sup>§</sup>	45 ± 3	41 ± 3 <sup>*</sup>	46 ± 3 <sup>*§</sup>
DRAP, mm Hg	1.5 ± 0.5 <sup>‡§</sup>	-0.9 ± 1.1 <sup>‡</sup>	1.6 ± 0.8 <sup>*</sup>	0.1 ± 0.8 <sup>*§</sup>
ETP, mm Hg	1.5 ± 0.2 <sup>†§</sup>	0.2 ± 0.4 <sup>†</sup>	1 ± 0.3	0.3 ± 0.2 <sup>§</sup>
ICP, mm Hg	21 ± 1	21 ± 1	22 ± 1	22 ± 1
CPP, mm Hg	18 ± 1 <sup>†§</sup>	22 ± 3 <sup>*</sup>	25 ± 2 <sup>†¶</sup>	30 ± 2 <sup>*¶§</sup>
Total CPP, area/min (mm Hg × sec)	4567 ± 367 <sup>†‡§</sup>	5283 ± 513 <sup>†‡</sup>	9245 ± 722 <sup>*†</sup>	10356 ± 871 <sup>*‡§</sup>
CerPP, mm Hg	16 ± 3 <sup>§</sup>	24 ± 3	18 ± 3 <sup>*</sup>	23 ± 3 <sup>*§</sup>
Carotid blood flow (mL/min)	48 ± 5 <sup>†‡§</sup>	64 ± 5 <sup>†‡</sup>	82 ± 5 <sup>*†</sup>	112 ± 7 <sup>*‡§</sup>
Compressions/min	66 ± 2 <sup>†§</sup>	65 ± 2 <sup>*</sup>	84 ± 2 <sup>†</sup>	85 ± 1 <sup>*§</sup>
No compression time (sec/min)	20 ± 1 <sup>†§</sup>	20 ± 2 <sup>*</sup>	10 ± 1 <sup>†</sup>	9 ± 1 <sup>*§</sup>
Cardiac output (L/min)	0.7 ± 0.1 <sup>§</sup>	0.8 ± 0.1	0.95 ± 0.1	1.1 ± 0.1 <sup>§</sup>

ITD, impedance threshold device; SBP, systolic blood pressure; DBP, diastolic blood pressure; MAP, mean arterial pressure; DRAP, diastolic right atrial pressure; ETP, endotracheal pressure; ICP, intracranial pressure; CPP, coronary perfusion pressure; CerPP, cerebral perfusion pressure.

<sup>†‡¶§</sup>Statistically significant differences between values with the same symbol in the same row with a *p* value of <.05.

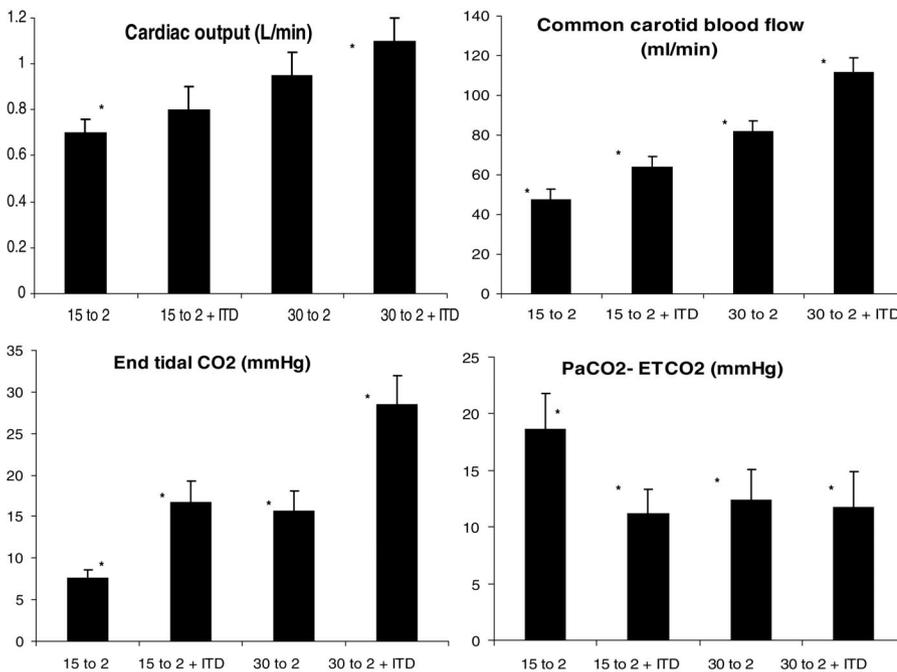


Figure 1. Mean ± SEM values for major blood flow parameters during all four interventions: 15:2, 15:2 + impedance threshold device (ITD), 30:2, and 30:2 + ITD (ET, end-tidal). \*Statistically significant differences between 15:2 and other treatments with *p* <.05.

**Subjects and Methods.** We implemented a randomized, prospective, blinded clinical trial involving subjects who were ≥18 yrs old, certified in BLS, completed a CPR course within the last 24 months, and signed an informed consent. Excluded were subjects who were current or past CPR instructors and any individual with health-related problems that limited their physical job performance. Subjects were told investigators were evaluating different CPR techniques but not that the study was assessing fatigue and specific components of CPR performance. The Human Research Review Committee at the Medical College of Wisconsin approved the study.

To evaluate CPR performance, study subjects performed single-rescuer CPR on a Lae-

rdal Skill Reporter CPR manikin. The manikin software was configured to consider any decompression that did not return to within 2 mm of baseline as incomplete. Depth of compression was defined as adequate if it was between 38 and 51 mm (1.5 to 2 inches). A pressure sensor incorporated into the lower half of the manikin's sternum measured proper hand placement. Compressions performed outside the boundaries of the pressure sensor (left, right, high, and low) were recorded as incorrect hand placement.

To evaluate fatigue, subjects' heart rate was continuously recorded (Lead II, ECG Zoll M Series monitor, Zoll Medical) before, during, and after each method of CPR. Perceived fatigue and discomfort was evaluated by self-

reported survey results with use of a Likert Scale (0 = none; 25 = slight; 50 = moderate; 75 = severe; 100 = very severe).

**Experimental Protocol.** Subjects were attached to a continuous electrocardiography (ECG) monitor, and baseline heart rate was recorded. Then, a research team member (a certified CPR instructor) verbally and comprehensively reviewed standard CPR with the subject. By means of a computerized randomization list, subjects were randomized to first perform 5 mins of single-rescuer CPR at either a 15:2 or 30:2 ratio. To ensure a stable and consistent baseline for measurement of the compression/decompression cycle by the recording manikin, ventilations were always simulated. Subjects were instructed to open the manikin's airway by the head tilt–chin lift technique and to state the words *breath, breath* over approximately 4 secs (the recommended time for delivery of two breaths during CPR). No coaching occurred during CPR, and a metronome was used to help maintain a rate of 100 compressions per minute. Peak heart rate was recorded during CPR performance. Following completion of 5 mins of continuous single-rescuer CPR at the randomized C:V ratio, subjects were allowed to rest for 20 mins. The time to return to baseline heart rate was recorded during this rest period. Subjects also completed a perceived fatigue and discomfort survey during this time. This process was then repeated with the alternate C:V ratio.

**Statistical Analysis.** Comparisons of performance between CPR techniques and fatigue measurements were made by paired Student's *t*-tests. A *p* value of <.05 was considered statistically significant.

**Findings.** Subjects (*n* = 20; 10 males) had an average age of 27.5 ± 9.7 yrs (range, 20–62 yrs). The mean height was 67.1 ± 3.7 inches and the average weight was 176.6 ± 39.5 lb. Education level ranged from less than a high school diploma to a 4-yr college degree. The 10 EMS providers (seven BLS-trained) had an

Table 2. Mean  $\pm$  SEM values for arterial and venous blood gases

Blood Gas Parameters	15:2	15:2 + ITD	30:2	30:2 + ITD
Arterial pH	7.47 $\pm$ 0.08 <sup>§</sup>	7.36 $\pm$ 0.05	7.42 $\pm$ 0.03 <sup>†</sup>	7.23 $\pm$ 0.04 <sup>†§</sup>
Paco <sub>2</sub>	26.14 $\pm$ 4 <sup>§</sup>	28 $\pm$ 2.5 <sup>*</sup>	28.1 $\pm$ 4.2 <sup>†</sup>	40.4 $\pm$ 4.4 <sup>*†§</sup>
PaO <sub>2</sub>	122.8 $\pm$ 17.1 <sup>§</sup>	146.5 $\pm$ 19.8	131.3 $\pm$ 19.91 <sup>†</sup>	150.7 $\pm$ 22 <sup>†§</sup>
Art HCO <sub>3</sub>	17.31 $\pm$ 1.5	15.65 $\pm$ 1.04	17.2 $\pm$ 1.2	17.7 $\pm$ 0.9
Art base excess	-6.87 $\pm$ 2.11	-9.83 $\pm$ 1.6	-7.2 $\pm$ 1.16	-9.86 $\pm$ 1.21
%Sat O <sub>2</sub>	98.4 $\pm$ .8	97.0 $\pm$ 2.74	96 $\pm$ 2	99.3 $\pm$ 2.21
ETCO <sub>2</sub>	7.7 $\pm$ 0.9 <sup>†¶§</sup>	16.8 $\pm$ 2.5 <sup>*¶</sup>	15.7 $\pm$ 2.4 <sup>†¶</sup>	28.5 $\pm$ 3.5 <sup>†*§</sup>
Paco <sub>2</sub> -ETCO <sub>2</sub>	18.7 $\pm$ 3.1 <sup>†¶§</sup>	11.2 $\pm$ 2.1 <sup>‡</sup>	12.4 $\pm$ 2.7 <sup>¶</sup>	11.9 $\pm$ 3.1 <sup>§</sup>
Venous pH	7.19 $\pm$ 0.03 <sup>‡</sup>	7.09 $\pm$ 0.03 <sup>‡</sup>	7.20 $\pm$ 0.02 <sup>†</sup>	7.13 $\pm$ 0.02 <sup>†</sup>
PvCO <sub>2</sub>	63 $\pm$ 9 <sup>§</sup>	73.5 $\pm$ 11.4 <sup>‡</sup>	71.5 $\pm$ 4.74	75 $\pm$ 3.89 <sup>§</sup>
PvO <sub>2</sub>	22.7 $\pm$ 2.16 <sup>§</sup>	25 $\pm$ 3.31	27.33 $\pm$ 2.57 <sup>†</sup>	31.1 $\pm$ 2.18 <sup>†§</sup>
VHCO <sub>3</sub>	26.22 $\pm$ 1.65	24.8 $\pm$ 1.2	27.44 $\pm$ 1.19 <sup>†</sup>	24.85 $\pm$ 1 <sup>†</sup>
V base excess	-2.0 $\pm$ 2	-3.87 $\pm$ 1.5	-0.67 $\pm$ 1.2 <sup>†</sup>	-4.31 $\pm$ 1.28 <sup>†</sup>
% Sat MvO <sub>2</sub>	26.33 $\pm$ 5.5 <sup>†§</sup>	25.5 $\pm$ 5.47 <sup>‡</sup>	35.67 $\pm$ 5.12 <sup>†</sup>	38.6 $\pm$ 4.12 <sup>‡§</sup>

ITD, impedance threshold device; Art, arterial; Sat, saturated; ETCO<sub>2</sub>, end-tidal carbon dioxide (mm Hg); MvO<sub>2</sub>, mixed venous oxygen.

<sup>†‡¶§</sup>Statistically significant differences between values with the same symbol in the same row with a *p* value of <.05.

average of 3.9  $\pm$  3.3 yrs of experience (range, 1–11 yrs). Five of the EMTs had never performed CPR on a patient, and two EMTs had performed CPR >20 times. Ten subjects were laypersons. Three laypersons worked in service professions, one was retired, and six listed “other” as their job description. They had been certified in CPR an average of 4.7  $\pm$  5.7 yrs (range, 1–17 yrs), and their last class was 14.2  $\pm$  5.0 months before testing (range, 8–24 months). None of them had performed CPR in a rescue situation.

When all 20 subjects were considered together, there was a significant increase in chest compressions delivered during 5 mins of CPR with a C:V ratio of 30:2 vs. 15:2 (401.8  $\pm$  13.7 vs. 339.8  $\pm$  8.5; *p* < .001). There were no significant differences between the two groups in the quality of CPR delivered. Only about half of the chest compressions in both groups were of adequate depth, and less than half had complete chest wall recoil (Table 3). There also were no significant differences between the groups in fatigue measurements (Table 4).

When comparing the quality of CPR delivered with a C:V ratio of 30:2 vs. 15:2 by EMS providers and laypersons, we found no significant differences except percentage of complete chest wall recoil (17.7  $\pm$  5.6% vs. 33.7  $\pm$  8.5%; *p* < .004) (Table 3). When comparing fatigue measurements, there were no significant differences except peak heart rate, which averaged about 5 beats per minute higher in laypersons performing CPR at a C:V ratio of 30:2 (Table 4).

## DISCUSSION

In recent years, it has become apparent that the traditional approaches to CPR are inadequate, providing only 10% to 20% of normal blood flow to the heart and brain (13, 14). These findings, combined with dismal survival rates, have

stimulated new approaches to improve circulation during CPR. A re-examination of the 15:2 ratio, recognition that uninterrupted chest compressions are essential, and development of the ITD are new ways to increase vital organ perfusion during CPR (20). The results of this study clearly demonstrate that a C:V ratio of 30:2 significantly improves vital organ perfusion pressures, cardiac output, and common carotid artery blood flow and that BLS-certified rescuers perform CPR with a ratio of 30:2 as easily and as well as that with a 15:2 ratio.

Increasing the ratio from 15:2 to 30:2 generated a 30% higher cardiac output and doubled common carotid artery blood flow in the animal study. The higher number of compressions and fewer interruptions for ventilations contributed to this finding. It has been reported that the average rescuer in out-of-hospital resuscitation delivers two breaths over 14–16 seconds, potentially further decreasing the comparative number of compressions delivered with the 15:2 ratio (21).

ITD use improved every hemodynamic parameter in both ratios studied, with the largest benefit observed with a 30:2 ratio. The ITD has been reported in numerous studies to improve hemodynamics and survival outcomes for animals and humans (13–18, 22). These results support the hypothesis and recent computer modeling work showing that the ITD will further increase vital organ perfusion, even with higher C:V ratios and after prolonged resuscitative efforts (4)

There were no differences in arterial blood gases between the two ratios. Addition of an ITD at the 30:2 ratio resulted in higher Po<sub>2</sub>, lower pH, and higher Paco<sub>2</sub> as a consequence of a combination of better ventilation/perfusion match (shown from the low Paco<sub>2</sub> – ETCO<sub>2</sub> value) and relative hypoventilation. Hypoventilation with a ratio of 30:2 + ITD occurs because of the elimination of free gas exchange (occult ventilation) during each compression/decompression cycle caused by inspiratory impedance. The higher Paco<sub>2</sub> levels seen in this study have been shown to be beneficial for oxygen delivery to tissues, especially the brain (23–28). Improved pulmonary circulation and ventilation/perfusion match also contributed to the improvement in PaO<sub>2</sub> observed with the 30:2 + ITD treatment.

In this study, we also addressed the feasibility of implementing a ratio of 30:2 in clinical practice. Although the quality of CPR delivered with both ratios was poor, there was a significant increase in the number of chest compressions delivered with the 30:2 ratio, without decreased CPR performance or increased fatigue. This provides strong confirmatory evidence that a ratio of 30:2 is practicable and would be well tolerated and easily implemented by professional and nonprofessional BLS-certified rescuers.

## Limitations

There are limitations to the clinical study. First, the controlled CPR testing of BLS-certified rescuers was performed in a single city and with a limited number of subjects using a recording manikin. Second, ventilation was simulated to optimize measurement of chest compressions on the recording manikin. In actual practice, rescuers are likely to take >4 secs to deliver two breaths. As a result, this study probably underestimated the differences between the number of chest compressions delivered at the 15:2 and 30:2 ratios. Third, the comparison was limited to 5 mins of CPR, and for that reason we cannot exclude that longer periods of CPR might cause more fatigue when a 30:2 ratio is used.

There are limitations to the porcine animal study. First, the use of intubated animals in our model may have allowed for occult ventilation that might have influenced the blood gas results. However, there is no good established animal model to simulate the unique human upper-airway response to CPR with decom-

Table 3. Clinical study cardiopulmonary resuscitation (CPR) performance measurements (SEM)

CPR Component	Combined (n = 20)			Laypersons (n = 10)			EMT (n = 10)		
	15:2	30:2	p	15:2	30:2	p	15:2	30:2	p
Total compressions	339.8 (8.45)	401.80 (13.74)	<.001	338.6 (15.59)	395.20 (24.62)	<.001	341.00 (7.65)	408.40 (13.43)	<.001
Duty cycle	38.80 (1.07)	39.05 (1.33)	.69	38.3 (1.54)	37.70 (2.20)	.54	39.3 (1.55)	40.4 (1.47)	.15
%Complete recoil	42.32 (7.40)	37.56 (7.38)	.35	50.95 (11.92)	57.42 (10.56)	.42	33.69 (8.52)	17.70 (5.59)	.004
%Adequate depth	47.92 (8.19)	50.45 (8.11)	.67	44.76 (13.05)	40.18 (12.62)	.23	51.10 (10.53)	60.72 (9.76)	.40
Mean compression depth, mm	40.08 (2.06)	39.46 (1.98)	.47	36.95 (3.16)	37.34 (3.42)	.71	43.21 (2.41)	41.59 (1.95)	.24
%Hand placement accuracy	92.08 (4.54)	85.15 (6.41)	.09	92.44 (6.98)	88.91 (8.71)	.18	91.72 (6.20)	81.38 (9.72)	.20

Table 4. Clinical study fatigue measurements (SEM)

Fatigue Measurements	Combined (n = 20)			Lay person (n = 10)			EMT (n = 10)		
	15:2	30:2	p	15:2	30:2	p	15:2	30:2	p
Fatigue rating <sup>a</sup>	22.88 (3.23)	24.24 (3.62)	.59	25.89 (5.06)	27.61 (6.19)	.67	19.88 (4.07)	20.81 (3.8)	.76
Discomfort rating <sup>a</sup>	14.6 (2.99)	14.16 (2.98)	.87	18.76 (5.08)	18.91 (4.93)	.91	10.92 (2.99)	9.42 (2.86)	.5
Time to peak heart rate, sec	212.3 (14.97)	193.45 (17.55)	.38	219.9 (20.77)	204.1 (21.47)	.3	204.7 (22.41)	182.8 (28.54)	.61
Peak heart rate	136.55 (3.27)	138.15 (3.6)	.38	137.8 (5.86)	143.1 (5.94)	.028	135.3 (3.22)	133.2 (3.74)	.41
Time to return to baseline heart rate, sec	174.85 (42.63)	176.65 (27.61)	.97	243.2 (79.09)	238.5 (45.46)	.95	106.5 (19.54)	114.8 (17.37)	.76

<sup>a</sup>Rating on a Likert scale (0 = none; 25 = slight; 50 = moderate; 75 = severe; 100 = very severe).

pression obstruction. Moreover, there were no adverse effects on oxygenation with use of the 30:2 ratio and inspiratory impedance with the ITD, even though it was consistently applied late in this experimental model. Taken as a whole, this study's findings suggest that the 30:2 ratio provides acceptable gas exchange during resuscitation from cardiac arrest.

Second, we used delivery of oxygen-enriched tidal volumes to model professional resuscitation attempts. The study was not designed to evaluate survival. The return to spontaneous circulation rates reported is an observation with minimal clinical significance. Additional studies are needed to determine the effects of 30:2 ratio on long-term survival and neurologic outcomes.

## CONCLUSION

Increasing the C:V ratio to 30:2 during CPR improved all hemodynamic parameters in a porcine model of cardiac arrest, doubled common carotid artery blood flow and ETco<sub>2</sub>, improved cardiac output by 35%, and had no negative effects on oxygenation and acid-base balance. Cardiac output was further improved by 135% with the addition of an ITD, even

after prolonged CPR efforts. BLS-certified rescuers performed a 30:2 ratio without significant worsening in CPR quality or fatigue. These data strongly support the contention that a C:V ratio of 30:2 is superior to one of 15:2 during manual CPR and that the ITD further enhances circulation with both C:V ratios.

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