Comparison of 15:1, 15:2 and 30:2 Compression-to-Ventilation Ratios for Cardiopulmonary Resuscitation in an Animal Model of a Simulated, Witnessed Cardiac Arrest

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묵묵히, 변함없는 사랑을 주시면서 여지껏 버팀목이 되주시는 부모님 들께도 감사의 말씀을 드립니다. 마지막으로 사랑하는 아내 나영과, 예쁜 휴나, 보나에게도 고마움을 전합니다.

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Abstract

Comparison of 15:1, 15:2 and 30:2 Compression-to-Ventilation Ratios for Cardiopulmonary Resuscitation in an Animal Model of a Simulated, Witnessed Cardiac Arrest

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Background: The compression-ventilation (C:V) ratio of 30:2 that is recommended in the 2005 guidelines for cardiopulmonary resuscitation (CPR) and emergency cardiovascular care is based on expert consensus rather than on any clear scientific evidence. Further validation of this guideline is therefore required.

Purpose: This experimental study was conducted to compare the effect of C:V ratios of 15:2, 30:2 and 15:1 on hemodynamics and survival in a canine model of a simulated, witnessed VF cardiac arrest.

Subjects and Methods: Thirty healthy dogs irrespective of species (19.2 ± 2.2 kg) were used in this study. The dogs received CPR and were divided into 3 groups based on their C:V ratios of 15:1, 15:2 and 30:2. Hemodynamics, arterial blood gas analysis and survival were measured. After 1 minute of ventricular fibrillation, 4 minutes of BLS CPR, defibrillation and ACLS CPR were performed.

Results: In the 15:1 and 30:2 groups, compressions delivered over 1 min were significantly greater than that of the 15:2 group (73.1±8.1 and 69.0±6.9 to 56.3±6.8; p=0.000). The time for ventilation in which compression was stopped at each minute was significantly reduced in the 15:1 and 30:2 groups (15.4±3.9 and 17.1±2.7 to 25.2±2.6 sec/min; p=0.000). During BLS and ACLS CPR, all the hemodynamic parameters, acid-base status were not significantly different between the 3 groups. Eight dogs (80%) of each group achieved ROSC during BLS and ACLS. The survival rate was not different between the 3 groups. The median time to ROSC was shorter in the 15:1 group and the median survival time after ROSC was longer in the 30:2 group, but the differences were not significant between the 3 groups.

Conclusion: In an animal model of witnessed VF using a simulated scenario, CPR with three C:V ratios, 15:1, 15:2, and 30:2 did not show any differences in hemodynamics, ROSC rate and short-term survival rate among the three groups. CPR with a C:V ratio of 15:1 provided increased compression delivery, fewer pauses for ventilation, and less interruption of chest compression more than 5 sec.

Key Words: cardiopulmonary resuscitation, compression-ventilation ratio, hemodynamics, ROSC, survival

I. Introduction

The compression-ventilation (C:V) ratio of 30:2 that is recommended in the 2005 guidelines for cardiopulmonary resuscitation (CPR) and emergency cardiovascular care is based on expert consensus rather than on any clear scientific evidence.[1] Further validation of this guideline is therefore required.

Past adult CPR with a 15:2 C:V ratio is essentially the same as the normal ratio of heart rate to breathing in a quietly resting adult.[2] Ideally, the 15:2 C:V ratio is a 5 sec pause for ventilation following every 15 chest compressions, but in practice the interruption of chest compression for rescue breathing by lay rescuers requires about 16 sec.[3,4] The 5 sec pause for ventilation following every 15 chest compressions has been shown to reduce coronary perfusion pressure (CPP) by 50% and interrupting chest compressions for rescue breathing can adversely affect hemodynamics during CPR for ventricular fibrillation (VF).[5] Actual interruption of chest compressions of more than 5 sec for 2 ventilations can produce further adverse effects on hemodynamics. Chest compression-only CPR has shown to be as good as standard CPR for successful outcome,[6] but the advantage of increased blood flow is offset by increasing hypoxia after more than a few minutes of CPR, with progressively less oxygen delivered to the body tissues.[7] Higher C:V ratios may improve hemodynamics by decreasing interruptions for ventilation and delivering more compressions during CPR.[8,9] In a recent investigation, a ratio of 30:2 was superior to 15:2 during manual CPR.[10]

This experimental study was conducted to compare the effect of C:V ratiosof 15:2, 30:2 and 15:1 on hemodynamics and survival in a canine model of a simulated, witnessed VF cardiac arrest. First, we hypothesized that a C:V ratio of 30:2 would be superior to 15:2. A C:V ratio of 15:1 is the same as 30:2, but chest compression is interrupted for less than 5 sec for 1 ventilation. Our

second hypothesis was that a C:V ratio of 15:1 would be superior to 30:2 and 15:2 by decreasing the interruption time to less than 5 sec for 1 ventilation.

II. Subjects and methods

1. Subjects

Thirty healthy dogs irrespective of species (19.2 ± 2.2 kg) were used in this study. The dogs received CPR and were divided into 3 groups based on their C:V ratios of 15:1, 15:2 and 30:2.

2. Methods

1) Preparation

The dogs were initially sedated with 400 mg of intramuscular ketamine (Ketalar®, Yuhan Corp, Korea) and 0.3 mg/kg of intramuscular xylazine (Rompun®, Bayer Animal Health Corp, Korea), and further sedated with 200 mg of intravenous ketamine. The thoraces of all dogs were shaved by razor for application of the automated external defibrillator (AED) pads to the chest. After sedation, endotracheal intubation was done with a cuffed, 7.0-mm, French endotracheal tube. Electrocardiographic monitoring was recorded continuously. Under aseptic conditions, the dogs were positioned supine and right femoral artery cannulation was done using the Seldinger method. Central aortic blood pressures were recorded continuously, with a micromanometer-tipped catheter (5 Fr, Millar Instruments, Inc., Houston, TX). An introducer sheath (7.5 Fr, Arrow International Inc., USA) was placed in the right external jugular vein, and right atrial pressures were recorded continuously. An introducer sheath was placed in the right femoral vein for infusion of normal saline and intravenous medications. Left femoral artery cannulation was performed for arterial blood gas analysis. The right common carotid artery was surgically exposed, and an ultrasonic flow

probe (T106, Transonic systems Inc., USA) was placed around it to quantify blood flow. All dogs were treated with a heparin bolus (100 units/kg IV) once catheters were in place for avoiding thrombosis formation. During the preparation, the dog enclosures were ventilated with room air via a volume control ventilator (MDS Matrix 3000, Hallowell, USA). The tidal volume was initially set at 12 mL/kg and the ventilator rate at 12 breaths per min. An outline of the preparation is shown in Fig. 1.

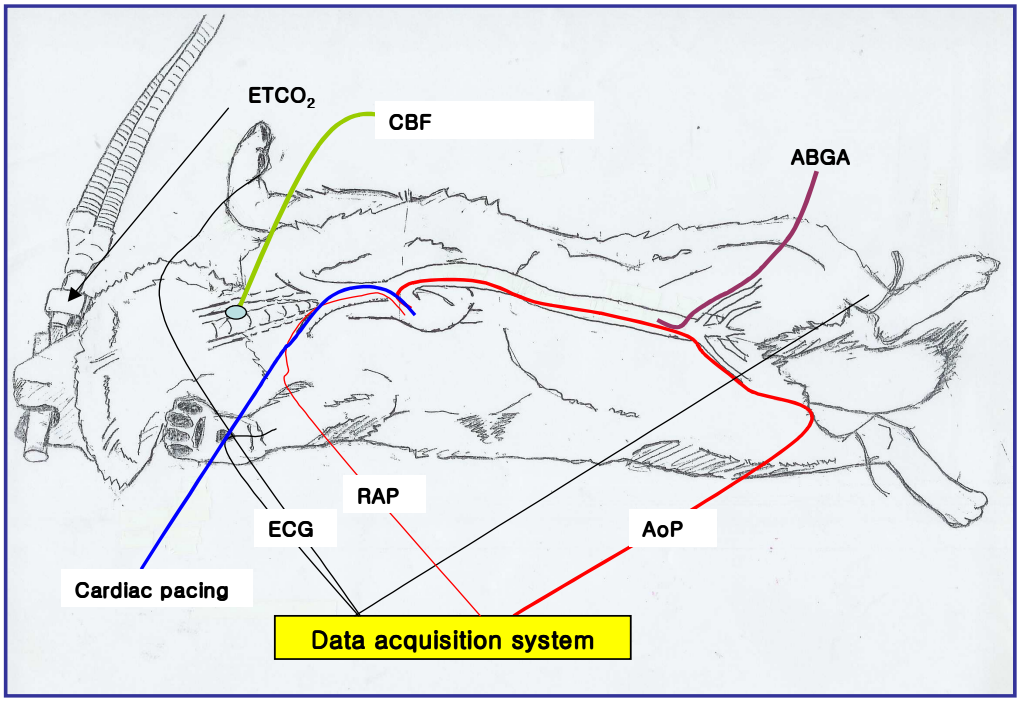

Fig. 1. Preparation and measurements of dogs

AoP: aortic pressure

RAP: right atrial pressure

CBF: cerebral blood flow

Pacing: pacing wire for delivering alternating current

ABGA: arterial blood gas analysis

$ETCO_2$: end-tidal CO_2

2) Measurements

Data were digitized by a digital recording system (Powerlab, AD Instruments, USA). All the parameters (aortic, right atrial, and common carotid blood flow) were recorded continuously and analyzedat baseline, between minutes 0 and 4 of CPR (1 to 5 minutes after VF cardiac arrest) and between minutes 4 and 14 of CPR (5 to 15 minutes after VF cardiac arrest), 2 hours after restoration of

spontaneouscirculation (ROSC) if ROSC was achieved. CPP during CPR was calculated by subtracting mid-diastolic right atrial pressure from mid-diastolic aortic pressure (Fig. 2).

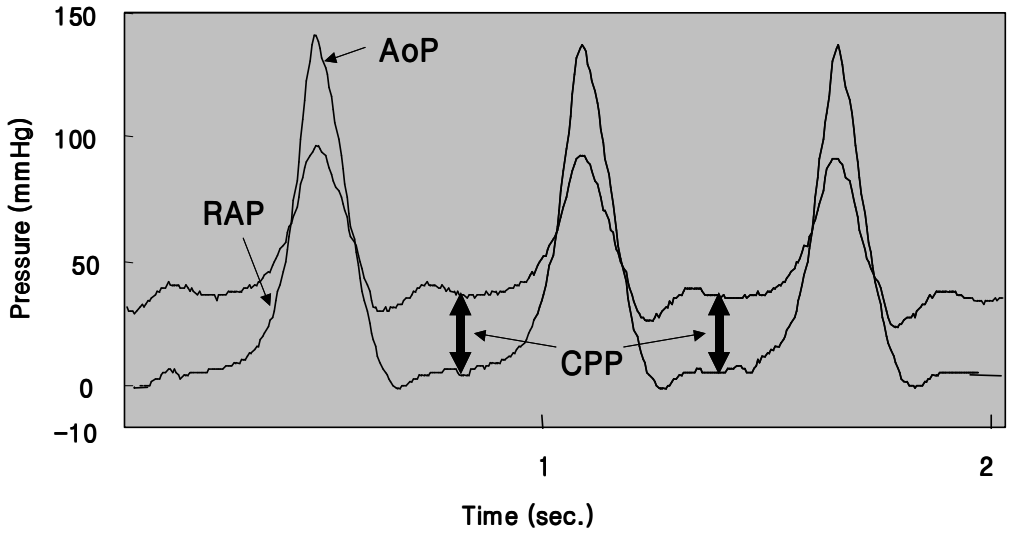

Fig. 2. Measurements of coronary perfusion pressures (CPP)

AoP: aortic pressure

RAP: right atrial pressure

CPP: coronary perfusion pressure

End tidal CO_2 ($ETCO_2$), tidal volumes, and oxygen saturation were continuously measured (CO_2SMO Plus®, Novametrix Medical Systems, Wallingford, CT). Arterial blood gas specimens were collected from the left femoral artery at baseline (before cardiac arrest), and at 1, 5, 9 and 15 minutes after cardiac arrest. Oxygen saturation, PCO_2, PO_2, pH, base excess, HCO_3^-, hemoglobin, and hematocrit were measured with a blood gas analyzer (i-STAT®1, Abbott Laboratories, Illinois, USA). Compressions delivered over 1 min, interruption time of compressions over 1 min and ventilation duration during basic life support (BLS) CPR were measured.

3) Experimental Protocol

After baseline data were collected, a pacing catheter (5 Fr, bipolar lead, Arrow International Inc., USA) was positioned in the right ventricle. VF was then induced by delivering alternating electrical current at 60 Hz to the endocardium and confirmed by the ECG waveform and a decline in aortic pressure. Once VF was induced, the ventilator was disconnected from the endotracheal tube. After 1-minute of untreated VF, mimicking the activity of a bystander recognizing cardiac arrest and calling for help, 4 minutes of BLS was performed. Positive bag valve ventilations were delivered with a resuscitator bag (Silicone resuscitator 870040, Laerdal, Norway) during BLS. The dogs were randomly assigned to three groups: (1) the 15:1 group, provided with arescue breath followed by 15 manual chest compressions at the metronome-guided rate of 100 compressions per minute, repeated sequentially, (2) the 15:2 group, provided with 2 manual rescue breaths followed by 15 manual compressions or (3) the 30:2 group, provided with 2 manual rescue breaths followed by 30 manual compressions. At the end of the 4-minute CPR, dogs were defibrillated with an AED (CU-ER1, CU Medical Systems Inc., Korea) delivering 80 J (≈4 J/kg) of energy. If VF persisted or asystole developed, 1mg of epinephrine was administered intravenously and all resuscitation efforts were started by the 2005 advanced cardiac life support (ACLS) pulseless arrest algorithm.[11] Shock delivery was performed every 2 minutes of CPR and 1 mg of epinephrine was administered intravenously every 3 minutes of CPR. ACLS CPR efforts were continued for 10 minutes unless ROSC was attained. ROSC was defined as an unassisted pulse with a systolic arterial pressure >50 mmHg and a pulse pressure >20 mmHg lasting >1 minute. If ROSC was not achieved despite all efforts for 10 minutes, the experiment was terminated. If ROSC was attained, dogs were observed for 2 hours of survival without any pharmacologic supports and then sacrificed by an intravenous injection of potassium chloride. The

experimental protocol is outlined in Fig. 3.

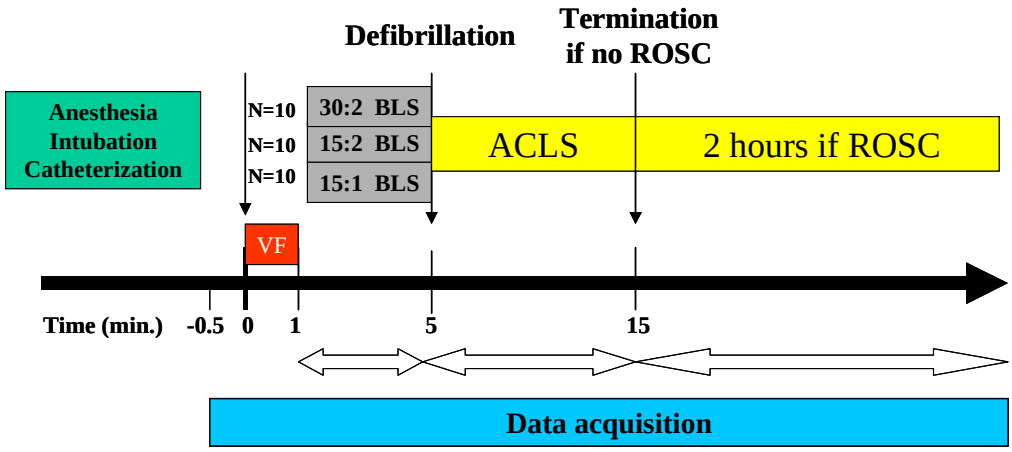

Fig. 3. Experimental protocol

VF: ventricular fibrillation

BLS: basic life support

ACLS: advanced cardica life support

3. Statistical analysis

Data were summarized as mean ± standard deviation (SD) and coded into a computerized data processing software package (SPSS for windows 12.0, SPSS Inc., Chicago, IL). One-way ANOVA was used when appropriate to compare the three groups with regard to the other aforementioned variables. Kruskal Wallis test was used to evaluate the differencesof survival time and time to ROSC between the three groups. Statistical differences of ROSC rate and 2-hour survival rate between the three groups were analyzed using the x^2-test. Independent t-test was used to compare two groups with ventilation time > 5 sec and < 5 sec. P-values below 0.05 were defined as statistically significant.

II. Results

1. Baseline demographic data of the dogs

The mean (±SD) weight of the dogs was 19.2 (±2.2 kg) with no significant difference between the 3 groups. Demographic baseline data of the dogs are presented in Table 1. There were no significant difference in systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MBP), right atrial pressure (RAP), CPP and cerebral blood flow (CBF) between the 3 groups before VF induction.

Table 1. Baseline demographic data of the dogs*

Characteristics	15:1 (n=10)	15:2 (n=10)	30:2 (n=10)	*P* value[a]
Weight (kg)	19.0±2.3	19.1±1.9	19.5±2.2	0.954
SBP[b] (mmHg)	136.3±25.6	123.2±27.2	129.7±26.2	0.546
DBP[c] (mmHg)	100.4±21.0	99.9±32.9	96.8±24.0	0.947
MBP[d] (mmHg)	112.3±21.9	107.7±25.4	107.8±24.4	0.882
RAP[e] (mmHg)	9.3±5.5	15.2±7.4	10.9±4.0	0.080
CPP[f] (mmHg)	99.0±20.3	96.0±32.9	94.8±23.6	0.934
CBF[g] (mL/min.)	88.7±26.5	74.3±24.5	72.8±19.6	0.248

* Mean±SD.

[a] one-Way ANOVA.

[b] SBP: systolic blood pressure.

[c] DBP: diastolic blood pressure.

[d] MBP: mean arterial pressure.

[e] RAP: right atrial pressure.

[f] CPP: coronary perfusion pressure.

[g] CBF: cerebral blood flow.

2. Comparison of 3 groups of C:V ratios for CPR

2.1 Hemodynamic parameters at BLS and ACLS CPR

During BLS and ACLS CPR, CBF tended to be higher in the 30:2 group than the other two groups, but the difference was not significant. None of the hemodynamic parameters were significantly different during BLS and ACLS CPR (Table 2).

Table 2. Hemodynamics at BLS and ACLS CPR*

Characteristics	15:1	15:2	30:2	*P* value[a]
BLS[b]				
SBP (mmHg)	85.7±18.7	94.0±21.0	71.7±18.6	0.546
DBP (mmHg)	25.2±7.2	31.3±20.0	24.5±10.8	0.947
MBP(mmHg)	42.1±8.5	46.6±21.7	37.5±15.6	0.467
RAP (mmHg)	96.4±33.1	78.6±19.0	82.5±13.0	0.217
CPP (mmHg)	22.4±13.7	22.1±19.5	17.3±12.8	0.722
CBF (mL/min.)	29.3±34.1	29.0±18.1	43.2±32.0	0.480
ACLS[c]				
SBP (mmHg)	83.6±23.6	90.0±30.2	101.5±30.1	0.451
DBP (mmHg)	25.5±10.6	30.4±18.1	27.4±17.3	0.810
MBP(mmHg)	44.9±13.8	50.3±21.1	52.1±17.7	0.707
RAP (mmHg)	85.5±49.4	75.4±26.8	86.1±20.0	0.768
CPP (mmHg)	18.3±13.0	20.9±24.8	18.9±16.1	0.956
CBF (mL/min.)	22.1±10.4	19.3±16.7	34.3±36.4	0.408

* Mean±SD.

[a] one-Way ANOVA.

[b] BLS: basic life support.

[c] ACLS: advanced cardiac life support.

2.2 Compression rate, pause of compression and ventilation duration during BLS CPR

In the 15:1 and 30:2 groups, compressions delivered over 1 min were significantly greater than that of the 15:2 group (73.1±8.1 and 69.0±6.9 to 56.3±6.8; p=0.000). The interruption of compression were reduced in the 15:1 and 30:2 groups (19.2±3.7 and 20.0±3.7 to 27.9±3.7 sec/min; p=0.000). The time for ventilation in which compression was stopped at each minute was significantly reduced in the 15:1 and 30:2 groups (15.4±3.9 and 17.1±2.7 to 25.2±2.6 sec/min; p=0.000). The time for ventilation at each cycle was reduced in the 15:1 group compared to that in the 15:2 and 30:2 groups (3.3±1.1 to 6.9±1.5 and 7.6±1.8 sec p=0.000) (Fig. 4).

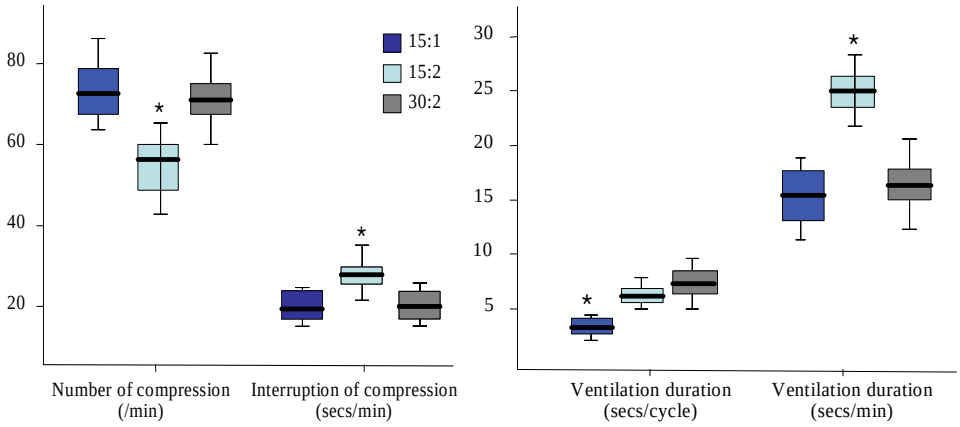

Fig. 4. Compression rate, interruption of chest compression and ventilation duration with 15:1, 15:2 and 30:2 C:V ratios during BLS. *Statistically significant differences between the 3 groups with $p < 0.05$.

2.3 ROSC and survival

Eight dogs (80%) of each group achieved ROSC during BLS and ACLS. The survival rate was not different between the 3 groups (Table 3). The median time to ROSC was shorter in the 15:1 group and the median survival time after ROSC was longer in the 30:2 group, but the differences were not significant between the 3 groups (Table 4).

Table 3. ROSC rate and 2 hours survival rate after ROSC*

Characteristics	15:1 (n=10)	15:2 (n=10)	30:2 (n=10)	*P* value[a]
ROSC[b]	8	8	8	1.000
Survival	6	6	7	0.866

* Mean±SD.

[a] x^2-test.

[b] ROSC: restoration of spontaneous circulation.

Table 4. Time to ROSC from ventricular fibrillation and survival time after ROSC*

Characteristics	15:1	15:2	30:2	*P* value[a]
Time to ROSC (sec)	405.4±182.2	465.6±195.9	432.5±264.3	0.298
Survival time (min)	95.3±45.9	95.9±45.1	117.8±6.4	0.690

* Mean±SD.

[a] Kruskal Wallis test.

2.4 Hemodynamic parameters after ROSC

There were no significant differencesin hemodynamics at immediately after ROSC, and at 30 and 60 minutes, and 2 hours after ROSC, between the 3 groups (Table 5).

Table 5. Hemodynamics after ROSC*

Characteristics	15:1	15:2	30:2	*P* value[a]
At ROSC				
SBP (mmHg)	94.1±24.5	79.3±26.7	80.1±29.0	0.436
DBP (mmHg)	50.6±43.6	49.0±35.8	37.2±28.0	0.671
MBP(mmHg)	65.4±28.4	64.7±33.1	64.9±31.8	0.999
RAP (mmHg)	18.7±24.1	32.1±37.6	31.0±43.2	0.654
CBF (mL/min.)	52.5±31.6	45.6±25.9	53.3±33.7	0.859
After 30 min				
SBP (mmHg)	64.6±32.2	80.9±31.7	75.0±26.1	0.596
DBP (mmHg)	39.9±27.9	48.3±25.8	42.4±23.8	0.822
MBP(mmHg)	49.5±35.4	51.4±29.3	51.3±29.1	0.992
RAP (mmHg)	9.1±5.0	12.7±7.1	10.0±5.1	0.492
CBF (mL/min.)	45.1±21.4	53.2±14.3	58.8±32.7	0.609
After 1 hours				
SBP (mmHg)	69.2±26.6	80.2±23.8	77.6±28.3	0.754
DBP (mmHg)	41.7±23.0	46.5±25.9	44.0±26.3	0.947
MBP(mmHg)	50.8±27.5	60.5±26.2	56.1±30.4	0.840
RAP (mmHg)	13.0±9.2	12.7±6.5	12.9±6.3	0.997
CBF (mL/min.)	45.8±21.8	48.4±27.3	59.4±34.0	0.672
After 2 hours				
SBP (mmHg)	92.7±21.9	78.6±35.5	103.3±15.8	0.256
DBP (mmHg)	58.8±17.8	42.0±33.1	60.4±14.9	0.338
MBP(mmHg)	75.6±17.9	64.5±32.7	71.9±23.7	0.671
RAP (mmHg)	10.0±5.1	12.0±7.2	14.2±7.6	0.576
CBF (mL/min.)	61.5±31.8	78.4±52.9	59.5±20.9	0.620

* Mean±SD.

[a] one-Way ANOVA.

2.5 Arterial blood gas analysis

There were no significant differences in arterial blood gas analysis at the time of baseline, immediately after VF and first defibrillation, 9 and 15 minutes after initiation of experiment between the 3 groups (Table 6).

Table 6. Arterial Blood Gases*

Characteristics	15:1	15:2	30:2	*P* value[a]
Baseline, before VF				
pH	7.50±0.13	7.46±0.18	7.48±0.16	0.861
SO_2	97.6±2.9	97.6±2.4	98.4±0.5	0.728
PO_2	101.5±18.1	100.7±20.4	99.4±11.4	0.968
PCO_2 (mmHg)	16.2±8.2	17.0±9.7	14.1±3.9	0.745
HCO_3^- (mmol/L)	11.8±3.1	11.5±4.1	10.5±2.6	0.693
Hemoglobin (g/dL)	8.6±2.5	8.5±3.3	9.6±4.6	0.812
1 min, VF				
pH	7.54±0.13	7.52±0.18	7.49±0.11	0.753
SO_2	96.3±3.9	97.7±2.1	97.9±0.6	0.406
PO_2	82.3±20.1	92.1±15.8	92.3±14.2	0.436
PCO_2 (mmHg)	13.7±6.8	10.2±3.4	12.1±4.5	0.432
HCO_3^- (mmol/L)	10.0±2.8	8.7±3.7	7.7±3.5	0.409
Hemoglobin (g/dL)	10.1±2.6	8.2±3.2	10.1±4.2	0.479
5 min, at defibrillation				
pH	7.34±0.15	7.31±0.16	7.39±0.13	0.538
SO_2	87.3±11.3	82.4±15.9	89.6±7.7	0.506
PO_2	63.8±16.3	63.1±27.4	66.8±23.1	0.945
PCO_2 (mmHg)	15.5±7.8	17.7±9.6	14.7±4.2	0.725
HCO_3^- (mmol/L)	7.7±3.6	8.6±3.7	8.5±1.5	0.850
Hemoglobin (g/dL)	9.9±10.9	10.9±4.0	10.1±4.3	0.876
9 min, during ACLS				
pH	7.29±0.20	7.17±0.14	7.25±0.18	0.303
SO_2	91.1±9.8	80.1±19.3	88.3±6.9	0.291
PO_2	80.7±18.4	65.0±23.0	66.3±14.8	0.256
PCO_2 (mmHg)	18.1±8.7	25.9±16.6	15.9±2.6	0.218
HCO_3^- (mmol/L)	7.9±1.6	8.7±3.5	7.0±1.7	0.384
Hemoglobin (g/dL)	10.2±2.1	9.9±3.6	10.5±3.4	0.937
15 min				
pH	7.25±0.15	7.14±0.13	7.22±0.12	0.261

SO_2	91.0±8.4	85.9±10.0	85.3±15.6	0.579
PO_2	79.0±19.4	75.1±23.3	67.0±17.8	0.491
PCO_2 (mmHg)	15.9±6.85	19.5±8.5	15.6±5.9	0.511
HCO_3^- (mmol/L)	6.6±1.0	6.4±2.9	6.1±1.5	0.893
Hemoglobin (g/dL)	9.1±2.1	8.5±3.0	9.5±3.2	0.801

* Mean±SD.

[a] one-Way ANOVA.

2.6 Comparison of Defibrillation

The success rate of defibrillation was 70% in the 15:1 group, and 60% in the 15:2 and 30:2 groups at the first shock. There were no significant differencesin the success rate of defibrillation during the ACLS CPR and hemodynamics during the compressions before defibrillation between the 3 groups (Table 7).

Table 7. Hemodynamics during compressions before defibrillation*

Characteristics	15:1	15:2	30:2	*P* value[a]
1st defibrillation	(n=10)	(n=10)	(n=10)	
SBP (mmHg)	89.3±28.2	89.0±32.6	86.6±31.3	0.977
DBP (mmHg)	23.0±10.4	25.9±15.3	20.4±6.5	0.560
MBP(mmHg)	45.1±13.2	46.9±20.2	42.3±10.2	0.830
RAP (mmHg)	79.0±35.5	73.5±30.4	81.1±33.1	0.869
CPP (mm Hg)	25.4±15.0	25.3±16.4	20.1±8.6	0.620
CBF (mL/min.)	13.2±14.4	23.0±22.1	33.5±29.4	0.157
2nd defibrillation	(n=3)	(n=4)	(n=4)	
SBP (mmHg)	76.0±24.3	84.3±39.5	96.8±13.5	0.634
DBP (mmHg)	22.7±15.0	27.3±26.8	38.8±7.4	0.515
MBP(mmHg)	40.4±18.0	46.3±31.0	58.1±9.2	0.564
RAP (mmHg)	51.7±28.3	72.5±37.1	89.3±11.6	0.264
CPP (mmHg)	18.7±13.8	27.5±33.0	35.8±8.3	0.613
CBF (mL/min.)	5.7±7.2	13.5±8.7	33.3±18.4	0053
3rd defibrillation	(n=3)	(n=2)	(n=2)	
SBP (mmHg)	72.7±23.2	82.0±14.1	107.0±39.6	0.441
DBP (mmHg)	17.7±15.5	32.5±20.5	25.0±9.9	0.623
MBP(mmHg)	36.0±18.1	49.0±18.4	52.3±20.0	0.616
RAP (mmHg)	44.3±20.8	64.0±9.9	98.5±38.9	0.170

CPP (mmHg)	14.7±12.0	31.5±26.2	30.5±16.3	0.533
CBF (mL/min.)	12.0±19.1	6.5±2.1	29.5±17.7	0.408
4th defibrillation	(n=3)	(n=2)	(n=2)	
SBP (mmHg)	72.3±23.7	77.5±19.1	87.5±41.7	0.848
DBP (mmHg)	19.0±15.5	34.0±24.0	18.0±5.7	0.582
MBP(mmHg)	36.8±17.8	48.5±22.4	41.2±17.7	0.806
RAP (mmHg)	47.0±31.4	59.5±3.5	72.5±29.0	0.612
CPP (mmHg)	15.3±12.2	29.5±31.8	20.5±19.1	0.764
CBF (mL/min.)	7.0±6.6	4.0±4.2	18.5±16.3	0.365
5th defibrillation	(n=3)	(n=2)	(n=2)	
SBP (mmHg)	69.3±34.0	78.5±31.8	90.0±18.4	0.769
DBP (mmHg)	16.0±16.1	29.0±25.5	15.0±4.2	0.674
MBP(mmHg)	33.8±45.5	45.5±27.6	40.0±3.3	0.831
RAP (mmHg)	40.3±22.5	55.5±4.9	79.0±7.1	0.142
CPP (mmHg)	14.0±15.4	29.0±29.7	21.5±7.8	0.703
CBF (mL/min.)	6.7±9.8	5.5±3.5	34.0±28.3	0.227
6th defibrillation	(n=2)	(n=2)	(n=2)	
SBP (mmHg)	68.5±43.1	68.0±46.7	98.5±43.1	0.754
DBP (mmHg)	18.5±23.3	29.5±29.0	20.0±5.7	0.866
MBP(mmHg)	35.2±29.9	42.3±34.9	46.2±18.2	0.928
RAP (mmHg)	28.5±7.8	41.5±13.4	83.5±31.8	0.144
CPP (mmHg)	14.5±19.1	26.0±29.7	27.5±20.5	0.842
CBF (mL/min.)	9.0±11.3	4.0±0.0	30.0±28.3	0.407

* Mean±SD.

[a] one-Way ANOVA.

The defibrillation success rate was 75% (18/24) and 67% (4/6) in the ROSC group, 83.3% (5/6) and 0% (0/5) in non-ROSC group at the first and second shocks, respectively. SBP of the ROSC group tended to be higher than that of the non-ROSC group after the second shock, but there were no significant difference in hemodynamics during the compression before defibrillation between the two groups (Table 8).

Table 8. Comparison of hemodynamics between ROSC and non-ROSC group during compression before defibrillation*

Characteristics	ROSC	non-ROSC	*P* value[a]
1st defibrillation	(n=24)	(n=6)	
SBP (mmHg)	86.1±25.7	97.2±44.4	0.424
DBP (mmHg)	23.0±9.6	23.7±17.3	0.892
MBP(mmHg)	44.0±13.2	48.2±23.4	0.544
RAP (mmHg)	76.5±30.2	83.5±41.4	0.639
CPP (mmHg)	24.3±12.1	21.0±19.2	0.607
CBF (mL/min.)	26.0±24.8	12.0±14.5	0.197
2nd defibrillation	(n=6)	(n=5)	
SBP (mmHg)	98.5±27.1	72.2±20.0	0.106
DBP (mmHg)	38.7±18.3	20.0±12.5	0.086
MBP(mmHg)	58.6±21.2	37.4±14.7	0.093
RAP (mmHg)	81.2±35.7	63.0±17.9	0.330
CPP (mmHg)	34.7±24.2	20.2±14.6	0.275
CBF (mL/min.)	24.2±20.0	11.8±10.0	0.242
3rd defibrillation	(n=2)	(n=5)	
SBP (mmHg)	117.0±25.5	72.4±14.1	0.026
DBP (mmHg)	33.5±2.1	20.2±15.9	0.315
MBP(mmHg)	61.3±7.1	37.6±15.1	0.096
RAP (mmHg)	73.5±74.2	62.2±8.8	0.709
CPP (mmHg)	34.5±10.6	19.8±17.9	0.340
CBF (mL/min.)	38.0±5.7	6.4±6.7	0.002
4th defibrillation	(n=2)	(n=5)	
SBP (mmHg)	102.0±21.2	68.6±19.1	0.097
DBP (mmHg)	28.0±8.5	21.0±18.0	0.634
MBP(mmHg)	52.7±1.4	36.9±17.7	0.287
RAP (mmHg)	56.0±52.3	58.6±14.7	0.912
CPP (mmHg)	30.0±5.7	17.2±20.3	0.442
CBF (mL/min.)	22.0±11.3	4.4±3.1	0.015
5th defibrillation	(n=2)	(n=5)	
SBP (mmHg)	103.5±0.7	67.6±24.2	0.104
DBP (mmHg)	23.0±15.6	18.0±17.1	0.736
MBP(mmHg)	49.8±10.6	34.5±19.1	0.349
RAP (mmHg)	54.0±42.4	56.4±16.4	0.909
CPP (mmHg)	29.0±2.8	17.0±19.2	0.443
CBF (mL/min.)	36.0±25.5	5.4±5.6	0.032
6th defibrillation	(n=2)	(n=4)	
SBP (mmHg)	114.0±21.2	60.5±30.8	0.098
DBP (mmHg)	29.5±7.8	19.3±21.3	0.564
MBP(mmHg)	57.7±1.9	33.0±24.2	0.246

RAP (mmHg)	64.5±58.7	44.5±13.9	0.507
CPP (mmHg)	35.0±9.9	16.5±20.9	0.319
CBF (mL/min.)	33.5±23.3	4.8±3.8	0.052

* Mean±SD.

[a] one-Way ANOVA.

IV. Discussion

This investigation compared the effect of different C:V ratios of 15:1, 15:2, and 30:2 in a caninemodel of a witnessed VF cardiac arrest with a simulated scenario. We formulated a scenario of resuscitation with BLS and an AED shock performed by a bystander followed by full support of ACLS by healthcare providers. The scenario comprised of one minute of no-CPR period for checking the response of the victim and activating the EMS system, 4 minutes of BLS CPR and the first defibrillation shock with an AED mimicking immediate response of a bystander, and 10 minutes of ACLS CPR mimicking treatment by healthcare providers. This scenario for experimental protocol represented a typical emergency medical response including the early BLS CPR and defibrillation within 5 minutes by bystander, and ACLS CPR by healthcare providers. The study results revealed no differences in hemodynamics, arterial blood gas profiles, and resuscitation outcome with this scenario of resuscitation between the 15:1, 15:2, and 30:2 C:V ratio groups.

Survival from prehospital cardiac arrest is critically dependent upon response time.[12] The three-phase CPR model, which reflects the time-sensitive progression of resuscitation physiology, includes electrical, circulatory and metabolic phases.[13] Early defibrillation is the most important therapy in the electrical phase, which extends from the time of cardiac arrest to approximately 4 minutes following the arrest. Early CPR followed by defibrillation is the most crucial lifesaving therapy in the circulatory phase, which extends from 4 minutes to approximately 10 minutes after cardiac arrest. During the metabolic phase (after 10 minutes of cardiac arrest), the effectiveness of both immediate defibrillation and CPR followed by defibrillation decreases rapidly and survival rates appear poor. Data from animal experiments support the concept of a circulatory phase, in which chest compression takes priority over

defibrillation.[14-19] Yakaitis et al[20] showed that immediate defibrillation was only optimal when performed within 3 minutes or less. In clinical studies, Cobb et al[21] showed that immediate defibrillation was superior to providing 90 sec of CPR within the first 3 minutes following cardiac arrest, but that after 3 minutes, providing 90 sec of CPR followed by defibrillation was superior. However, immediate defibrillation within 4 to 5 minutes is rarely perform in real resuscitation situations because an AED is not immediately available at most out-of-hospital cardiac arrests. Even if an AED is present, the clinically relevant time intervals were a mean of 3.5 minutes from collapse to AED attachment, and a further 0.9 minutes to the delivery of the first defibrillation shock.[22] Therefore, an experimental protocol for a witnessed, out-of-hospital, cardiac arrest is more realistic and relevant to the real clinical situation.

In this study, the compressions delivered over 1 minute were greater in the 30:2 and 15:1 groups than in the 15:2 group, and pauses for ventilation over 1 minute were reduced in the 30:2 and 15:1 groups. The mean ventilation duration over 1 minute was greater in the 15:2 group than in the 30:2 or 15:1 groups during BLS CPR. Interrupting chest compression for ventilation per cycle was less than 5 sec in the 15:1 group but more than 5 sec in the 15:2 and 30:2 groups. Interruption of chest compression of more than 5 sec might reduce the probability of resuscitation.[23] Among the three C:V ratios in this study, only 15:1 provided increased compression delivery, fewer pauses for ventilation, and less interruption of chest compression more than 5 sec. We therefore propose a C:V ratio of 15:1 for BLS CPR.

The American Heart Association Guidelines recommend that rescuers spend 4 ~ 5 sec for the interval for 2 rescue breaths. However, it has been reported that the average rescuer in out-of-hospital resuscitation delivers two breaths over 14-16 sec, potentially further deteriorating the hemodynamics.[3] Rescuers are unwilling to perform mouth-to-mouth ventilation due to concerns of contracting

infectious diseases,[24,25] which has lead to concern about the optimum C:V ratio during CPR. Mathematical analysis determined an optimum C:V ratio of near 30:2 for standard performance and 60:2 for actual lay rescuer performance in the field.[3] Based on theoretical analysis, Babbs and Kern[8] proposed that a ratio of 30:2 would be ideal with interruption in chest compressions of 2.5 sec per breath. Dorph et al[26] showed that a ratio of near 30:2 might be optimal for standard CPR in a study of three different C:V ratios. Changing the ratio from 15:2 to 30:2 increased cardiac output by 30% and doubled common carotid artery blood flow in an animal study.[10] This finding was due to increased compression and fewer interruptions for ventilations.

Our investigation showed that arterial blood gas profiles, including oxygen saturation, PO2, and PCO2, were not significantly different between the 3 groups during BLS and ACLS. This finding suggests that CPR can maintain adequate arterial oxygenation with any C:V ratio among 15:1, 15:2, or 30:2. Hypoxia and hypercarbia are important mediators of poor VF outcome.[27,28] Therefore, preventing hypoxia, hypercarbia and maintainingblood flow is significant for ROSC. Based on simulation study, Turner et al[7] suggested that an optimal C:V ratio of 20:1 in terms of blood flow, oxygen delivery and carbon dioxide clearance over a range of clinical settings and inspired oxygen concentrations. Dorph et al[26] showed that a C:V ratio of 15:2 with ideal basic CPR had a better pulmonary gas exchange and cerebral oxygen delivery than higher C:V ratios, including 50:5 or 50:2.

Our study did not include a group of continuous chest compression CPR (no ventilation group). Oxygen delivery depends both on blood flow, which is generated by compressions, and its oxygen content, which is increased by ventilation during CPR. It hasbeen demonstrated that maximal blood flow generated by continuous chest compressions is offset by ongoing hypoxia after more than a few minutes of CPR.[13,29]

Our study suffered some limitations. First, pauses for ventilation were not identical to those in a real resuscitation situation, although our experimental scenario did simulate a witnessed cardiac arrest with bystander CPR. Therefore, the data from our study cannot be extrapolated to layperson CPR. Secondly, our scenario simulated an ideal response to the victim with out-of-hospital cardiac arrest witnessed by bystanders. Not all emergency medical systems experience this kind of emergency response to victims with out-of-hospital cardiac arrest. Not all bystanders can perform CPR or defibrillate the victim with an AED. Therefore, our study results are limited to the situation when a bystander is familiar with CPR, AED is available and the emergency medical response is adequate.

V. Conclusion

In an animal model of witnessed VF using a simulated scenario, CPR with three C:V ratios, 15:1, 15:2, and 30:2 did not show any differences in hemodynamics, ROSC rate and short-term survival rate among the three groups. CPR with a C:V ratio of 15:1 provided increased compression delivery, fewer pauses for ventilation, and less interruption of chest compression more than 5 sec.

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Abstract in Korean

목격된 심실세동에 의한 동물 심정지 모델에서 15:1, 15:2, 30:2 의 흉부 압박 - 인공 호흡 비에 의한 심폐소생술 효과 비교

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김선휴

배경 및 목적 : 2005년 개정된 미국 심장 협회 심폐소생술 지침서에 따르면 흉부 압박 대 인공 호흡 비율을 30:2로 정하고 있다. 이러한 30:2 의 비율은 확실한 과학적 근거 보다는 전문가에 의한 합의하에 개정되었기에 이에 대한 추가적인 과학적 근거가 필요한 실정이다. 본 연구의 목적은 목격자가 있는 심실세동에 의한 심정지 조건에서, 기존의 15:2와 개정된 30:2, 추가적으로 30:2와 흉부 압박 대 인공 호흡 비율이 같은 15:1의 흉부 압박 대 인공 호흡 비율이 다른 세 군 간의 혈역동학적 지수, 생존률에 대해서 비교해보고자 하였다.

대상 및 방법 : 종에 관계 없이 건강한 30마리의 개(19 ± 2.6 kg)를 실험동물로 사용하였다. 마취후 기도 삽관을 하고 인공 호흡을 시행하였고 대동맥과 우심방에 도자를 삽입하여 대동맥압과 우심방압을 측정하였다. 우측 경동맥을 노출하여 뇌혈류량계를 장치하였고, 우심실에 인공 심박조율기를 삽입하여 직류 전원에 의한 심실 세동을 유도하였다. 각 실험군에 대해 혈압, 우심방압, 경동맥 뇌혈류량, 관상동맥 관류압, 호기말 이산화탄소분압 등의 혈역학적 지수를 측정하였고 동맥혈 가스 검사를 시행하였다. 심실 세동 유발후 1분간 방치하였고 이후에 무작위로 세 군

(15:1, 15:2, 30:2)으로 나누어 흉부 압박과 인공 호흡으로만 구성된 기본 심폐소생술을 4분간 시행하였다. 4분후 심전도를 확인하여 심실세동이 지속되고 있으면 자동제세동기에 의한 제세동을 1회 시행하였고, 이후 심실세동이 지속되고 있으면 약물 투여를 포함하는 전문 심장 구조술에 의한 심폐소생술을 10분간 진행하였고 중간에 매 2분 마다 심전도를 확인하여 제세동 여부를 결정하였다. 자발 순환이 회복되면 그 시점부터 하여 2시간 생존률을 측정하였다.

결과 : 15:1 과 30:2 군에서 1분간 시행된 흉부 압박 횟수가 15:2 군보다 많았고 (73.1±8.1 and 69.0±6.9 to 56.3±6.8; p=0.000), 인공 호흡을 위해 흉부 압박 시행이 중단 되었던 시간이 15:1 과 30:2 군에서 15:2 군보다 짧았다(15.4±3.9, 17.1±2.7 vs 25.2±2.6 secs/min; p=0.000). 기본 심폐 소생술과 전문 심장 구조술을 진행하는 동안의 혈역학적 지수, 산-염기 상태는 세 군간에 큰 차이는 없었다. 각 군 모두 8 마리(80%)에서 기본 심폐소생술과 전문 심장구조술을 진행하는 동안 자발 순환이 회복되었고, 2시간 생존률은 세 군간에 차이는 없었다. 자발 순환이 회복되기까지 평균 소요 시간은 15:1 군에서 짧았고, 자발 순환 회복후 평균 생존 시간은 30:2 군에서 길었지만 세 군간에 유의한 차이는 없었다.

결론 : 목격된 심실세동에 의한 심정지시 15:1, 15:2, 30:2 의 흉부 압박 대 인공 호흡 비율로 심폐 소생술을 시행하였을 때 세 군간에 혈역학적 지수, 자발 순환 회복, 생존률에 있어 큰 차이는 없었다. 기본인명구조술상의 심폐소생술 시행에서 1분간 시행된 흉부압박 횟수는 15:1, 30:2 군에서 15:2 군보다 많았다.

Key Words: cardiopulmonary resuscitation, compression-ventilation ratio, hemodynamics, ROSC, survival