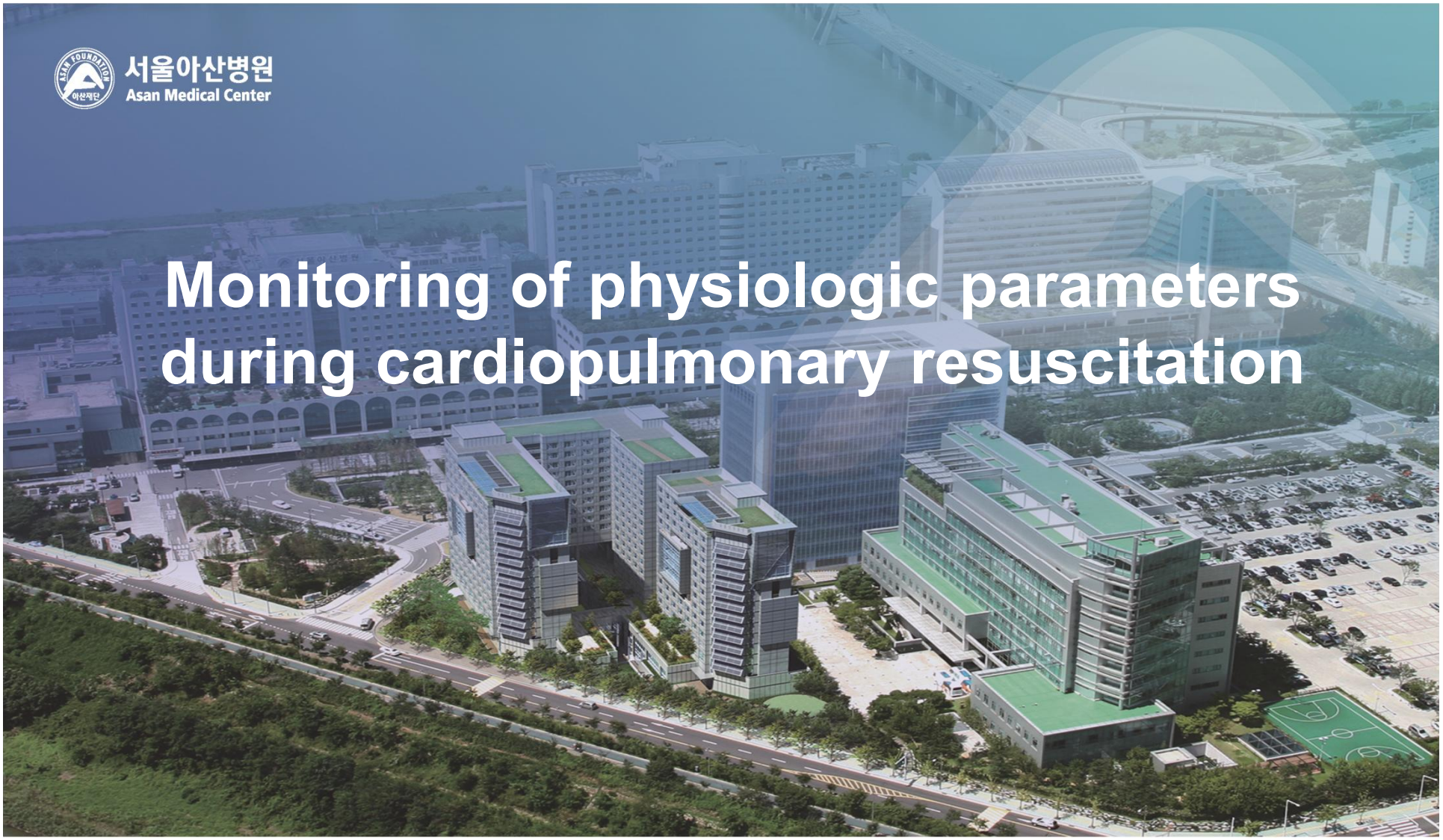


Won Young Kim, MD, PhD.

Asan Medical Center

Monitoring parameters during CPR (2010)

Real-time monitoring and optimization of CPR quality

1) ECG leads and pulse checks
2) End-tidal CO_2
3) Coronary perfusion pressure (CPP)
4) Central venous oxygen saturation (ScvO2)
5) Pulse oximetry
6) Arterial blood gases
7) Echocardiography

Pulse

- ❖ Palpate arterial pulses during chest compressions to assess the effectiveness of compressions ??

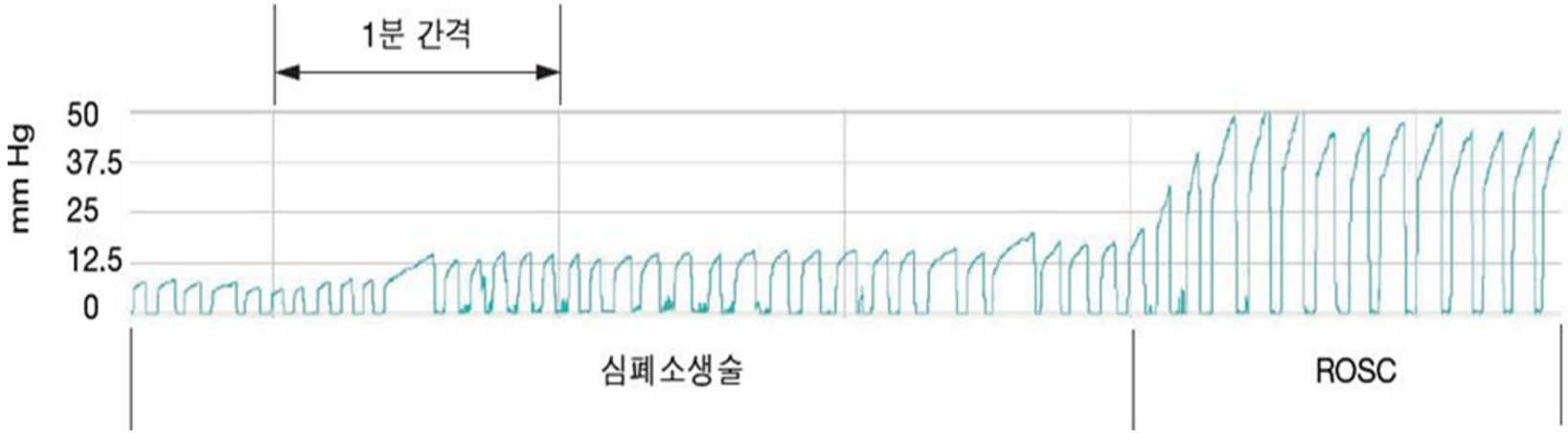
- ❖ Femoral pulse: retrograde blood flow may produce femoral vein pulsations → no value

- ❖ Carotid pulsations
  - during CPR: not indicate the efficacy of perfusion
  - paused CPR: a reliable indicator of ROSC

End-tidal CO_2

- as a partial pressure in mmHg ($ETCO_2$): 35-40 mmHg
  cardiac arrest: 0
  CPR: cardiac output is the major determinant

- If ventilation is relatively constant,
  $ETCO_2$ correlates well with cardiac output during CPR



End-tidal CO_2

❖ **$ETCO_2$<10 mmHg**
: trying to improve CPR quality by optimizing chest compression parameters (Class IIb, LOE C)

❖ If **$ETCO_2$ abruptly increases** to a normal value (35-40 mmHg): an indicator of ROSC (Class IIa, LOE B)

- Non-intubated patients(supraglottic airway):
  to monitor and optimize CPR quality and detect ROSC is uncertain (Class IIb, LOE C)

End-tidal CO_2

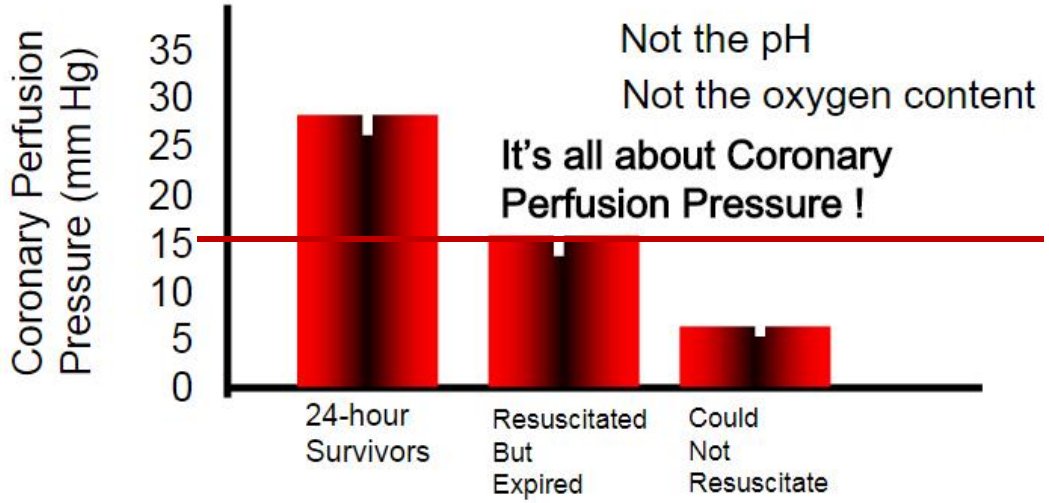
- specific target $ETCO_2$ value has not been established
- Confounding factors
  : arrest cause, minute ventilation, pulmonary pathology
- $ETCO_2$ transient increase
  : sodium bicarbonate, vasopressors
- Recommendations
  : against using $ETCO_2$ cutoff values alone as a mortality predictor or for the decision to stop a resuscitation (strong recommendation, low-quality evidence)

Coronary Perfusion Pressure

- CPP (coronary perfusion pressure)= aortic relaxation[“diastolic”] pressure - right atrial relaxation [“diastolic”] pressure) during CPR correlates with both myocardial blood flow and ROSC

  rarely available: require aortic and central venous pressure

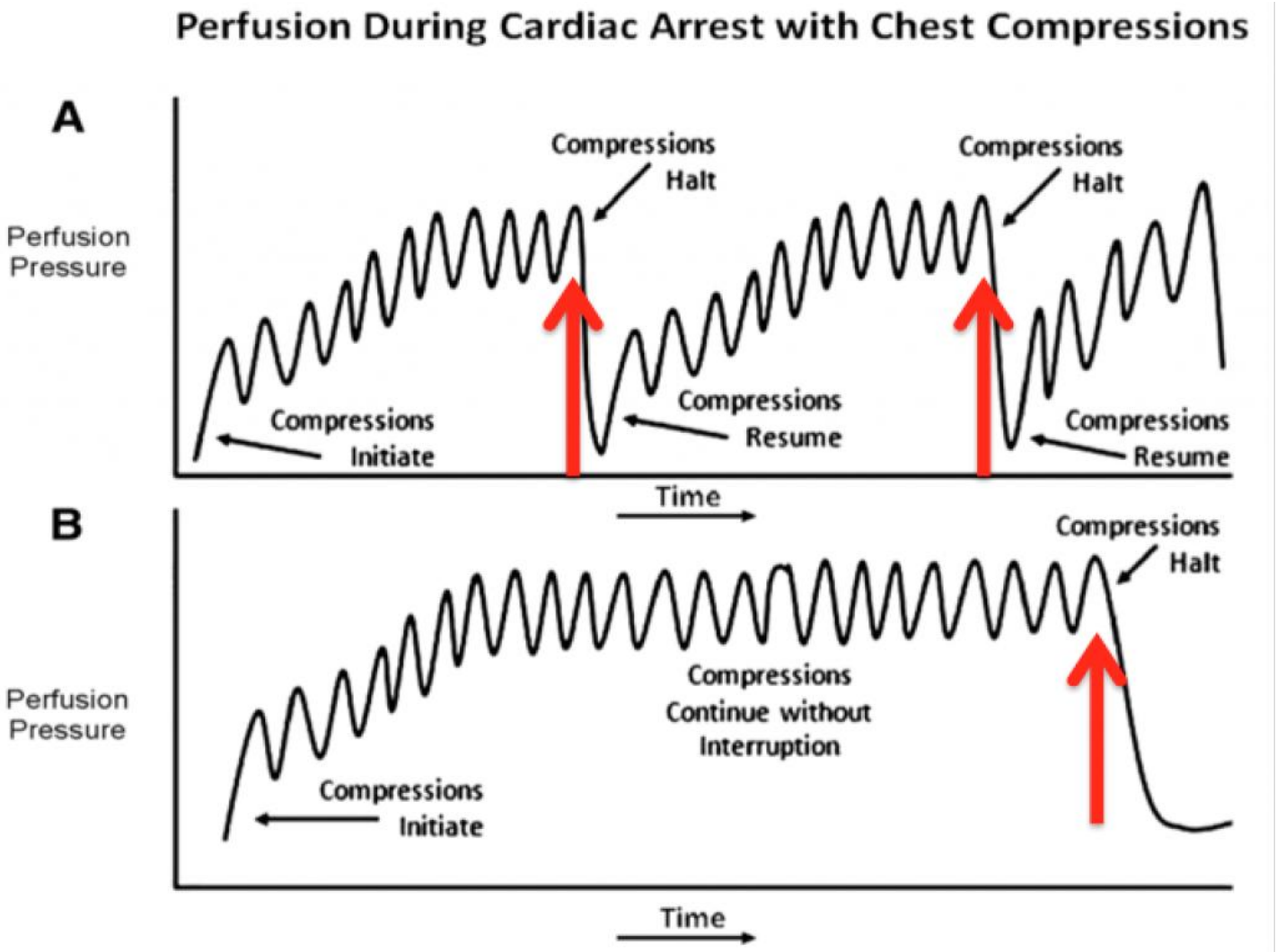
  Survival is related to CPP generated by chest compressions



Kern, Ewy, Voorhees, Babbs, Tacker *Resuscitation* 1988; 16: 241-250

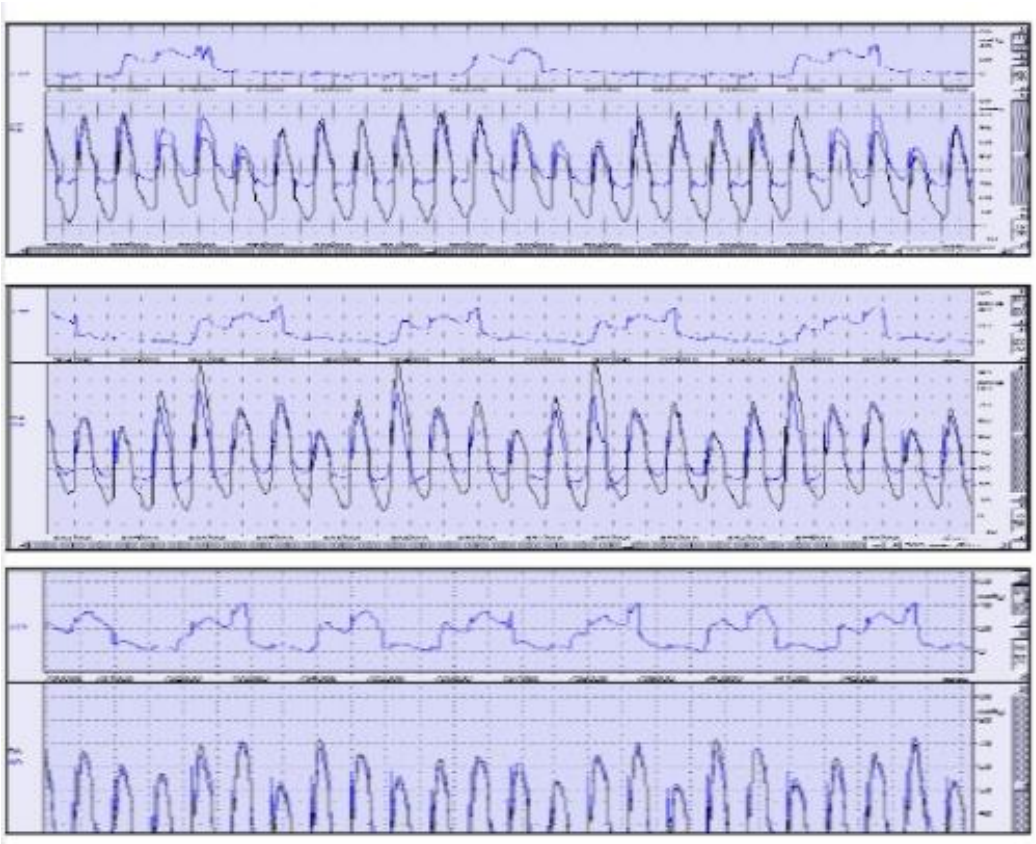
Paradis *et al. JAMA* 1990; 263:1106

Coronary Perfusion Pressure



Coronary Perfusion Pressure

❖ Effect of ventilatory rate on CPP



12 RR /minute

CPP 23.4 ± 1.0mmHg

MIP 7.1 ± 0.7 mmHg/min

20 RR /minute

CPP 19.5 ± 1.8 mmHg
MIP 11.6 ± 0.7
mmHg/min

30 RR /minute

CPP 16.9 ± 1.8 mmHg
MIP 17.5 ± 1.0
mmHg/min

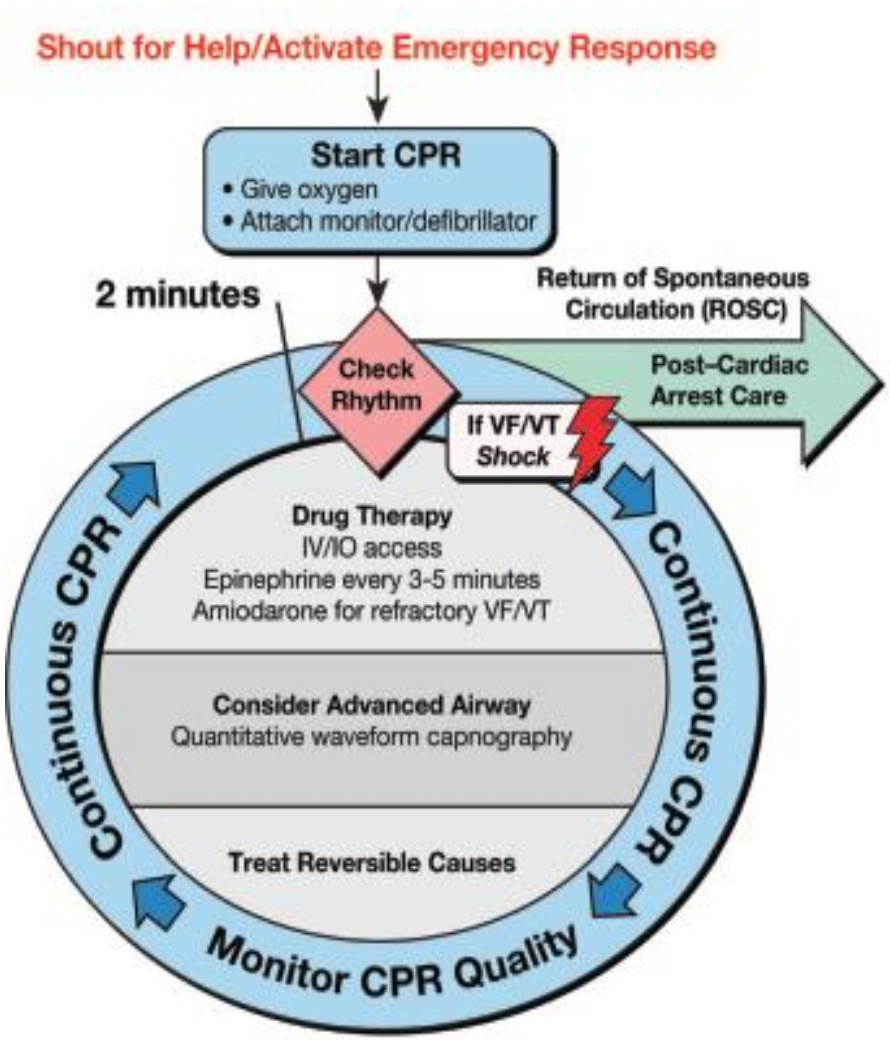
Coronary Perfusion Pressure (≒ Arterial Relaxation Pressure)

- ROSC did not occur unless a CPP≥15 mmHg was achieved during CPR

- Arterial relaxation (diastolic) pressure
  : surrogate for CPP during CPR
  ROSC was not achieved if diastolic pr. < 17mmHg

- "diastolic" pressure to monitor CPR quality, optimize chest compressions, and guide vasopressor therapy (Class IIb, LOE C). If "**diastolic" pressure <20 mmHg**, it is reasonable to consider trying to improve quality of CPR (Class IIb, LOE C)

Adult Cardiac Arrest



CPR Quality
- Push hard (≥2 inches [5 cm]) and fast (≥100/min) and allow complete chest recoil
- Minimize interruptions in compressions
- Avoid excessive ventilation
- Rotate compressor every 2 minutes
- If no advanced airway, 30:2 compression-ventilation ratio
- Quantitative waveform capnography
  - If P_{ETCO_2} <10 mm Hg, attempt to improve CPR quality
- Intra-arterial pressure
  - If relaxation phase (diastolic) pressure <20 mm Hg, attempt to improve CPR quality

Return of Spontaneous Circulation (ROSC)
- Pulse and blood pressure
- Abrupt sustained increase in P_{ETCO_2} (typically ≥40 mm Hg)
- Spontaneous arterial pressure waves with intra-arterial monitoring

Shock Energy
- **Biphasic:** Manufacturer recommendation (120-200 J); if unknown, use maximum available. Second and subsequent doses should be equivalent, and higher doses may be considered.
- **Monophasic:** 360 J

Drug Therapy
- **Epinephrine IV/IO Dose:** 1 mg every 3-5 minutes
- **Vasopressin IV/IO Dose:** 40 units can replace first or second dose of epinephrine
- **Amiodarone IV/IO Dose:** First dose: 300 mg bolus. Second dose: 150 mg.

Advanced Airway
- Supraglottic advanced airway or endotracheal intubation
- Waveform capnography to confirm and monitor ET tube placement
- 8-10 breaths per minute with continuous chest compressions

Reversible Causes
- **H**ypovolemia
- **H**ypoxia
- **H**ydrogen ion (acidosis)
- **H**ypo-/hyperkalemia
- **H**ypothermia
- **T**ension pneumothorax
- **T**amponade, cardiac
- **T**oxins
- **T**hrombosis, pulmonary
- **T**hrombosis, coronary

Contents lists available at ScienceDirect

Resuscitation

journal homepage: www.elsevier.com/locate/resuscitation

Experimental paper

A quantitative comparison of physiologic indicators of cardiopulmonary resuscitation quality: Diastolic blood pressure versus end-tidal carbon dioxide☆

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End-tidal carbon dioxide

ABSTRACT

Aim: The American Heart Association (AHA) recommends monitoring invasive arterial diastolic blood pressure (DBP) and end-tidal carbon dioxide ($ETCO_2$) during cardiopulmonary resuscitation (CPR) when available. In intensive care unit patients, both may be available to the rescuer. The objective of this study was to compare DBP vs. $ETCO_2$ during CPR as predictors of cardiac arrest survival.

Methods: In two models of cardiac arrest (primary ventricular fibrillation [VF] and asphyxia-associated VF), 3-month old swine received either standard AHA guideline-based CPR or patient-centric, BP-guided CPR. Mean values of DBP and $ETCO_2$ in the final 2 min before the first defibrillation attempt were compared using receiver operating characteristic curves (area under curve [AUC] analysis). The optimal DBP cut point to predict survival was derived and subsequently validated in two independent, randomly generated cohorts.

Results: Of 60 animals, 37 (61.7%) survived to 45 min. DBP was higher in survivors than in non-survivors (40.6 ± 1.8 mmHg vs. 25.9 ± 2.4 mmHg; $p < 0.001$), while $ETCO_2$ was not different (30.0 ± 1.5 mmHg vs. 32.5 ± 1.8 mmHg; $p = 0.30$). By AUC analysis, DBP was superior to $ETCO_2$ (0.82 vs. 0.60; $p = 0.025$) in discriminating survivors from non-survivors. The optimal DBP cut point in the derivation cohort was 34.1 mmHg. In the validation cohort, this cut point demonstrated a sensitivity of 0.78, specificity of 0.81, positive predictive value of 0.64, and negative predictive value of 0.89 for survival.

Conclusions: In both primary and asphyxia-associated VF porcine models of cardiac arrest, DBP discriminates survivors from non-survivors better than $ETCO_2$. Failure to attain a DBP >34 mmHg during CPR is highly predictive of non-survival.

Personalized resuscitation: “one-size-fits-all”

- Pilot study of AMCPR (Augmented-Medication CPR) for improving outcome in patient with cardiac arrest: Multi-center, double-blind, prospective randomized clinical trial

- Inclusion: non-traumatic OHCA + PEA/Asystole

- Exclusion
  : DNAR / Terminal stage cancer (PD state or no CTx plan), arrest to ED arrival > 60 min, Previous cognitive impairment (CPC 3, 4), ECPR case, A-line insertion over 6 min

ClinicalTrials.gov Protocol Registration and Results System (PRS) Receipt
Release Date: June 16, 2017

ClinicalTrials.gov ID: NCT03191240

Study Identification

Unique Protocol ID:	AMCPR
Brief Title:	AMCPR (Augmented-Medication CardioPulmonary Resuscitation) Trial for OHCA
Official Title:	AMCPR (Augmented-Medication CardioPulmonary Resuscitation) for Improving Outcome in Patient With Cardiac Arrest: Multi-center, Double-blind, Prospective Randomized Clinical Trial.
Secondary IDs:	

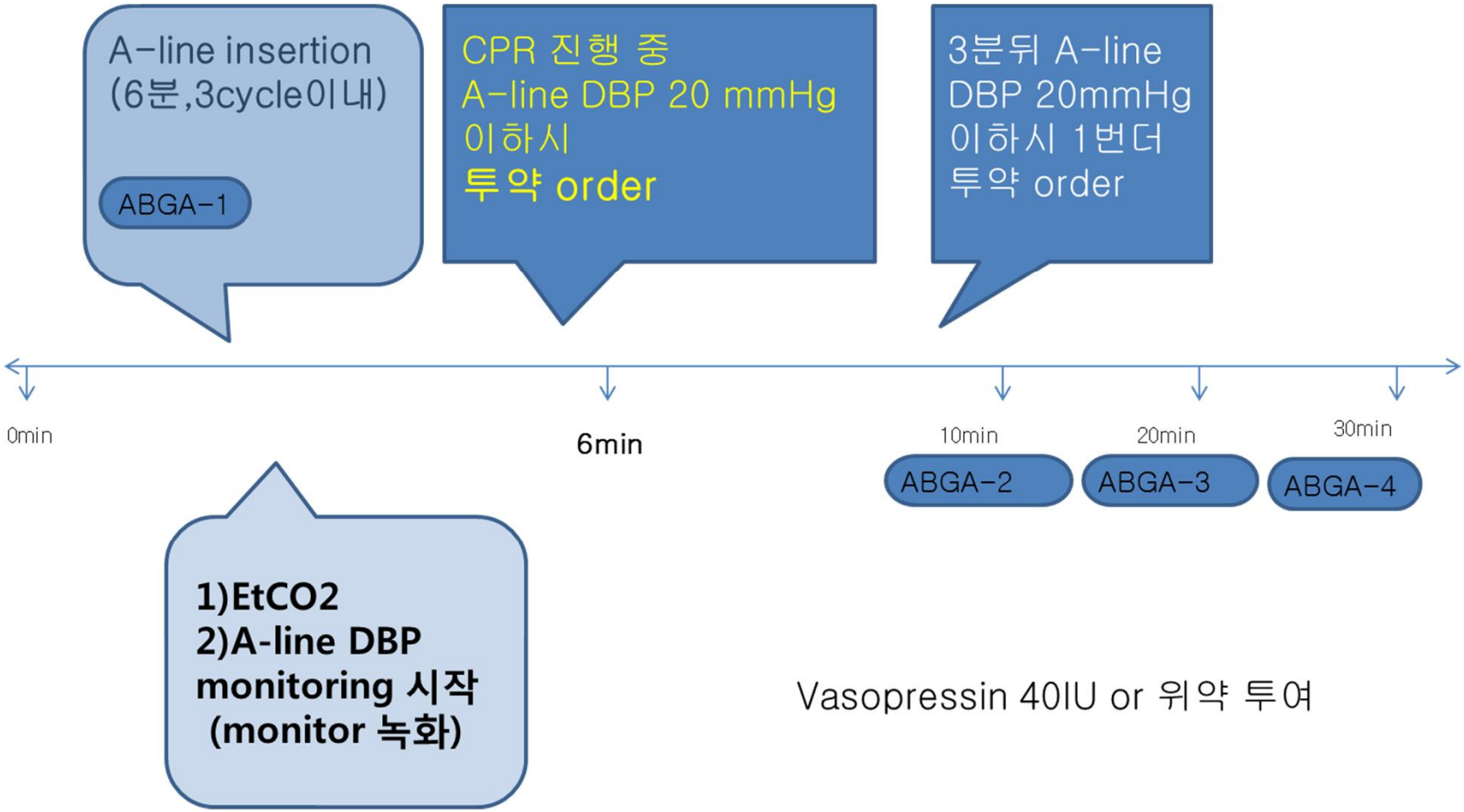
Study Status

Record Verification:	June 2017
Overall Status:	Not yet recruiting
Study Start:	July 1, 2017 [Anticipated]
Primary Completion:	June 30, 2018 [Anticipated]
Study Completion:	July 30, 2018 [Anticipated]

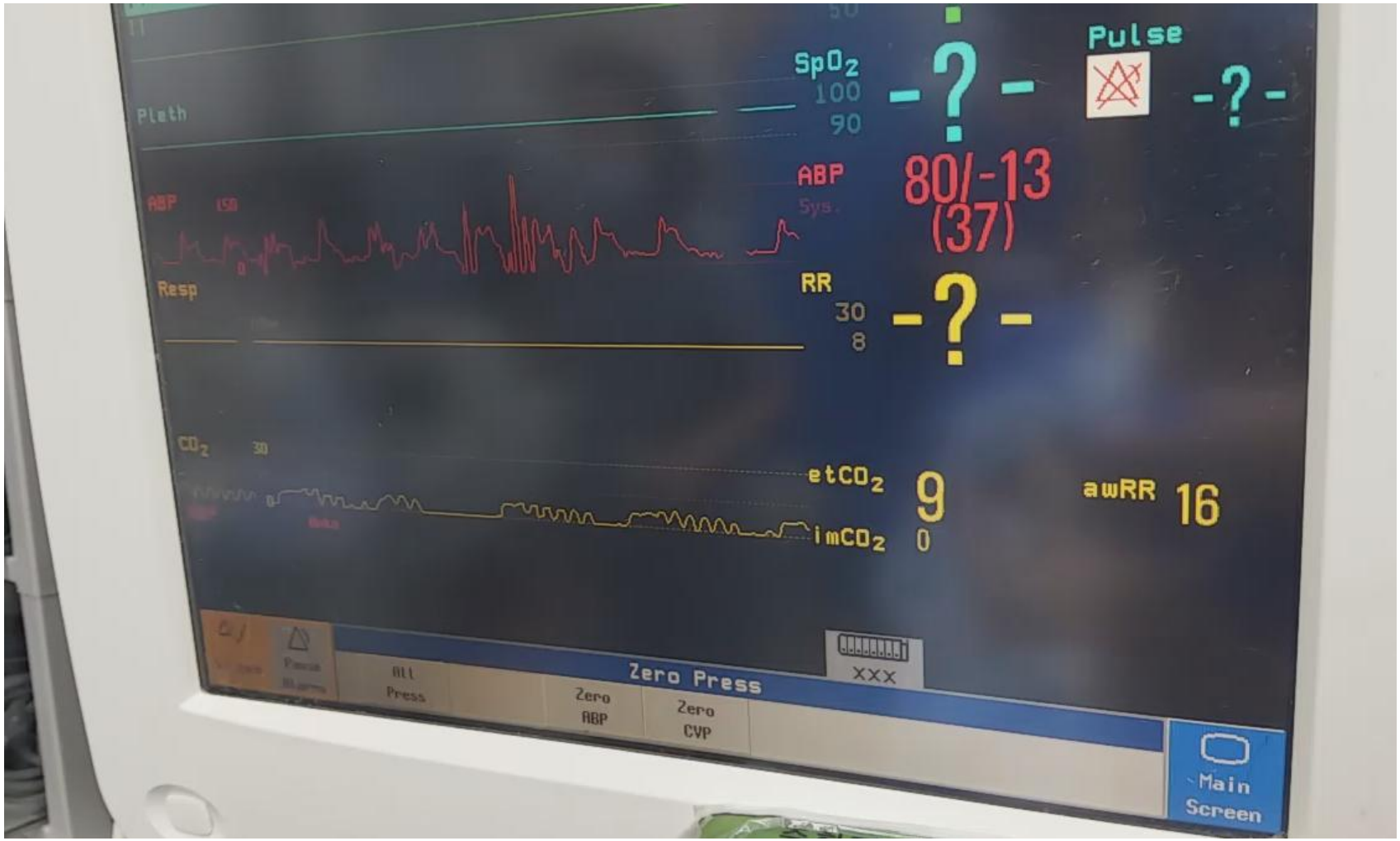
Sponsor/Collaborators

Sponsor:	Asan Medical Center
Responsible Party:	Principal Investigator Investigator: Won Young Kim [wykim] Official Title: Associate proffesor Affiliation: Asan Medical Center

AMCPR


A-line insertion
(6분,3cycle이내)
ABGA-1
CPR 진행 중
A-line DBP 20 mmHg
이하시
투약 order
3분뒤 A-line
DBP 20mmHg
이하시 1번더
투약 order
0min
6min
10min
20min
30min
ABGA-2
ABGA-3
ABGA-4
1)EtCO2
2)A-line DBP
monitoring 시작
(monitor 녹화)
Vasopressin 40IU or 위약 투여


Screen A
13:01
Adult
Not Admitted
Rzone2
ECG LEADS OFF
Arrhythmia Off
HR
-?-
SpO2
100
90
Pleth
Pulse
-?-
-?-
ABP
150
0
ABP
124/35
(59)
CO2
40
0
etCO2
12
imCO2
0
awRR
13


SpO2
100
90
Pleth
Pulse
ABP
Sys.
80/-13
(37)
ABP
150
0
Resp
RR
30
8
CO2
30
0
etCO2
9
imCO2
0
awRR
16
Zero Press
All
Press
Zero
ABP
Zero
CVP
XXX
Main
Screen

Central venous oxygen saturation (ScvO2)

- Changes in ScvO2 reflect changes in oxygen delivery by means of changes in cardiac output (normal 60-80%)
- During cardiac arrest and CPR: range from 25-35%
- If ScvO2 is <30%, it is reasonable to consider trying to improve the quality of CPR by optimizing chest compression parameters (Class IIb, LOE C)

etcs

❖ Pulse oximetry
- during cardiac arrest, not provide a reliable signal (inadequate pulsatile flow in peripheral tissue beds)
- the presence of a waveform: valuable in detecting ROSC

❖ Arterial Blood Gases
- not a reliable indicator of the severity of tissue hypoxemia, hypercarbia, or tissue acidosis
- routine measurement of ABGA during CPR has uncertain value (Class IIb, LOE C)

Observational Study

Medicine®

OPEN

Role of blood gas analysis during cardiopulmonary resuscitation in out-of-hospital cardiac arrest patients

Youn-Jung (MD), You Jin Lee (MD), Seung Mok Ryoo (MD), Chang Hwan Sohn (MD), Shin Ahn (MD, PhD), Dong-Woo Seo (MD, PhD), Kyoung Soo Lim (MD, PhD), Won Young Kim (MD, PhD)*

Abstract

To determine the relationship between acid–base findings, such as pH, pCO_2, and serum lactate levels, obtained immediately after starting cardiopulmonary resuscitation and the return of spontaneous circulation (ROSC).

A prospective observational study of adult, nontraumatic out-of-hospital cardiac arrest (OHCA) patients was conducted at an urban academic teaching institution between April 1, 2013 and March 31, 2015. Arterial blood sample for acid–base data was taken from all OHCA patients on arrival to the emergency department. Of 224 OHCA patients, 88 patients with unavailable blood samples or delayed blood sampling or ROSC within 4 minutes were excluded, leaving 136 patients for analysis.

The pH in the ROSC group was significantly higher than in the non-ROSC group (6.96 vs. 6.85; $P=0.009$). pCO_2 and lactate levels in the ROSC group were significantly lower than those in the non-ROSC group (74.0 vs. 89.5 mmHg, $P<0.009$; 11.6 vs. 13.6 mmol/L, $P=0.044$, respectively). In a multivariate regression analysis, pCO_2 was the only independent biochemical predictor for sustained ROSC (OR 0.979; 95% CI 0.960–0.997; $P=0.025$) and pCO_2 of <75 mmHg was 3.3 times more likely to achieve ROSC (OR 0.302; 95% CI 0.146–0.627; $P=0.001$).

pCO_2 levels obtained during cardiopulmonary resuscitation on ER arrival was associated with ROSC in OHCA patients. It might be a potentially marker for reflecting the status of the ischemic insult. These preliminary results need to be confirmed in a larger population.

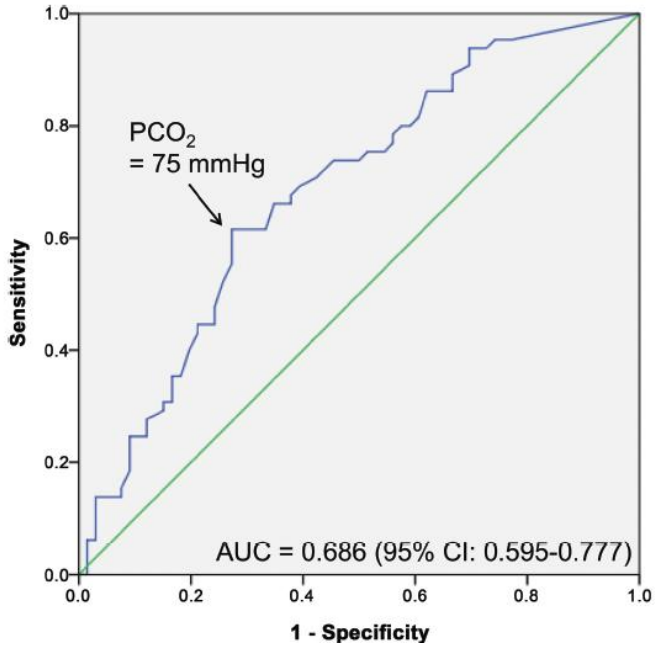
Table 1

Blood Gas Analysis of Out-of-Hospital Cardiac Arrest Patients.

Parameter	Median (IQR)	Minimum	Maximum	Could Not Be Evaluated (%)
pH	6.89 (6.80–7.02)	6.80	7.52	0 (0%)
pO_2 (mmHg)	17.5 (9.0–45.8)	0.0	355.0	0 (0%)
pCO_2 (mmHg)	78.0 (63.0–98.0)	13.0	115.0	5 (3.7%)
Bicarbonate (mmol/L)	18.0 (14.0–21.5)	6.0	60.0	43 (31.6%)
Base excess (mmol/L)	−15.6 (−18.6 – −11.5)	−37.0	2.1	43 (31.6%)
Sodium (mmol/L)	137 (132–140)	100	149	5 (3.7%)
Potassium (mmol/L)	6.4 (5.0–7.8)	3.0	12.0	6 (4.4%)
Glucose (mg/dL)	216 (131–319)	20	500	5 (3.7%)
Lactate level (mmol/L)	12.5 (9.2–15.0)	3.7	15.0	0 (0%)

Multivariate Analysis for Factors Predicting Sustained Return of Spontaneous Circulation.*

Variable	ORs (95% CI)	*P*
Age (years)	0.996 (0.973–1.018)	*0.702*
Male	0.450 (0.200–1.008)	*0.052*
pH	1.291 (0.019–87.061)	*0.905*
pCO_2 (mmHg)	0.979 (0.960–0.997)	*0.025*
Serum lactate level (mmol/L)	0.888 (0.764–1.031)	*0.119*



Resuscitation

journal homepage: www.elsevier.com/locate/resuscitation

EUROPEAN RESUSCITATION COUNCIL

Letter to the Editor

Emerging role of arterial blood gases during cardiopulmonary resuscitation: Another reason for invasive arterial pressure monitoring

Sir,

We have read with great interest and excitement the recent article by Spindelboeck et al.[1] published online in *Resuscitation* regarding arterial blood gas characteristics during out-of-hospital cardiopulmonary resuscitation (CPR). The authors reported that acidosis was present in nearly every patient during CPR and that no patient with an $AaDCO_2$ ($PaCO_2$-$EtCO_2$) greater than 33.5 mmHg survived to hospital admission. Although $EtCO_2$ has been used as a reliable indicator of cardiac output during CPR, several factors, such as hyperventilation and administration of adrenaline (epinephrine), may affect $EtCO_2$ levels.[2] Alternatively, the $AaDCO_2$ may serve as a global indicator of cardiorespiratory function for optimal management during CPR. Recently, there has been increasing evidence that extracorporeal membrane oxygenation is an effective therapeutic option for patients experiencing out-of-hospital cardiac arrest (OHCA) who are refractory to classical resuscitation.[3] A recent study from our group suggested a prognostic value of $PaCO_2$ obtained during early CPR. A patient with $PaCO_2$ <75 mmHg was 3.3 times more likely to achieve return of spontaneous circulation (ROSC; odds ratio 0.302; 95% confidence interval 0.146–0.627; $P = 0.001$).[4] As the probability of survival with good neurologic outcomes declines rapidly during CPR, early prediction of the probability of achieving sustained ROSC in patients experiencing OHCA is necessary for clinical decision-making. Although further studies will be needed to clarify the role of $AaDCO_2$ and $PaCO_2$, these measures might be used as indicators for early use of extracorporeal CPR in patients experiencing OHCA. In addition, these findings may provide potential evidence that monitoring and optimizing cardiorespiratory function with capnography and arterial blood gas analysis should be considered and that it is feasible. Although insertion of an arterial line during chest compressions can be technically difficult, it is worth trying. Invasive arterial pressure monitoring to measure arterial diastolic pressure during CPR may have potential benefits.[5]

However, we would like to highlight one point of concern. Hypercapnia contributed to acidosis in many cases even though all patients were intubated and mechanically ventilated. The authors suggested that hyperventilation resulting in hypocapnia is not a major problem during CPR because hypocapnia was present in only 6% of cases. However, hypercapnia during CPR results from dead space ventilation caused by reduced perfusion during compression. Hyperventilation would result in brain vasoconstriction and secondary ischemic brain insults, and therefore, it should be avoided according to recent guidelines.[5]

Further studies examining the dynamic changes of acid–base metabolism during CPR are warranted to confirm the association between arterial blood gas analysis and sustained ROSC.

Conflict of interest statement

The authors have disclosed that they do not have any potential conflicts of interest.

Funding

None declared.

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E-mail address: wonpia73@naver.com (W.Y. Kim)

4 August 2016

Bicarbonate

❖ Consider sodium bicarbonate (50 ml of an 8.4% solution)
1) life-threatening hyperkalaemia
2) preexisting metabolic acidosis or tricyclic, barbiturates overdose
3) prolonged intervals of CPR

❖ Disadvantage
- exacerbates intracellular acidosis
- negative inotropic effect on ischemic myocardium
- a large, osmotically active, sodium load to an already compromised circulation and brain
- a shift to the left in the oxygen dissociation curve, further inhibiting release of oxygen to the tissues

Sodium bicarbonate on severe metabolic acidosis during prolonged cardiopulmonary resuscitation: a double-blind, randomized, placebo-controlled pilot study

Shin Ahn[1*], Youn-Jung Kim[1*], Chang Hwan Sohn[1], Dong Woo Seo[1], Kyoung Soo Lim[1], Michael W. Donnino[2], Won Young Kim[1*]

Background: Sodium bicarbonate administration during cardiopulmonary resuscitation (CPR) is controversial. Current guidelines recommend sodium bicarbonate injection in patients with existing metabolic acidosis, but clinical trials, particularly, those involving patients with acidosis, are limited. We aimed to evaluate the efficacy of sodium bicarbonate administration in out-of-hospital cardiac arrest (OHCA) patients with severe metabolic acidosis during prolonged CPR.

Methods: Prospective, double-blind, randomized placebo-controlled pilot trial was conducted between January 2015 and December 2015, at a single center emergency department (ED). After 10 minutes of CPR, patients who failed to achieve return of spontaneous circulation (ROSC) and with severe metabolic acidosis (pH<7.1 or bicarbonate <10 mEq/L) were enrolled. Sodium bicarbonate (n=25) or normal saline (n=25) were administered. The primary end point was sustained ROSC. The secondary end points were the change of acidosis and good neurologic survival.

Results: Sodium bicarbonate group had significant effect on pH (6.99 *vs.* 6.90, P=0.038) and bicarbonate levels (21.0 *vs.* 8.0 mEq/L, P=0.007). However, no significant differences showed between sodium bicarbonate and placebo groups in sustained ROSC (4.0% *vs.* 16.0%, P=0.349) or good neurologic survival at 1 month (0.0% *vs.* 4.0%, P=1.000).

Conclusions: The use of sodium bicarbonate improved acid-base status, but did not improve the rate of ROSC and good neurologic survival. We could not draw a conclusion, but our pilot data could be used to design a larger trial to verify the efficacy of sodium bicarbonate.

Trial Registration: NCT02303548 (http://www.ClinicalTrials.gov).

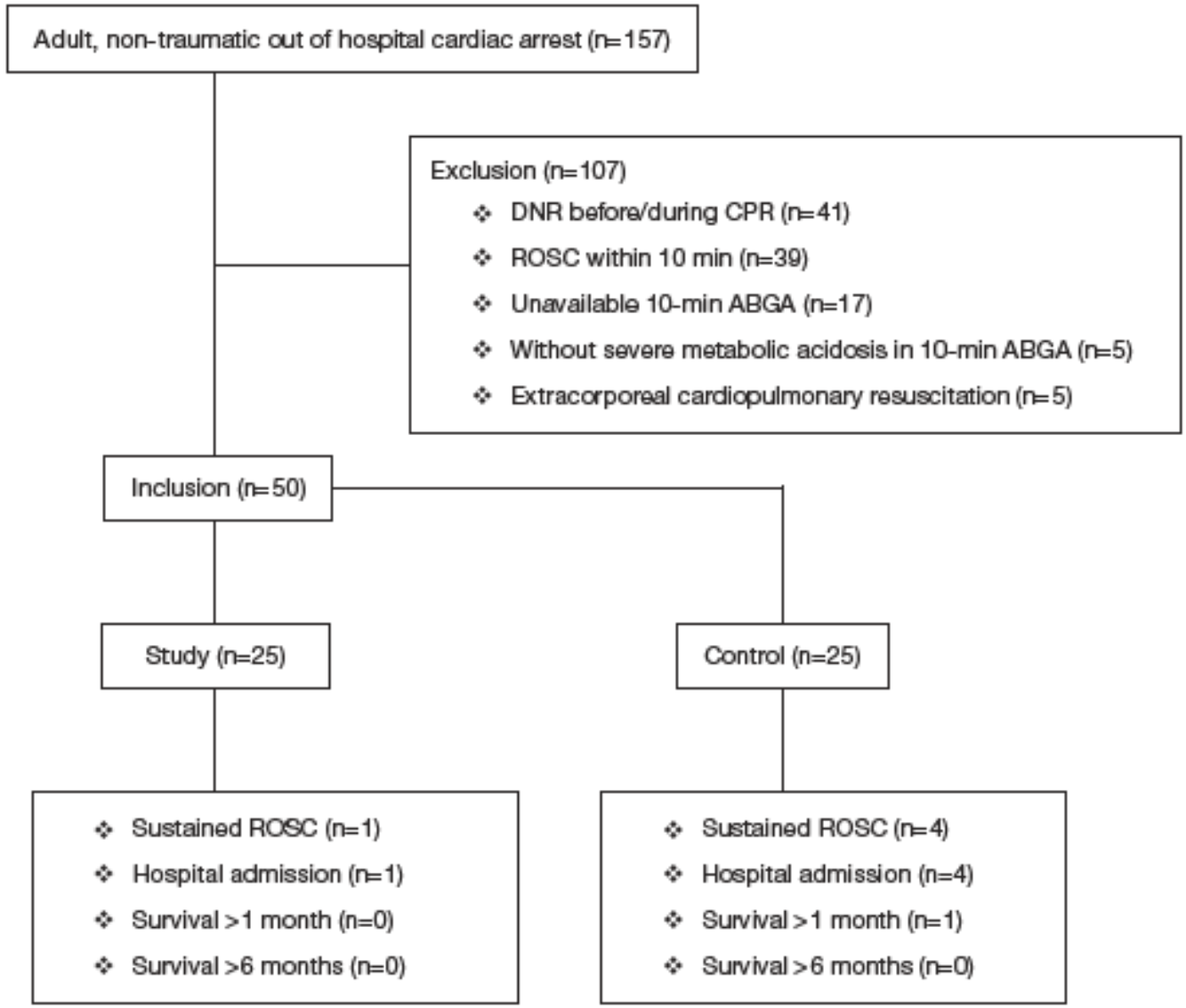
Adult, non-traumatic out of hospital cardiac arrest (n=157)
Exclusion (n=107)
DNR before/during CPR (n=41)
ROSC within 10 min (n=39)
Unavailable 10-min ABGA (n=17)
Without severe metabolic acidosis in 10-min ABGA (n=5)
Extracorporeal cardiopulmonary resuscitation (n=5)
Inclusion (n=50)
Study (n=25)
Control (n=25)
Sustained ROSC (n=1)
Hospital admission (n=1)
Survival >1 month (n=0)
Survival >6 months (n=0)
Sustained ROSC (n=4)
Hospital admission (n=4)
Survival >1 month (n=1)
Survival >6 months (n=0)

Table 2 Differences in blood gas analysis results between the study and control groups

Outcome	Study group (n=25)	Control group (n=25)	P
Blood gas analysis at 10 minutes			
Lactic acid	12.00 (10.10–14.90)	11.90 (7.85–15.00)	0.725
pH*	6.86 (6.84–7.00)	6.93 (6.84–7.00)	0.888
HCO_3^- †	16.00 (10.00–20.00)	10.00 (7.45–13.00)	0.358
pCO_2 ‡	76.00 (58.50–89.00)	49.50 (34.75–65.00)	0.579
pO_2	64.00 (21.50–89.50)	33.50 (19.00–82.00)	0.398
Blood gas analysis at 20 minutes			
Lactic acid	12.00 (10.35–15.00)	14.05 (7.85–14.78)	0.965
pH*	6.99 (6.92–7.12)	6.90 (6.85–6.94)	0.038
HCO3$^-$ †	21.00 (15.85–28.75)	8.00 (3.30–14.00)	0.007
pCO_2 ‡	82.00 (73.00–94.50)	86.00 (51.50–112.75)	0.908
pO_2	41.00 (25.00–58.00)	49.00 (29.25–74.00)	0.806

Data are presented as median with interquartile range. Wilcoxon Signed Rank Test: Study group, *, P=0.330; †, P=0.036; ‡, P=0.053 *vs.* Control group; *, P=0.068; †, P=0.141; ‡, P=0.308.

Table 3 Differences in outcome between the study and control groups

Outcome	Total (n=50)	Study group (n=25)	Control group (n=25)	P
ROSC	9 (18.0)	3 (12.0)	6 (24.0)	0.469
Sustained ROSC (>20 minutes)	5 (10.0)	1 (4.0)	4 (16.0)	0.349
Hospital admission	5 (10.0)	1 (4.0)	4 (16.0)	0.349
Good neurologic survival >1 month	1 (4.0)	0 (0.0)	1 (4.0)	1.000
Good neurologic survival >6-month	0 (0.0)	0 (0.0)	0 (0.0)	–

Monitoring parameters during CPR (2015)

- It may be reasonable to use physiologic parameters (quantitative waveform capnography, arterial relaxation diastolic pressure, arterial pressure monitoring, and central venous oxygen saturation) when feasible to monitor and optimize CPR quality, guide vasopressor therapy, and detect ROSC (Class IIb, LOE C-EO)

- Previous guidelines specified physiologic parameter goals; however, precise numerical targets for these parameters during resuscitation have not as yet been established, these were not specified in 2015

Summary: Monitoring parameters during CPR

- ECG leads and pulse checks
- End-tidal CO2
- Coronary perfusion pressure (CPP)
  : diastolic pressure
- Central venous oxygen saturation (ScvO2)
- Arterial Blood Gas Analysis

Thank you for your attention