Haemodynamic parameters during cardiopulmonary resuscitation in estimating the degree of ischaemic contracture

Se-hyeok Park, Yong Deok Lim, Yong Hun Jung, [Kyung Woon Jeung](#)
Department of Emergency Medicine, Chonnam National University Hospital, Gwangju, Republic of Korea

Myocardial ischemic contracture, which refers to a progressive LV wall thickening following ischemia, occurs frequently in patients with prolonged cardiac arrest. Myocardial ischemic contracture inevitably results in reduction in the LV chamber volume. Thus, the volume of blood ejected by chest compression decreases as myocardial ischemic contracture progresses, ultimately precluding successful resuscitation (Fig. 1). In experimental settings, echocardiographic assessment with transesophageal or transmediastinal approaches has been used to observe the development of ischemic contracture during CPR. However, the echocardiographic assessments using these approaches are usually impractical or unavailable in clinical settings.

Fig. 1. Transmediastinal echocardiography demonstrating severe LV ischemic contracture in a pig undergoing prolonged CPR

In the present study, we investigated, in a swine model of out-of-hospital cardiac arrest, whether hemodynamic parameters, including arterial pressure and ETCO₂, can serve as predictive factors for the severity of ischemic contracture during CPR of consistent quality, and therefore can be utilized as a tool to estimate the severity of ischemic contracture during ACLS conducted according to resuscitation guidelines.

Methods

We retrospectively analyzed data derived from a previous study in Yorkshire/Landrace cross pigs weighing 25.2 ± 2.9 kg (Fig. 2). The IACUC of our university approved the study (CNU IACUC-H-2017-18).

Fig. 2. Experimental timeline. Lightning marks indicate the onset of a 10-second pause in chest compressions for rhythm analysis and a 150-J shock, if indicated.

Echocardiograms were obtained by an experienced investigator five minutes before induction of VF, immediately after initiation of VF, and in one-minute intervals during ACLS (Fig. 3). LV wall thickness and EDV during CPR were measured at the frame showing maximal chamber dimension of the LV following release of chest compression. EDV, which was the primary outcome measurement of the present study, was expressed as a percent of measured EDV relative to EDV at the onset of VF (%EDV), to correct for its substantial inter-animal variation.

Fig. 3. Echocardiography during cardiopulmonary resuscitation in the present study.

Results

Eight animals underwent the 12 minute-ACLS period without achieving ROSC. Thus, a total of 96 sets of ETCO₂, arterial pressure, LV wall thickness, and %EDV data were collected (12 times with one-minute intervals in eight animals).

Fig. 4. LV wall thickness (A), %EDV (B), systolic arterial pressure (C), diastolic arterial pressure (D), and ETCO₂ (E) during cardiopulmonary resuscitation. Data are presented as the median and interquartile ranges. LV, left ventricular; EDV, end-diastolic volume; ETCO₂, end-tidal carbon dioxide.

Each repeatedly measured response variable, including LV wall thickness and %EDV, were significantly correlated with each predictor variable, including systolic and diastolic arterial pressure, and ETCO₂ (Table 1).

Table 1. Correlations between repeatedly measured variables			
Variables	Correlation coefficient	95% CI	<i>P</i>
LV wall thickness			
ETCO ₂	-0.55	-0.681 – -0.383	<0.001
Systolic arterial pressure	-0.476	-0.624 – -0.295	<0.001
Diastolic arterial pressure	-0.477	-0.625 – -0.297	<0.001
%EDV			
ETCO ₂	0.303	0.098 – 0.483	0.004
Systolic arterial pressure	0.405	0.212 – 0.567	<0.001
Diastolic arterial pressure	0.437	0.250 – 0.593	<0.001

Tables 2 and 3 show the fixed effects of time and each predictor variable on LV wall thickness and %EDV in linear mixed effect models, respectively. After adjustment for baseline covariates, systolic and diastolic arterial pressure were not predictive factors for LV wall thickness or %EDV, while ETCO₂

was a significant predictive factor for both LV wall thickness (*P* = 0.014) and %EDV (*P* = 0.013).

Throughout our models, time was the most significant predictive factor for all response variables. For example, in the model including time and %EDV, time explained 52.9% of total variance of %EDV when adjusted for baseline covariates (semi-partial *R*² = 0.529; 95% confidence interval [CI], 0.407 – 0.642; *P* < 0.001), while ETCO₂ explained 14.2% of total variance of %EDV (semi-partial *R*² = 0.142; 95% CI = 0.040 – 0.283, *P* = 0.003).

In conclusion, systolic and diastolic arterial pressure were not predictive factors for the severity of ischemic contracture. While ETCO₂ was a significant predictive factor for the severity of ischemic contracture, it explained only a small proportion of the variance of ischemic contracture severity.

Table 2. Fixed effects of time and hemodynamic parameters on LV wall thickness in mixed effect models

	Coefficient	SE	<i>P</i>	Semi-partial <i>R</i> ²	95% CI
Time	0.514	0.044	<0.001	0.405	0.269 – 0.538
ETCO ₂ (mmHg)	-0.069	0.043	0.11	0.056	0.002 – 0.173
Time*	0.509	0.04	<0.001	0.629	0.526 – 0.721
ETCO ₂ (mmHg)*	-0.08	0.032	0.014	0.061	0.003 – 0.180
Time	0.546	0.045	<0.001	0.414	0.279 – 0.546
Systolic arterial pressure (mmHg)	-0.004	0.014	0.782	0.001	0 – 0.056
Time*	0.572	0.04	<0.001	0.682	0.591 – 0.762
Systolic arterial pressure (mmHg)*	0.011	0.009	0.225	0.015	0 – 0.100
Time	0.525	0.045	<0.001	0.403	0.268 – 0.537
Diastolic arterial pressure (mmHg)	-0.049	0.042	0.248	0.012	0 – 0.091
Time*	0.543	0.044	<0.001	0.619	0.514 – 0.714
Diastolic arterial pressure (mmHg)*	-0.017	0.041	0.676	0.002	0 – 0.060

* Baseline variables used to adjust the mixed effect model included right atrial pressure, heart rate, PaCO₂, PaO₂, SaO₂, and troponin. SE, standard error; CI, confidence interval; ETCO₂, end-tidal carbon dioxide.

Table 3. Fixed effects of time and hemodynamic parameters on %EDV in mixed effect models

	Coefficient	SE	<i>P</i>	Semi-partial <i>R</i> ²	95% CI
Time	-5.512	0.402	<0.001	0.410	0.275 – 0.543
ETCO ₂ (mmHg)	-1.239	0.399	0.003	0.146	0.043 – 0.288
Time*	-5.477	0.4	<0.001	0.529	0.407 – 0.642
ETCO ₂ (mmHg)*	-1.174	0.389	0.003	0.142	0.040 – 0.283
Time	-5.032	0.424	<0.001	0.377	0.240 – 0.514
Systolic arterial pressure (mmHg)	-0.114	0.129	0.378	0.008	0 – 0.082
Time*	-4.961	0.418	<0.001	0.491	0.363 – 0.611
Systolic arterial pressure (mmHg)*	-0.074	0.123	0.549	0.006	0 – 0.074
Time	-4.719	0.421	<0.001	0.345	0.209 – 0.486
Diastolic arterial pressure (mmHg)	0.198	0.395	0.617	0.002	0 – 0.060
Time*	-4.699	0.42	<0.001	0.454	0.322 – 0.580
Diastolic arterial pressure (mmHg)*	0.234	0.391	0.552	0.003	0 – 0.066

* Baseline variables used to adjust the mixed effect model included ETCO₂ and troponin. SE, standard error; CI, confidence interval; ETCO₂, end-tidal carbon dioxide.