Original Article

Investigation of Myocardial Stunning after Cardiopulmonary Resuscitation in Pigs*

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Abstract

Objective To investigate cardiac function and myocardial perfusion during 48 h after cardiopulmonary resuscitation (CPR), further to test myocardial stunning and seek indicators for long-term survival after CPR.

Methods After 4 min of untreated ventricular fibrillation, fifteen anesthetized pigs were studied at baseline and 2 h, 4 h, 24 h, and 48 h after restoration of spontaneous circulation (ROSC). Hemodynamic data, echocardiography and gated-single photon emission computed tomography myocardial perfusion images were carried out.

Results Mean arterial pressure (MAP), coronary perfusion pressure (CPP) and cardiac troponin I (CTNI) showed significant differences between eventual survival animals and non-survival animals at 4 h after ROSC (109.2±10.7 mmHg *vs.* 94.8±12.3 mmHg, *P*=0.048; 100.8±6.9 mmHg *vs.* 84.4±12.6 mmHg, *P*=0.011; 1.60±0.13 ug/L *vs.* 1.75±0.10 ug/L, *P*=0.046). Mitral valve early-to-late diastolic peak velocity ratio, mitral valve deceleration time recovered 24 h; ejection faction and the summed rest score recovered 48 h after ROSC.

Conclusion Cardiac systolic and early active relaxation dysfunctions were reversible within survival animals; cardiac stunning might be potentially adaptive and protective after CPR. The recovery of MAP, CPP, and CTNI could be the indicators for long-term survival after CPR.

Key words: Myocardial stunning; Systolic and diastolic dysfunctions; Cardiopulmonary resuscitation

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INTRODUCTION

Myocardial stunning after cardiopulmonary resuscitation (CPR) is well known to be characterized after successful CPR[1-3] and belonged to post–cardiac arrest syndrom[4], with documented time-dependent systolic and diastolic dysfunction in previous studies in animals and humans[5-8].

Studies have usually employed a single method to demonstrate the cardiac dysfunction after CPR (e.g. floating catheter, echocardiography, isotope imaging). Echocardiography was considered to be a quantitative indicator for early post-resuscitation myocardial stunning occurring in experimental models of CPR[9]. In recent years, technetium-99m methoxyisobutylisonitrile (^{99m}TcMIBI) gated-single photon emission computed tomography (G-SPECT), which allows assessment of perfusion and functional parameters, has been successfully used in the detection of myocardial stunning[10]. We combined Swan-Ganz catheter, echocardiography, and G-SPECT myocardial perfusion images for further investigating myocardial stunning during the 48 h after CPR and tried to find which phase of cardiac diastolic functions were involved and the indicators

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for long-term survival after cardiac arrest and CPR.

MATERIALS AND METHODS

Animal Preparation

The Institutional Committee on the Care and Use of Animals of Beijing Chaoyang Hospital (Beijing, China) approved the study protocol. Fifteen male domestic pigs (2-3 months; 30±5 kg) were used in the present study. Pigs were fasted overnight but had free access to water. Anesthesia was induced by midazolam (0.5 mg/kg, i.m.) and then by injection of propofol (1.0 mg/kg) into the ear vein. Anesthesia was maintained with intravenous infusion of pentobarbital (8 mg/kg per h). A cuffed 6.5-mm endotracheal tube was advanced into the trachea. Pigs were mechanically ventilated with a volume-controlled ventilator (PB-7200, Nellcor Puritan Bennett Incorporated, USA) using a tidal volume of 15 mL/kg, respiratory frequency of 12 breaths per minute, and room air. The end-tidal partial pressure of carbon dioxide ($EtPCO_2$) was measured by an in-line infrared capnograph (COSMO plus monitor, Respironics Incorporated, USA). Respiratory frequency was adjusted to maintain $EtPCO_2$ between 35 mmHg and 40 mmHg before inducing cardiac arrest and after return of spontaneous circulation (ROSC). Room temperature was adjusted at 26 °C. A Swan-Ganz catheter (7 F, Edwards Life Sciences, USA) was advanced from the right femoral vein and flow-directed into the pulmonary artery for the measurement of right atrial pressure (RAP) through a pressure transducer (Biosensors International Corporation, Singapore); continuous cardiac output (CCO) and mixed venous blood oxygen saturation (SvO_2) with a CCO monitor (Vigilance II, Edwards Life Sciences). An angiographic catheter was inserted from the femoral artery into the aortic arch for measurement of arterial blood pressure (ABP) and analyses of arterial blood gases (GEM Premier 3 000 Blood Gas Analyzer, Instrumentation Laboratory, USA). Electrocardio graphy (ECG) was carried out and ABP, RAP and $EtPCO_2$ measured with a Hewlett-Packard monitor (M1165, Hewlett- Packard, USA). Another catheter was inserted into the left external jugular vein to place an electrode catheter to induce ventricular fibrillation with a programmed electrical stimulation instrument (GY-600A, Kai Feng Huanan Instrument Limited Company, China).

Experimental Protocol

After surgery, pigs were allowed to acclimatize for 30 min to achieve stable resting level. Ventricular fibrillation was induced by programmed electrical stimulation, and was confirmed by ECG and the presence of profound hypotension, ventilation was stopped and the ventilator was disconnected from the endotracheal tube. After 4 min of untreated ventricular fibrillation, pigs received 30:2 (compression-to-ventilation ratios) CPR[11].

Defibrillation shocks were administered at 200 J (Smart Biphasic). If the first defibrillation was unsuccessful, epinephrine (1 mg) was administered IV. Additional epinephrine (if needed), was given at 3-min intervals. Mechanical ventilation began at the beginning of the first attempt at defibrillation with 100% oxygen and continued until successful resuscitation, after which room air was used[12]. If an organized cardiac rhythm with a mean aortic pressure of $\geqslant$60 mmHg persisted for an interval of $\geqslant$1 min, pigs were regarded to be successfully resuscitated. Resuscitation continued for a maximum of 15 min until pigs were successfully resuscitated[13]. After successful resuscitation, the animals underwent a 4-h intensive care period. The vascular sheaths and endotracheal tube were removed after a 4-h intensive care period. Then they were placed in observation cages, and monitored until 48 h after resuscitation. Water, but no specific hemodynamic treatment was given during the observation period. 24 h and 48 h after ROSC, pigs were re-anesthetized with same method mentioned above, and echocardiography and SPECT imaging measurements repeated. After the final measurement had been completed, pigs were euthanized with pentobarbital (150 mg/kg, i.v.).

Rest acquisition was carried out after injection of 20 mCi ^{99m}Tc-MIBI at baseline and 2 h, 4 h, 24 h, 48 h after ROSC. Data acquisitions were done with a single-head SPECT system (Philips) equipped with a low-energy, high-resolution collimator. A 15% window around the 140-keV energy peak of ^{99m}Tc was used. The acquisition matrix size was 64×64×16. 64 projections (step-and-shoot mode, 40 s per projection) were obtained over a 180° circular orbit. Acquisitions were gated for 16 frames per cardiac cycle (acceptance window, 50%). The left ventricular ejection fraction (EF) and left ventricular volume was calculated using previously validated and commercially available automated software from the G-SPECT images.

Echocardiographic studies were carried out with a Philips iE33 machine (Philips, Bothell, WA, USA) at baseline and 2 h, 4 h, 24 h, 48 h after ROSC immedi

ately following gated SPECT imaging using a 1-5-MHz transthoracic device (Philips). Images were obtained in long-axis and short-axis parasternal views with two-dimensional (2D) and M-mode modalities. A pulsed-waved Doppler ultrasound transducer was used at each examination site. The main experimental procedure was seen in Figure 1.

Semi-quantitative analysis of the rest SPECT studies was performed on 17 segments model of the left ventricle[14] (including six basal, six mid-ventricular and four apical segments in short axis slices and one additional mid-ventricular apical slice in the vertical long axis) with four-point scoring system (0-3 for normal-to-absent tracer uptake).

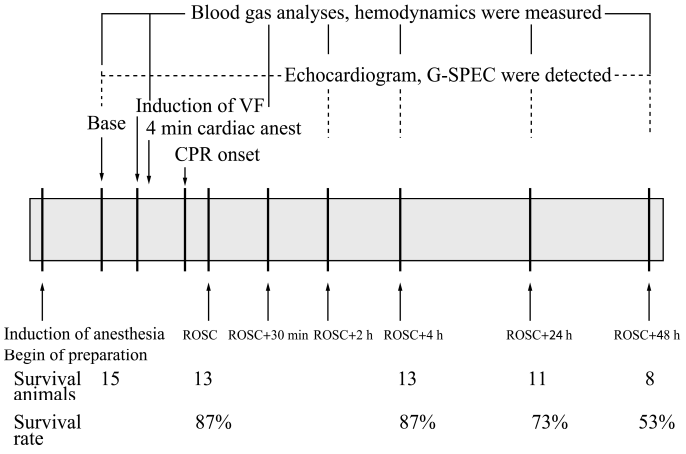

Figure 1. Main experimental procedure and number of survival animal. CPR: cardiopulmonary resuscitation; ROSC: restoration of spontaneous circulation; VF: ventnicular fibrillation.

Measurements

Hemodynamic data coronary perfusion pressure (CPP;CPP=aortic diastolic pressure-right atrial diastolic pressure), mean arterial pressure (MAP), CO, HR, and ECG (lead II) were recorded. Arterial, mixed venous blood gases and arterial blood lactic acid (Lac), together with cardiac troponin I (CTNI) were measured before and at 30 min, 2 h, 4 h, 24 h, and 48 h after ROSC. EF, mitral valve early-to-late diastolic peak velocity ratio (MV-E/A), and mitral valve deceleration time (MV-DT) were detected by transth oracic echocardiography, the summed rest score (SRS) reflecting the myocardial perfusion defect and EF were calculated by gated SPECT imaging at baseline and 2 h, 4 h, 24 h, and 48 h after ROSC.

Semi-quantitative analyses of SPECT studies were carried out using a 17-segment model of the left ventricle with a four-point scale[15]: perfusion no defects were scored as 0; perfusion mildly deduced uptake was 1; perfusion severely reduced uptake was 2 and perfusion absent uptake was 3. Cardiac blood perfusion defects could therefore be drawn according to the different coronary artery area by using the SRS (Figure 2).

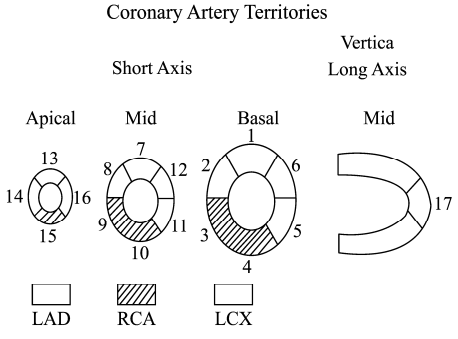

Figure 2. Coronary artery territories by SPECT segment.

Statistical Analyses

Data are $\bar{x} \pm s$. Comparisons between eventual survival animals and non-survival animals were performed using independent-samples t test in hemodynamics and G-SPECT of Table 1. Comparisons between groups were made with one-way analysis of variance in Table 2 and Table 3, with Pearson correlation coefficient for correlation comparisons in Table 4. $P<0.05$ was considered statistically significant. The Statistical Package for Social Sciences (SPSS 13.0) was used for statistical analysis.

Table 1. Comparison Hemodynamics and G-SPECT at ROSC 4h between Eventual Survival Animals and Non-survival Animals ($\bar{x} \pm s$)

	Eventual Survival Animals	Eventual Non-survival Animals	*P* Value
	n=8	*n*=5	
pH	7.48±0.07	7.48±0.05	0.86
PaO_2 (mmHg)	69.38±9.40	63.8±5.63	0.65
$PaCO_2$ (mmHg)	45.88±3.72	43.2±3.63	0.23
SvO_2 (%)	60.4±8.99	53.2±6.65	0.15
Lac (mmol/L)	3.70±1.24	4.3±0.89	0.37
HR(/min)	150±17	168±10	0.059
CTNI(ug/L)	1.60±0.13	1.75±0.10	0.046
MAP (mmHg)	109.2±10.7	94.8±12.3	0.048
CPP (mmHg)	100.8±6.9	84.4±12.6	0.011
CO (L/min)	4.08±0.46	3.54±0.46	0.066
EF (%)	27±5	24±4	0.31
SRS	9.0±1.60	11.0±2.45	0.10

Note. G-SPECT: gated myocardial perfusion single photon emission computed tomography ROSC: restoration of spontaneous circulation, PO_2: partial pressure of oxygen, PCO_2: partial pressure of carbon dioxide, SvO_2: mixed venous oxygen saturation, Lac: Lactic acid, CTNI : cardiac troponin I, MAP: Mean arterial pressure, CPP: coronary perfusion pressure, CO: cardiac output.

Table 2. Blood Gas Analyses, Hemodynamics during ROSC 48 h ($\bar{x} \pm s$)

	Baseline	VF 4 min	ROSC30 min	ROSC 2 h	ROSC 4 h	ROSC 24 h	ROSC 48 h
pH	7.44±0.05	7.30±0.09[b]	7.33±0.06[b]	7.38±0.04[a]	7.48±0.07	7.49±0.03	7.48±0.04
PaO_2 (mmHg)	89.13±6.40	49.00±7.25[b]	52.5±5.98[b]	63.1±5.38[b]	69.38±9.40[b]	79.50±11.39	83.38±6.89
$PaCO_2$ (mmHg)	43.63±4.81	34.63±3.20[b]	38.50±3.07[a]	39.50±2.56[a]	45.88±3.72	41.25±3.62	45.38±3.58
SvO_2 (%)	72.4±8.37	42.0±7.39[b]	52.3±11.89[b]	55.8±10.40[b]	60.4±8.99[a]	64.9±7.75	71.3±8.81
Lac (mmol/L)	1.89±0.66	5.60±1.02[b]	7.54±1.16[b]	6.88±1.10[b]	3.70±1.24[b]	2.21±0.49	2.11±0.49
HR (/min)	107±7	-	141±14[b]	150±17[b]	136±11[b]	117±9	111±8
CTNI(ug/L)	1.32±0.10	1.36±0.09	1.72±0.18[b]	1.85±0.18[b]	1.60±0.13[b]	1.47±0.10[a]	1.34±0.07
MAP (mmHg)	107.5±17.0	50.9±7.0[b]	92.5±10.9[a]	96.6±7.7	109.2±10.7	-	-
CPP (mmHg)	103.9±5.9	25.5±5.4[b]	92.8±8.7[b]	96.0±5.2[a]	100.8±6.9	-	-
CO(L/min)	4.72±0.46	1.50±0.50[b]	2.42±0.62[b]	3.27±0.55[b]	4.08±0.46[a]	-	-

Note. [a] $P \leqslant 0.05$, [b] $P < 0.01$ *vs.* baseline. ROSC: restoration of spontaneous circulation, PO_2: partial pressure of oxygen, PCO_2: partial pressure of carbon dioxide, SvO_2: mixed venous oxygen saturation, Lac: Lactic acid, CTNI : cardiac troponin I, MAP: Mean arterial pressure, CPP: coronary perfusion pressure, CO: cardiac output.

Table 3. Myocardial Function and Summed Rest Score by Echocardiogram and G-SPECT during ROSC 48 h ($\bar{x} \pm s$)

	Baseline	ROSC 2 h	ROSC 4 h	ROSC 24 h	ROSC 48 h
Echocardiogram					
EF (%)	62±7	24±3[b]	29±5[b]	50±3[b]	58±3
MV-E/A	1.28±0.13	0.87±0.10[b]	1.03±0.18[b]	1.22±0.16	1.33±0.10
MV-DT(ms)	44.63±5.63	54.00±7.71[a]	55.75±8.07[b]	46.13±7.70	43.25±6.09
G-SPECT					
EF (%)	56±4	23±4[b]	27±5[b]	49±5[b]	54±4
SRS	1.0±0.76	10.38±1.51[b]	9.0±1.60[b]	2.38±0.92[a]	1.5±0.93

Note. [a] $P \leqslant 0.05$, [b] $P < 0.01$ *vs.* baseline G-SPECT: gated myocardial perfusion single photon emission computed tomography, ROSC: restoration of spontaneous circulation, EF: ejection fraction, MV-E/A: mitral valve early-to-late diastolic peak velocity ratio, MV-DT: mitral valve deceleration time, SRS: summed rest score.

Table 4. Correlations of EF between Echocardiogram and G-SPECT during ROSC 48 h ($\bar{x} \pm s$)

		Baseline	ROSC 2 h	ROSC 4 h	ROSC 24 h	ROSC 48 h
EF (%)	Echocardiogram	62±7	24±3	29±5	50±3	58±3
	G-SPECT	56±4	23±4	27±5	49±5	54±4
Correlation Coefficient		0.80	0.90	0.91	0.86	0.84
P value		0.018	0.020	0.021	0.007	0.009

Note. EF: ejection fraction, G-SPECT: gated myocardial perfusion single photon emission, computed tomography, ROSC: restoration of spontaneous circulation.

RESULTS

13 out of 15 pigs returned to spontaneous circulation and survived for 4 h; 11 of the 13 survived for 24 h. The 11 pigs had free access to water and a small amount of food as per protocol, but the remaining eight pigs were observed for 48 h after ROSC (Figure 1). MAP, CPP and CTNI had significant difference between eventual survival animals (n=8) and non-survival animals (n=5) at 4 h after ROSC (Table 1).

Hemodynamic Data

The arterial partial pressure of oxygen (PO_2), SvO_2 decreased (P<0.01) and arterial partial pressure of carbon dioxide (pCO_2) decreased (P<0.05) 30 min after ROSC. PO_2, SvO_2, and PCO_2 increased to normal at 24 h, and 4 h after ROSC (P>0.05). Arterial pH was found to be at baseline levels 4 h after ROSC (P>0.05). The level of Lac and CTNI were maximal 30 min and 2 h after ROSC (P<0.01); a significant difference was not observed 24 h, 48 h after ROSC compared with baseline levels (P>0.05) (Table 2).

MAP, CPP, and CO increased slowly from 30 min to 4 h after ROSC. MAP and CPP returned to baseline levels 4 h after ROSC (P>0.05). CO increased but remained different from the baseline level (P<0.05) 4 h after ROSC. HR was increased 30 min and 4 h after ROSC; a significant difference was not observed 24 h after ROSC (P>0.05) (Table 2).

Echocardiography and Gated SPECT Myocardial Perfusion Imaging

EF detected by echocardiography was significantly decreased at 2 h (P<0.01) and increased slowly from 4 h to 24 h after ROSC; a significant difference was not found 48 h after resuscitation (P>0.05). MV-E/A was significantly decreased at 2 h (P<0.01); MV-DT was increased 4 h (P<0.01) after resuscitation, and both values returned to baseline levels 24 h after ROSC (P>0.05) (Table 3).

EF detected by G-SPECT was significantly reduced from 2 h to 24 h after resuscitation (P<0.01); the SRS was significantly increased from 2 h (P<0.01) and recovered 48 h after ROSC (P>0.05) (Table 3).

The total myocardial perfusion defect SRS 4 h after ROSC was 72. The left anterior descending artery, right coronary artery and left circumflex coronary artery SRS were 46, 8 and 18, respectively, and accounted for 63.9%, 11.1%, and 25%, respectively. We presented the examples of porcine cardiac perfusion on G-SPECT at baseline and after ROSC (Figure 3): the perfusion defect 4 h after ROSC was in the anteroseptal wall of the left anterior descending artery and posterolateral wall of the left circumflex coronary artery. The perfusion defect 24 h after ROSC was in the posterolateral wall of the left circumflex coronary artery and posteroseptal wall of the right coronary artery. The perfusion defect 48 h after ROSC was virtually absent.

The correlations of EF between echocardiogram and G-SPECT during 48 h after ROSC were showed in Table 4.

DISCUSSION

Hemodynamic Analysis

In our study, CO was did not return to normal during the monitoring period, but it clearly increased 4 h after resuscitation, then PO_2, SvO_2, and lactic acid levels returned to normal 24 h after ROSC. And oxygen dynamics and aerobic metabolism would be recovered after an improvement in coronary blood perfusion. An animal study showed identical outcomes which suggested that myocardial microcirculatory function was <50% of its pre-arrest baseline level at 30 min post-resuscitation, this dysfunction persisted for at least 4 h, and a parallel decline in LV function and coronary flow reserve was evident[16]. The reduction of PO_2 post-CPR might due to low cardiac output, ischemia reperfusion injury and pulmonary injury after resuscitation that resulted in cardiac pulmonary edema and gas-exchange dysfunction. In addition, the levels of cTnI in our study increased and topped at 2 h after ROSC, this might indicate a structural damage in the heart during and after resuscitation, and then severe myocardial injury recovered at 48 h after ROSC.

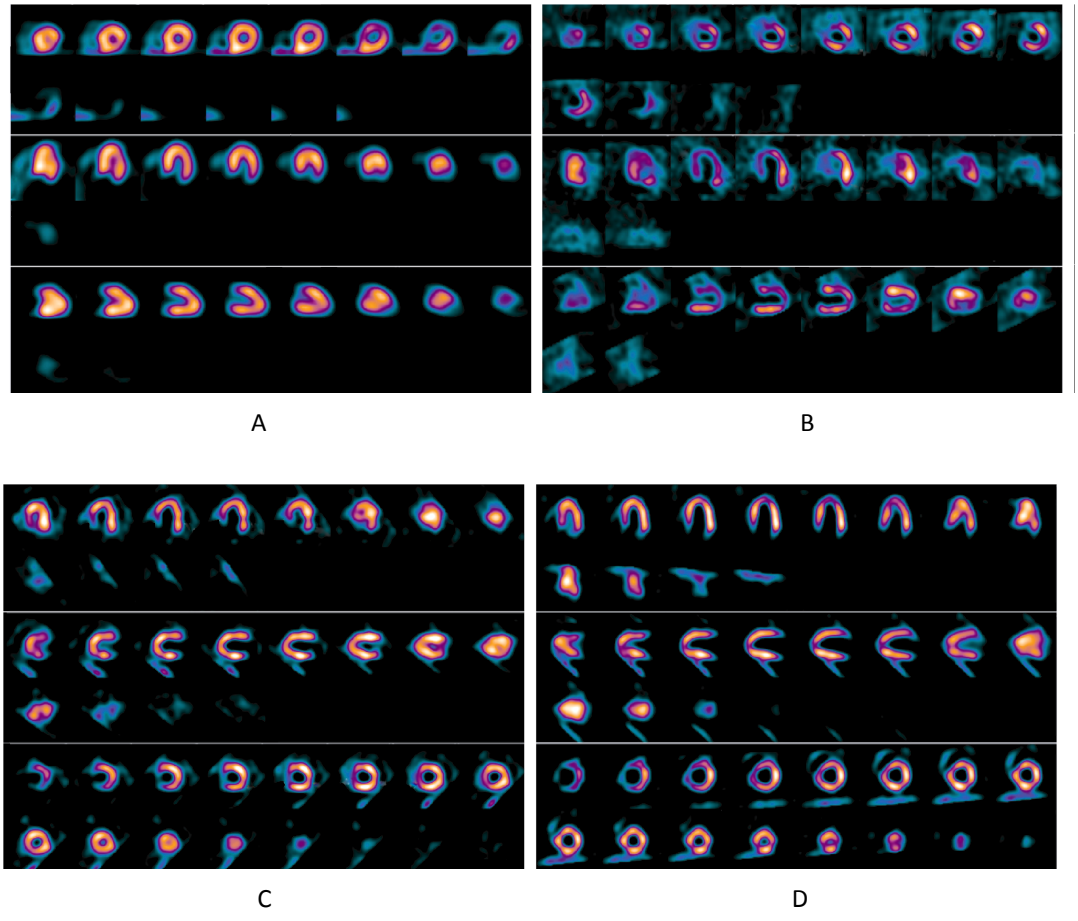

Figure 3. Examples of porcine cardiac perfusion on rest ^{99}mTc-MIBI gated single photon emission computed tomography at baseline and after restoration of spontaneous circulation.

A: Normal. No sparse distribution and defect of radioactivity in short axis, horizontal long axis and vertical long axis.

B: 4 h after ROSC. White arrow signed sparse distribution and defect of radioactivity in anteroseptal wall; red arrow signed sparse distribution and defect of radioactivity in postero-lateral wall.

C: 24 h after ROSC. White arrow signed sparse distribution and defect of radioactivity in posteroseptal wall; red arrow signed sparse distribution and defect of radioactivity in postero-lateral wall that has better than 4 h after ROSC.

D: 48 h after ROSC. Sparse distribution and defect of radioactivity recovered comparing to 24 h after ROSC.

CPP and MAP are highly relevant to myocardial perfusion, but are also reliable indicators of success of CPR[17]. As we finished study, we further compared hemodynamics and G-SPECT between eventual survival animals (*n*=8) and non-survival animals (*n*=5) at 4 h after ROSC, only MAP, CPP and CTNI had significant difference. So we could show the recovery of MAP, CPP and CTNI would be indicators for long-term survival after cardiac arrest and CPR. Kilgannon also demonstrated that early exposure to arterial hypotension after ROSC was common and an independent predictor of death[18]. Among the five pigs that eventual non-survival animals, only two pigs had an echocardiogram performed at 4 h after ROSC, so the difference of echocardiogram was not observed. The reasons for five non-survival animals death were cardiac failure (*n*=1), arrhythmia (*n*=2), ARDS (*n*=1) and hemorrhagic shock (*n*=1) following 44 h after ROSC.

Echocardiography and Gated SPECT Myocardial Perfusion Imaging

The progressive impairment of diastolic function

has been described by three abnormal transmitral Doppler patterns. These are the, "delayed relaxation" pattern, "pseudonormal" filling pattern, and "restrictive" filling pattern. The different patterns are dependent upon the degree of myocardial damage. In our study, based on MV-E/A decrease to 0.87±0.10, MVDT compensation was prolonged to 54.00±7.71, and the delayed relaxation pattern was observed 2 h after ROSC. This indicated post-resuscitation myocardial diastolic dysfunction may be an early diastolic dysfunction that is characterized by a decrease in active relaxing. MV-E/A was slightly more than 1, and MVDT increased 4 h after ROSC, which indicated that diastolic dysfunction improved gradually and did not evolve to a "pseudonormal" filling pattern 4 h after ROSC. Then MV-E/A and MVDT returned to normal 24 h after ROSC. Other study for postresuscitation LV diastolic function[19] showed increased isovolumic relaxation time (IVRT) was significantly longer in patients with noncardiac causes and initial rhythm of non-ventricular fibrillation/tachycardia. MV-DT reflected 80% of diastolic blood flow after opening of the mitral valve and responsed to the increased hardness of the left ventricle, which is a very effective indicator to reduce compliance. When LV stiffness increases, MVDT will be extended. So we selected MV-DT to assess LV diastolic function after resuscitation.

Klouche[5] described the term stone heart in animals with untreated VF, they showed that with increasing duration of untreated VF, progressive reductions in LV diastolic and stroke volume and increases in LV free-wall thickness were documented. Stony heart is a severe form of ischemic contracture in which a progressive impairment in diastolic function during CPR precedes evolution of the "stone heart" after failure of or prolonged CPR[1,5]. In our study, we indicated it would have a great impact on the early active relaxation function after successful CPR, and diastolic dysfunction could recover earlier (24 h after ROSC) than systolic dysfunction (48 h after ROSC). The reversibility of cardiac systolic and diastolic functions after CPR, which is called cardiac stunning, might be potentially adaptive and protective in survival animals after CPR.

The significant defect of myocardial perfusion which was indicated by increase of perfusion indices (SRS) showed no-reflow phenomenon, poorer LV function during CPR and ROSC. The no reflow phenomenon which is a notorious problem in postischemic tissue in general, but particularly after cardiac arrest, it also would specifically leads to microvascular damage and myocyte necrosis and apoptosis, which is widely known as reperfusion injury[20]. The left anterior descending artery perfusion defect accounted for 63.9% of total myocardial perfusion. The most serious hypoperfusion was in the left anterior descending artery and disproportionate coronary artery perfusion occurred after CPR. The cardiac perfusion defect on G-SPECT was more serious at 4 h than that at 24 h after ROSC; the perfusion defect at 48 h after ROSC was virtually absent. Myocardial hypoperfusion, which induced cardiac systolic and diastolic dysfunction, could be reversible in survival pigs. Therefore, cardiac stunning included systolic dysfunctions, early active relaxation dysfunctions and myocardial hypoperfusion, which could be reversed during 48 h after CPR.

We found the value of LVEF with SPECT was a little lower to that with echocardiogram, but the correlation between them was good. Little higher LVEF values were quoted in previous studies with SPECT than that with echocardiogram[21-22]. The reason of the little difference might be due to not quite accurate geometrical tangential capture of LV in its long axis in individual apical echocardiographic projections leading into underestimation of echocardiographic measurement of LV volumes comparing to gated-SPECT. Another reason could be a lower space resolution of gated-SPECT compared to echocardiography[21].

Several limitations of this study should be discussed. First, we want to investigate cardiac function during 48 h after CPR, and try to find the indicators for long-term survival after CPR, so we selected 4 min of untreated ventricular fibrillation. But, a 4-min period of cardiac arrest is relatively short and may not be representative of clinical scenarios. We will extend the time of VF in future studies. Second, due to the difficulty in collecting data of all non-survival pigs, further study between survival animals and non-survival animals was not available. Third, echocardiogram and perfusion indices of SPECT are done by visual review of data, which are subjective and possible source of error. Finally, in clinical cardiac arrest, the majority of patients have coronary disease or pre-existing left ventricular dysfunction, so direct applicability to human cardiac resuscitation must be our further clinical study.

CONCLUSIONS

A Swan-Ganz catheter, echocardiography, and G-SPECT myocardial perfusion images were applied together to demonstrate myocardial stunning after CPR, it showed LV systolic and diastolic function (early active relaxation function) were impaired after cardiac arrest and CPR, and diastolic dysfunction could be reversed earlier than systolic dysfunction (24 h than 48 h after ROSC). Cardiac stunning might be potentially adaptive and protective within survival animals after CPR. The left anterior descending artery hypoperfusion was most serious and the recovery of MAP, CPP, and CTNI could be indicators for long-term survival after cardiac arrest and CPR.

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