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Experimental paper

Coronary perfusion pressure during external chest compression in pseudo-EMD, comparison of systolic versus diastolic synchronization☆

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ABSTRACT

Background: The fraction of cardiac arrest patients presenting with pulseless electrical activity is increasing, and it is likely that many of these patients have pseudo-electromechanical dissociation (P-EMD), a state in which there is residual cardiac contraction without a palpable pulse. The efficacy of cardiopulmonary resuscitation (CPR) with external chest compression synchronized with the P-EMD cardiac systole and diastole has not been fully evaluated.
Hypothesis: During external chest compression in P-EMD, the coronary perfusion pressure (CPP) will be greater with systolic synchronization compared with diastolic phase synchronization.
Methods: A porcine model of P-EMD induced by progressive hypoxia with peak aortic pressures targeted to 50 mmHg was used. CPR chest compressions were performed by either load distributing band or vest devices. Paired 10 s intervals of systolic and diastolic synchronization were performed randomly during P-EMD, and aortic, right atrial and CPP were compared.
Results: Stable P-EMD was achieved in 8 animals, with 2.6 ± 0.5 matched synchronization pairs per animal. Systolic synchronization was association with increases in relaxation phase aortic pressure (41.7 ± 8.9 mmHg vs. 36.9 ± 8.2 mmHg), and coronary perfusion pressure (37.6 ± 11.7 mmHg vs. 30.2 ± 9.6 mmHg). Diastolic synchronization was associated with an increased right atrial pressure (6.7 ± 4.1 mmHg vs. 4.1 ± 5.7 mmHg).
Conclusion: During P-EMD, synchronization of external chest compression with residual cardiac systole was associated with higher CPP compared to synchronization with diastole.

1. Introduction

The incidence of pulseless electrical activity (PEA) as the initial presenting out-of-hospital cardiac arrest rhythm appears to be increasing.[1] Although this may reflect a relative decrease in the incidence of ventricular fibrillation, it is also possible that the changing substrate of atherosclerotic heart disease, in particular the increasing number of patients surviving in the population with varying degrees of ventricular dysfunction, may be causing secular increases in the absolute number of patients suffering PEA. As the incidence of PEA arrest increases, the need to further study PEA and to create new therapies that address this clinical condition becomes important.

We have previously shown that PEA is a heterogeneous clinical entity encompassing various levels of physiologic derangement,[2] and emergent cardiac ultrasonography of patients in apparent cardiac arrest has confirmed residual cardiac wall motion in many.[3,4] Subsequent invasive blood pressure measurement revealed that PEA-associated hemodynamics range from complete absence of cardiac wall motion resulting in true electromechanical dissociation (EMD), through to states of hypotension with a measurable blood pressure. We have termed cardiac arrest with measurable pulse pressures or coordinated left ventricular mechanical function to be pseudo-EMD (P-EMD). Prehospital investigations indicate that a majority of patients in PEA may actually be in P-EMD with detectable cardiac motion for some portion of resuscitation.[5]

The discovery of P-EMD made possible that CPR could be administered in a pattern synchronized with the intrinsic cardiac

Abbreviations: CPP, coronary perfusion pressure; CPR, cardiopulmonary resuscitation; ECG, electrocardiogram; ROSC, return of spontaneous circulation; EMD, electromechanical dissociation; PEA, pulseless electrical activity; P-PEA, pseudo-pulseless electrical activity; MBF, myocardial blood flow.

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contractions, and that such synchronization might improve outcome. Of particular import is the possibility that unsynchronized compressions may interfere with the ventricular filling of P-EMD and significantly decrease function cardiac output degrade the efficacy of CPR.

The present study was intended to evaluate the potential benefit of CPR synchronization during P-EMD. We hypothesized that maximal benefit would occur when external chest compression was synchronized with the systolic ejection phase of P-EMD, and maximal decrement might occur when it was synchronized with diastolic filling. Using a porcine asphyxial model of P-EMD, we compared the coronary perfusion pressure (CPP)[6] during chest compression synchronized with residual systole and diastole.

2. Methods

The application of external chest compression CPR during P-EMD cardiac arrest requires the defining of new terminology:

P-EMD systole: The period of ventricular ejection, detectable by arterial pressure measurement.
P-EMD diastole: The period of ventricular filling, defined as the intervals between P-EMD systoles.
P-EMD rate: The number of P-EMD systoles per minute.
P-EMD systolic synchronization: Execution of the external chest compression downstroke coincident with P-EMD systole, with or without a brief offset delay.
P-EMD diastolic synchronization: Execution of the external chest compression downstroke coincident with P-EMD diastole.
P-EMD CPR compression phase: The period of external chest compression during P-EMD starting at onset of the downstroke and lasting until the onset of the upstroke.
P-EMD CPR relaxation phase: The period of external chest compression during P-EMD starting at the onset of the upstroke and lasting until the next downstroke.
Electrical trigger: Electrical signal obtained either by surface ECG, or intracardiac electrode, utilized for triggering and synchronization of chest compression.
Synchronization offset: A delay between the onset of P-EMD systole and the onset of synchronized chest compression.

The study was conducted in accordance with the guidelines of the American Physiological Society for the Care and Use of Laboratory Animals and with the approval of the Johns Hopkins University Institutional Animal Care and Use Committee. The model utilized was a variation on previously described porcine asphyxial P-EMD preparation.[7]

Domestic Yorkshire swine weighing 18–20 kg were fasted over-night with free access to water and then sedated with an intramuscular injection of ketamine (30 mg/kg). After endotracheal intubation, anesthesia was initially be maintained with isoflurane (0.5–4%) and oxygen (1–3 L/min). Ventilation was provided by a volume-controlled ventilator (Draeger EV-A, Lübeck, Germany) with 100% O_2 (tidal volume of 15–20 cm^3/kg and ventilation rate of 8–15 breaths/min). Ventilation rate and tidal volume were initially adjusted to maintain normocapnia (the end-expiratory partial pressure of CO_2 between 35 and 45 mmHg) as measured continuously by a capnometer (NPB-75, Nellcor Puritan Bennett, Boulder, CO) placed in the airway. Arterial blood gases (ABL80, Radiometer, Denmark) were analyzed to confirm adequate baseline ventilation. Throughout the experiment, the animals were monitored using ECG, end-tidal CO_2, and arterial blood pressure. In addition, depth of anesthesia was continuously assessed. The animals were paralyzed using pancuronium (0.1 mg/kg) to minimize respiration effect on blood pressure during CPR.

The animals were secured in a supine position and were given normal saline at a rate of 10 mL/(kgh) through a vein to maintain a central venous pressure of ~5 mmHg. Through ultrasound guided percutaneous cannulations, three micromanometer single pressure sensors (SPC-350, Millar Instruments, Houston, TX) and a pressure sensor with a lumen (SPC-471A, Millar Instruments) were placed into: (1) the right atrium via the femoral vein, (2) the descending aorta through the femoral artery for pressure measurements, and (3) the left ventricle via the carotid artery for pressure measurement. All catheters were positioned under fluoroscopic guidance, and unfractionated Heparin 100 units/kg was given to prevent catheter clotting.

After instrumentation, baseline measurements were obtained for all variables including blood gas analyses. Analog outputs of the physiological parameters were digitized and stored in data files on a personal computer for further analysis using a 16-channel computerized data-acquisition system at a sampling rate of 400 Hz (Powerlab 16SP, ADInstruments, Castle Hill, Australia). Raw data channels included ECG, aortic pressure, right atrial pressure, left ventricular pressure, end-tidal CO_2, continuous CO_2 level, and CPR depth. Coronary perfusion pressure (CPP), defined as the average difference between the aortic pressure and right atrial pressure during the release phase of chest compression, was calculated using Chart 5 software (ADInstruments, Castle Hill, Australia).

Animals were converted to continuous intravenous anesthesia using ketamine (50 mcg/kg/min) and fentanyl (0.45 mcg/kg/min), isoflurane was gradually discontinued, and depth of anesthesia were maintained as determined by baseline blood pressure and heart rate readings. The animals were maintained in this plane of anesthesia for 15 min to allow isoflurane washout and to establish a stable level of continuous IV anesthesia prior to initiation of the arrest protocol. Intermittent boluses of ketamine (2 mg/kg), fentanyl (0.1 mcg/kg), and pancuronium (0.5 mg/kg) were administered if needed to maintain adequate anesthesia and paralysis.

Arterial blood gas (pH, pCO_2, pO_2, HCO_3, %saturation), serum sodium, serum potassium, serum glucose, hemoglobin, hematocrit, serum ionized calcium, and base excess (BE) were measured and recorded at regular intervals.

Once adequate anesthesia has been confirmed, the animals were ventilated with a hypoxic gas mixture of O_2/N_2 for the duration of the experiment. Gas concentrations were measured using an oxygen concentration analyzer (Oxygen Analyzer S-3A/II, Applied Electrochemistry, VMETEK) and the concentration of O_2 was adjusted to achieve stable P-EMD. Onset of pseudo-EMD was defined as an aortic systolic pressure $\leq$60 mmHg recorded by the aortic catheter in the presence of an organized cardiac rhythm.

Chest compressions were performed by two devices: (1) a custom stepper motor based system that is under full computer control (Labview) and intended to provide the mechanics of an animal adapted load distributing band CPR device[8] and (2) a pneumatic vest previously demonstrated to provide excellent CPR hemodynamics.[9] Both devices allow rapid and accurate control of chest compression mechanics.

Once stable P-EMD has been achieved, paired 10 s periods of systolic and diastolic synchronization were applied in a random order with minimal delay between alternative methods (Fig. 1). The desired synchronization pattern was achieved using either the ECG, intraventricular electrogram, or the aortic pressure as a trigger, along with an appropriate synchronization offset needed achieve either systolic or diastolic synchronization (Fig. 2). The number of paired 10 s periods per animal was ad hoc and based on the length of time stable P-EMD was maintained.

If ROSC occurred (peak systolic pressure > 60 mmHg without chest compressions), the oxygen was adjusted downward to again achieve pseudo-EMD with pressures in the desired range.

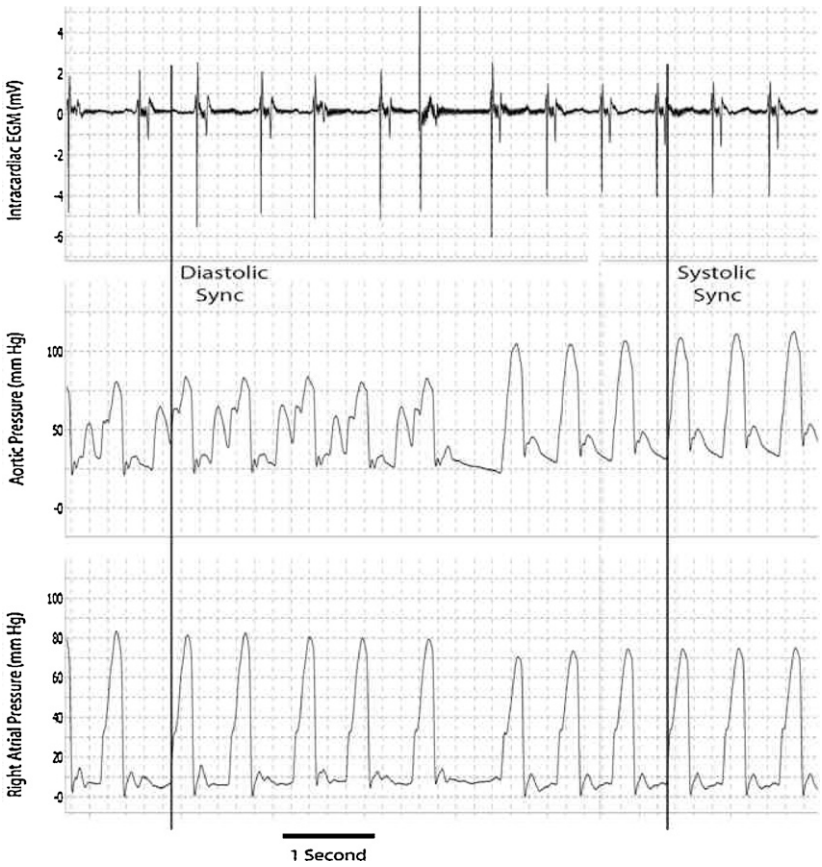

Fig. 1. Aortic and right atrial pressures during the transition from diastolic to systolic synchronization. The vertical lines indicate where a chest compression starts, and are timed to the intracardiac electrograms. With diastolic synchronization, the peak aortic pressure during intrinsic mechanical systole is just to the left of the vertical line, while the peak pressure with chest compression is just to the right of the vertical line. With systolic synchronization, the peak aortic pressure during intrinsic mechanical systole and during chest compression are coincident, and just to the right of the vertical line.

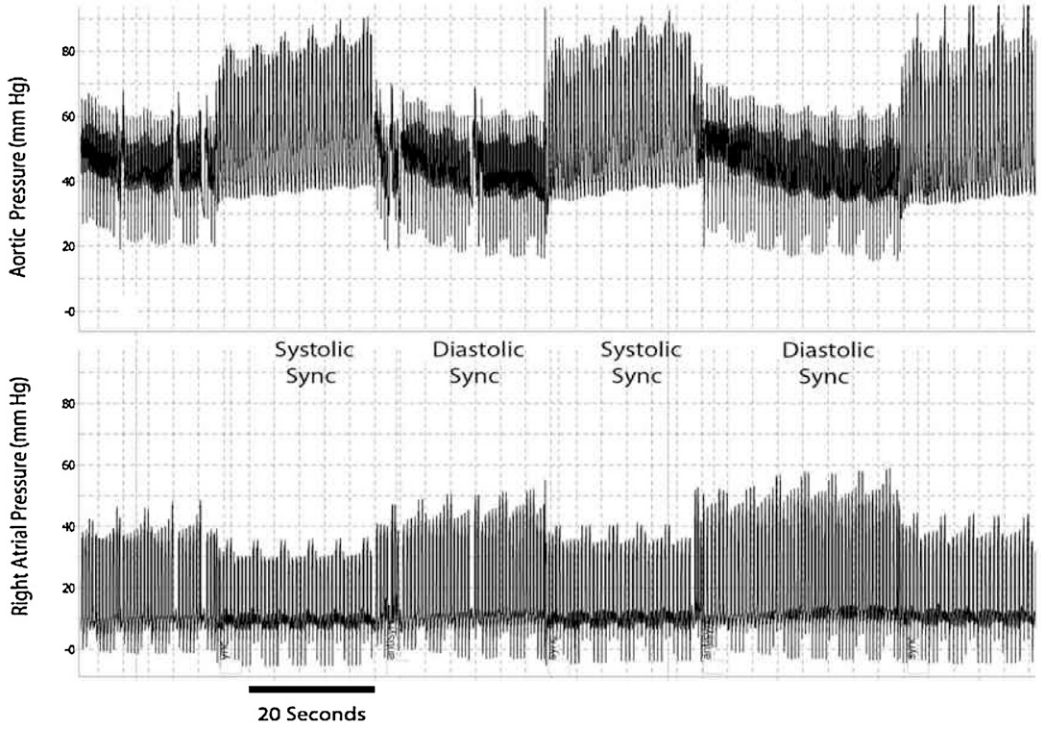

Fig. 2. Aortic and right atrial pressures during alternating brief periods of systolic and diastolic synchronization.

Table 1
Baseline hemodynamics (M ± SD, mmHg as appropriate).

Variables	Mean ± SD	95% CI of mean
Heart rate	78.6 ± 12.1	68.5–88.8
Ao systole pressure	123 ± 31.7	96.5–149.5
Ao diastole pressure	91.4 ± 27.5	68.4–114.4
RA pressure	1.0 ± 2.8	−1.3–3.3

During P-EMD and CPR, aortic and right atrial pressures were measured, and coronary perfusion pressures during CPR was defined as the aortic minus the right atrial pressure during the relaxation phase of external chest compression.[10,11] Because diastolic synchronization places the CPR pressure impulse during the beginning of the phase in which the aortic and right atrial pressures are measured for calculation of the coronary perfusion pressure, these pressures were obtained from the portion of the relaxation phase between the end of the CPR compression and the upstroke of the intrinsic systolic pressure.

After completion of the protocol, the animals were euthanized with pentobarbital and potassium chloride. Postmortem examination was performed for identification of visceral and thoracic organ injuries and histology.

Data analysis was performed using SAS version 9.2 (SAS Institute Inc., North Carolina USA), and reported as mean ± standard deviation ($M \pm SD$) along with confidence intervals (CI) unless otherwise noted. A linear repeated measures regression model which accounted for multiple measurements per animal was used to determine differences in the treatment effect. The model was fit via maximum likelihood and a compound symmetric covariance structure was used. A p value <0.05 was considered statistically significant.

3. Results

Stable P-EMD was achieved in 8 animals, with 2.6 ± 0.5 matched synchronization pairs per animal. Baseline and P-EMD hemodynamics are shown in Tables 1 and 2, respectively.

Typical waveforms during diastolic and systolic synchronization are shown in Fig. 1. The chest compressions were synchronized to the intracardiac electrograms. The vertical lines show where respective chest compression started. The synchronization offset between the onset of P-EMD systole and the onset of synchronized chest compression averaged 14 ms during systolic synchronization and 313 ms during diastolic synchronization.

Peak aortic pressure was increased with systolic synchronization. In addition, aortic relaxation phase pressure was increased, and relaxation phase right atrial pressure was decreased, with systolic synchronization (Fig. 2). These changes in relaxation phase aortic and right atrial pressures resulted in an increase in coronary perfusion pressure with systolic synchronization.

Pressures during successive periods of systolic and diastolic synchronization in a single animal are shown in Fig. 2.

For the entire group of animals, systolic synchronization was associated with increases in relaxation phase aortic pressure, and coronary perfusion pressure (Table 3). Diastolic synchronization was associated with an increased right atrial pressure. The systolic CPP and peak aortic pressure were higher in 16 of 17 systolic–diastolic synchronization pairs that were evaluated. Subgroup analysis for sequences with each of the two different devices showed no differences or trends. However, these data sets were small.

Table 2
P-EMD hemodynamics (M ± SD, mmHg as appropriate).

Variables	Mean ± SD	95% CI of mean
P-EMD heart rate	89.7 ± 21.9	77.6–101.9
P-EMD Ao systole	59.1 ± 16.7	50.8–67.3
P-EMD Ao diastole	38.6 ± 12.4	32.4–44.7
P-EMD RA	6.3 ± 4.3	3.9–8.7

4. Discussion

As the proportion of arrested patients with PEA increases, therapies that may improve the outcome in PEA become increasingly important. PEA has no intervention, analogous to defibrillation, that can completely and promptly extricate the patient from what is a life threatening pathophysiologic state. Clinicians must resuscitate the patient out of an extreme low-flow state through improved coronary perfusion. Thus, hemodynamic interventions are of central importance in PEA.

Improving the outcome of patients with PEA may be made considerably more difficult by the significant fraction of such patients who are actually in P-EMD, and the lack of animal models for the study of this entity. Earlier animal models include asphyxiation and post-countershock, both of which tend to result in only brief periods PEA.[12] Spreng et al. found only a brief duration from loss of femoral pulses (onset of pseudo-EMD) to loss of all aortic fluctuations (true-EMD) in the total asphyxial swine model of PEA arrest.[13] Such brief periods of P-EMD are inadequate for comparison of interventions or delineation of mechanisms, and we developed this partial asphyxiation model of P-EMD to address this need.

We have achieved relatively prolonged and hemodynamically stable P-EMD through sequential decreases in FIO2. In general, P-EMD was achieved with an oxygen concentration of about 10%, but there was significant variability. Once P-EMD was achieved, external chest compression was synchronized with the systolic and diastolic phases using the surface ECG, intraventricular electrogram or aortic pressure waveform as triggering events. Systolic synchronization was associated with increased relaxation phase aortic pressure, and decreased relaxation phase right atrial pressure, both of these would contribute to higher coronary perfusion pressure during systolic synchronization.

The cardiac arrest state of P-EMD is qualitatively different from ventricular fibrillation, asystole and true-EMD, in that it is associated with varying degrees of intrinsic hemodynamics.[2] Although CPP during external chest compression is classically believed to be limited to the relaxation phase of CPR,[10,11] this may not be the case during P-EMD, during which the intrinsic hemodynamics of P-EMD may result in varying degrees of forward aortic flow during systole. In this setting, the probability of ROSC may be a function of both the CPR diastolic CPP and the P-EMD hemodynamics.

With the aggregate hemodynamics during P-EMD and external chest compression possibly reflecting varying contributions from each component, it was reasonable to hypothesize that one of the multiple temporal relationships that might exist between the intrinsic hemodynamic cycle of P-EMD, and the induced hemodynamics created by external chest compression, might be optimal.

Synchronization during the relaxation phase of external chest compression initially seemed attractive since it appeared to augment CPR CPP directly. However, because the pulse pressure that is generated by the intrinsic left ventricular ejection of P-EMD is dependant on ventricular filling, we actually hypothesized that the external chest compression synchronized so as not to interfere – and even possibly augment – ventricular filling would be optimal. This turned out to be the case, with higher aortic and lower RA pressures during systolic synchronization.

In stable preparations, the effects of synchronization on aortic and right atrial pressures appeared to increase over time (Fig. 2), and in general the absolute effects on aortic pressure were greater than those on right atrial pressure.

Table 3
Comparison of CPR systolic and diastolic synchronization (mmHg, $M \pm SD$).

	Sys-Sync	Dias-Sync	Delta	%delta	*P* value
Relaxation Ao	41.7 ± 8.9	36.9 ± 8.2	4.8 ± 5.7	12	0.0009
Relaxation RA	4.1 ± 5.7	6.7 ± 4.1	−2.6 ± 3.5	−63	0.002
CPP	37.6 ± 11.7	30.2 ± 9.6	7.4 ± 7.0	20	0.0001
Peak Ao	86.7 ± 16.4	69.3 ± 14.1	17.5 ± 10.3	20	<0.0001

The equation for CPP in a beating heart is the diastolic arterial blood pressure minus left ventricular end diastolic pressure. During cardiac arrest, there tends to be no gradient between left ventricular pressure and right atrial pressure, and the equation appears to collapse to aortic minus right atrial pressure during the relaxation phase of external chest compression. It seems likely that in the hybrid state of P-EMD and CPR, in which each component may contribute to perfusion to a greater or lesser extent, that the equation for CPP may be a hybrid of the spontaneous circulation and cardiac arrest equations. Increasing the complexity, the equation for CPP under these circumstances may be variable depending on the intrinsic P-EMD pressures and the efficacy of CPR. When the P-EMD pressures are very low, below 30 mmHg for instance, most forward flow may be from CPR. When the intrinsic blood pressure is relatively higher, say above 50 mmHg, then the contribution from external chest compression might be relatively small and the equation for perfusion similar to normal hemodynamics. Between the low-intrinsic and high-intrinsic conditions, the equation would have varying contributions from each source of hemodynamics.

This line of reasoning leads to the hypothesis that there may be an optimal range of P-EMD pressures for augmentation of CPP with systolic synchronization, and that there may be states of extreme shock just above PEA, in which systolic synchronization may be beneficial. Our current study does not address these issues.

Limitations: Our results have a number of limitations beyond those that are standard for preliminary data generated in an animal model. Most important of these is the use of CPP as a surrogate for outcome. Although the aortic to right atrial pressure difference during relaxation phase of CPR has been found, to a greater or lesser extant, to be predictive of outcome and likely associated with myocardial blood flow, its utility during P-EMD and CPR has not yet even been studied.

Although alterations in CPP may be valid when measured in an asphyxial model of P-EMD, the state of the ventricular fibrillation is likely qualitatively different, and actual clinical outcome would likely be different in the setting of P-EMD associated with coronary occlusion.

Our study will need to be followed by a series of investigations in which the relationships between P-EMD hemodynamics and CPR hemodynamics are related to actual myocardial blood flow measured by microspheres, and then actual ROSC. Because microsphere blood flow measurements require relatively long periods of stable hemodynamics, the asphyxial P-EMD model may be ideal in this setting.

Our comparison of systolic to diastolic synchronization may indicate the maximal effect of synchronization on CPP. In clinical practice, chest compressions are applied randomly with respect to the intrinsic hemodynamics of P-EMD. In all likelihood, there will be varying periods of systolic and diastolic synchronization by chance, and a comparison with controlled synchronization might be less dramatic. Alternatively, further investigations may identify more optimal patterns for onset and withdrawal of synchronization, enhancing the effect.

Measurement of CPP when compressions are being performed during the diastolic phase was difficult, and to some extent our measurement of CPP was interpreted. This likely degraded the accuracy of this measurement.

The small number of experiments performed, and the limited number paired comparisons, also limit the utility and applicability of our results.

In conclusion: In this porcine model, progressive hypoxia resulted in relatively prolonged periods of P-EMD. Synchronization of external chest compression to the systolic phase of P-EMD resulted in higher aortic and lower right atrial pressures, with resultant increases in CPP, compared with diastolic phase synchronization. Synchronization of CPR may improve outcomes in PEA.

Conflict of interest statement

Dr. Paradis holds U. S. Patent 7,645,247 "A device for synchronizing the parameters of resuscitative therapies to residual myocardial activity during cardiopulmonary resuscitation," which has been licensed to ZOLL, Inc. the sponsor of the study. Dr. Paradis and Dr. Barash are consultants to ZOLL. Dr. Henry Halperin is a consultant of ZOLL Medical Corporation, and his financial interests are governed by policies of Johns Hopkins University. Drs. Quan and Freeman are employees of ZOLL Inc.

Role of the funding source

This work was supported by ZOLL Inc. and its employees and consultants participated in the research.

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