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Practical application of pressure-volume loop analysis in a swine model of medical cardiac arrest

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ABSTRACT

INTRODUCTION: Cardiac arrest (CA) is the sudden cessation of cardiac activity, leading to hemodynamic collapse and high mortality. Advanced cardiac life support (ACLS) standardizes resuscitation, but the post-return of spontaneous circulation (ROSC) period remains turbulent and poorly understood. Pressure-volume loop (PVL) analysis offers insight into load-independent cardiac biomechanics. The aim of this study is to explore the feasibility PVL analysis in a swine model of CA.

MATERIALS AND METHODS: This swine study utilized a ventricular fibrillation (VF) cardiac arrest model in combination with PVL analysis. Swine underwent anesthesia and instrumentation. Ventricular fibrillation was induced using an electrophysiology catheter. Subjects were divided into 3- and 6- minutes of CA before commencement of ACLS for 30 mins. If ROSC was obtained, animals underwent a 3-hr critical care period. Cardiac indices were compared between baseline and end-of-study values.

RESULTS: Eight Yorkshire swine were studied, with VF successfully induced and PVL data collected. All animals in the 3-minute ACLS group achieved ROSC compared to one in the 6-minute group. Post-ROSC metabolic changes included acidemia, elevated lactate and potassium, partially resolving by study end. Right ventricle PVL data was unreliable, while left ventricle PVL was reliable in 3 of 5 ROSC animals. Preload-recruitable stroke work proved most trustworthy, showing a hyperdynamic circulation post-resuscitation.

CONCLUSIONS: This study demonstrates that PVL analysis can be applied to a post-ROSC swine model of CA, although there are technical limits. Unsurprisingly, warm ischemic time predicted ROSC; however, PVL analysis provided insights into load-independent parameters and has the potential to guide future therapeutic targets for post-ROSC intervention.

KEY WORDS: Cardiac arrest, pressure-volume loops, animal model, cardiac physiology, pre-recruitable stroke work

INTRODUCTION

American Heart Association defines cardiac arrest (CA) as sudden cessation of cardiac activity resulting in a hemodynamic collapse, which results in death without reversal. In CA, Ventricular fibrillation (VF) accounts for 50–80% of cardiopulmonary arrests, while pulseless electrical activity (PEA) and asystole contribute to 20–30% [1]. CA affects around 700 000 people in the United States annually, highlighting the need for standardized guidance, which has been effectively addressed by the American Heart Association Advanced Cardiac Life Support (ACLS) guidelines [2]. The implementation of ACLS measures has proven successful in improving patient outcomes in in-hospital CA scenarios, with studies demonstrating that adherence to the proposed algorithm is associated with higher rates of return of spontaneous circulations (ROSC) and better neurological outcomes, while deviations are linked with poorer results [3-5].

However, up to 50% of out-of-hospital CA patients who initially achieve ROSC subsequently experience rearrest, a grave event strongly associated with high mortality [6-8]. Rearrest typically occurs within minutes of ROSC, with one study of nearly 1,200 patients indicating a median (IQR) time of 3.1 (1.6-6.3) minutes [7]. It is, therefore, of utmost importance to better understand the immediate post-ROSC period. One of the major conditions contributing to rearrest is post-cardiac arrest syndrome (PCAS). It is defined as a complex condition affecting patients achieving ROSC and is related to multi-systemic insults caused by whole body ischemia that occurred during CA, and subsequent reperfusion injury after ROSC [9]. The key components of PCAS are brain injury, myocardial dysfunction, and systemic ischemia-reperfusion injury in addition to the precipitating pathology [10]. Most of the cardiac arrest research has focused on increasing ROSC rates, leading to significant advancements. However, many of these interventions do not improve long-term survival [10]. Strong evidence to indicate that any specific therapy target significantly modifies PCAS to improve outcomes is lacking [9].

Pressure-volume loop (PVL) analysis is a nascent technology that allows for comprehensive cardiac biomechanics by illustrating the relationship between ventricular pressure and volume throughout the cardiac cycle [11,12]. It is considered the gold standard for assessing load-dependent and load-independent measures of ventricular function [13]. This is yet to explored in the post-ROSC period. Our group has recently developed a method of PVL insertion for application in a closed chest swine model, providing a comprehensive approach for determining hemodynamic parameters and offering a novel method for analyzing raw PV loop data beyond individual cardiac cycles [14]. The aim of the current study is to apply PVL analysis to a swine model of CA to understand the practical application of PVL in the post-ROSC period to determine practicality and identify any potential targets for future therapies in this critical period.

MATERIALS AND METHODS

Study design and overview

This is a swine study using a model of VF cardiac arrest and PVL surveillance. Before commencing the experimental protocol, approval from the Institutional Animal Care and Use Committee (IACUC) of

Mayo Clinic (Rochester, MN, USA) was obtained. The study was conducted between November and December 2023, at Mayo Clinic, Rochester Minnesota. Eight Yorkshire swine weighing between 45-70kg were enrolled in the study. The animals were acclimatized to the environment for a minimum of 24 hours under the care of professional veterinary staff. They had access to food and water until the night prior to surgery. The experiment consisted of four phases: animal preparation, induction of VF, resuscitation and post-return of spontaneous circulation (ROSC) care (Fig. 1). Hemodynamic data was collected throughout the experiment. At the end of the study, swine were euthanized with a pentobarbital injection.

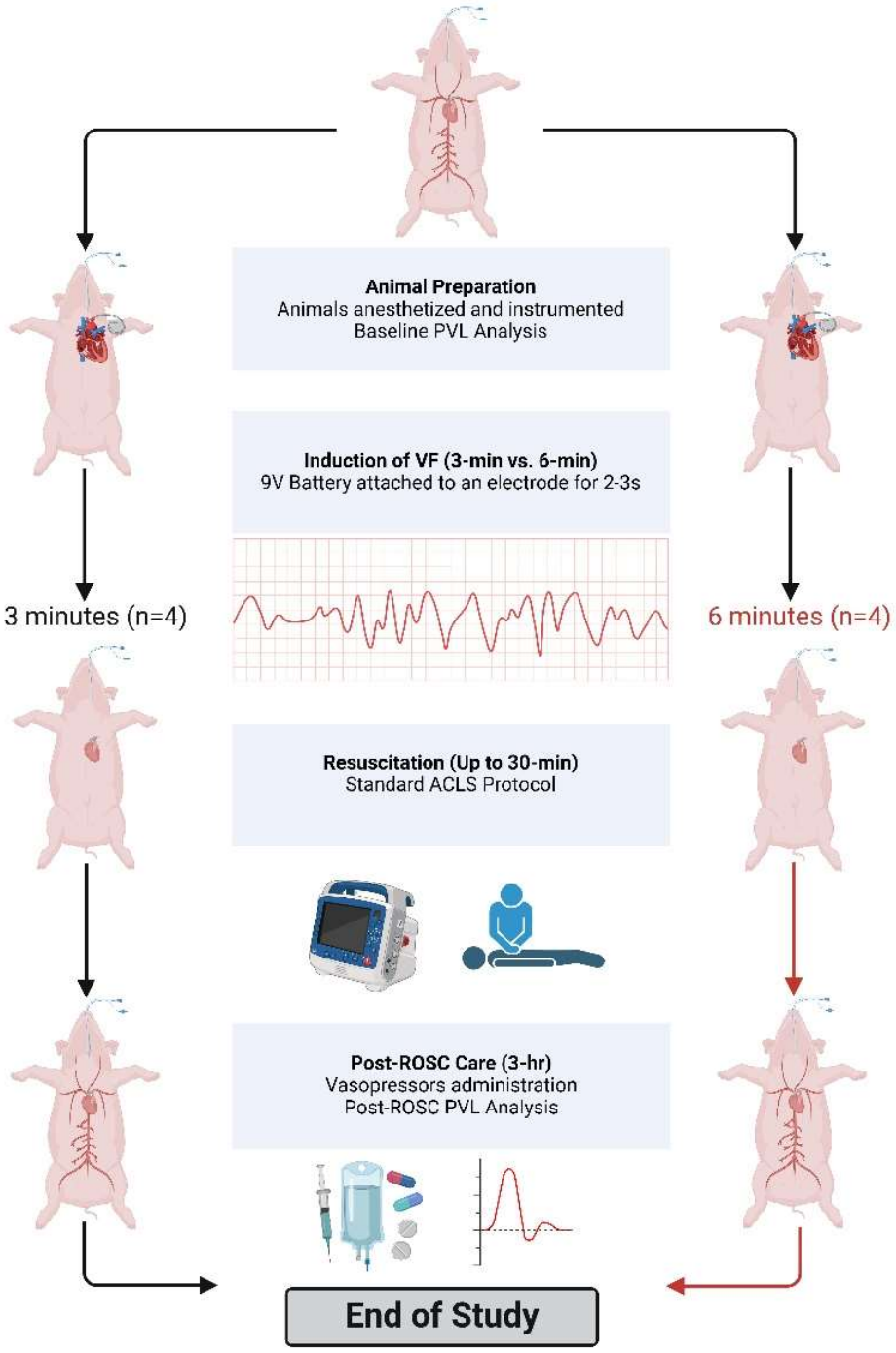

Figure 1. Experiment layout.

Animal preparation

The animals were initially sedated with telazol (5 mg/kg) and xylazine (2 mg/kg) via intramuscular injection. The animals were transported to the laboratory and general anesthesia was induced with isoflurane administered via facemask, followed by orotracheal intubation. Buprenorphine was given for analgesia (0.12mg/kg). Anesthesia was maintained with 1%-5% isoflurane in 40%-100% oxygen. The animals were mechanically ventilated using a volume-controlled mode set to 12-15 mL/kg and a respiratory rate of 10-15 breaths per minute. Ventilation parameters were adjusted to maintain an end-tidal pCO2 of 35-45 mmHg. A warming blanket, set to 38°C, was used to ensure normothermia in all animals. The animal was positioned in dorsal recumbency. Intravenous catheter was inserted into the ear vein for a bolus of fluids (1L Ringer Lactate with 5% Dextrose), followed by maintenance sodium chloride at 100 cc/hr. Bilateral carotid arteries, jugular veins, femoral arteries, and the right femoral vein were accessed percutaneously using the Modified Seldinger technique. Seven Fr sheaths (Terumo Medical Corporation, Elkton, MD) were inserted at each access site. The right carotid access was used for the proximal aortic root pressure probe and blood sampling. The left carotid artery was utilized for the temperature probe, thermodilution, and left ventricular pressure-volume (LV PV) loop, inserted using the Stonko method [14]. The right jugular vein was used for the right atrial pressure probe and drug administration. The left jugular vein facilitated the right ventricular pressure-volume (RV PV) loop. A distal aortic root pressure probe was placed through the left femoral artery. The right femoral vein was employed for an endocavitary electrode, powered by a 9V battery, to induce VF, while the left femoral vein was used for an inferior vena cava (IVC) occlusion catheter. Finally, a mini-laparotomy was completed to place a urinary cystostomy.

Induction of VF

The PV Loops from both ventricles were removed. The endocavitary electrode position in the RV was confirmed with fluoroscopy. The pigs were arrested by attaching the electrode to a 9V battery, for about 2 to 3 seconds. Once the arrest was confirmed and rhythm identified, a timer was started.

Resuscitation

The animals were enrolled into one of two groups: 3-minute (n=4) and 6-minute (n=4) arrest. A standard ACLS protocol was delivered and consisted of CPR (between 100-120 compressions per minute), asynchronous ventilation, defibrillation with external paddle delivering 360J biphasic (if shockable rhythm), and medication administration, depending on cycle - 300 micrograms of epinephrine, 300 milligrams of amiodarone, 1g of calcium gluconate, 5g of magnesium, 50 milliequivalents of bicarbonate.

Post-ROSC Critical Care

If the animal survived, it was enrolled in the post-ROSC Critical Care phase. Sustainable ROSC was confirmed with pulsatile flow in the aortic root. PV Loops were reintroduced. Norepinephrine and dobutamine infusion were started to maintain MAP over 40 mmHg, up to the maximum dose of 2mg/kg/min and 20 mcg/kg/min, respectively. Electrolyte abnormalities were corrected, and the animal was monitored for 3 hours, until euthanasia.

Data collection

Physiological data were continuously monitored using the PowerLab system and recorded in LabChart (ADInstruments). Parameters collected included arterial blood pressure (mm Hg), heart rate (beats per minute), temperature (°C), electrocardiography traces (mV), as well as the previously mentioned aortic pressures, and PV loops in both ventricles. Resuscitation data, such as survival, time to ROSC, undertaken ACLS steps and serial serum and ABG blood tests were recorded.

Data analysis

All continuous variables are summarized by mean (SD), and categorical variables are summarized by frequencies. GraphPad Prism v8.0 (GraphPad Software Inc, San Diego, CA, USA) and Python 3.12 (Python Software Foundation, Wilmington, Delaware, USA) were used for visual representation of data and statistical analysis.

RESULTS

Eight adult (seven male and one female) Yorkshire swine were enrolled in the study. The mean weight was 50.2 ± 2.9 kg. All animals were successfully instrumented apart of one pig, due to technical issues with the equipment. Endocavitary electrode was then successfully placed, and VF induction was induced in all animals, proving successful development of a porcine VF cardiac arrest model. Characteristics of the experimental animals are displayed in Table 1.

Table 1. Overview of the animal characteristics. ROSC – return of spontaneous circulation; LVPVL – left ventricular pressure-volume loop; RVPVL – right ventricular pressure-volume loop.

Animal ID	**Sex**	**Weight** (kg)	**ROSC?** (Y/N)	**LVPVL?** (Y/N)	**RVPVL?** (Y/N)	**ROSC Cycle**	**Recovery Time** (hours)
ACLS3-01	M	53	Yes	No	No	1	3
ACLS3-02	M	51	Yes	Yes	Yes	2	3
ACLS3-03	M	54	Yes	Yes	Yes	1	3
ACLS3-04	M	46	Yes	No	No	3	0.67
ACLS6-01	M	53	Yes	Yes	Yes	4	3
ACLS6-02	M	48	No	-	-	-	-
ACLS6-03	M	51	No	-	-	-	-
ACLS6-04	F	47	No	-	-	-	-

During the resuscitation phase, 100% of animals from the ACLS 3-minute group achieved ROSC, whereas only one animal achieved ROSC in the ACLS 6-minute cohort. Amongst the 3-minute cohort, it has taken 1 to 3 cycles to achieve ROSC, whereas the 6-minute cohort animal achieved ROSC in the 4th cycle. Kaplan-Meier survival analysis revealed a longer survival time in ACLS 3-minute compared to ACLS 6-minute animals. The median survival was 180 minutes in the ACLS 3-minute group and 15 minutes in the ACLS 6-minute group. The difference between the two groups was not statistically significant (log-rank test, $p = 0.12$) (Fig. 2).

During the recovery phase, one animal from the 3-minute group was unable to achieve sustained ROSC and was euthanized early at the 40-minute mark due to hemodynamic instability, arrythmias and rearrest. In this animal, no PVL data was recorded.

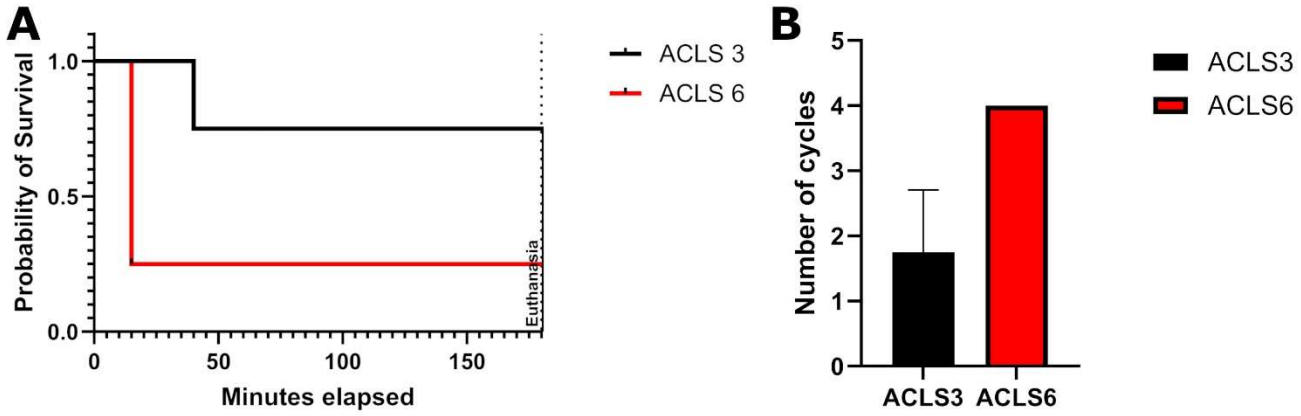

Figure 2. (A) Kaplan-Meier curve showing the probability of survival over time for both cohorts (3-minute vs. 6-minute). (B) Number of cycles required to achieve ROSC in the ACLS 3-minute and 6-minute cohorts.

Post-ROSC, significant metabolic changes were observed. pH decreased from 7.51 ± 0.04 at baseline to 7.18 ± 0.08, indicating acidemia (p=0.016), before improving to 7.47 ± 0.07 at the end of the study. Lactate levels rose markedly from 2.6 ± 0.7 mmol/L to 8.5 ± 1.4 mmol/L (p=0.004), post-ROSC but later decreased to 4.0 ± 1.8 mmol/L. Potassium increased from 3.4 ± 0.2 mmol/L to 6.5 ± 1.4 mmol/L post-ROSC (p=0.013) before stabilizing at 4.4 ± 0.2 mmol/L at the study's conclusion.

With regards to PVL, they were successfully inserted in all animals but one, however, meaningful data was recorded in only three animals. One-minute periods where pressure and volume measurements were stable were identified for evaluation of preload recruitable stroke work (PRSW), yielding 100-200 high-fidelity loops per period, depending on subject heart rate. This procedure was repeated before and after ROSC for each surviving animal. In the LV, the PRSW slope increased by a significant amount for all animals (p<0.001) following ROSC, indicating a load-independent increase in contractility (Tab. 2). A sample PVL and a PRSW slope from animal ACLS3 03 has been depicted in Figure 3.

Table 2. Comparison of left ventricle PRSW at baseline, and post-ROSC. Statistical test: Paired t-test.

Animal ID	LV PRSW Slope pre-ROSC (95% CI) [mmHg]	LV PRSW Slope post-ROSC (95% CI) [mmHg]	% Increase	p-value
ACLS3 02	46 (41, 51)	95 (90, 100)	51	<0.001
ACLS3 03	35 (28, 42)	53 (50, 56)	34	<0.001
ACLS6 01	71 (64, 78)	92 (87, 97)	23	<0.001

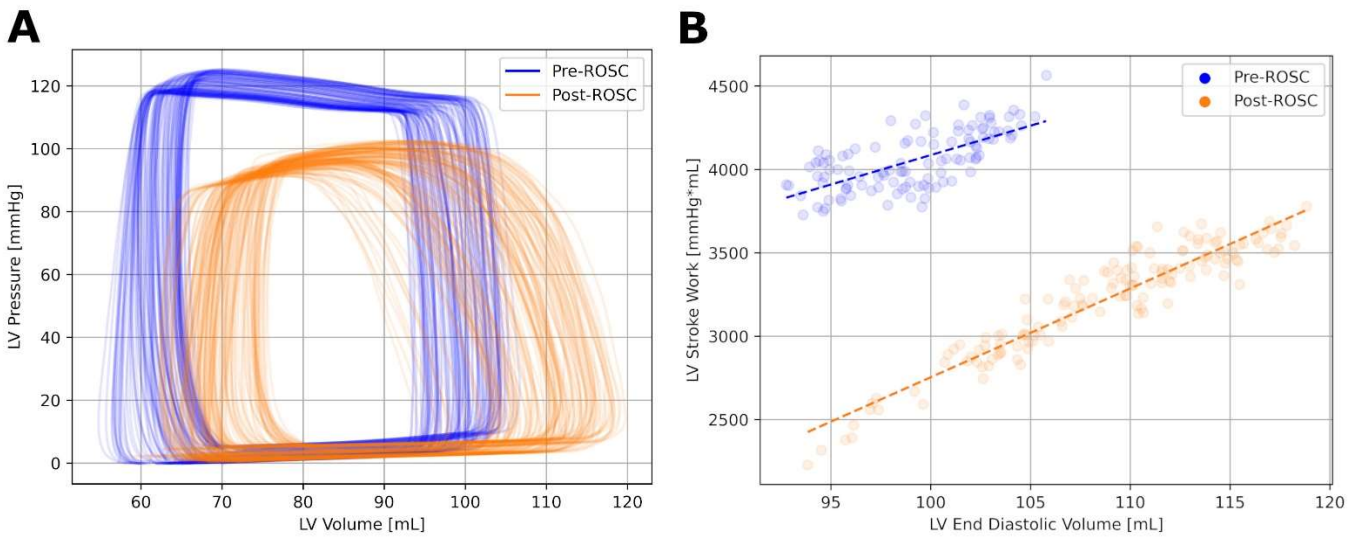

Figure 3. Represents PVL data from ACLS3 03 animal, where (A) depicts PVL pre and post ROSC, and (B) represents the PRSW slope before and after ROSC.

DISCUSSION

Despite the clinical relevance of cardiac arrest and extensive studies, effective therapeutic strategies remain underdeveloped, with high mortality rates and limited data on optimal post-ROSC care. Due to the complex nature of the condition and the absence of advanced animal models, the study of physiological changes related to post-CA has been difficult [15]. Our study introduces a novel swine VF cardiac arrest model, combined with PVL analysis, to address this gap. This model closely replicates human physiology and offers an unprecedented opportunity to study hemodynamic changes after resuscitation.

In our study, we observed striking differences between the 3-minute and 6-minute cardiac arrest groups. All animals in the 3-minute group achieved ROSC with fewer cycles, while only one animal regained circulation in the 6-minute group. This highlights the critical impact of early intervention, as longer arrest durations require more cycles and are linked to poorer outcomes, with multiple studies confirming this finding [16-18]. However, a limitation of our experiment is the small sample size, which may introduce bias in comparing the 3-minute and 6-minute cohorts, particularly as only one animal in the 6-minute group survived.

Following resuscitation, notable trends were observed in metabolic markers. pH levels decreased, reflecting acidosis associated with prolonged cardiac arrest. Lactate levels increased significantly, indicating tissue hypoxia and anaerobic metabolism during the arrest. Potassium levels also rose, likely due to cell membrane depolarization during ischemia and reperfusion. These shifts underscore the physiological strain on the body during and after resuscitation [19-21]. The shift in these parameters is meaningful, as higher potassium and lactate levels are associated with decreased survival and poorer neurological outcomes [22].

Initially, our group planned to investigate the End Systolic Pressure Volume Relationship (ESPVR) during preload-reduction via inferior vena cava (IVC) occlusion. However, there were intermittent data quality issues observed throughout the experiment. Considering that the IVC occlusion is necessarily quite brief (~30 seconds), the intrusion of data quality degradation into this period prevents faithful evaluation of the ESPVR. As an alternative, we evaluated the PRSW, which is defined as the linear relationship between stroke work expended during the cycle (area of the PV loop), and the preload (indexed by End Diastolic Volume) [23]. It has been established that an increase in inotropy/contractility results in an increase in the slope of the PRSW relationship [24]. PRSW was well resolved before and after ROSC in the left ventricle (LV) for all surviving animals. It was also determined that PRSW could be evaluated during baseline. In the presented data, PRSW slope increased by a statistically significant amount for all animals, however, we cannot make statistically powerful statements whether the resuscitation method affects the magnitude of the change given the small number of animals in each group. We do make the passing remark that in the data we do have, the 3-minute ACLS group appears to experience the largest change, which can likely be explained by exogenous epinephrine administration, as well as circulating endogenous catecholamines.

In this study, we primarily focused on LV PVL due to technical challenges encountered with RV measurements. The readings in the RV were frequently unstable, ranging out of physiological regions, likely due to fluctuations in right-sided pressures and other physiological factors that were difficult to control for in the experimental setup (Fig. 4). These instabilities prevented us from identifying suitable periods for evaluating PRSW in the RV. As a result, RV analysis was omitted from this study. Given the above, it is possible that recording RV PVL may be inherently difficult to reliably obtain in this model.

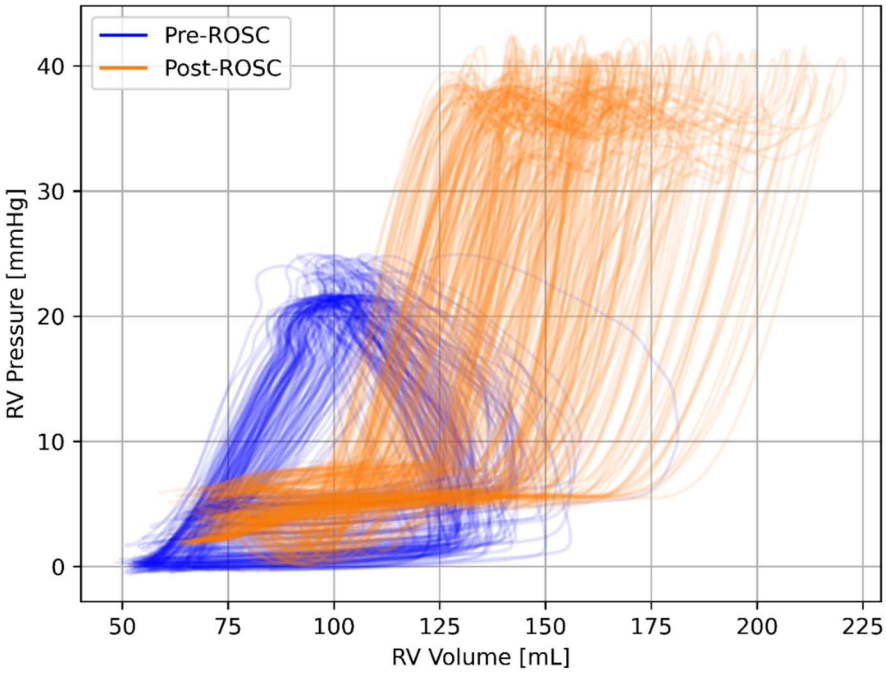

Figure 4. Sample RV PVL data obtained in the experiment.

LIMITATIONS

This study has several limitations. The small sample size limited statistical power and precluded robust subgroup analysis. Technical challenges resulted in incomplete PVL data, particularly for the right ventricle. The short monitoring period post-ROSC may not fully capture longer-term hemodynamic changes. The experimental swine model, while physiologically relevant, may not fully replicate human cardiac arrest pathophysiology. Finally, survival bias, given that only animals achieving ROSC were included in post-ROSC analysis, may have influenced results.

CONCLUSIONS

We developed and applied a swine model of VF cardiac arrest combined with PVL analysis to characterize hemodynamic changes in the immediate post-ROSC period. PRSW measurement was feasible in the left ventricle and revealed significant increases in contractility after ROSC. These findings demonstrate the potential utility of PVL analysis in post-arrest research and highlight possible targets for therapeutic intervention. Larger studies are warranted to confirm these results and expand the analysis to both ventricles.

SUPPLEMENTARY INFORMATION

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Institutional Review Statement: *The study was conducted according to the guidelines of the Declaration of Helsinki.*

Informed Consent Statement: *Informed consent was obtained from all subjects involved in the study.*

Data Availability Statement: *The datasets generated and analyzed during the current study are available from the corresponding author on reasonable request.*

Conflicts of Interest: *The authors declare no conflicts of interest.*

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