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ORIGINAL ARTICLE

Haemodynamic implications of VA-ECMO vs. VA-ECMO plus Impella CP for cardiogenic shock in a large animal model

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Abstract

Aims Veno-arterial extracorporeal membrane oxygenation (VA-ECMO) with profound left ventricular (LV) failure is associated with inadequate LV emptying. To unload the LV, VA-ECMO can be combined with Impella CP (ECMELLA). We hypothesized that ECMELLA improves cardiac energetics compared with VA-ECMO in a porcine model of cardiogenic shock (CS).

Methods and results Land-race pigs (weight 70 kg) were instrumented, including a LV conductance catheter and a carotid artery Doppler flow probe. CS was induced with embolization in the left main coronary artery. CS was defined as reduction of ≥50% in cardiac output or mixed oxygen saturation (SvO_2) or a SvO_2 < 30%. At CS VA-ECMO was initiated and embolization was continued until arterial pulse pressure was <10 mmHg. At this point, Impella CP was placed in the ECMELLA arm. Support was maintained for 4 h. CS was induced in 15 pigs (VA-ECMO *n* = 7, ECMELLA *n* = 8). At time of CS MAP was <45 mmHg in both groups, with no difference at 4 h (VA-ECMO 64 mmHg ± 11 vs. ECMELLA 55 mmHg ± 21, *P* = 0.08). Carotid blood flow and arterial lactate increased from CS and was similar in VA-ECMO and ECMELLA [239 mL/min ± 97 vs. 213 mL/min ± 133 (*P* = 0.6) and 5.2 ± 3.3 vs. 4.2 ± 2.9 mmol/ (*P* = 0.5)]. Pressure-volume area (PVA) was significantly higher with VA-ECMO compared with ECMELLA (9567 ± 1733 vs. 6921 ± 5036 mmHg × mL/min × 10^{-3}, *P* = 0.014). Total diureses was found to be lower in VA-ECMO compared with ECMELLA [248 mL (179–930) vs. 506 mL (418–2190); *P* = 0.005].

Conclusions In a porcine model of CS, we found lower PVA, with the ECMELLA configuration compared with VA-ECMO, indicating better cardiac energetics without compromising systemic perfusion.

Keywords Cardiogenic shock; VA-ECMO; Impella; ECMELLA; Pressure-volume loops

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Introduction

In cardiogenic shock (CS) due to acute myocardial infarction (AMI), mechanical circulatory support (MCS) is a treatment option in the most severe or refractory cases. MCS can provide temporary circulatory support and serve as a bridge to recovery, long-term MCS, heart transplantation, or a decision to withdraw MCS and aim for palliation. There are various types of MCS and the choice of intervention is based on the presenting pathophysiology,[1] but with biventricular failure, refractory cardiac arrest, or concomitant lung injury, veno-arterial extracorporeal membrane oxygenation (VA-ECMO) is often preferred. VA-ECMO offers restoration of cardiac output (CO) and oxygenation but has also the capability to deliver higher flowrates than transvalvular microaxial flow pumps, such as the Impella system. With peripheral cannulation, VA-ECMO will reverse oxygenated blood flow in the aorta ensuring that good end-organ oxygenation can be achieved

but at the expense of increased resistance to the left ventricle (LV).[2] In case of severely depressed LV myocardial function, the LV may not overcome this increase with consequently reduced or absent aortic valve opening and LV congestion, clinically seen as no or low pulse pressure and/or LV dilatation on echocardiography. Inadequate LV emptying will lead to increased LV end-diastolic pressure (LVEDP), which may result in severe pulmonary congestion.[3] One of several proposed options to avoid and treat this is to unload the LV with an axial flow pump draining the LV and in theory reduce LV pressures. The combination of VA-ECMO and Impella® (ECMELLA), has recently been associated with better survival compared with VA-ECMO alone in retrospective observational studies, but at the cost of more complications.[4–6] The aim of the current study was to evaluate whether ECMELLA improves cardiac energetics without compromising end-organ perfusion compared with VA-ECMO alone in a porcine model of CS with severely reduced LV function.

Methods

Fifteen Danish female landrace pigs with a weight of approximately 75 kg were included in this study. The study was conducted in accordance with current guidelines from the Danish Animal Experiments Expectorate which also approved the study (Study ID: 2006-15-00951). The model has previously been described in detail,[7] but in summary, all animals were anaesthetized and mechanically ventilated. All animals received a bolus of 300-mg amiodarone intravenously followed by continuous infusion to avoid malignant arrhythmias. Anticoagulation on VA-ECMO was achieved by 20.000 IE unfractionated heparin administered every 2 h.

Instrumentation was done percutaneously except for access to the left carotid artery, where surgical cut down was used to place a 4-mm Doppler flow probe for continuous measurement of carotid blood flow (MEDISTIM SonoQ TTFM Probe, Emtec GmbH, Finning Germany). In the right internal jugular vein, a pulmonary artery catheter was inserted through a multi-lumen-sheath (Arrowg+ard Blue® MAC, Teleflex, NC, USA) for continuous measurements of CO, mixed venous saturation, and pulmonary artery pressures. In the right carotid artery, an 8-Fr sheath was placed for introduction of a conductance catheter in the LV (Ventri-cath 510 PV Loop Catheter, Millar Inc. TX, USA) and monitoring of peripheral arterial blood pressure. In the groin, a 17-Fr arterial cannula and 21-Fr venous cannula were placed on the left and right side, respectively, for VA-ECMO support. In VA-ECMO alone animals, the right femoral artery was cannulated with a 7-Fr sheath placed for coronary angiogram and in ECMELLA animals a 14-Fr Impella sheath was placed for angiography and Impella placement. In the left femoral vein, an 8-Fr sheath was placed for introduction of a Swan-Ganz catheter to the renal vein for blood sampling and pressure monitoring.

The conductance catheter was connected to a MPVS Ultra Pressure-Volume loop system (Millar Inc., Houston, TX, USA). This was further connected to a Powerlab 16/35 (ADInstruments, Dunedin, New Zealand) for the recording of data in Labchart Pro (ADInstruments, Dunedin, New Zealand).

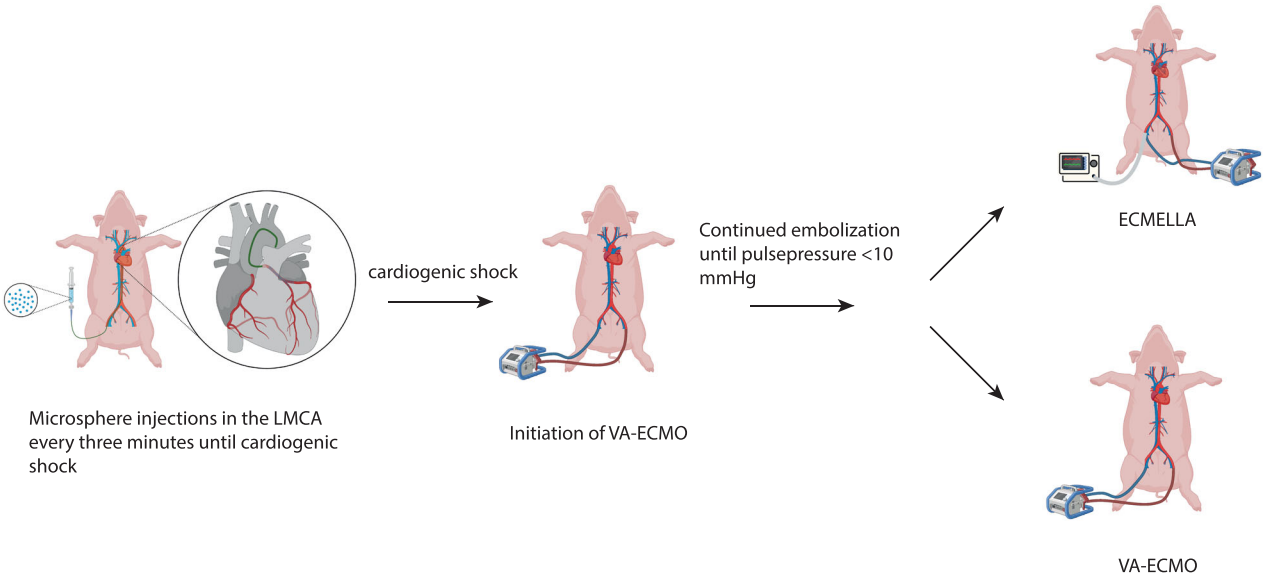
At baseline, an inferior caval vein occlusion using a 20-mm balloon catheter was performed to determine V_0, a theoretical volume where no pressure is generated in the LV. V_0 was kept constant throughout the study for single-beat estimation of end-systolic elastance (EES).[8] For volume calibration, we infused hypertonic saline to determine parallel wall conductance and alfa calibration was performed with the measured CO from the PA catheter. From the conductance catheter, we measured the following: stroke volume (mL), CO (L/min), stroke work (mmHg × mL), end-systolic and end-diastolic pressure, and volume and arterial elastance (EA) (mmHg/mL). The ventriculo-arterial coupling was calculated as the ratio of EA to EES.

Potential energy (PE) was calculated as LV end-systolic pressure (LVESP) × minimal LV volume × V_0 × 0.5.[9] All values for V_0 were converted to absolute values. Pressure volume area (PVA) was calculated as PE + stroke work and further multiplied by heart rate (HR) to obtain total cardiac work.

Intervention

The left main coronary artery (LMCA) was catheterized using a JL 3.5 catheter (Launcher, Medtronic Inc., Minneapolis, MN, USA) and this was used for injecting polyvinyl alcohol microspheres to create local embolism (*Figure 1*). Each injection consisted of 1 mL of a mixture of isotonic saline (10 mL), contrast (10 mL), and polyvinyl alcohol flakes (125 μg; Contour™; Boston Scientific, MA, USA). Embolization was repeated every 3 min until CS was established. For this study, CS was defined as a ≥50% reduction in CO and/or mixed venous saturation compared with baseline or an absolute mixed venous saturation of ≤30%. If mean arterial blood pressure (MAP) drifted below 45 mmHg, an infusion of norepinephrine was initiated. When CS was achieved, VA-ECMO was started at a flow of approximately 50 mL/kg and embolization was continued until systemic arterial pulse pressure was ≤10 mmHg. However, if there was no change in arterial pulse pressure in three consecutive embolizations, further injections were deemed futile and embolization was stopped. In ECMELLA-treated animals, an Impella CP (Abiomed, Danvers, MA, USA) was placed and started, when the pulse pressure target was reached. The Impella was set to the highest possible performance level without causing suctions events. The interventions were running for 4 h until euthanization of animals was performed. Blinding of allocation groups was not possible and studies were done in blocks of two animals shifting between VA-ECMO and ECMELLA treatment.

Figure 1 Study workflow. Schematic overview of workflow and interventions. Partly created using BioRender (www.biorender.com). LMCA, left main coronary artery; VA-ECMO, veno-arterial extracorporeal membrane oxygenation.



Blood samples were drawn from the PA catheter, renal vein, and arterial line in the right carotid artery for analysis of blood gases and lactate at baseline, CS, and every hour after the start of VA-ECMO.

Statistics

Data are presented as mean with standard deviation or median and 25th and 75th percentiles as appropriate. Analysis of differences in treatment effect with the two interventions was done with a mixed effects model. The model used random intercept and an independent covariance matrix. The measurements at CS were included in the analysis and thereby the model adjusted for any differences between interventions at CS. Baseline values were excluded from the mixed model. Further, time was included in the model as a categorical variable. The *P* value presented is the interaction between the interventions at 4 h. A *P* value of <0.05 was considered significant. All analysis was performed with STATA 17 (StataCorp, College Station, TX, USA).

Results

CS was induced in 15 animals, seven treated with VA-ECMO and eight treated with ECMELLA. Conductance data were lost in one animal due to a hardware defect, leaving seven animals for pressure-volume analysis in each treatment arm. Animals had an average weight of 72.3 ± 4.1 kg. CS was induced in all animals, leading to a mean reduction in CO of 60 ± 16% in both groups and a reduction in mixed venous saturation to an absolute mean of 48 ± 9% in the VA-ECMO arm and 52 ± 3% in the ECMELLA group (*Table 1*, data from all timepoints provided in *Table* S1). When CS was established stroke work was reduced by 80% in both groups (*Table 1*). Three animals required a norepinephrine infusion during shock induction; one in the VA-ECMO group and two in the ECMELLA group.

After the final embolization on VA-ECMO, pulse pressure was reduced to 11 ± 4.2 mmHg and four of seven animals reached the target of ≤10 mmHg in the VA-ECMO arm. In the ECMELLA arm pulse pressure was 9 ± 2.4 mmHg and seven of eight animals reached the pulse pressure target. VA-ECMO flow was significantly higher in the VA-ECMO arm compared with ECMELLA (3.7 ± 0.4 L/min vs. 3.0 ± 1.1 L/min, *P* = 0.001; *Table 1*). Estimated Impella flow increased from 1.2 ± 1.0 L/min at 1 h to 1.7 ± 0.8 L/min at 4 h; thus, the total MCS flow was lower with VA-ECMO than ECMELLA (3.7 ± 0.4 L/min vs. 4.7 ± 1.1 L/min; *P* = 0.016). Norepinephrine was required in three animals in the VA-ECMO arm (average infusion in treated animals was 0.12 ± 0.15 microgram/kg/min and one animal on the ECMELLA arm (requiring 0.4 μg/kg/min) at 4 h (*P* = 0.2). We did not observe harlequin phenomenon in any animals.

Cardiac energetics

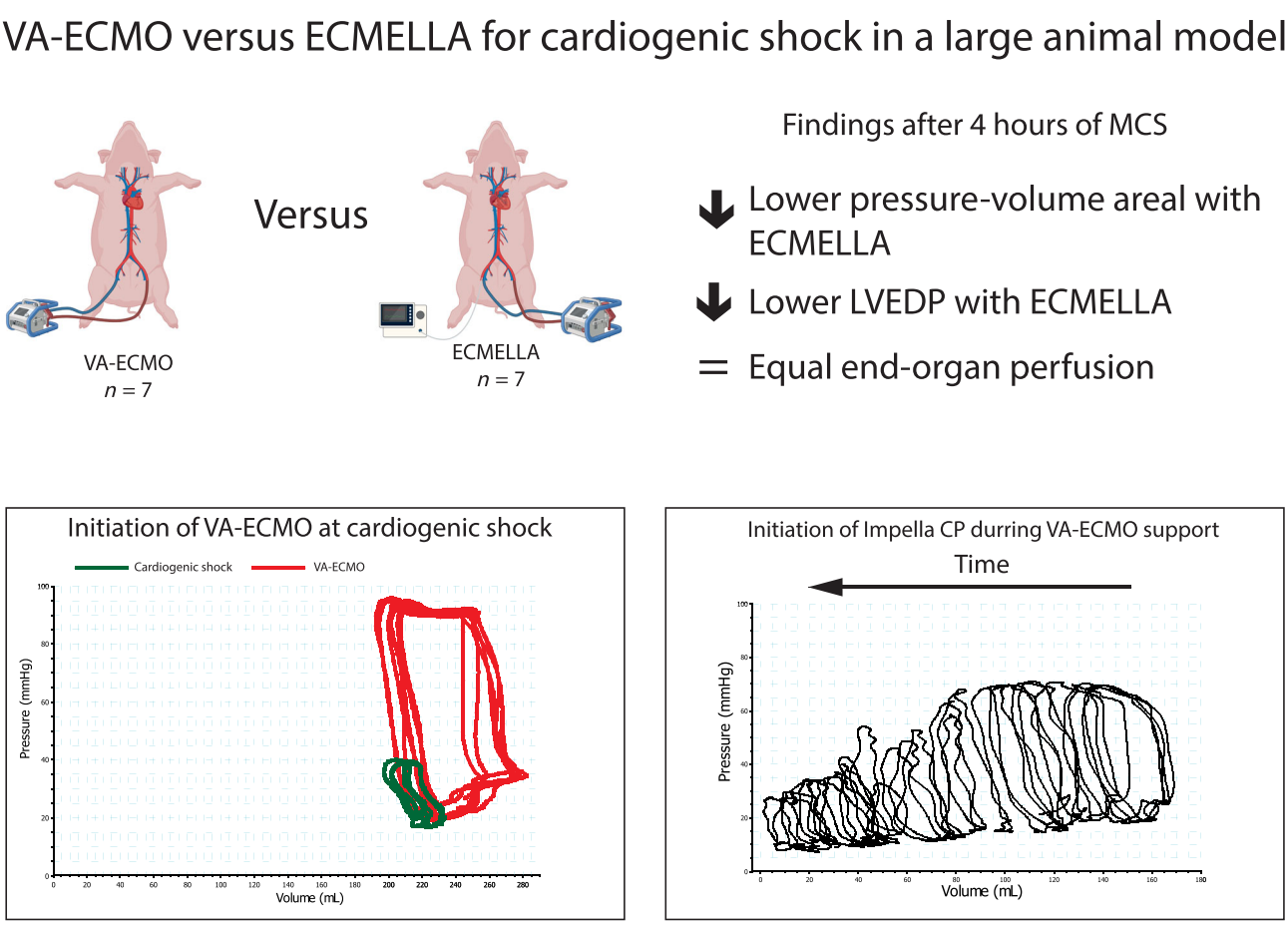
With the initiation of VA-ECMO, a marked increase in LV pressure was observed in all animals. However, with implantation

Table 1 Haemodynamics from baseline to end of study

	Baseline				Cardiogenic shock				MCS 4 h				
	VA-ECMO		ECMELLA		VA-ECMO		ECMELLA		VA-ECMO		ECMELLA		*P* value
Heart rate (beats per min)	82	(8)	72	(10)	77	(4)	68	(7)	64	(11)	55	(21)	0.087
MAP (mmHg)	70	(11)	74	(15)	42	(8)	39	(9)	65	(10)	69	(21)	0.599
Pulse pressure (mmHg)	35.7	(15.3)	41.4	(11.8)	27.0	(4.9)	24.6	(6.2)	11.1	(3.3)	5.6	(1.1)	0.064
PAP sys (mmHg)	31.6	(3.8)	35.4	(4.2)	33.8	(7.0)	33.6	(5.6)	33.4	(11.1)	34.2	(6.2)	0.752
PAPdia (mmHg)	16.6	(6.9)	22.8	(4.0)	22.0	(6.0)	25.4	(5.0)	25.1	(10.4)	25.8	(5.1)	0.543
CVP (mmHg)	11.6	(5.0)	11.5	(2.4)	14.0	(3.7)	14.5	(1.1)	11.4	(4.2)	9.9	(3.2)	0.215
Renal venous pressure (mmHg)	11.7	(4.2)	7.2	(2.2)	17.8	(13.1)	9.5	(5.0)	10.9	(3.6)	6.6	(1.4)	0.295
Carotid flow (mL/min)	276	(68)	253	(99)	121	(28)	68	(49)	239	(97)	213	(133)	0.446
Mixed Venous Saturation (%)	61.9	(10.5)	58.8	(16.5)	37.3	(13.7)	27.2	(6.8)	65.7	(21.1)	75.3	(13.0)	0.380
Stroke volume (mL)	74	(11)	74	(11)	39	(23)	30	(12)	14	(6)	21	(9)	0.274
CO (mL/min)	6.1	(0.8)	5.2	(0.6)	2.5	(1.1)	2.0	(0.7)	0.9	(0.4)	1.1	(0.3)	0.497
Pressure volume area (mmHg × mL)	15 553	(4292)	13 161	(4023)	8923	(1975)	8436	(2973)	9567	(1733)	6921	(5036)	0.014
Stroke Work (mmHg × mL)	5761	(1540)	5049	(911)	1138	(593)	989	(557)	774	(364)	954	(570)	0.518
Potential energy (mmHg × mL)	9792	(3595)	8112	(3360)	7785	(2275)	7447	(2690)	8794	(1714)	5967	(4764)	0.005
Total cardiac work (PVA × HR × 10^{-3})	1274	(371)	941	(300)	686	(167)	565	(192)	618	(161)	380	(323)	0.008
LVEDP (mmHg)	20.9	(2.2)	18.4	(1.7)	23.0	(3.7)	22.4	(1.2)	22.5	(5.7)	12.0	(3.6)	0.000
LVESP (mmHg)	91.0	(15.0)	83.3	(13.0)	58.4	(4.4)	53.3	(9.9)	73.0	(12.0)	53.7	(20.1)	0.023
EA (mmHg/mL)	0.8	(0.1)	0.8	(0.3)	0.9	(0.3)	0.8	(0.5)	1.9	(1.3)	1.6	(0.8)	0.633
EES (mmHg/mL)	0.47	(0.18)	0.47	(0.15)	0.25	(0.12)	0.20	(0.07)	0.33	(0.15)	0.31	(0.15)	0.847
Urin output (ml/h)									110	(154)	192	(213)	0.714
ECMO flow (L/min)					3.3	(0.2)	3.4	(0.2)	3.7	(0.4)	3.0	(1.1)	0.001
Impella flow (L/min)											1.7	(0.8)	
Total MCS flow									3.7	(0.4)	4.6	(1.1)	0.016

Table showing main haemodynamic and organ perfusion measures at baseline, cardiogenic shock and at the end of the study after 4 h. *P* values from mixed effects model.
CO, cardiac output; CVP, central venous pressure; Ea, arterial elastance; EES, end-systolic elastance; LVEDP, left ventricular end-diastolic pressure; MAP, Mean arterial pressure; MCS, mechanical circulatory support; NE, norepinephrine; PAP, pulmonary artery pressure; Pes, left ventricular end-systolic pressure; VA-ECMO, veno-arterial extracorporeal membrane oxygenation.

Figure 2 Pressure and volume changes to VA-ECMO and ECMELLA initiation. Examples of pressure and volume changes when initiating VA-ECMO at cardiogenic shock (left) and initiation of Impella during VA-ECMO support in ECMELLA-treated animals (right). Note: The downward shift in volume when initiating VA-ECMO at CS is due to interference with the thermodilution cardiac output measures. Partly created using BioRender (www.biorender.com). LV, left ventricle; VA-ECMO, veno-arterial extracorporeal membrane oxygenation.



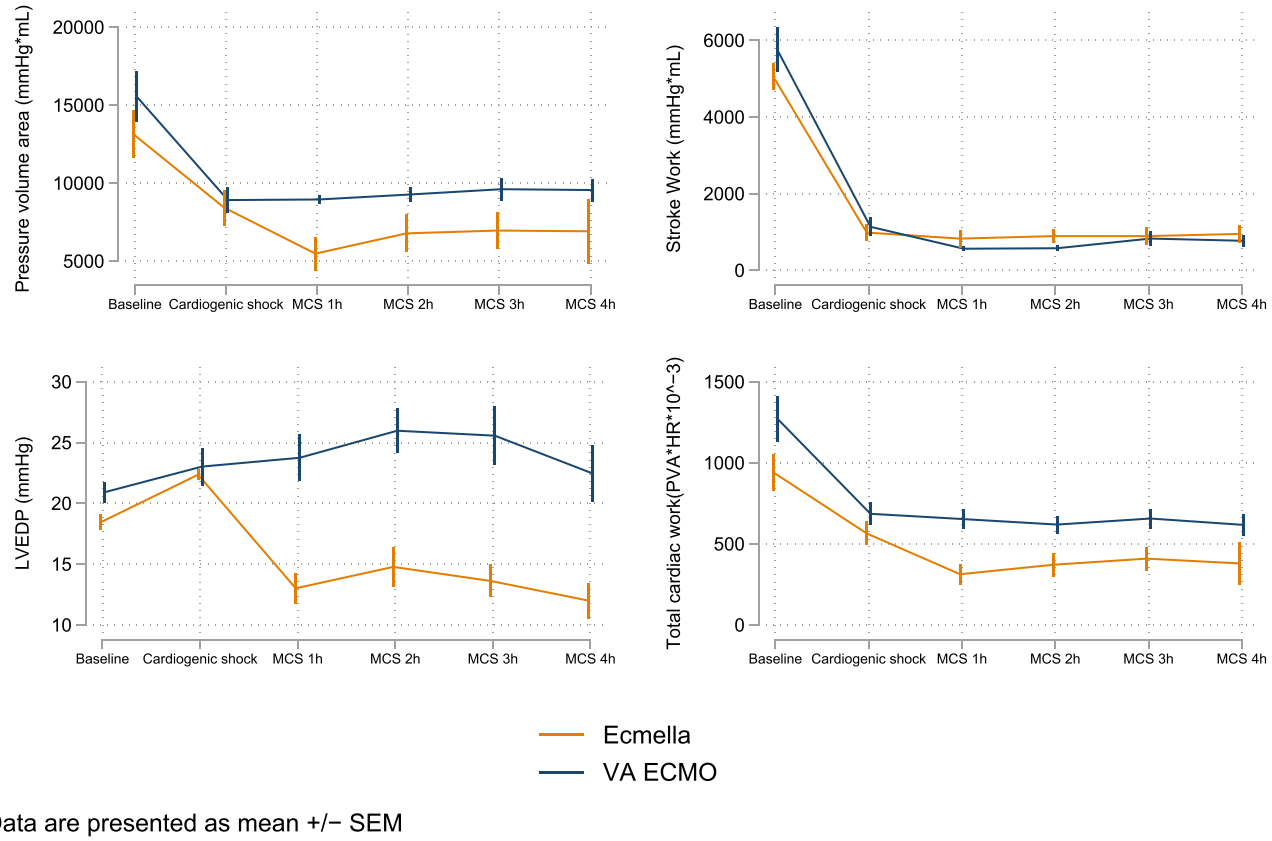
of the Impella device, ventricular volume and pressure declined towards baseline in the ECMELLA group (*Figure 2*). MAP increased in both groups after establishment of mechanical support without significant differences between groups after 4 h (*Table 1*). PVA was significantly higher with VA-ECMO alone (9567 ± 1733 mmHg × mL vs. 6921 ± 5036 mmHg × mL; $P = 0.014$), whereas stroke work was similarly low in the two groups ($P = 0.5$; *Table 1*). Because HR was comparable in the two groups, the total cardiac work was also significantly higher in VA-EMCO treated animals (618 ± 161 PVA × HR × 10^{-3} vs. 380 ± 323 PVA × HR × 10^{-3}; $P = 0.008$) (*Table 1* and *Figure 3*). LVEDP increased during CS induction in both groups and remained elevated in VA-ECMO-treated animals while ECMELLA-treated animals developed LVEDP lower than baseline. LVEDP was significantly lower in ECMELLA animals at 4 h, (22.5 ± 5.7 mmHg vs. 12.0 ± 3.6 mmHg; $P < 0.001$) (*Table 1*).

End-organ perfusion

MCS lead to an increase in mixed venous saturation in both intervention groups without any significant difference at 4 h (65 ± 21% vs. 75 ± 13%; $P = 0.38$) (*Table 2*, data from all timepoints provided in *Table S2*). Arterial lactate continued to increase with both interventions after CS without any significant differences (*Table 2* and *Figure 4*).

Venous renal pressure increased during shock induction (*Table 1*), which was accompanied by a decrease in venous renal saturation (*Table 2*). Both interventions lead to a reduction in renal venous pressure and improved venous renal saturation without any difference between interventions at 4 h. Hourly urine output was numerically lower with VA-ECMO, but the difference was not statistically significant (*Table 2*). Consequently total urine output was significantly lower with VA-ECMO [median 248 mL (179–930) vs. 506 mL

Figure 3 Cardiac energetics. Plot of pressure volume area, stroke work, LVEDP, and total cardiac work over time. Data are presented as mean with standard error. LVEDP, left ventricular end-diastolic pressure; MCS, mechanical circulatory support; VA-ECMO, veno-arterial extra corporal membrane oxygenation.



(418–2190); $P = 0.005$] from initiation of VA-ECMO to end of the observation period.

Blood flow in the left carotid artery decreased with shock induction and MCS led to increased blood flow in both interventions. Numerically higher flow rates were seen with VA-ECMO compared with ECMELLA, although the difference was not statistically significant ($P = 0.6$) (*Table 1* and *Figure 4*).

Discussion

In this translational study of severe infarct related CS, induced by embolization of the LMCA, we examined VA-ECMO or immediate ECMELLA as MCS strategies, in the presence of reduced pulse pressure on VA-ECMO. With VA-ECMO alone, we found a higher PVA and LVEDP suggestive of lower myocardial oxygen consumption with ECMELLA driven by reduced PE. There was no difference in end-organ perfusion, although cumulated diuresis in the VA-ECMO arm was lower than in the ECMELLA-treated animals.

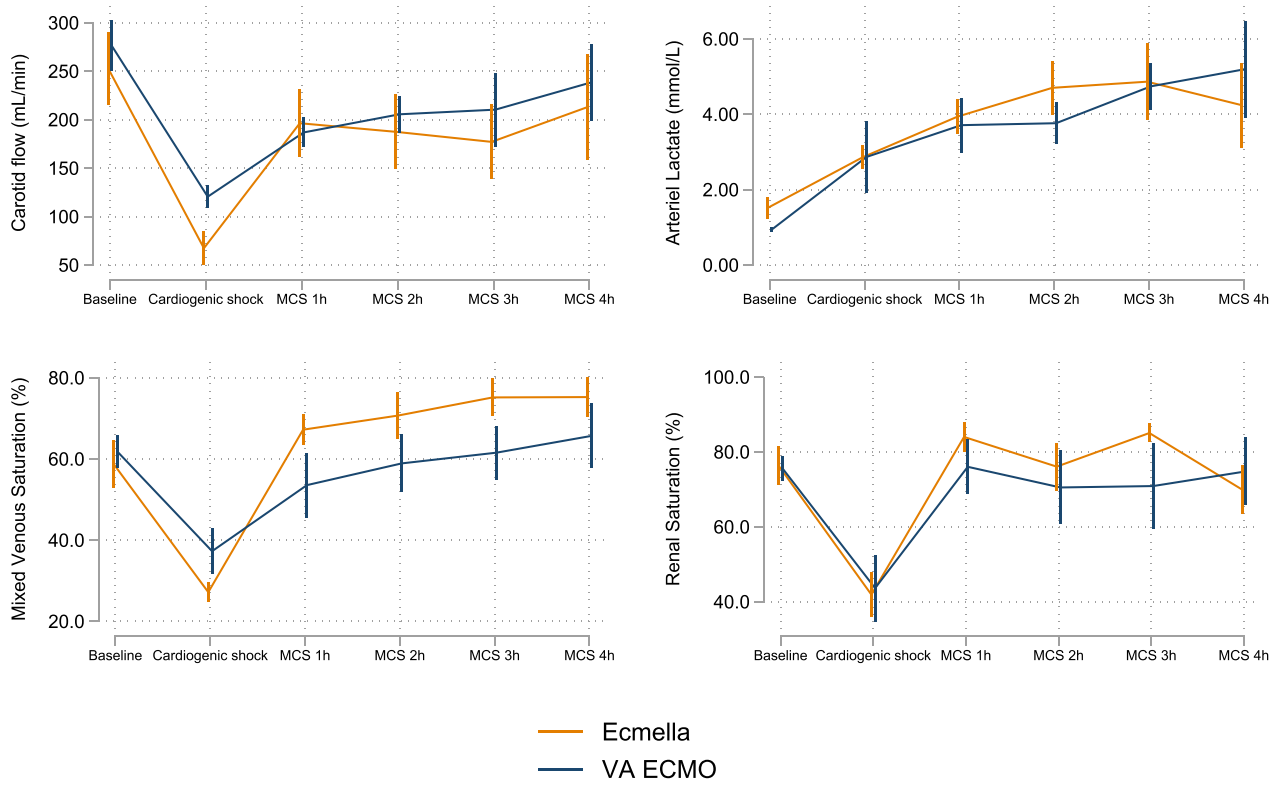
Our findings confirm that the LV is exposed to significant strain during VA-ECMO treatment. This has been shown in previous experimental studies[2] and can lead to the clinical condition with LV distention and development of pulmonary oedema during VA-ECMO support.[3,10] The underlying pathophysiology is a combination of severe LV dysfunction, contin-

Table 2 Blood gas analysis

	Baseline				Cardiogenic shock				MCS 4 h				
	VA-ECMO		ECMELLA		VA-ECMO		ECMELLA		VA-ECMO		ECMELLA		*P* value
Arterial pH	7.45	(0.1)	7.45	(0.1)	7.44	(0.1)	7.55	(0.1)	7.38	(0.1)	7.45	(0.1)	0.504
Arterial lactate (mmol/L)	0.9	(0.2)	1.5	(0.80)	2.9	(2.1)	2.9	(0.9)	5.2	(3.4)	4.2	(3.0)	0.623
Renal saturation (%)	75.7	(7.9)	76.3	(14.7)	43.7	(19.9)	41.9	(16.8)	74.8	(23.8)	70.0	(17.3)	0.504
Mixed venous saturation (%)	61.9	(10.5)	58.8	(16.5)	37.3	(13.7)	27.2	(6.9)	65.7	(21.1)	75.3	(13.0)	0.380

Results of blogas analysis from baseline to end of study.
ECMO, extracorporeal membrane oxygenation; VA, veno-arterial.

Figure 4 End-organ perfusion. Plot of mean carotid blood flow, mixed venous saturation, arterial lactate and renal venous saturation from baseline to end-of study after 4 h. MCS, mechanical circulatory support; VA-ECMO, veno-arterial extracorporeal membrane oxygenation.



Data presented as mean +/− SEM

ued flow to the LV and the resistance generated by VA-ECMO. The initiation of Impella CP in the ECMELLA group, immediately lowered LV pressure and volume resulting in unloading of the LV and consequently, lowered PVA while stroke work remained similar between the groups. There are two considerations when interpreting the similar stroke work within the two groups. First, the stroke work after 4 h of intervention is very low in both groups with very little intrinsic function in the LV (13% and 10% of baseline values in VA-ECMO and ECMELLA, respectively), which is likely the result of the continued embolization to mimic a Society for Cardiovascular Angiography and Interventions class E shock[11] situation. Second, stroke work is the product of stroke volume and intraventricular pulse pressure and as such, there is no information on the placement of the PV-loop in this single parameter.[12,13] As PVA is the sum of PE and stroke work, the lower PVA was mainly driven by a reduction in PE, thus a leftward and downward shift of the pressure-volume loop. In agreement with this, LVEDP and LVESP were significantly reduced in the ECMELLA-treated animals. PVA has in several previous studies shown strong correlation with myocardial oxygen consumption[14,15] and may be considered a surrogate marker of cardiac energetics. The lower PVA in ECMELLA-treated animals thus indicates better unloading of the LV that remained significant for total cardiac work (PVA × heart rate).

The main indication for MCS in AMICS is to restore systemic flow and secure adequate tissue perfusion. In our model, we found only a modest increase in lactate when CS was present, and with both VA-ECMO and ECMELLA lactate increased throughout the study period; without any difference between groups. In a clinical context, the lack of improvement in lactate levels is disturbing as high lactate levels are associated with 30-day mortality in patients with CS due to AMI.[16] However, lactate conversion may be different in pigs compared with humans, and as opposed to clinical practice, we did not provide distal perfusion in the femoral arteries leading to moderate limb ischaemia. These two factors could explain our finding of increasing lactate levels.

Interestingly, we found higher cumulated diuresis with ECMELLA compared with VA-ECMO pointing towards better kidney function. Also, we registered numerically lower renal pressures and numerically higher MAP in the ECMELLA group compared with VA-ECMO treated animals, although without any statistical significance for either. Further, the total flow rate was higher with ECMELLA compared with VA-ECMO. In

sum, this points towards better renal perfusion and could explain our finding of higher diuresis.

Although VA-ECMO and ECMELLA support equally increased carotid blood flow, VA-ECMO mainly provided carotid blood flow from the retrograde perfusion because forward flow from the heart was minimal. With ECMELLA it is likely that carotid flow at least in some animals would be forward flow from the Impella CP, but the exact localization of the watershed area could not be assessed in this model. We did not see any signs of Harlequin phenomena due to ejection of poorly oxygenated blood from the heart to upper body; however, Harlequin phenomena have previously been reported during ECMELLA support.[17] In case of acute lung injury with compromised pulmonary gas exchange, despite reducing LVEDP the ECMELLA configuration will eject less well-oxygenated blood into the aorta that will mix with 100% oxygenated blood from the VA-ECMO. To overcome this a veno-arterial–venous configuration or a different venting strategy may be required.[18]

This study provides mechanistic observations demonstrating improved cardiac energetics with ECMELLA compared with VA-ECMO alone in CS. We further found comparable effects on end-organ perfusion between the two groups and a signal towards better kidney function with higher cumulated urine output with ECMELLA. This supports the observational propensity matched data indicating better 30-day mortality with ECMELLA compared with ECMO alone.[19] Ultimately, this needs to be tested in a controlled randomized trial like the UNLOAD ECMO (www.clinicaltrials.gov NCT05577195) trial now enrolling patients, which will provide important insights into the effect of ECMELLA on clinical endpoints.

Limitations

Some limitations should be noted within the study. Animals are young and healthy at the beginning of the study without any comorbidities which is seldom the case in the clinical setting. Further, the systemic response to VA-ECMO and Impella might differ between species and the medication used might also differ in effect. As noted earlier, we only have 4 h of support and our findings should not be extrapolated to time points beyond this.

For estimating V_0, we performed preload reduction with caval occlusion at baseline and kept the acquired V_0 throughout the study as it was not feasible to perform preload occlusion with ongoing VA-ECMO support. From other animal experimental studies, it has been shown that V_0 increases after induction of myocardial ischaemia,[20] and the calculated EES and PVA may thereby be lower for EES and higher for PVA, than the true EES and PVA. However, this applies to both groups.

Conclusions

In infarct related CS with severely impaired LV function, ECMELLA showed better cardiac energetics with lower cardiac work compared with VA-ECMO alone. We found higher urine output in ECMELLA-treated animals, but otherwise, the end-organ perfusion was similar between the two intervention groups. The observed positive effects of ECMELLA should be balanced against the increased complexity and risk of complications.

Conflict of interest

Jacob E. Møller has received an unrestricted research grant from Abiomed Inc.

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Supporting information

Additional supporting information may be found online in the Supporting Information section at the end of the article.

Table S1. Hemodynamics from baseline to end of study.
Table S2. blood gas analysis.

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