Available online at www.sciencedirect.com

Resuscitation

journal homepage: www.elsevier.com/locate/resuscitation

Clinical paper

# Survival and neurological outcome with extracorporeal cardiopulmonary resuscitation for refractory cardiac arrest caused by massive pulmonary embolism: A two center observational study

*Loes Mandigers*[a,*], *Erik Scholten*[b], *Wim J.R. Rietdijk*[a], *Corstiaan A. den Uil*[a,c], *Robert J. van Thiel*[a], *Sander Rigter*[b], *Bram G.A.D.H. Heijnen*[b], *Diederik Gommers*[a], *Dinis dos Reis Miranda*[a]

[a] *Department of Adult Intensive Care, Erasmus MC University Medical Center, Rotterdam, The Netherlands*
[b] *Department of Intensive Care, Sint Antonius Hospital, Nieuwegein, The Netherlands*
[c] *Department of Cardiology, Erasmus MC University Medical Center, Rotterdam, The Netherlands*

## Abstract

**Background:** Cardiac arrest (CA) due to pulmonary embolism (PE) is associated with low survival rates and poor neurological outcomes. We examined whether Extracorporeal Cardiopulmonary Resuscitation (ECPR) improves the outcomes of patients who suffer from CA due to massive PE.

**Methods:** We retrospectively included 39 CA patients with proven or strongly suspected PE in two hospitals in the Netherlands, in a 'before/after'-design. 20 of these patients were treated with Conventional Cardiopulmonary Resuscitation (CCPR) and 19 patients with ECPR.

**Results:** The main outcomes of this study were ICU survival and favourable neurological outcome, defined as Cerebral Performance Category (CPC) score 1–2. The ICU survival rate in CCPR patients was 5% compared to 26% in ECPR patients ($p < 0.01$). Survival with favourable neurological outcome was present in 0/20 (0%) CCPR patients compared to 4/19 (21%) of the ECPR patients ($p < 0.05$).

**Conclusion:** ECPR seems a promising treatment for cardiac arrest patients due to (suspected) massive pulmonary embolism compared to conventional CPR, though outcomes remain poor.

## Introduction

Massive pulmonary embolism (PE) as an obstructive shock may lead to right ventricular overload and haemodynamic instability.[1–3] Eventually, this causes cardiac arrest (CA) in 32–48% of the patients.[4–6] Outcomes in CA patients due to massive PE are extremely poor, with survival rates around 0.4–18% despite urgent administration of thrombolytics.[3,7,8]

Veno-arterial extracorporeal membrane oxygenation (V-A ECMO) decreases right ventricular overload[9] and supports the entire circulation, bypassing both lungs and heart. Especially in patients with refractory CA caused by massive PE, V-A ECMO started during cardiopulmonary resuscitation (CPR) could improve the survival

* Corresponding author at: Department of Adult Intensive Care, Erasmus MC, Rotterdam, Doctor Molenwaterplein 40, 3015 GD, Rotterdam, The Netherlands.
E-mail address: l.mandigers@erasmumc.nl (L. Mandigers).

https://doi.org/10.1016/j.resuscitation.2018.12.008
Received 8 October 2018; Received in revised form 28 November 2018; Accepted 10 December 2018
Available online xxx

rates. Previous studies in massive PE patients without CA showed a higher survival rate in patients who received V-A ECMO compared to conventional treatment, 83% versus 58%, respectively.[9–12] However most of these previous studies included haemodynamic unstable patients without CA or patients after CA, who regained return of spontaneous circulation (ROSC) before V-A ECMO placement. Little is known of patients cannulated during CPR.

The aim of this study is to examine whether extracorporeal cardiopulmonary resuscitation (ECPR) improves outcome of patients who suffer from CA due to massive PE. To our knowledge no previous studies were performed in CA patients due to PE, comparing CCPR with ECPR therapy. In this retrospective cohort study we analysed the data of CA patients due to (suspected) PE from two extracorporeal membrane oxygenation (ECMO) expertise centers in the Netherlands.

## Methods

We performed a retrospective study in a 'before-after' fashion in two hospitals in the Netherlands. The Erasmus MC Rotterdam (EMC) is a tertiary referral academic hospital and the Sint Antonius Hospital (SAZ) Nieuwegein is a major teaching hospital and pulmonary hypertension expertise center. Both hospitals are referral centers for cardiothoracic surgery (including surgical pulmonary embolectomy), experienced in ECMO therapy and ECPR. These hospitals started with formal implementation of ECPR therapy in August 2014.

### Patients

We included adult patients treated with ECPR due to a (suspected) PE in the period of August 1st, 2014 till March 31st, 2017 (the 'after'-period). And, as a control group, we included patients treated with conventional CPR (CCPR) due to a (suspected) PE in the time period January 1st, 2012 till July 31st, 2014 (the 'before'-period).

CCPR was defined as conventional CPR, where ECPR was defined as ECMO cannulation during CPR. We defined a CA due to (suspected) PE if one of the next four criteria were present: PE proven with Computer Tomography (CT)-scan, PE proven through pathologic examination (PA), severe right ventricular dilatation during CA with a history compatible with (suspected) PE, or administration of thrombolysis during arrest due to suspicion of massive PE. We included both in-hospital cardiac arrest (IHCA) and out-of-hospital cardiac arrest (OHCA) patients. The criteria for initiation of ECPR were witnessed CA (last seen $<5$ min) in patients below 70 years, with good quality of basic life support (BLS)/advanced life support (ALS) (leading to an end-tidal $CO_2 > 10$ mmHg) during at least 15 min and a low flow time of $<60$ min.

### ECMO procedure

ECMO placement in all patients of both the EMC and the SAZ, is done by intensivists, interventional cardiologists or cardiothoracic surgeons, depending of which location the CA occurs. If the CA occurs in the operating room (OR) the cardiothoracic surgeon will place it, if it occurs in the ICCU or catherization laboratory (cath. lab.), the interventional cardiologist will insert the ECMO and in all other cases the intensivists will do the ECPR procedure. The location of the ECPR will be where the patient is (e.g. OR, cath. lab., ICU, hospital ward, emergency department). There is no routine left heart catherization or left ventricular venting done in patients who receive ECMO for PE in the EMC and all ECPR patients get a 6 Fr distal reperfusion catheter of the cannulated leg, primarily implanted by the cardiothoracic or vascular surgeon.

### Measured variables

Our primary outcome, ICU survival, was retrospectively collected by chart review. As a secondary outcome we analysed favourable neurological outcome. This favourable outcome was defined as a Cerebral Performance Category (CPC) score of 1–2. A CPC score of 3–5 was defined as unfavourable neurological outcome. For prospectively measuring the latter outcome, all surviving patients were contacted and asked for written informed consent and to fill in a CPC questionnaire.[13]

Besides the primary and secondary outcomes, complications (e.g. bleeding [defined as major bleeding in need of transfusion/intervention or major bleeding limiting the treatment options such as thrombolysis or continuing of CPR], CVVH use, cerebral haemorrhage, vascular/ischemic limb complications and infection [defined as any infection with positive culture in general, during admission for the CCPR group and during ECMO for the ECPR group]) were included as outcome measurements and collected by chart review. These complications were only registered in Emergency Department (ED) survivors.

### Statistical analysis

Continuous variables were reported using median and 25–75% quartiles and categorical variables were reported using numbers and percentages. We used the Mann–Whitney U test and a Fisher's exact test to test for statistical significant differences for continuous and categorical variables, respectively.[14]

For our primary outcome ICU survival, we plotted Kaplan–Meier curves and we compared these survival distributions using the Log-rank test. For our secondary outcome, we dichotomised the CPC value into favourable outcome and unfavourable outcome.

During chart screening of the CA patients, we found some patients treated with ECPR in the 'before'-period and CCPR patients in the 'after'-period. We decided to perform a sensitivity analysis on the complete sample of CA patients due to (suspected) massive PE treated with CCPR or ECPR. One of the ECPR patients treated in the 'before'-period was $>18$ years at time of the questionnaire, however $<18$ years at time of CA. Because this patient was treated at the adult ICU, with adult cannulas, and having an 'adult' frame, we decided to include this data in the sensitivity analysis. A p-value $<0.05$ was defined as statistically significant. IBM SPSS Statistics version 24 was used to perform the statistical analyses.

### Ethics

The Medical Ethics Committee of the ErasmusMC Rotterdam, the Netherlands, reviewed and approved the study protocol (number MEC-2017-305). All surviving patients were asked consent for participation. This consent consisted of answering the questionnaire and approving the use of their documented clinical data. For non-surviving patients, proxy consent for their chart review was not required.

## Results

Charts of 2618 patients were reviewed and 39 patients were included in the 'before/after'-analyses (Fig. 1). Of these 39 included patients, 20 were treated with CCPR and 19 with ECPR.

### *Baseline characteristics*

Patients in the CCPR group were significantly older at time of CA compared to the patients in the ECPR group. In only 60% of the CCPR patients, thrombolytic therapy was given, whereas in 95% of the ECPR patients (Table 1). In the ECPR group, all but one patient were treated with thrombolytic therapy prior to ECMO cannulation. No significant differences were seen the CCPR versus ECPR groups, concerning OHCA (42% versus 68%), time between CA and start of CPR (both median 0 min), median total duration of CPR (60 versus 77 min), and worst blood gas values during cardiac arrest (Table 1). The median duration of ECMO treatment in the ECPR group was 3 days (IQR 2–5 days). The median ICU length of stay in surviving ECPR patients is 19 days (IQR 10–35.5 days). In the non-surviving patients the median time on ECMO until death is 3 days (IQR 1–5 days).

### *Outcomes*

A Log-rank test showed a significantly lower cumulative ICU survival in CCPR patients compared to ECPR patients (5% vs 26%, $p < 0.01$) (Fig. 2). Survival with favourable neurological outcome was present in 0 (0%) CCPR patients versus four (21%) ECPR patients ($p < 0.05$). Four out of five (80%) ECPR survivors had a favourable neurological outcome. One patient (20%) had severe cerebral disability with a CPC score of 3.

Furthermore, the complication rate of bleeding and infection appear more frequently in the ECPR group ($p < 0.05$). Bleeding occurred in 5% in the CCPR group versus 74% in the ECPR group ($p < 0.01$). A bleeding complication was documented in 13/30 (43%) of the patients who received thrombolytic therapy versus 2/9 (22%) of the patients who did not receive thrombolytic therapy ($p = 0.44$). Of all ECPR patients, seven (37%) had access site related bleeding and two (11%) had an intracranial/cerebral haemorrhage. The other bleeding locations were abdominal, thoracic, pulmonary, and ear/nose/throat.

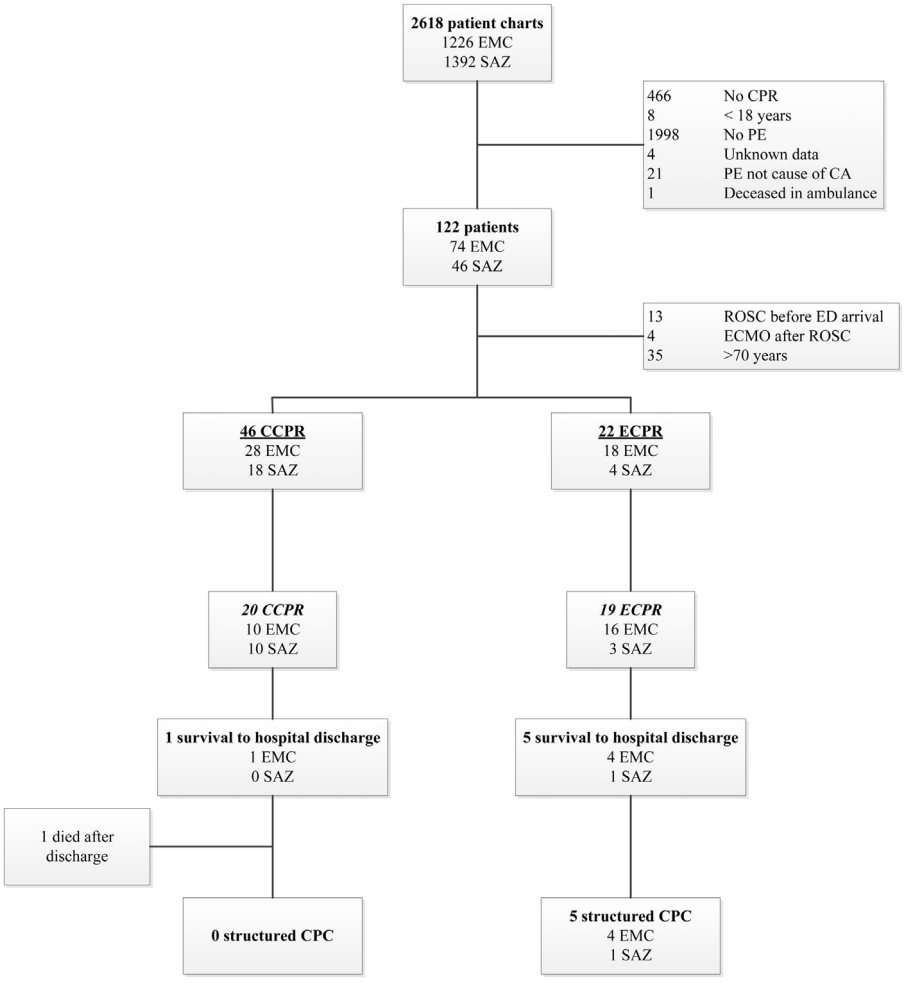


**Fig. 1 – Flow chart. Bold values represent the totals. The 'before/after'-analyses are done in the *italicized* patient numbers and the 'total sample'-analyses are done in the underlined patient numbers. CA cardiac arrest, CPR cardiopulmonary resuscitation, CCPR conventional cardiopulmonary resuscitation, ECPR extracorporeal cardiopulmonary resuscitation, ED emergency department, PE pulmonary embolism. EMC indicates patients from the Erasmus Medical Center, SAZ indicates patients from the Sint Antonius Hospital.**

**Table 1 – Baseline characteristics and outcomes for CCPR versus ECPR 'before/after'.**

| | CCPR (N=20) | ECPR (N=19) | p-Value |
|---|---|---|---|
| Baseline characteristics | | | |
| Patient characteristics | | | |
| Median age, years (IQR) | 56 (46–64) | 40 (30–60) | **0.04** |
| Gender male (%) | 8 (40) | 8 (42) | 1.00 |
| Medical history with cardiac diseases (%, total N=ECPR 19, CCPR 21) | 4 (21) | 0 (0) | 0.11 |
| Medical history with pulmonary diseases (%, total N=ECPR 19, CCPR 21) | 5 (26) | 1 (5) | 0.18 |
| Use of medication (%, total N=ECPR 18, CCPR 18) | 11 (65) | 11 (61) | 1.00 |
| Clinical characteristics | | | |
| OHCA (%) | 8 (42) | 13 (68) | 0.19 |
| CPR delay, in minutes (IQR) (total N=CCPR 13, ECPR 18) | 0 (0–0) | 0 (0–2.25) | 0.22 |
| Total duration CPR, in minutes (IQR) (total N=CCPR 19, ECPR 16) | 60 (45–90) | 77 (39–98) | 0.26 |
| Mechanical compressions (e.g. LUCAS) (%) | 7 (35) | 9 (47) | 0.52 |
| Thrombolysis (%) | 12 (60) | 18 (95) | **0.02** |
| Laboratory results | | | |
| Median pH (IQR)[a] (total N=CCPR 15, ECPR 16) | 6.85 (6.74–6.95) | 6.80 (6.68–6.87) | 0.28 |
| Median PO2 in kPa (IQR)[a] (total N=CCPR 14, ECPR 14) | 12.8 (6.2–20.7) | 14.7 (3.5–26.2) | 0.96 |
| Median PCO2 in kPa (IQR)[a] (total N=CCPR 14, ECPR 14) | 9.3 (5.8–14.4) | 9. 9 (7.1–12.4) | 0.96 |
| Median lactate in mmol/L (IQR)[a] (total N=CCPR 14, ECPR 14) | 14.3 (10.3–18.9) | 14.1 (11.3–16.6) | 0.91 |
| Median arterial saturation in mol/mol (IQR)[a] (total N=CCPR 13, ECPR 13) | 67 (43.5–96.5) | 76 (16–98.5) | 0.84 |
| Outcomes | | | |
| Primary outcome | | | |
| ICU survival (%)[b] | 1 (5) | 5 (26) | <**0.01** |
| Secondary outcome | | | |
| Survival with favourable neurological outcome (%) | 0 (0) | 4 (21) | <**0.05** |
| Favourable neurological outcome in ICU survivors (%) (CCPR N=1, ECPR N=5) | 0 (0) | 4 (80) | 0.33 |
| Complications | | | |
| Complication; bleeding (%) | 1 (5) | 14 (74) | <**0.01** |
| CVVH use (%) | 2 (10) | 7 (37) | 0.07 |
| Complication; infection with positive culture (%) | 0 (0) | 5 (28) | <**0.05** |
| Complication; intracranial bleeding (%) | 0 (0) | 2 (11) | 0.23 |
| Complication; vascular ECPR complications (%) | 0 (0) | 0 (0) | – |

[a] All the median blood gas values are the worst values 6 h before ECMO/ROSC.
[b] As there were no differences between ICU and hospital survival, we only note the ICU survival. The p-value represents the Log-rank test.

Use of CVVH showed a trend in favour of CCPR patients and no significant difference was seen in intracranial/cerebral haemorrhage between the two groups (Table 1). Besides the access site related bleedings, no other vascular or ischemic complications due to ECPR therapy, such as limb ischemia, compartment syndromes of the legs or fasciotomies occurred.

When performing the analyses on only the patients treated with thrombolytic therapy, we found comparable results. The cumulative ICU survival was 8% in CCPR patients vs 28% in ECPR patients ($p < 0.01$) with a survival with favourable neurological outcome of 0% vs 22% respectively ($p = 0.13$).

### *Sensitivity analysis*

In our sensitivity analysis, we included the 39 patients from the 'before/after'-setting, the remaining three ECPR patients from the 'before'-period, and the remaining 26 CCPR patients from the 'after'-period. The reasons why the CCPR patients of the 'after'-group included in sensitivity analysis were not treated with ECPR therapy are explained in Supplementary material, Table 3. Of the total 68 included patients, 46 patients had CCPR and 22 patients got treatment with ECPR therapy.

The baseline characteristics of the total sample were comparable to the results of the 'before/after'-setting. The only difference, was a significantly higher percentage of patients with a medical history of pulmonary disease prior to the CA in the CCPR group in comparison with the ECPR group, 26% (n=11) versus 5% (n=1), respectively ($p < 0.05$) (Supplementary material, Table 2). This pulmonary disease consisted of Chronic Obstructive Pulmonary Disease (COPD), pulmonary emphysema, bronchial asthma, mesothelioma, pulmonary metastasis of colon carcinoma, pulmonary metastasis of breast cancer, stage IV pulmonary carcinoma, or pulmonary embolism. Table 4 in Supplementary material shows the diagnostics used to confirm/suspect diagnosis of PE.

The primary and secondary outcome results of the sensitivity analysis were also comparable to the results of the 'before/after'-setting (Table 2 and Fig. 3, Supplementary material). There were two notable differences. First the difference in survival with favourable neurological outcome between the CCPR and ECPR groups became nonsignificant (Table 2, Supplementary material). However a trend to significance remained, with a p-value of 0.05. Second the trend in the use of CVVH became significantly lower in CCPR versus ECPR patients (Table 2, Supplementary material). The reasons for death in non-survivors in this study in the CCPR group (n=41, out of total

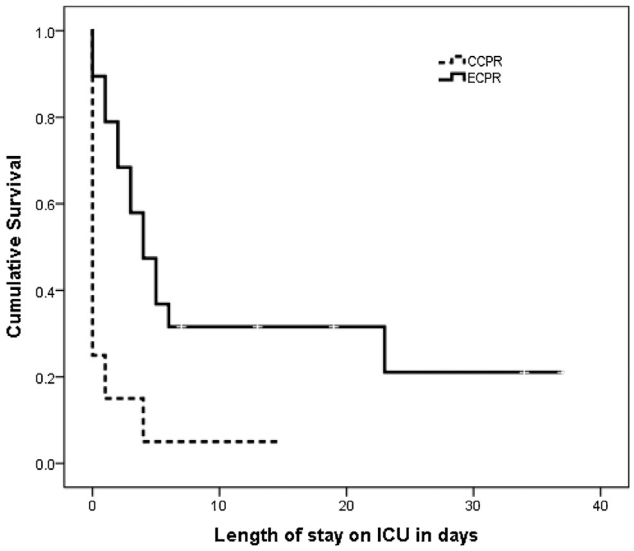


**Fig. 2 – Survival curve for CCPR versus ECPR of the 'before/after' analysis (n=39).**

n=46) mostly because of unsuccessful resuscitation (n=39) and two because of neurological reasons. Most of the ECPR patients (total n=22) died from neurological causes (n=11), e.g. brain death or termination of treatment due to poor neurological prognosis. Two patients died from multi-organ failure and two from haemorrhagic shock.

## Discussion

The present study showed that patients with CA due to (suspected) massive PE treated with CCPR had a lower ICU survival rate and a worse favourable neurological outcome as compared to patients treated with ECPR. To our knowledge, this is the first retrospective study that attempts to compare the survival and neurological outcomes of the CCPR and ECPR treatments in (suspected) PE patients.

In line with previous studies in CA patients due to massive PE, the survival rate of our control group (CCPR group) was very low (5%). Er et al.[15] showed a somewhat higher survival rate of 18.3%, but they included only IHCA patients. Comess et al.[16] also found a slightly higher survival rate (22.2%). However, only patients with unknown reason for CA received a transoesophageal echocardiography (TEE) and were included in their study. Thereby they excluded the other CA patients due to PE, what probably resulted in selection bias. Leitner et al.[17] included only patients that received thrombolytic agents and found a survival rate of 19%.

In contrast, two other studies found lower survival rates. In a study comparing thrombolytic therapy against no thrombolytic therapy in CA due to PE, Yousuf et al.[8] found a combined survival rate of 9.5% (10.5% vs 8.7%, respectively). In a slightly larger study, Kurkciyan et al.[7] also compared thrombolysis versus no thrombolysis in CA due to PE. This study showed a survival rate of 5%.[7] These rates are comparable with our controls, with a survival rate of 5% in the CCPR group in the 'before/after' design and 11% in the total sample. Previous studies concerning thrombolytic therapy in CA patients due to PE, show both significantly and non-significantly lower survival rates in patients not treated with thrombolytic agents.[8,18] Thus, the significant difference in administration of thrombolytic therapy in our study, may contribute to the difference in survival outcomes between the two groups.

The survival rate in our ECPR group is comparable with previous studies.[9,11,19–23] Although, the survival rates in studies describing V-A ECMO after massive PE show large variation.[9,11,19–23] In these studies, demonstrating a relatively high survival rate (25–83%), only patients who regained ROSC before implantation of V-A ECMO were included.[9,11,19–23] In other studies examining V-A ECMO during resuscitation, survival rates were between 0 and 60%, however, these studies only included between two and seven patients.[24–26] Two recent, larger studies[27,28] showed a survival rate of 38.5–46% in patients with cardiogenic shock or CA due to massive PE. In contrast with those studies, we did not include patients with ROSC before ECMO placement.

Patients who are treated with ECPR for cardiac arrest due to PE, seem to have a good neurological outcome. Pasrija et al.[23] found a good neurological outcome in both of the ECPR patients included in their study. A slightly larger study by Hashiba et al.[11] included 12 patients with CA due to PE who received V-A ECMO. 6 patients were treated with ECPR and 6 patients treated with V-A ECMO after ROSC. They found a CPC-score of 1–2 at discharge in 70% of the surviving patients,[11] comparable to our results with 80% of the survivors having a favourable neurological outcome.

There are several limitations to this study, related to the observational design that need to be addressed. First, two potential biases that may influence the generalizability of the results are confounding by indication and selection bias. The three patients in the 'before'-period who were treated with ECPR. The decision to treat with ECPR took place at the discretion of the clinical physician present. We tried to minimize these two biases as much as possible by performing a pure 'before/after'-analysis and a sensitivity analysis with the total sample of CCPR and ECPR patients. This sensitivity analysis showed similar results in comparison with the 'before/after'-analysis which reduces the selection bias.

Second, we tried to establish two comparable and homogeneous groups by excluding all patients above 70 years old of the CCPR group. However, we were not able to perform propensity matching, nor multivariate analysis due to the small sample size. To minimize confounding by indication as well as countering the limitations of this study, a randomized controlled trial is required in the future.

Next, diagnosis of PE in patients without ROSC is difficult. Therefore, we defined criteria which had to be met, to apply for the diagnosis suspected PE as closest approximation of the diagnosis PE during CA. To obtain a definitive diagnosis PE, a CT-scan, ventilation-perfusion scan or pulmonary angiography is needed, which was not feasible in CA patients without ROSC. In our study, only one patient had a history of (stable) decreased RV function. However, this patient had a cardiac arrest 20 days after a surgical procedure, therefore we included this patient with diagnosis high suspicion of PE. All other included patients had no known history of RV failure/dilatation.

Another important limitation of this study is the relative small sample size and thereby the inability of performing any modelling in which probable confounders (i.e., the application of thrombolytic therapy) could be included. Therefore, in the future it is necessary to perform a study in a larger sample size, to measure the effect of confounders.

Thus, ECPR therapy has promising results in comparison with the CCPR therapy, although the overall survival rate in the ECPR group remains low and significant complications may occur. With this study we have shown that a large percentage of patients who survive using ECPR, have a favourable neurological outcome. With these results, and taking the high risk of complications into account, ECPR could be a potential treatment option in CA patients due to (suspected) PE.

## Conclusion

In our two-center experience, we conclude that survival rate and favourable neurological outcome in patients with CA due to (suspected) massive PE is improved using ECPR as compared to CCPR. However, further research is needed in larger samples to confirm these encouraging findings.

## Conflicts of interest

DRM declares having received speaking fees from Xenios GmbH and HillRom GmbH.

DG is a member of the medical advisory board of Xenios GmbH and received travel expenses and speakers fees from Xenios and Maquet GmbH.

## Acknowledgment

None.

## Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at https://doi.org/10.1016/j.resuscitation.2018.12.008.

## REFERENCES

1. Fedullo PF, Yung GL, et al. Pulmonary thromboembolic disease. In: Grippi MA, Elias JA, Fishman JA, editors. Fishman's pulmonary diseases and disorders. New York, NY: McGraw-Hill Education; 2015.
2. Aissaoui N, Konstantinides S, Meyer G. What's new in severe pulmonary embolism? Intensive Care Med 2018;1–3, doi:http://dx.doi.org/10.1007/s00134-018-5199-4.
3. Logan JK, Pantle H, Huiras P, Bessman E, Bright L. Evidence-based diagnosis and thrombolytic treatment of cardiac arrest or periarrest due to suspected pulmonary embolism. Am J Emerg Med 2014;32:789–96.
4. Bailen MR, Cuadra JA, Aguayo De Hoyos E. Thrombolysis during cardiopulmonary resuscitation in fulminant pulmonary embolism: a review. Crit Care Med 2001;29:2211–9.
5. Kadner A, Schmidli J, Schonhoff F, et al. Excellent outcome after surgical treatment of massive pulmonary embolism in critically ill patients. J Thorac Cardiovasc Surg 2008;136:448–51.
6. Tokyo CCUNSC. Latest management and outcomes of major pulmonary embolism in the cardiovascular disease early transport system: Tokyo CCU Network. Circ J 2010;74:289–93.
7. Kurkciyan I, Meron G, Sterz F, et al. Pulmonary embolism as a cause of cardiac arrest: presentation and outcome. Arch Intern Med 2000;160:1529–35.
8. Yousuf T, Brinton T, Ahmed K, et al. Tissue plasminogen activator use in cardiac arrest secondary to fulminant pulmonary embolism. J Clin Med Res 2016;8:190–5.
9. Maggio P, Hemmila M, Haft J, Bartlett R. Extracorporeal life support for massive pulmonary embolism. J Trauma 2007;62:570–6.
10. Goldhaber SZ, Visani L, De Rosa M. Acute pulmonary embolism: clinical outcomes in the International Cooperative Pulmonary Embolism Registry (ICOPER). Lancet 1999;353:1386–9.
11. Hashiba K, Okuda J, Maejima N, et al. Percutaneous cardiopulmonary support in pulmonary embolism with cardiac arrest. Resuscitation 2012;83:183–7.
12. Yusuff HO, Zochios V, Vuylsteke A. Extracorporeal membrane oxygenation in acute massive pulmonary embolism: a systematic review. Perfusion 2015;30:611–6.
13. Mak M, Moulaert VR, Pijls RW, Verbunt JA. Measuring outcome after cardiac arrest: construct validity of Cerebral Performance Category. Resuscitation 2016;100:6–10.
14. Field A. 3rd ed.. Discovering statistics using SPSS. Sage Publications; 2009.
15. Er F, Nia AM, Gassanov N, Caglayan E, Erdmann E, Hoppe UC. Impact of rescue-thrombolysis during cardiopulmonary resuscitation in patients with pulmonary embolism. PLoS One 2009;4:e8323.
16. Comess KA, DeRook FA, Russell ML, Tognazzi-Evans TA, Beach KW. The incidence of pulmonary embolism in unexplained sudden cardiac arrest with pulseless electrical activity. Am J Med 2000;109:351–6.
17. Leitner JM, Jilma B, Spiel AO, Sterz F, Laggner AN, Janata KM. Massive pulmonary embolism leading to cardiac arrest is associated with consumptive coagulopathy presenting as disseminated intravascular coagulation. J Thromb Haemost 2010;8:1477–82.
18. Spohr F, Wenzel V, Bottiger BW. Thrombolysis and other drugs during cardiopulmonary resuscitation. Curr Opin Crit Care 2008;14:292–8.
19. Bougouin W, Marijon E, Planquette B, et al. Pulmonary embolism related sudden cardiac arrest admitted alive at hospital: management and outcomes. Resuscitation 2017;115:135–40.
20. George B, Parazino M, Omar HR, et al. A retrospective comparison of survivors and non-survivors of massive pulmonary embolism receiving veno-arterial extracorporeal membrane oxygenation support. Resuscitation 2018;122:1–5.
21. Munakata R, Yamamoto T, Hosokawa Y, et al. Massive pulmonary embolism requiring extracorporeal life support treated with catheter-based interventions. J Int Heart 2012;53:370–4.
22. Omar HR, Miller J, Mangar D, Camporesi EM. Experience with extracorporeal membrane oxygenation in massive and submassive pulmonary embolism in a tertiary care center. Am J Emerg Med 2013;31:1616–7.
23. Pasrija C, Kronfli A, George P, et al. Utilization of veno-arterial extracorporeal membrane oxygenation for massive pulmonary embolism. Ann Thorac Surg 2018;105:498–504.
24. Swol J, Buchwald D, Strauch J, Schildhauer TA. Extracorporeal life support (ECLS) for cardiopulmonary resuscitation (CPR) with pulmonary embolism in surgical patients — a case series. Perfusion 2016;31:54–9.
25. Corsi F, Lebreton G, Brechot N, et al. Life-threatening massive pulmonary embolism rescued by venoarterial-extracorporeal membrane oxygenation. Crit Care 2017;21:76.
26. Kawahito K, Murata S, Adachi H, Ino T, Fuse K. Resuscitation and circulatory support using extracorporeal membrane oxygenation for fulminant pulmonary embolism. Artif Organs 2000;24:427–30.
27. Meneveau N, Guillon B, Planquette B, et al. Outcomes after extracorporeal membrane oxygenation for the treatment of high-risk pulmonary embolism: a multicentre series of 52 cases. Eur Heart J 2018;39(47):4196–204.
28. Al-Bawardy R, Rosenfield K, Borges J, et al. Extracorporeal membrane oxygenation in acute massive pulmonary embolism: a case series and review of the literature. Perfusion 2018;34(1):22–8.