# Supportive Therapy
## Extracorporeal Membrane Oxygenation

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**KEYWORDS**

- Extracorporeal membrane oxygenation (ECMO) • Pulmonary embolism (PE)
- Hemodynamic instability

**KEY POINTS**

- The spectrum of pulmonary embolism (PE) ranges from subclinical microemboli to massive embolism causing immediate cardiac arrest.
- Venoarterial extracorporeal membrane oxygenation can be used in the management of high-risk PE with hemodynamic instability as a bridge to treatment or recovery.

### INTRODUCTION

After witnessing the sudden death of a patient from acute pulmonary embolism (PE), Dr John Gibbon worked decades to develop cardiopulmonary bypass (CPB).[1] Now, approximately 70 years after his rudimentary CPB circuit first supported open heart surgery, modern versions are used worldwide daily for cardiac surgery and extracorporeal membrane oxygenation (ECMO). ECMO is a modified CPB circuit that

1. Drains venous blood
2. Pumps the blood through a membrane oxygenator where up to full gas exchange occurs
3. Returns the blood to either venous circulation for respiratory support only (venovenous [VV]-ECMO) or arterial circulation for both respiratory and hemodynamic support (venoarterial [VA]-ECMO). Although available for more than 40 years, over the past decade, ECMO has gained popularity for PE resuscitation in the most severe acute PE cases.

Acute PE is a relatively common emergency, with an annual incidence of approximately 1 per 1000 people in the United States.[2,3] Clinical presentation of acute PE

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ranges from absent or mild symptoms (chest pain and cough) to life-threatening hemodynamic deterioration.[4] Acute PE is classified as high risk, intermediate risk, or low risk, based on the degree of hemodynamic compromise.[4] High-risk PE causes hemodynamic instability (shock or hypotension: systolic blood pressure <90 mm Hg or heart rate <40 bpm); intermediate-risk PE causes myocardial strain (identified by echocardiography or elevated plasma troponin or natriuretic peptide levels); and low-risk PE does not cause hemodynamic compromise.

The preferred method of diagnosis for all risk levels of PE is computed tomography (CT) angiography.[4] If CT angiography is unavailable or if a patient is too unstable to be transported to the radiology suite, bedside echocardiography is performed. Low-risk and intermediate-risk PE require anticoagulants, which decrease new thrombus formation and permit existing clot to naturally dissolve.[5] High-risk hemodynamically unstable PE may be treated either with thrombolytics to dissolve clot or embolectomy to remove clot. Current guidelines suggest systemic or catheter-directed thrombolytics as first-line therapy for high-risk PE.[4] Thrombolytics, however, carry a substantial risk of major bleeding and intracerebral hemorrhage and use typically is considered on a case-by-case basis.[6–8] In patients with refractory circulatory collapse or cardiac arrest, ECMO is considered in combination with catheter-directed treatment or surgical embolectomy.[4] Resuscitation or stabilization with VA-ECMO (which provides up to total cardiopulmonary support) prior to surgical embolectomy has improved outcomes over embolectomy alone. See **Table 1** for details. Likewise, several single-center and multicenter reports have demonstrated success with ECMO as a bridge to decision: either surgical embolectomy or as definitive treatment.[9,10] This article reviews the growing literature of ECMO management of PE.

## CARDIAC ARREST

Cardiac arrest is an ever-present threat in high-risk PE. When emboli obstruct blood flow to the lungs, the increased impedance and resistance elevates right heart pressure, which can rapidly progress to right heart failure with cardiac arrest. The rapidity of deterioration is directly related to the size and amount of obstruction and the degree of stabilization achieved by compensatory physiology. Hemodynamic deterioration to cardiac arrest can occur with large saddle emboli obstructing the bifurcation of the pulmonary artery (**Fig. 1**).[11]

Cardiac arrest during high-risk PE that is refractory to fluids, inotropes, and CPR usually requires immediate CPB or VA-ECMO for salvage.[12,13] ECMO during cardiopulmonary resuscitation (CPR) is known as ECPR and can be initiated in or out of the hospital.[14,15] The goal of ECPR is maintain tissue perfusion during refractory cardiac arrest in order to prevent long-term ischemic damage. Prehospital cardiac arrest has a poor overall survival rate of 6% to 15%[16–18] whereas in-hospital cardiac arrest from PE has a survival rate of 25% when supported with ECPR.[19] As in all major series, improved survival is related directly to timely initiation of resuscitation and ECMO.

As discussed previously, the most important determinant of ECPR outcome is early initiation of quality chest compressions to generate a modest cardiac output (low-flow) that supplies coronary blood flow, facilitating the return of spontaneous circulation (ROSC).[20–22] ECPR should be considered only when CPR is initiated within 5 minutes of cardiac arrest.[15] A 6-year (2005–2011) retrospective registry study capturing all prehospital cardiac arrests in Denmark (Danish Cardiac Arrest Registry) demonstrated bystander CPR initiated within 5 minutes of arrest doubled 30-day survival from 6.3% (no bystander CPR; 95% CI, 5.1–7.6) to 14.5% (95% CI, 12.8–16.4).[20,23]

**Table 1**
**Recent reports of extracorporeal membrane oxygenation for pulmonary embolism**

| Author | Date | Extracorporeal Membrane Oxygenation (n) | Indications for Extracorporeal Membrane Oxygenation | Pre–extracorporeal Membrane Oxygenation Cardiac Arrest (n) | Pre–extracorporeal Membrane Oxygenation Treatment (n) | Survival, % (n) | Definitive Therapy, n (Discharged [n]) |
|---|---|---|---|---|---|---|---|
| Al-Bawardy et al,[42] 2019 | 2012–2019 | 13 | RV dilatation and RV hypokinesis | 13 | NR | 69 (7/13), 30-d overall | 1 anticoagulation (1, 90-d)<br>8 systemic thrombolytics (3, 90-d)<br>3 catheter-directed thrombolytics (1, 90-d)<br>4 surgical embolectomy (2, 90-d) |
| Ius et al,[26] 2019 | 2012–2018 | 36 | Cardiac arrest or refractory hemodynamic instability | 15 | 19 thrombolytics or catheter-directed therapy | 67 (24/36) to discharge | 16 anticoagulation (5)<br>9 failed on ECMO<br>7 decannulated<br>20 surgical embolectomy (19) |

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|---|---|---|---|---|---|---|---|
| Kjaergaard et al,[43] 2019 | 2004–2017 | 22 | Cardiac arrest | 22 | 5 thrombolytics | 92 (13/14) never ECMO, 30-d<br>54 (12/22) ECMO, 30-d | 10 anticoagulation (4, 30-d)<br>1 failed on ECMO from incorrect cannulation<br>7 thrombolytics (2, 30-d)<br>5 surgical embolectomy (3, 30-d)<br>14 thrombolytics, never ECMO (13, 30-d) |
| Kmiec etal,[41] 2020 | 2006–2017 | 75<br>VA-ECMO 46<br>VV-ECMO 29 | VA: cardiac arrest, RV failure with refractory hemodynamic instability<br>VV: respiratory failure refractory to mechanical ventilation | 49 | 23 thrombolytics | 47 (35/75) to discharge | 28 anticoagulation<br>7 thrombolytics<br>8 interventional thrombectomy<br>10 surgical embolectomy |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| Pasrija et al,[9] 2018 | Protocol | 2015–2017 | 27 | Massive PE with unknown neurologic status or end-organ dysfunction | 6 | 6 thrombolytics | 97 (28/29) to discharge | 15 anticoagulation (14)<br>1 confirmed neurologic death<br>12 surgical embolectomy (12)<br>2 surgical embolectomy, never ECMO |
| | Historic | 2011–2015 | 6 | Cardiac arrest before planned surgical embolectomy | 6 | NR | 82 (22/27) to discharge | 6 surgical embolectomy<br>27 surgical embolectomy, never ECMO |
| Pasrija et al,[10] 2018 | 2014–2016 | | 20 | Massive PE with unknown neurologic status or end-organ dysfunction | 5 | 7 thrombolytics | 95 (19/20) to discharge | 8 anticoagulation (7)<br>1 confirmed neurologic death<br>11 surgical embolectomy (11)<br>1 catheter-directed thrombolytics (1) |
| George et al,[19] 2018 | 2012–2015 | | 32 | Massive PE with hemodynamic instability or end-organ dysfunction | 15 | NR | 53 (17/32) to discharge | 5 systemic thrombolysis (0)<br>15 catheter-directed thrombolytics (11)<br>4 aspiration thrombectomy (3)<br>2 surgical embolectomy (0) |

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|---|---|---|---|---|---|---|---|
| Meneveau et al,[29] 2018 | 2014–2015 | 52 | Cardiac arrest, hemodynamic instability, contraindication to of failure of other therapies, failure to wean CPB | 39 | 17 thrombolytics<br>10 surgical embolectomy | 38, 30-d | 18 anticoagulation (4)<br>7 surgical embolectomy (4) |
| Corsi et al,[11] 2017 | 2006–2015 | 17 | Cardiac arrest, cardiogenic shock | 15 | 8 thrombolytics<br>2 surgical embolectomy<br>1 catheter-directed thromboaspiration | 47 (8/17) to discharge | 6 anticoagulation<br>1 catheter-directed thromboaspiration<br>1 surgical embolectomy |
| Swol et al,[44] 2016 | 2008–2014 | 5 | Cardiac arrest | 5 | All surgical patients | 40 (2/5) to discharge | 3 systemic thrombolytics (1)<br>1 surgical embolectomy (0) |

*Abbreviation:* NR, not reported.

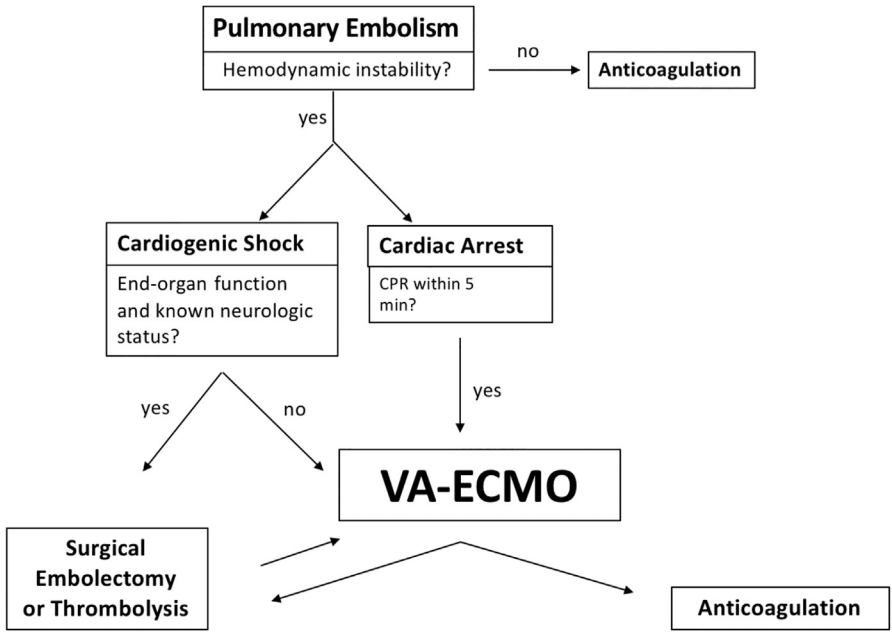


**Fig. 1.** Hemodynamic deterioration to cardiac arrest can occur with large saddle emboli obstructing the bifurcation of the pulmonary artery. (*Data from* Corsi F, Lebreton G, Bréchot N, et al. Life-threatening massive pulmonary embolism rescued by venoarterial-extracorporeal membrane oxygenation. Crit Care. 2017;21:76.)

When time between arrest and CPR increased to 10 minutes, 30-day survival dropped to 6.7% (95% CI, 5.4–8.1) but showed a 3-fold survival benefit compared with no bystander CPR. After 13 minutes, the association between survival and bystander CPR was no longer significant.[20]

If CPR fails to achieve ROSC after 10 minutes of refractory arrest in qualified patients, tissue reperfusion with ECPR cannulation should occur with VA-ECMO.[15,24] Reynolds and colleagues[21] showed if ROSC is not achieved within 16 minutes of CPR, survival with good neurologic outcome drops below 2%. Likewise, Sakuma and colleagues[25] reported ECPR survival less than 10% when CPR duration was longer than 30 minutes. These data support the concept that just a few minutes either way are critical to quality survival.

## CARDIOGENIC SHOCK

Survival drops below 10% when ECMO is initiated 30-minutes postarrest,[25] yet survival as high as 76% has been reported when ECMO is started before cardiac arrest during progressive cardiogenic shock.[19] Attempts to stabilize rapid deterioration during PE-induced cardiogenic shock can counterintuitively compound deterioration. During a PE, induction of general anesthesia with endotracheal intubation counteracts the body's vasoactive compensatory mechanisms, causing vasodilation and decreased mean blood pressure. Additionally, positive pressure ventilation decreases venous return and further accelerates hypotension with risk of sudden cardiac arrest.[12,26] A 10-year single-center chart review of 57 consecutive PE patients reported 19% experienced immediate hypotension and cardiac arrest after induction of general

anesthesia.[12] Likewise, a 4-year (2008–2012) single-center chart review of 40 PE patients showed 12.5% arrested after general anesthesia was induced. Of those who did not arrest, 17% later experienced cardiac collapse from a combination of positive pressure breathing and pericardial opening.[13]

In contrast to general anesthesia with intubation, VA-ECMO can be initiated with local anesthesia only to preserve the active compensatory physiologic mechanisms and avoid an unpredictable cardiac arrest. VA-ECMO with systemic anticoagulation and heparin-bonded circuits achieves hemodynamic stabilization and total gas exchange and enhances existing recovery mechanisms by tipping the balance toward thrombus resolution. Pulmonary vascular resistance normalizes with clot resolution, but the distended right ventricle (RV) needs time to recover before the underlying threat of arrest from right heart failure resolves. VA-ECMO allows the RV to decompress by removing volume from the inferior and/or superior vena cava before it reaches the right heart. Blood then is returned to the systemic circulation, usually by femoral artery access. If the right heart does not decompress, additional venous drainage, higher flow, and trans-septal left atrial decompression are options.[27]

The benefits of VA-ECMO are remarkable; 52% of massive PE patients bridged to surgical embolectomy had thrombus resolution and recovered RV function after 3 days of VA-ECMO support, including systemic heparinization.[9] When VA-ECMO fails to resolve thrombus and RV dysfunction, by stabilizing the patient's hemodynamics and gas exchange prior to surgery, outcomes of a subsequent surgical embolectomy may improve. Pasrija and colleagues[10] showed massive PE supported with VA-ECMO (n = 20) before surgical embolectomy had 95% in-hospital survival (100% survival after decannulation), with 40% of patients recovering with VA-ECMO support alone. Systemic thrombolytics typically are not given concomitantly with VA-ECMO; however, catheter-directed thrombolysis while on VA-ECMO has been reported.[19] ECMO also has been used successfully for stabilization before catheter-directed thrombolysis.[28]

**Table 1** summarizes the current literature regarding ECMO support for acute PE. Studies include case series and cohort studies, ranging from 5 patients to 75 patients. There currently are no randomized control trials comparing VA-ECMO to medical therapy or surgical embolectomy alone for acute PE. The table illustrates the complex decision making and lack of consensus regarding patient selection and timing of VA-ECMO. The most common indication for VA-ECMO in acute PE was cardiac arrest with study groups containing 25% to 100% of pre-ECMO cardiac arrest. Once ECMO was initiated, patients were bridged to anticoagulation, systemic thrombolytics, catheter-directed thrombolytics, and surgical embolectomy, alone or in combination. Survival ranged from 38% to 97%.[9,29]

## PREGNANCY

PE is a leading cause of death during pregnancy.[30] ECMO should be considered for massive PE during pregnancy because the stability of the mother and the survival of the child depend on adequate perfusion and gas exchange. Maternal blood gases of $Pa_{O_2}$ greater than or equal to 70 mm Hg, oxygen saturation greater than or equal to 95%, and $Pa_{CO_2}$ 30 mm Hg to 32 mm Hg[31] are proposed to ensure survival of both child and mother. Through decades of experience with CPB during pregnancy, and multiple cases of ECMO, the use of heparin anticoagulation during pregnancy has been established as relatively safe.[32–35] Low-molecular-weight heparin and unfractionated heparin do not cross the placenta and are the drugs

of choice for PE during pregnancy.[35] In contrast, thrombolytics are relatively contraindicated in pregnancy and are associated with higher postpartum hemorrhage.[36]

## CANNULATION STRATEGIES

VA-ECMO is the most common ECMO strategy for supporting massive PE. For patients with hemodynamic instability, cannulation for VA-ECMO often is performed emergently. The most commonly employed cannulation strategy utilizes femoral venous drainage and femoral arterial return. This strategy is popular because it can be accomplished at the bedside either by a percutaneous approach or with a surgical cutdown for insertion. To prevent vascular complications in the groin when attempting to establish urgent access, preplacement of suture-mediated closure devices (ie, Perclose ProGlide, Abbott Vascular, Santa Clara, CA) has been reported.[37] After femoral arterial access is obtained, an additional distal perfusion cannula (5–6 French) should be routinely placed to prevent distal limb ischemia.[38,39] The importance of distal perfusion is elevated in patients with a history of peripheral vascular disease due to their increased likelihood of vascular complications.[40]

Recently, VV-ECMO has also been used to support PE patients with RV strain secondary to respiratory failure.[41] VV-ECMO requires only venous access to oxygenate and ventilate central venous blood through the ECMO circuit. Although lacking in cardiac support, total gas exchange often stabilizes the patient by relieving pulmonary vasoconstriction and also provides therapeutic systemic anticoagulation, without the increased risk of arterial access. If VV-ECMO fails to

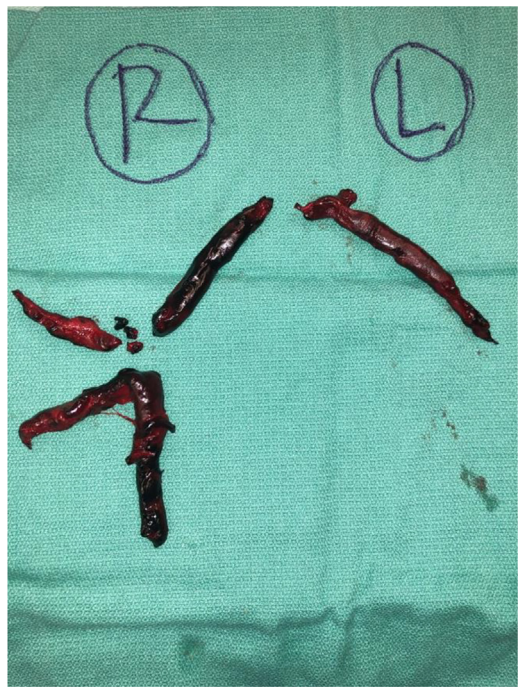


**Fig. 2.** Saddle embolus surgically removed from right and left pulmonary vessels.

relieve right heart strain and reverse the respiratory failure, conversion to VA-ECMO is accomplished by additional arterial access previously described to achieve cardiac support.

## PULMONARY EMBOLISM RESPONSE TEAM

PE response teams (PERTs) are interdisciplinary teams developed to optimize care for PE. The teams may consist of pulmonologists, cardiologists, radiologists, cardiothoracic surgeons, and critical care and emergency medicine physicians.[26,42] Decisions to use advanced treatments are made by the PERT in collaboration with the primary treatment team and the patient's family.[42] The risk-benefit analysis for treatment with thrombolysis, surgical embolectomy, and ECMO is complex and made on a case-by-case basis. The authors have placed the literature experience in a table to illustrate the wide variability of practices, preferences, techniques, and outcomes (see **Table 1**). From this table, it can readily be appreciated that patient selection, use of thrombectomy or anticoagulation prior to ECMO, use of thrombectomy or catheter-based thrombolytics, and patient outcomes are extremely variable, yet promising, toward improved care of saddle embolus. A review of these articles shows the benefit of an algorithm-based management approach of PE (**Fig. 2**). Additional studies are required to define the best algorithms to utilize during different patient presentations.

## SUMMARY

Massive PE represents a minority of PE cases but is associated with high mortality. ECMO can stabilize cardiac output and allows gas exchange while simultaneously providing systemic anticoagulation to prevent clot propagation and allow natural thrombolytics to progress. ECMO can be implanted in an awake patient, thereby avoiding hemodynamic collapse from anesthesia. ECMO is indicated as either a bridge to recovery or a decision to perform thrombectomy or thrombolysis.

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