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Supportive organ management in acute right ventricular failure secondary to pulmonary embolism: a narrative review

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Abstract

Background. Pulmonary embolism (PE) is among the most common cardiovascular syndromes and a leading cause of acute right ventricular failure (ARVF), which drives hemodynamic and respiratory deterioration and high mortality. While causal treatment removes the thrombus, supportive organ management keeps patients alive until it takes effect, yet clear algorithms are lacking.

Aim. To review the pathophysiology of ARVF in PE and the available supportive (hemodynamic, oxygenation, ventilation) strategies, appraising their efficacy, safety and evidence quality.

Material and methods. Narrative review of PubMed and Google Scholar (2016–2026, with justified exceptions), using Boolean combinations of terms on right ventricular failure, PE and organ support. Thirty-six studies were narratively synthesized, each assessed for design, sample size and methodology.

Results. ARVF in PE stems from right ventricular (RV) pressure overload uncoupling contractility from afterload, causing RV dilatation, septal shift, left ventricular (LV) underfilling and ischemia. Supportive care targets preload, afterload and contractility. High-flow oxygen therapy appears superior to low-flow and possibly comparable to non-invasive ventilation; positive-pressure ventilation risks hemodynamic harm. Fluids require cautious titration guided by central venous pressure (CVP) and venous excess ultrasound (VExUS). Norepinephrine and dobutamine are preferred; levosimendan and milrinone reduce pulmonary vascular resistance but risk hypotension. Inhaled vasodilators (notably nitric oxide) and oxygen show promise but are of unproven benefit. Mechanical circulatory support (mainly veno-arterial extracorporeal membrane oxygenation, VA-ECMO) is a bridging option, best combined with embolectomy.

Conclusions. Management of ARVF in PE demands combined causal and supportive treatment, but much supportive care rests on extrapolated or low-quality evidence; PE-specific studies are needed.

Keywords: pulmonary embolism; acute right ventricular failure; hemodynamic support; oxygen therapy; mechanical circulatory support

1 Introduction

Venous thromboembolism (VTE), with pulmonary embolism (PE) as one of its clinical forms, is one of the most frequent cardiovascular syndromes, affecting millions of people a year and being responsible for up to 10% of in-hospital deaths (Konstantinides et al., 2020; Shah et al., 2022). PE, which is characterized by occlusion of a pulmonary artery by embolic material, affects both circulation and gas exchange. The main risk factors are major trauma, surgery, lower limb fractures, malignancies, estrogen-containing oral contraceptive agents and infection (Konstantinides et al., 2020). The primary cause of death in PE is acute right ventricular failure (ARVF), one of the most frequent causes of which is PE itself (Giannakoulas et al., 2025; Konstantinides et al., 2020; McGuire et al., 2024).

ARVF is a rapidly progressing condition characterized by right ventricular (RV) dysfunction, decreased cardiac output (CO), systemic congestion and hemodynamic decompensation or cardiogenic shock (Giannakoulas et al., 2025). It is a common condition treated in intensive care units (ICUs) and is associated with a high mortality rate (up to 70% when mechanical circulatory support is needed) (Manca et al., 2025). This, combined with the fact that precise management algorithms are lacking and the optimal approach remains ambiguous (Manca et al., 2025), underscores the clinical relevance of the topic. ARVF in the course of PE requires causal treatment to remove the thrombus responsible for the hemodynamic deterioration. This is achieved through methods such as systemic thrombolysis and percutaneous or surgical thrombectomy (Konstantinides et al., 2020). However, supportive management that keeps the patient alive and allows time for causal treatment to take effect is also crucial. Such management, focused on optimizing hemodynamics, oxygenation and ventilation, can involve oxygen therapy, mechanical ventilation, fluid therapy, inotropes, vasopressors or pulmonary vasodilators, and mechanical circulatory support (MCS) (Harjola et al., 2016; McGuire et al., 2024; Sorathia et al., 2025).

Providing comprehensive management of ARVF in PE requires a thorough understanding of both the pathophysiology of these conditions and the available therapeutic methods. That is the focus of this narrative review.

2 Material and methods

This study is a narrative literature review based on a comprehensive search and analysis of the available scientific research on right ventricular failure secondary to pulmonary embolism and related conditions. Particular attention was paid to the pathophysiology and etiology of such right ventricular failure; to hemodynamic and respiratory support, especially the available pharmacological agents, oxygen therapy and mode of ventilation; and to other advanced methods of organ support, together with their effectiveness, safety and the weight of the evidence supporting them. The literature search was structured and performed across the PubMed and Google Scholar electronic databases.

The strategy involved searching for articles by combining the keywords below with Boolean operators, with terms applied to either the Title/Abstract or All fields to broaden retrieval. The strings included “acute right ventricle failure”, “Right ventricular dysfunction”, “Acute right heart failure”, “Acute pulmonary embolism”, “Hemodynamics”, “Fluid resuscitation”, “Fluid”, “Inotropes”, “critical care”, “intensive care”, “Anesthetic”, “Clinical practice”, “mechanical ventilation”, “organ support”, “mechanical circulatory support”, “vasopressors”, “vasodilators”, “thrombectomy”, “surgical”, “catheter-directed”, “thrombolysis”. Several supplementary sources were also consulted to provide a more comprehensive overview of specific topics.

Inclusion criteria were as follows:

- Research published in the years 2016–2026 (with a few exceptions justified by the significance of the data for the advancement of knowledge in the field)
- Articles that were guidelines of European or American scientific associations, randomized controlled trials, meta-analyses, case series, and systematic and narrative reviews

- Articles in Polish or English

Exclusion criteria were as follows:

- Studies published before 2016 unless they provided fundamental data
- Single case studies, case studies with insufficient clinical data
- Non-peer-reviewed articles.

After the initial search and a supplementary search, 36 studies were included. The data were narratively synthesized, with particular emphasis on current pathophysiological models of acute right ventricular failure in pulmonary embolism and their application to clinical reasoning, on the available supportive management methods (hemodynamics, respiration, oxygenation), and on their efficacy and safety. Each study was analyzed in the context of its design, sample size and methodology.

3 Results

3.1 Pathophysiology of ARVF and PE

Acute right ventricular failure (ARVF) is a rapidly progressive condition caused by RV dysfunction leading to reduced cardiac output, systemic congestion and decompensated heart failure or cardiogenic shock (Giannakoulas et al., 2025; Manca et al., 2025). Its central pathophysiological mechanism is the uncoupling of RV contractility (Ees) and pulmonary artery elastance (Ea), which reflects the relationship between RV afterload and the ventricle's ability to overcome it (Giannakoulas et al., 2025). Understanding these mechanisms is essential for interpreting the hemodynamic and imaging findings in ARVF.

The right ventricle (RV) pumps blood returning from the systemic circulation into the low-pressure, low-resistance pulmonary circulation (Manca et al., 2025). Unlike the left ventricle (LV), it has two layers of cardiomyocytes (longitudinal and circumferential). Because of the

aforementioned properties of the pulmonary circulation (low resistance), the RV has thinner walls and is lighter than the LV (approximately one sixth of its mass). For this reason it can accommodate large variations in venous return with a relatively constant stroke volume (SV) (Giannakoulas et al., 2025). The two ventricles are, however, interconnected, as they occupy the same pericardial space and share the interventricular septum. This is why 20–40% of RV pressure generation is dependent on the LV. LV function is also crucial to the RV in the context of right coronary artery (RCA) perfusion. It is worth mentioning that the marginal branches of the RCA are perfused in both diastole and systole, and not, as in the case of their LV equivalents, only in diastole (Giannakoulas et al., 2025).

These properties of the RV and the pulmonary circulation have consequences that are crucial for the pathophysiology of right ventricular failure (RVF). The RV is adapted to work under low afterload. This is highlighted by the “hang-out” phenomenon, in which blood continues to flow from the RV to the pulmonary artery (PA) even after systole has finished. It occurs when PA pressure decreases faster than RV pressure (Luk et al., 2025). The main way for the RV to adapt to increasing afterload (PA pressure) is to increase its diastolic volume, which, according to Starling's law, increases SV. On the one hand the RV is dependent on preload; on the other hand its filling is limited by the pericardium, the cytoskeleton and right atrial (and caval vein) pressure (once this equals RV pressure, filling has to stop). When PA pressure rises, the RV becomes volume-limited, meaning that it can neither generate enough force to overcome the afterload nor increase filling to do so. This is well visualized on pressure–volume graphs (see Figure 1). Such graphs show the changes in the pressure and volume of the ventricle during systole and diastole (Giannakoulas et al., 2025; Luk et al., 2025). More importantly, they allow the contractility of the ventricle and its elastance to be visualized. Elastance is the change in pressure generated by a given change in volume (the opposite of compliance). The parameter that is especially important for RVF pathophysiology is the end-systolic elastance (Ees) of the RV (Giannakoulas et al., 2025). On the aforementioned graphs, Ees is represented by a line drawn through the end-systolic pressures of several cardiac cycles under different preloads. The slope of this line defines the contractility of the ventricle. The other crucial parameter is pulmonary artery elastance (Ea). Under normal conditions these two should be at least equal; most of the time the RV works with an Ees/Ea of 1.5–2.0 (which is the most energy-efficient). This ratio is referred to as RV–PA coupling. When Ees/Ea falls below 0.7–1.0, RVF occurs (Giannakoulas et al., 2025).

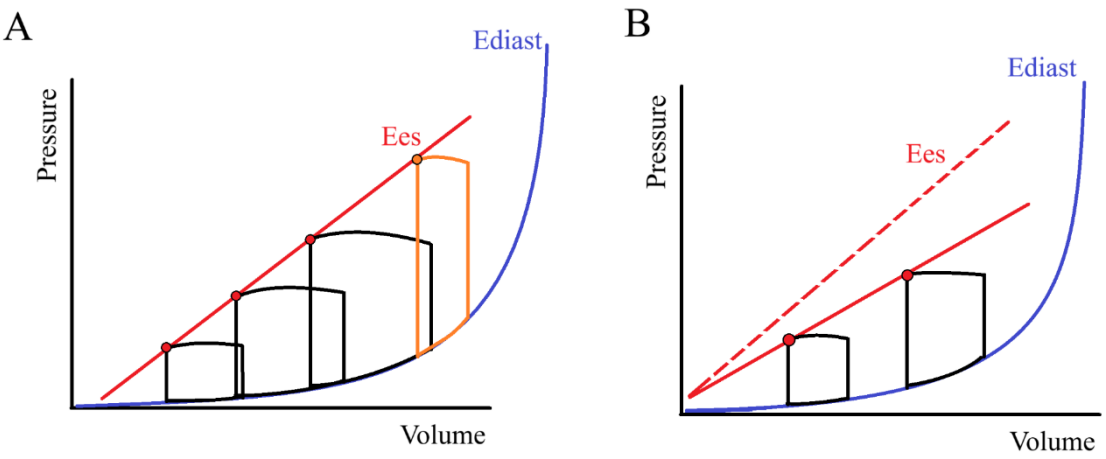

Figure 1 Pressure–volume graphs visualizing changes in end-diastolic pressure and right ventricular end-systolic elastance under different conditions.

A – Normal pressure–volume loops (black); as end-diastolic pressure increases, end-systolic volume and pressure also increase. "Ediast" denotes the end-diastolic elastance line. The orange loop shows a situation with increased pulmonary artery pressure (increased afterload). The right ventricle is "volume-limited": filling is increased almost to its maximum and stroke volume (the difference between end-diastolic and end-systolic volumes) is reduced because of the increased PA pressure limiting it. B – Pressure–volume loops in a situation of impaired right ventricular contractility (the slope of the Ees line is shallower, and the end-diastolic pressures and volumes are lower than in the normal situation, which is visualized by the dashed line).

RVF occurs through three main pathophysiological mechanisms: volume overload (i), ventricular injury (ii) and pressure overload (iii), or a mixture of these (Giannakoulas et al., 2025; Manca et al., 2025).

Volume overload is related to increased preload (venous return, VR) of the RV and in most cases cannot cause RVF alone, because of the aforementioned RV tolerance to increased VR. However, an increased volume load of the RV can be seen, for example, in the central venous pressure (CVP) or right atrial pressure (RAP) waveform as flattening of the X descent, C–V fusion and large V-waves (Giannakoulas et al., 2025). The key point, however, is that such

indicators of venous congestion do not constitute definitive evidence of volume overload as the primary mechanism of RVF, as they reflect the interaction between fluid status and right ventricular function rather than either factor in isolation. Furthermore, a patient with confirmed venous stasis may still be fluid-responsive (Pierre-Grégoire, 2025).

Ventricular injury caused by infarction or cardiac surgery leads to uncoupling of the RV from the pulmonary circulation by primarily decreasing its contractility. Impaired RV function decreases the arterial pulse pressure (the difference between systolic and diastolic PA pressure) and, retrogradely, increases CVP. The ratio of these two is called the pulmonary arterial pulsatility index (PAPi) and is considered a useful hemodynamic indicator of RV function (Giannakoulas et al., 2025).

Pressure overload is associated with increased afterload of the RV. As described above, to characterize this situation appropriately, both its resistive and its pulsatile components must be considered. The resistive part of the afterload, expressed as the transpulmonary gradient divided by CO, is not sufficient, because of the large pulmonary recruitment reserve, which blunts that parameter when engaged. A better indicator appears to be pulmonary arterial capacitance (PAC), defined as SV divided by pulmonary pulse pressure. This ratio reflects the stroke volume of the pulmonary circulation accommodated per unit of pulmonary pulse pressure. These considerations are particularly important in RVF caused by PE, as the main hemodynamic manifestation in such cases is pressure overload (Giannakoulas et al., 2025; Luk et al., 2025; Manca et al., 2025).

It is worth mentioning that, eventually, severe pressure overload leads to circulatory collapse with a secondary decrease in RV contractility, so the evolution of these hemodynamic manifestations is also important in establishing the etiopathology of RVF.

In PE, emboli obstructing the pulmonary arteries generate pressure overload: the increased afterload of the RV leads secondarily to increased right ventricular end-diastolic volume (RVEDV) and right ventricular end-diastolic pressure (RVEDP). These conditions lower RV CO and cause acute dilatation of the right ventricle (as it is thin-walled), bowing of the

interventricular septum and systemic congestion (Giannakoulas et al., 2025). Furthermore, as both ventricles occupy the same pericardium, RV dilatation and septal displacement lead to LV underfilling. The lower RV CO also enhances this phenomenon, as the CO of both ventricles tends to equalize over time (if there is no right-to-left shunt, e.g. through a patent foramen ovale) (McGuire et al., 2024). As a result, mean arterial pressure falls, as does O2 delivery. This is especially harmful for the RV: on the one hand, its O2 demand increases as afterload rises (Giannakoulas et al., 2025; Guarnieri et al., 2025; Manca et al., 2025); on the other hand, its perfusion is impaired because of a reduced myocardial perfusion gradient (right coronary artery diastolic perfusion depends on the difference between diastolic arterial pressure and RVEDP, which is increased) (McGuire et al., 2024). This situation promotes RV ischemia, which worsens its performance and aggravates RVF through all of the mechanisms described above. This cycle eventually causes hypoperfusion, circulatory collapse, multiorgan failure (MOF) and death (Giannakoulas et al., 2025; Guarnieri et al., 2025; Manca et al., 2025; McGuire et al., 2024).

Venous thromboembolism is globally the third most frequent cardiovascular syndrome (after myocardial infarction and stroke) (Konstantinides et al., 2020), with approximately 10 million cases a year (Shah et al., 2022). Clinically, it presents as deep vein thrombosis (DVT) and pulmonary embolism (PE). Up to 10% of patients with VTE develop PE, and PE in turn accounts for up to 10% of all in-hospital deaths (Shah et al., 2022). A number of VTE risk factors have been identified. It is important to distinguish between patient-related (usually non-modifiable) and setting-related (usually modifiable) factors, because PE is considered to result from an interaction between them. It is also useful to identify strong (odds ratio [OR] > 10), moderate (OR 2–9) and weak (OR < 2) risk factors (Konstantinides et al., 2020).

The best-recognized risk factors are major trauma, surgery, lower limb fractures, malignancies, estrogen-containing oral contraceptives and infection. In this context, the surgical procedures carrying the highest risk are joint replacements. Although cancer in general carries a risk of PE, the highest risk has been recognized for pancreatic, brain, lung and gastric cancers and hematological malignancies. Among the aforementioned contraceptives, the most dangerous are those containing both estrogen and progestogen (combined oral contraceptives) (Konstantinides et al., 2020). All of the recognized predisposing factors are listed in Table 1.

Table 1. PE predisposing factors (based on Konstantinides et. al., 2020).

High risk factors • Fracture of lower limb • Hospitalization for heart failure or atrial fibrillation/flutter (within previous 3 months) • Hip or knee replacement • Major trauma • Myocardial infarction (within previous 3 months) • Previous VTE • Spinal cord injury
Moderate risk factors • Arthroscopic knee surgery • Autoimmune diseases • Blood transfusion • Central venous lines • Intravenous catheters and leads • Chemotherapy • Congestive heart failure or respiratory failure • Erythropoiesis-stimulating agents • Hormone replacement therapy (depends on formulation) • In vitro fertilization

- Oral contraceptive therapy
- Post-partum period
- Infection (specifically pneumonia, urinary tract infection, and HIV)
- Inflammatory bowel disease
- Cancer (highest risk in metastatic disease)
- Paralytic stroke
- Superficial vein thrombosis
- Thrombophilia

Weak risk factors

- Bed rest >3 days
- Diabetes mellitus
- Arterial hypertension
- Immobility due to sitting (e.g. prolonged car or air travel)
- Increasing age
- Laparoscopic surgery (e.g. cholecystectomy)
- Obesity
- Pregnancy
- Varicose veins

3.2 Diagnostics

The clinical presentation of RVF is not always clear. Diagnosis therefore requires vigilance, especially in patients with numerous comorbidities. PE is the most common cause of RVF; however, not all cases are associated with it (Giannakoulas et al., 2025). The diagnostic process must focus on identifying both PE and alternative causes.

Initial triage should include the medical history, physical examination, blood-gas analysis and electrocardiogram (ECG). This should be followed by laboratory biomarkers (troponins, BNP, NT-proBNP, D-dimer, liver and kidney markers), imaging studies (echocardiography, computed tomography [CT], chest X-ray) (Giannakoulas et al., 2025) and, if RVF is strongly suspected, hemodynamic monitoring (Manca et al., 2025). These techniques allow confirmation of RV dysfunction and of the hemodynamic consequences of right ventricular failure, which, together with the dynamic course of the condition, form the core of the diagnosis of ARVF. One of the most important diagnostic modalities is imaging, which allows visualization of RV overload. The quickest method to perform is echocardiography. The most important signs are RV dilatation, an RV basal diameter > 41.0 mm, an inferior vena cava diameter > 21.0 mm with inspiratory collapse < 50%, a ventricle-to-ventricle ratio > 1.0, decreased tricuspid annular plane systolic excursion (TAPSE) < 17 mm, septal shift with a D-shaped LV, a TAPSE/systolic pulmonary artery pressure ratio < 0.55 mm/mmHg and pericardial effusion (Giannakoulas et al., 2025; Sorathia et al., 2025). Although the best imaging method with regard to RV function and tissue characterization is cardiac magnetic resonance, it is time-consuming and requires the patient's cooperation (Manca et al., 2025). CT is performed more frequently and shows the morphology and allows measurement of RV diameters, which helps in diagnosing some etiologies of RVF (pulmonary hypertension, chronic obstructive pulmonary disease [COPD], congenital heart diseases). CT pulmonary angiography (CTPA), on the other hand, is very useful in diagnosing PE (Manca et al., 2025).

For PE, the most important diagnostic methods are bedside echocardiography, D-dimer measurement and CTPA. Their use, however, depends on the clinical probability of PE. The most recognized and best-validated tools for estimating this probability are the Wells and Geneva scales. Both are recommended by the 2019 ESC guidelines on the management and treatment of PE (Konstantinides et al., 2020). It is worth noting that the Geneva scale uses two systems for determining clinical probability: a three-level score (low, intermediate and high) and a two-level score (PE-unlikely and PE-likely). Another term worth knowing in the context of PE diagnosis is “high-risk PE”, which is defined by the ESC guidelines as PE in hemodynamically unstable patients (Konstantinides et al., 2020). The simplified rules for using D-dimer measurement, echocardiography and CTPA according to clinical presentation and probability are shown in Figure 2.

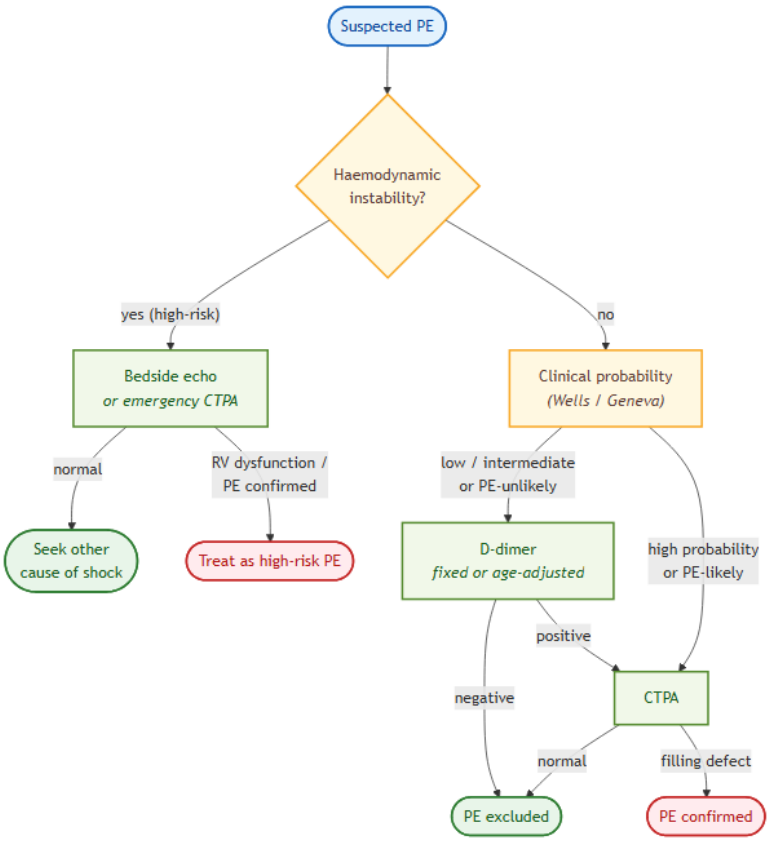

Figure 2 Diagnosing PE, based on (Konstantinides et al., 2020)

Once PE is confirmed, it is especially important to perform risk stratification, as this guides further management (described briefly later in the text). Depending on the score obtained with the Pulmonary Embolism Severity Index (PESI), the mortality risk is classified as very high, high, moderate, low or very low. Taking other factors into account (such as hemodynamic instability, RV dysfunction and elevated cardiac troponin), the early mortality risk of PE is divided into high, intermediate (intermediate-high and intermediate-low) and low (Konstantinides et al., 2020). It is important not to confuse these categories with the aforementioned high, intermediate and low clinical probability of PE.

3.3 Causal treatment

Treatment of PE, on the one hand, depends on the clinical presentation (the risk of early mortality) and, on the other hand, consists of treating the underlying cause (anticoagulation, thrombolysis and embolectomy) and supporting oxygenation and hemodynamics (Shah et al., 2022). This article focuses on the second aspect; however, causal treatment is addressed first.

Anticoagulation is the mainstay of most PE treatment (Shah et al., 2022). The 2019 European Society of Cardiology (ESC) Guidelines on Acute Pulmonary Embolism recommend initiating this therapy without delay in every case of PE (class I, level C). However, the risk of early death, as well as existing contraindications, can affect the choice between unfractionated heparin (UFH), low-molecular-weight heparin (LMWH), direct oral anticoagulants (DOACs) and vitamin K antagonists (VKAs). Generally, UFH is preferred in high-risk PE (I, C). Anticoagulation should be administered in cases of intermediate or high clinical probability of PE while the diagnostic workup is in progress (I, C). When parenteral anticoagulation is started, LMWH or fondaparinux is preferred over UFH (I, A), and a DOAC is preferred in all other cases unless there are contraindications (I, A) — the most important of which are renal failure, pregnancy, lactation and antiphospholipid antibody syndrome, in which case VKAs or LMWH should be used (Konstantinides et al., 2020).

An important complement to anticoagulation is reperfusion treatment (systemic thrombolysis and surgical or percutaneous embolectomy). Thrombolytic therapy improves RV hemodynamics compared with UFH alone. Surgical embolectomy and systemic thrombolysis appear to have similar efficacy, defined as 30-day mortality, but thrombolysis is associated with a higher risk of stroke and reinterventions. It is, however, much better documented (Konstantinides et al., 2020). A more detailed list of recommendations is provided in Table 2.

Table 2 ESC recommendations on reperfusion treatment in PE. The class and level of the recommendation are indicated in parentheses (Konstantinides et al., 2020)

Reperfusion strategy	**Indications**

Systemic thrombolysis	Recommended in high-risk PE (I, B) Recommended in low/moderate PE in case of hemodynamic deterioration (I, B) Not recommended as routine treatment in low/moderate PE (III, B).
Surgical embolectomy	Recommended in high-risk PE when systemic thrombolysis is contraindicated or failed (I, C) For consideration in low/moderate PE in case of hemodynamic deterioration as an alternative to systemic thrombolysis (IIa, C)
Percutaneous embolectomy	For consideration in high-risk PE when systemic thrombolysis is contraindicated or failed (IIa, C) For consideration in low/moderate PE in case of hemodynamic deterioration as an alternative to systemic thrombolysis (IIa, C)

Another strategy, this time straddling the line between causal treatment and prevention, is the use of inferior vena cava (IVC) filters. The facts that this method is associated with common and potentially serious complications (e.g. perforation of the venous wall) and that it is not well documented are reflected in the lower class and level (IIa and C) of the recommendation for its use. Indications include recurrent PE despite therapeutic anticoagulation and absolute contraindications to anticoagulation (Konstantinides et al., 2020).

The concept of multidisciplinary PE teams is also worth addressing. PE response teams (PERTs), which bring together specialists from different fields, are intended to accelerate decisions and improve their effectiveness. The ESC recommends considering the establishment of such teams, depending on the resources and expertise available in the hospital (IIa, C). The idea is gaining increasing recognition (Konstantinides et al., 2020). Data from the literature suggest that this approach can yield tangible benefits. A retrospective study conducted at Baylor Scott & White Heart Hospital in Plano showed non-significant trends toward reduced in-hospital (2% vs 5%, p = 0.2), 30-day (2% vs 8%, p = 0.06) and 12-month (7% vs 8%, p = 0.7) mortality when a PERT was activated. It also halved the median number of days spent in the ICU and reduced the rates of cardiac arrest (2% vs 7%, p = 0.09), vasopressor requirement (9% vs 18%, p = 0.02) and major bleeding (2% vs 7%, p = 0.09) (Russell et al., 2023). A cohort study conducted at the University Medical Center of the Johannes Gutenberg University Mainz (PERT Mainz), comparing patients after and before the implementation of a PERT using univariate regression analysis, found that all-cause mortality (OR 0.37 [95% confidence interval (CI) 0.18–0.77]; p = 0.009) was reduced and that PE-related mortality (OR 0.54 [95% CI 0.24–1.18]; p = 0.121) showed a downward trend in the latter group (Sagoschen et al., 2024). The multidisciplinary composition of such teams, including intensivists alongside interventionists (radiologists, cardiologists, cardiac surgeons), reflects the fact that the management of PE is based not only on causal therapy but also on hemodynamic support, which is the focus of this article.

3.4 Supportive management

Supportive care in ARVF in the course of PE consists of managing preload and afterload while enhancing cardiac contractility (fluid administration, vasopressors, inotropic support, pulmonary vasodilators), managing hypoxemia (oxygenation, ventilation) and providing mechanical circulatory support (MCS). It is also crucial to identify the correct cause of RVF, because both the causal and the hemodynamic approaches differ (Giannakoulas et al., 2025; Manca et al., 2025; McGuire et al., 2024).

Oxygenation and ventilation management

Patients with PE often present with hypoxemia, so oxygen therapy is commonly required. It is initiated when arterial oxygen saturation falls below 90%, and the therapeutic target is to keep this parameter above that value. The main underlying mechanism is ventilation-to-perfusion mismatch, although in most cases supplemental oxygen therapy alone is sufficient to overcome it. High-flow oxygen therapy (HFT, i.e. nasal high-flow oxygen therapy) is preferred (Konstantinides et al., 2020; McGuire et al., 2024; Sorathia et al., 2025). Severe hypoxemia (or hypercapnia) refractory to this form of therapy may be explained by a right-to-left shunt through a foramen ovale or another atrial septal defect (Konstantinides et al., 2020). Other available methods are non-invasive ventilation (NIV) and invasive ventilation (intubation). Because of the adverse hemodynamic effects described below, invasive mechanical ventilation should be avoided and used only when adequate oxygenation or ventilation cannot be achieved with NIV (Konstantinides et al., 2020; McGuire et al., 2024; Sorathia et al., 2025).

The simplest methods available when oxygen therapy is indicated are conventional supplemental oxygen therapy (low-flow) and, as an alternative to it, HFT. In recent years HFT has gained popularity and become an important resource in respiratory units. This method allows the generation of a gas flow (up to 70 L/min) with an FiO2 of 21 to 100%. It also generates a slight positive end-expiratory pressure (PEEP), enhancing CO2 washout and clearance of secretions, and overall reducing respiratory effort. The effectiveness of this method is increasingly being highlighted in respiratory failure, in weaning from NIV in COPD and even as a proposed alternative to NIV in patients with poor tolerance (González Ramos et al., 2024; Turnbull, 2022). These characteristics are reflected in studies comparing high- and low-flow oxygen therapy. A small randomized controlled trial (RCT) conducted in Erzurum (Turkey) in 2021 compared the improvement in oxygenation obtained with HFT and with low-flow therapy in 58 patients with PE and acute hypoxemic respiratory failure. Whereas at baseline PaO2 and SpO2 were comparable between the two groups, at 1 h both were significantly higher in the HFT group. Although there was no analysis of hard endpoints such as mortality, the trial suggests the superiority of HFT over low-flow therapy in improving blood-gas parameters and, according to the authors, that it can be used safely as primary treatment in PE (Aksakal et al., 2021). Trials on HFT in heart failure secondary to PE are, however, rare. Nevertheless, some indirect evidence comes from acute heart failure (AHF), in which this kind of therapy is more beneficial than conventional therapy. A large systematic review with meta-analysis published in 2023, including 16 studies and 1333 patients, showed that HFT (nasal high-flow oxygen

therapy) reduced the intubation rate (odds ratio [OR] 0.29, 95% CI 0.14–0.58, $p < 0.05$) and hospital stay (standardized mean difference [SMD] −0.73, 95% CI −0.99 to −0.47; $p < 0.00001$) and improved oxygenation parameters such as PaO2 (SMD 0.88, 95% CI 0.70–1.06, $p < 0.00001$) and SpO2 (SMD 0.70, 95% CI 0.34–1.06, $p = 0.0001$) compared with low-flow therapy. Moreover, there was no difference in this respect between HFT and NIV (Yan et al., 2023). In addition to its benefits over conventional supplemental oxygen therapy, the value of HFT in ARVF in the course of PE lies in the fact that mechanical ventilation (NIV or intubation) with positive pressure carries a risk of unfavorable hemodynamic effects. By increasing intrathoracic pressure, these methods reduce venous return and RV preload and consequently increase both pulmonary vascular resistance (PVR) and RV afterload. They are therefore associated with a risk of RV overload and ischemia, which applies to HFT to a much lesser extent (Giannakoulas et al., 2025; Konstantinides et al., 2020; McGuire et al., 2024). Taking all of this into consideration, HFT appears to be a safe and appropriate primary oxygen therapy, better than conventional therapy and potentially safer than NIV (provided, of course, that the hypoxemia does not require the initiation of ventilation).

As mentioned above, intubation should be performed only when necessary (when HFT or NIV is not sufficient or when patients cannot tolerate these methods). This is important not only because of the adverse hemodynamic effects but also because of the frequent hemodynamic instability and the patient's sensitivity to general anesthetics (Konstantinides et al., 2020; McGuire et al., 2024; Pérez-Nieto et al., 2023). An experimental study published in 2025 showed how invasive mechanical ventilation can affect RV afterload. In this study, 11 pigs underwent induction of PE. Their PAP was then measured under different conditions: various levels of PEEP, minute ventilation and FiO2, and bicarbonate-induced alkalosis. Mean PAP was significantly reduced by decreasing PEEP, increasing minute ventilation, alkalosis and increasing FiO2 (which is consistent with the vasodilatory properties of oxygen described later in the text) (Baltsen et al., 2025). In this context, the recommendations to keep the tidal volume at 6 mL/kg, the end-inspiratory plateau pressure at a maximum of 30 cm H2O and PEEP as low as possible (0 cm H2O if tolerated) are more reasonable (Konstantinides et al., 2020; Pérez-Nieto et al., 2023). Induction of general anesthesia carries a risk of hemodynamic deterioration. This is why drugs such as propofol should be avoided or used with great caution, and etomidate or ketamine are preferable. The most important measure, however, is to maintain intravenous

access and access to vasoactive drugs in order to keep the mean arterial pressure (MAP) adequate (Konstantinides et al., 2020; McGuire et al., 2024; Pérez-Nieto et al., 2023).

Fluid administration

Fluid optimization is widely discussed in numerous studies on RVF. Appropriate fluid administration is crucial for right ventricular function because of the aforementioned physiological, pathophysiological and anatomical circumstances (the dependence of the RV on preload, together with a simultaneous reduction in its tolerance to preload due to pressure overload). Excessive preload (especially in a pressure-overloaded RV) leads to RV distension, increased wall tension, tricuspid regurgitation, worsening systemic congestion and reduced LV filling (Giannakoulas et al., 2025; Luk et al., 2025). On the other hand, patients with ARVF have diastolic RV dysfunction, which means that a higher-than-normal RAP is needed to maintain optimal CO. Fluid therapy is therefore aimed at achieving an optimal balance (Manca et al., 2025). This is made all the more difficult by the fact that hypotensive patients with RVF often present with a combination of pressure and volume overload rather than a pure form of either (Dodi & Jacobs, 2025).

The tools available to clinicians for assessing the appropriateness of a fluid bolus are venous excess ultrasound (VExUS) and CVP (Giannakoulas et al., 2025; Luk et al., 2025; Manca et al., 2025). The easiest to interpret appears to be CVP measurement, because of the ease of reading the result and the ability to set a cut-off threshold. Numerous studies have shown higher mortality in critically ill patients with a CVP > 10 mmHg and worsening renal function with increased mortality in cardiac patients with a CVP > 6 mmHg (Manca et al., 2025). A frequently cited target of fluid therapy is a CVP between 8 and 12 mmHg (Guarnieri et al., 2025; Luk et al., 2025; Manca et al., 2025); however, this value, derived from a trial by Rivers et al., lacks strong supporting evidence. Recent studies suggest that the CVP value alone is a poor indicator of fluid responsiveness (Luk et al., 2025). Nevertheless, any sudden rise in CVP from baseline during rapid fluid administration should draw attention, as continuing the bolus is then unlikely to improve SV (Giannakoulas et al., 2025).

A small intravenous bolus (250 mL) is justified if the preload is suspected to be insufficient, but the volume status and CO must be monitored (Luk et al., 2025). The ESC recommends using saline or Ringer's lactate in a volume of less than 500 mL over 15–30 min (Konstantinides et al., 2020). Monitoring the latter falls within the scope of hemodynamic monitoring, whereas for the former, combining CVP with VExUS offers an attractive alternative to measuring CVP on its own (Dodi & Jacobs, 2025; Giannakoulas et al., 2025). Although point-of-care ultrasound (POCUS) enables the assessment of variables such as inferior vena cava dilatation and absent respiratory variation, it has poor diagnostic accuracy on its own (Dodi & Jacobs, 2025). The VExUS score, which integrates pulse-wave Doppler measurements of the hepatic, portal and renal veins, provides quantifiable and more objective variables for assessing venous congestion and fluid status. The score (grades 0–3) correlates with RAP (Longino et al., 2023), with longer ICU stays and a greater need for renal replacement therapy (RRT) (Dodi & Jacobs, 2025), and even with mortality in patients with decompensated heart failure (Landi et al., 2024). A low CVP (< 8–12 mmHg) and no signs of congestion on VExUS indicate that fluid administration (ideally with reevaluation and CO monitoring) may be reasonable. Conversely, a situation in which CVP exceeds 8–12 mmHg (or pulmonary artery wedge pressure, PAWP) and VExUS shows mild or severe congestion indicates a need for fluid removal (Giannakoulas et al., 2025). This is all the more important because renal blood flow is often decreased in ARVF and diuretic resistance is common, so early and aggressive diuretic therapy is frequently needed (Dodi & Jacobs, 2025). For patients who are unresponsive to the addition of a second diuretic, rapid escalation to RRT is recommended (Giannakoulas et al., 2025).

Vasopressors and inotropes

Adequate fluid administration does not always maintain blood pressure in PE. In such situations, vasopressors may be required to augment cardiovascular function (Ajah, 2024). The Heart Failure Association and the Working Group on Pulmonary Circulation and Right Ventricular Function of the ESC list norepinephrine (0.2–1.0 µg/kg/min) and dobutamine (2–20 µg/kg/min) as agents that are often needed, in parallel with or as a bridge to causal treatment (Harjola et al., 2016). The ESC guidelines, however, describe norepinephrine as a drug whose use should be limited to patients with cardiogenic shock, in whom it improves RV perfusion and inotropy, with the important caveat that peripheral perfusion may deteriorate in the event of excessive vasoconstriction. The indication for dobutamine is a low cardiac index with

maintained normal blood pressure (because of the risk of vasodilatation caused by this agent, which, alongside tachyarrhythmias, is its main side effect) (Konstantinides et al., 2020).

The use of vasopressors and inotropes is intended to keep the MAP at a level sufficient to sustain peripheral perfusion (Dodi & Jacobs, 2025; Giannakoulas et al., 2025; Luk et al., 2025; Manca et al., 2025). In terms of hemodynamic targets, a MAP of 65 mmHg is considered sufficient for adequate organ perfusion (Dodi & Jacobs, 2025; Luk et al., 2025). However, recent studies suggest that some adjustment of this threshold may be beneficial. For example, a retrospective study conducted in Connecticut (USA) showed that patients with a dynamic MAP target defined as 60 + CVP had lower in-hospital mortality (16.7% vs 43.3%, $p = 0.047$) and a lower incidence of acute kidney injury (AKI) (3.3% vs 73.3%, $p < 0.001$) than patients treated with a static target of 65 or 70 mmHg (Moallem et al., 2023). The size of the study and of the control group ($n = 30$ in each) may raise doubts about its reproducibility, but the observation shows that some kind of individualized approach to hemodynamics might be advantageous. More details on hemodynamic targets are given later in the text.

Another agent that may seem promising is levosimendan. Its effects are defined by two actions: it acts as a calcium sensitizer and it opens ATP-sensitive potassium channels. The first is responsible for the unique effect of levosimendan, namely enhancing cardiac contractility without increasing oxygen demand. The second causes vasodilatation (by opening these channels in smooth muscle) and provides cardioprotection (by activating mitochondrial channels). For these reasons, levosimendan appears to be an attractive agent in cardiogenic shock, and potentially also in the course of pulmonary embolism (Susilo et al., 2024). It has a better inotropic effect on the RV than dobutamine (Luk et al., 2025) and reduces PVR through vasodilatation, as dobutamine can also do but not to the same extent (Giannakoulas et al., 2025; Luk et al., 2025; Manca et al., 2025). Moreover, dobutamine at the doses that improve CO best (5–15 μg/kg/min) does not affect PVR at all (Giannakoulas et al., 2025). In this context, levosimendan appears particularly promising, because it not only enhances cardiovascular function to maintain systemic perfusion but also allows the afterload of the RV to be managed. However, its side effects — systemic hypotension and tachyarrhythmias (Giannakoulas et al., 2025) — are a significant limitation. In PE, modulating the increased afterload of the RV should be of great benefit, but levosimendan (administered systemically) used to this end has not

shown clear clinical benefit despite promising experimental results, as emphasized by the ESC guidelines (Konstantinides et al., 2020). The reason for this may be that the aforementioned reduction in systemic vascular resistance (SVR) is particularly detrimental to blood flow to the right ventricle, which — in cases where inotropes must be used during PE — is most often dilated and has an increased oxygen demand. As described in the section on pathophysiology, the RV is perfused in both systole and diastole, and the relationship between blood pressure and wall stress is crucial to the function of the wall.

There is one more drug that could be considered as an afterload-management agent: milrinone. It is a phosphodiesterase-3 inhibitor with a hemodynamic profile similar to that of levosimendan (Giannakoulas et al., 2025; Manca et al., 2025). It lowers both RV and LV filling pressures and causes tachyarrhythmias less often than dobutamine, but it has a longer half-life (Giannakoulas et al., 2025).

Inotropes with a greater impact on the pulmonary circulation (lowering PVR) might offer greater benefit when RVF is secondary to LV failure (Giannakoulas et al., 2025). However, in cases of pressure overload, e.g. pulmonary hypertension (pre-capillary overload, similar to PE), hypotension as a side effect significantly limits their use, as stated earlier (Giannakoulas et al., 2025).

Pulmonary vasodilators

In the previous subsection, the concept of modulating RV preload was introduced. As PE is by definition an RV pressure overload (Giannakoulas et al., 2025), this idea is particularly interesting for managing this cause of ARVF. The side effect of SVR reduction described above, which is harmful for the ischemic RV in PE, could potentially be reduced by changing the route of administration of the vasoactive agents. More importantly in this context, some dilators, e.g. levosimendan, may have a higher efficacy (maximal dilatation potential) on arteries other than the pulmonary ones (e.g. the mesenteric arteries), even though they are more specific for the latter (Bedo & Grignola, 2024).

Although vasodilators are a widely recognized treatment in pulmonary hypertension (PH), in acute PE the data are limited (McGuire et al., 2024). The best known of these agents used in

PH is inhaled nitric oxide (iNO). Other medications, such as inhaled epoprostenol, iloprost, treprostinil, milrinone and levosimendan, may be similar to iNO (Liu et al., 2021).

The simplest vasodilator, not mentioned above but widely used in the treatment of PE, is oxygen (McGuire et al., 2024). A pilot randomized trial comparing patients with stable PE, without hypoxemia and with an enlarged RV (assessed by echocardiography), who received either anticoagulation plus supplemental oxygen or anticoagulation alone, showed an improvement in echocardiographic findings after 48 h in the former group. A normal RV size was non-significantly more frequent in the patients who received oxygen (42.4% vs 21.6%, $p = 0.08$). In this group there was also a significant improvement in the RV-LV ratio between baseline and the values measured after 48 hours of treatment (1.28 ± 0.28 vs 1.01 ± 0.16, $p < 0.001$). However, a significant reduction in this parameter was also observed in the anticoagulation-only group (1.21 ± 0.18 vs 1.08 ± 0.19, $p < 0.01$). As their authors stated, this analysis did not demonstrate that oxygen therapy alone improves right ventricular echocardiographic parameters in PE; it supports such a hypothesis and the need for further research in this area (Barrios et al., 2024). In the context of ARVF in PE, the vasodilatory properties of oxygen are more of a curiosity, since most patients will require supplementation anyway because of hypoxemia. It is, however, a low-risk procedure with potential therapeutic benefit (McGuire et al., 2024).

It is worth mentioning that a randomized, double-blind controlled trial of oxygen as a vasodilator is to be conducted at the Massachusetts General Hospital (USA). The primary outcome of the study will be the change in pulmonary artery systolic pressure with and without supplemental oxygen. The secondary endpoints will be echocardiographic and metabolic measures. Forty patients in the study group will alternate for 180 min between 21% and 60% FiO2, thus acting as their own control group. A study designed in this way should make it possible to demonstrate the vasodilatory properties of oxygen (Lyhne et al., 2024).

As described above, the most recognized agent used as an inhaled pulmonary vasodilator in PH is iNO. It is used, with varying frequency, in conditions such as persistent pulmonary hypertension of the newborn, bronchopulmonary dysplasia, PH in congenital heart disease,

cardiothoracic surgery and extracorporeal membrane oxygenation (ECMO), acute respiratory distress syndrome (ARDS) and COPD (Liu et al., 2021). As cited in a review article (Ajah, 2024), iNO improved pulmonary and systemic blood pressures and gas exchange in patients with massive PE. A recent small (n = 14) randomized trial showed that iNO reduces dyspnea and improves exercise tolerance in patients after pulmonary embolism (Phillips et al., 2026). A multicenter randomized controlled trial conducted in 2019 sought to assess the effectiveness of iNO in improving hemodynamics in patients with PE and RV dysfunction without systemic hypotension. Patients were treated with standard anticoagulation and either oxygen with nitrogen (placebo, n = 38) or oxygen plus NO (n = 38) for 24 h. The primary endpoint was a normal RV on echocardiography together with a plasma troponin T concentration < 14 pg/mL. iNO showed a numerically higher but statistically non-significant rate of achieving this (13% vs 24%, respectively, p = 0.375). However, 29% more patients in the iNO group had resolution of RV hypokinesis or dilatation after 24 h of treatment (p = 0.01, Cochran's Q test) (Kline et al., 2019). iNO is a promising and probably the best-known pulmonary vasodilator in PE, one that could aid rehabilitation or improve hemodynamics in selected patients, but its effectiveness remains unclear.

Other popular pulmonary vasodilators used in PH are aerosolized prostacyclin and its analogs (e.g. iloprost, epoprostenol, treprostinil) (Liu et al., 2021). The literature on their use in PE is rather limited, consisting mostly of uncontrolled case series and small RCTs with results that are not particularly promising. A small randomized trial conducted in 2010 reported that patients with PE and RV overload who received epoprostenol (n = 7) had no improvement in right ventricular dilatation compared with placebo (n = 7). The difference in change from baseline between the two groups was −8.0% in favor of epoprostenol, but the 95% confidence interval was −21.2 to 5.2 (p = 0.21) (Kooter et al., 2010). In this study, however, the medication was administered intravenously. McGuire et al. describe two case series focused on iloprost in intermediate-risk PE. The patients' RV status improved over 3 to 90 days; however, the lack of a control group makes it impossible to assess the actual contribution of iloprost to this improvement (McGuire et al., 2024). Inodilators (levosimendan and milrinone) also act as pulmonary vasodilators. They were discussed in the section on vasoactive and inotropic agents.

Knowledge about pulmonary vasodilators in PE is still insufficient, which is reflected in the ESC guidelines describing only inhaled NO as a solution potentially able to improve hemodynamics, but with neither efficacy nor safety validated (Konstantinides et al., 2020).

Mechanical circulatory support (MCS)

MCS in PE includes direct and indirect RV bypass. The indirect form is veno-arterial ECMO (VA-ECMO), in which blood is drawn from the caval vein and returned to the descending aorta. Direct bypass uses mechanical pumps (e.g. Impella RP, or ProtekDuo with an additional oxygenator) whose ends are located in the atrium and the pulmonary artery. This type of therapy should serve as a bridge to the restoration of normal right ventricular function or to lung transplantation. It should be discontinued 5–10 days after it is started (Giannakoulas et al., 2025). In cases where the LV is overloaded after implantation of VA-ECMO (LV distension), it is also possible to use an Impella CP for additional LV support (the ends of the pump are placed in the LV and the aorta) (Mentzel et al., 2025). The 2019 ESC guidelines state that the use of MCS may be helpful in patients with high-risk PE and circulatory collapse or cardiac arrest, but that it involves potential complications and that its effectiveness depends on the experience of the center. They focus mostly on VA-ECMO, mentioning only a few cases of use of the Impella catheter. Moreover, in 2019 there were no randomized controlled trials (RCTs) analyzing the effectiveness of VA-ECMO (Konstantinides et al., 2020). This situation has not changed to this day, and knowledge of VA-ECMO is based on case series, observational studies and meta-analyses of these.

Table 3 Comparison of MCS methods in PE, based on (Konstantinides et al., 2020; McGuire et al., 2024; Mentzel et al., 2025)

Method	VA-ECMO	Impella RP	ProtekDuo
Principle of operation	Indirect RV bypass — blood drawn from the caval vein and	Direct RV bypass — blood drawn from the right atrium and	Direct RV bypass (like the Impella RP) with additional oxygenation

	pumped into the descending aorta	pumped into the pulmonary artery	
Hemodynamic effect	Increased LV afterload; decreased RAP and LV stroke volume. Potentially a mild decrease in native CO.	Increased RV and PA pressure. Increased LVEDP, LV stroke volume and native CO. Decreased RAP.	Increased RV and PA pressure. Increased LVEDP, LV stroke volume and native CO. Decreased RAP.
Advantages	The most widely used MCS in patients with PE. Can be deployed outside the operating room or catheterization laboratory.	Only venous access is needed — no surgical procedure required.	Only venous access is needed — no surgical procedure required. Provides additional oxygenation.
Disadvantages and risks	Elevated risk of bleeding and limb ischemia.	May be ineffective in severely obstructed pulmonary arteries due to lack of additional oxygenation. Elevated risk of pulmonary hemorrhage.	Elevated risk of pulmonary hemorrhage.

One example of such a study is a retrospective analysis of patients with PE treated with pulse thrombolysis complemented by ECMO. Twenty-two of the 29 patients included in the study survived to hospital discharge, while the 90-day mortality was 24.1%; 6 patients died from refractory shock. The RV/LV ratio decreased from 1.31 ± 0.17 to 0.92 ± 0.11 in the patients who survived (Dumantepe & Ozturk, 2022). In 2021, however, a systematic review with meta-analysis of survival rates in patients with PE treated with ECMO was published. This paper includes data from 29 observational studies with a total of 1947 patients (VA-ECMO n = 1138, control n = 809). The meta-analysis showed no difference in short-term survival between the

groups (relative risk [RR] 0.91, 95% CI 0.71–1.15). Moreover, the study revealed that in patients aged > 60 years survival in the ECMO group was lower than in younger patients (RR 0.72, 95% CI 0.52–0.99) and that surgical embolectomy combined with ECMO was associated with a higher chance of survival than ECMO alone (RR 1.96, 95% CI 1.39–2.76). However, the paper was based on numerous heterogeneous observational studies with small samples, which is a real limitation. Even though the study provided some low-quality evidence that VA-ECMO may be associated with higher short-term survival rates, this is still insufficient and points to the need for prospective studies (Karami et al., 2021).

The literature data on direct RV bypass MCS are even more limited and, as with ECMO, are based on individual cases or case series. A recent literature review of case reports of Impella RP use in massive PE points, however, to the potentially positive effects of this device. The initiation of MCS was associated with hemodynamic improvement: for example, mean systolic blood pressure increased from 94.5 ± 12.62 mmHg to 142.67 ± 23.67 mmHg and mean diastolic blood pressure increased from 65 ± 10.66 mmHg to 89 ± 11.4 mmHg. Heart rate, on the other hand, decreased from 107.62 ± 11.58 to 85.8 ± 11.8 bpm. This, combined with an improvement in cardiac index (from 1.67 ± 0.46 L/min/m2 to 2.8 ± 1.2 L/min/m2) and a 100% survival rate, may suggest that the Impella RP is a valuable treatment option in circulatory-decompensated PE (Pandey et al., 2025). However, the methodology of this study (the lack of a control group and of hard endpoints) prevents any stronger statement from being made on this matter.

MCS remains a rapid bridging option that has not been studied in sufficient depth, although it offers benefits, as emphasized in the 2019 ESC guidelines. These benefits depend, however, on combining MCS with surgical embolectomy, on the level of expertise of the team and on an appropriate duration of therapy (Konstantinides et al., 2020). Every decision to initiate or wean MCS should be made by a multidisciplinary team after an individual assessment of each case on its own merits (Mentzel et al., 2025).

4 Discussion

Venous thromboembolism is the third most common cardiovascular syndrome, and pulmonary embolism, which is one of its forms, may account for as much as 10% of in-hospital deaths. Pulmonary embolism is the most frequent cause of acute right ventricular failure, which is

crucial for the prognosis in PE, as it leads to hemodynamic and respiratory deterioration and is associated with higher mortality rates (Giannakoulas et al., 2025; Konstantinides et al., 2020; McGuire et al., 2024; Shah et al., 2022). Effective management of ARVF in PE requires a dual approach: causal treatment (anticoagulation, thrombolysis, embolectomy) and temporary organ support (RV preload and afterload optimization, and hemodynamic, oxygenation and respiratory management). The two are closely connected, as causal treatment addresses the underlying obstruction while organ support maintains viability long enough for that treatment to be delivered and to take effect.

Despite the interconnection of these two approaches, the evidence regarding the effectiveness and safety of the specific approaches and methods within each varies widely. The 2019 ESC Guidelines for the diagnosis and management of acute pulmonary embolism (Konstantinides et al., 2020) are one of the most important sources for analyzing such evidence for PE specifically. Whereas causal treatment is accompanied by extensive commentary, including on large-scale studies devoted to these methods, supportive management is discussed in much less detail and in more general terms. The section on anticoagulation cites diverse and large studies (including prospective cohort studies, RCTs with up to thousands of participants and meta-analyses with up to twenty thousand patients). The evidence base for reperfusion treatment is somewhat more uneven in this respect, with a more solid foundation for systemic thrombolysis than for mechanical methods. Nonetheless, in this section of the ESC guidelines we can find numerous studies, including RCTs with several dozen patients, retrospective case-control studies with over 2000 patients and meta-analyses with up to several hundred patients. As for supportive management, the summary of oxygen and ventilation is based on scientific correspondence and a case report. The recommendations on fluid therapy, vasopressors and inodilators in the ESC guidelines refer to experimental animal studies, experimental studies without a control group conducted on a dozen or so patients, and even a narrative review. The issue of vasodilators mainly concerns iNO and is based on a case series involving several patients and on a systematic review that includes no RCTs. The recommendations on MCS refer to literature reviews and case series. This situation is reflected in the levels and grades of the recommendations for the individual interventions. The recommendations on anticoagulation are classified as Class I (Level A for the choice of therapy and Level C for immediate initiation in moderate- and low-risk PE and for the choice of UFH in high-risk PE). The recommendations on systemic thrombolysis are classified as Class I, Level B for PE of any risk level. For mechanical

thrombectomy methods, the grades are lower, ranging from IIa to IIb depending on the indication and method, and all carry a Level C, reflecting the more limited and less robust evidence. Organ support methods, however, have a maximum of Class IIa, Level C for the use of norepinephrine or dobutamine, or Class IIb, Level C when ECMO is used in conjunction with surgical thrombectomy. Finally, fluid therapy and vasodilators are not included in the recommendations with specific levels or classes at all. The guidelines clearly focus primarily on causal treatment. At present, there are no dedicated guidelines for ARVF in PE in the context of organ support, or even for PE in general. Such guidelines may be supplemented by Contemporary Management of Acute Right Ventricular Failure: A Statement from the Heart Failure Association and the Working Group on Pulmonary Circulation and Right Ventricular Function of the European Society of Cardiology (Harjola et al., 2016). However, this is an expert opinion on a broader issue (ARVF in general), which in itself indicates a lower level of evidence for organ support methods in the condition in question.

In this context, several of the gaps in knowledge and evidence observed in this review become more understandable. The section on oxygenation and respiration was based on several literature reviews (McGuire et al., 2024; Sorathia et al., 2025) but also on a large systematic review with meta-analysis (Yan et al., 2023). The latter is particularly valuable, as its results suggest that HFT is a better and safer method of oxygenation than low-flow therapy and could even be preferable to NIV when sufficient. However, it was dedicated to AHF, which makes its applicability to PE only indirect. The only study found on HFT in PE was rather small, but it was an RCT (Aksakal et al., 2021). It is a highly relevant trial, but the lack of hard endpoints (such as mortality), the sample size and the absence of other similar studies keep the issue open, especially regarding the comparison of HFT with NIV and the timing of the transition between these modalities. A similar situation applies to mechanical ventilation. Apart from one experimental animal study (Baltsen et al., 2025), no PE-specific trial was found. The recommendations on this topic are based on physiological knowledge and analogies with similar conditions (Giannakoulas et al., 2025; Konstantinides et al., 2020; McGuire et al., 2024). In terms of hemodynamic management, a wide range of studies is available for right ventricular failure, but far fewer specifically target PE. There is relative consensus on the selection of vasoactive and inotropic agents, with the recognition that norepinephrine or dobutamine should be preferred despite the potential benefits of levosimendan or milrinone, which, however, carry a higher risk of hypotension (Guarnieri et al., 2025; Konstantinides et al., 2020; McGuire et al.,

2024; Susilo et al., 2024). Nevertheless, the inhaled use of these latter two compounds, as well as of other vasodilators administered by this route to reduce PVR, remains unclear and may be an attractive avenue for further research in this field. The issue of hemodynamic targets in ARVF in the course of PE is still a matter of research. A universal MAP of 65 mmHg is proposed as suitable in ARVF (Dodi & Jacobs, 2025). The CVP value, particularly when correlated with VExUS, is reported to be a valuable factor in the decision to continue fluid therapy or to induce diuresis (Dodi & Jacobs, 2025; Giannakoulas et al., 2025). Of particular interest here is the study by Moallem et al. (2023), which reported better outcomes, in the form of lower in-hospital mortality, in patients with a dynamic MAP target (CVP + 60). This suggests that an individualized approach to hemodynamic support in patients with ARVF due to PE may be beneficial — an approach that has not yet gained widespread attention in the literature on this topic and that may be a direction worthy of future research. Studies on MCS in ARVF are mostly case series (Dumantepe & Ozturk, 2022; Pandey et al., 2025), but one large meta-analysis was also found (Karami et al., 2021). It showed that age, the combination of ECMO with surgical embolectomy, and the type of PE are important for the outcome. This suggests that the use of MCS in PE is a complex issue that requires further study.

It should also be noted that this study has certain limitations. As it is a narrative review, the literature search was selective (although it was based on current guidelines and reviews), and there was no systematic search or evaluation of sources. The very design of the study means that works relevant or interesting to the topic may have been overlooked. Excluding papers published before 2016, on the one hand, ensures up-to-date information but, on the other hand, could result in the omission of important trials from that period.

5 Conclusion

The management of acute right ventricular failure in pulmonary embolism requires hemodynamic and respiratory support (including oxygen therapy, ventilation, fluid therapy, vasopressors, inotropes and MCS), which is an important addition to causal treatment. The two approaches are interdependent: organ support sustains the patient until causal treatment can take effect. Yet much of this supportive management still rests on extrapolated or low-quality evidence, and stronger, PE-specific data are needed to guide it.

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Author Contributions

Conceptualization: AU, MP, RP, KK

Methodology: AU, MW, JW, KK

Investigation: AU, KW, RP

Resources: AU, MW, WZ

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Data Availability Statement

Not applicable.

Conflicts of Interest

The authors report no conflicts of interest.

Declaration of the use of generative AI

During the preparation of this work, the authors used AI tools for the purpose of language editing and stylistic refinement to improve clarity and readability of the manuscript. After using this tool, the authors reviewed and edited the content as needed and take full responsibility for the accuracy, integrity and conclusions of the publication

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