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American Journal of Interventional Radiology

Review Article *Vascular Interventions*

Pulmonary artery saturation as a primary endpoint for mechanical thrombectomy

Andrew F. Talon[1], Suresh Uppalapu[1], Mariel Ma[2], Abdul Ahad Khan[1], Ramachandra Rao Sista[3], Karim El-Kersh[3], Manoj Mathew[4], Nafis Shamsid-Deen[3]

Departments of [1]Pulmonary/Critical Care and [2]Internal Medicine, University of Arizona College of Medicine, [3]Department of Pulmonary/Critical Care, Program Director of Lung Transplantation, University of Arizona College of Medicine, Banner Lung Institute, [4]Department of Pulmonary/Critical Care, Banner Lung Institute, Pulmonary Hypertension Center, [5]Department of Pulmonary/Critical Care, Veteran Affairs Medical Center, Phoenix, Arizona, United States.

***Corresponding author:**
Andrew F. Talon,
Department of Pulmonary/Critical Care, University of Arizona College of Medicine, Phoenix, Arizona, United States.

atalonmd@gmail.com

Received: 01 June 2024
Accepted: 27 July 2024
Published: 03 September 2024

DOI
10.25259/AJIR_26_2024

Quick Response Code:

ABSTRACT

As catheter-directed mechanical thrombectomies become more prevalent to treat intermediate-risk pulmonary embolisms (PE), an objective measure of efficacy and risk stratification is needed. Studies currently use indirect measures of afterload reduction, such as the right ventricle (RV)/left ventricle ratio and mean pulmonary pressure. They lack nuance in identifying patients who have reestablished RV compensation or right ventricular-pulmonary artery (PA) coupling. In this review, we discuss the potential benefits of monitoring the pulmonary artery saturation (SvO_2) for predicting the success of mechanical thrombectomy for intermediate-risk PE patients. As an index of cellular oxygen delivery, PA saturation has been established as a critical component of understanding complex shock states and hemodynamic instability. In addition, it can be readily measured in these patients during PA instrumentation, requiring minimal additional procedure time.

Keywords: Pulmonary embolism, Thrombectomy, Pulmonary artery saturation

INTRODUCTION

There is an expanding role for catheter-directed mechanical thrombectomy in the treatment of intermediate-risk pulmonary embolisms (PEs). Several clinical trials have been conducted on catheter-directed interventions in the context of PE in recent years. According to the American College of Chest Physicians, catheter-assisted mechanical thrombectomy is recommended when a patient's condition does not improve despite systemic thrombolytics, bleeding risk is high, or hemodynamic degeneration continues, which is likely to result in death.[1] Considering risks and carefully selecting candidates is therefore essential. A prospective interventional study (FlowTriever All-Comer Registry for Patient Safety and Hemodynamics (FLASH) Registry) in the field of PE has already demonstrated that catheter-assisted mechanical thrombectomy with the FlowTriever System (Inari Medical, Irvine, CA; NCT03761173) can be safely used to treat intermediate- and high-risk PE patients with immediate benefits in hemodynamics and symptoms.[2] A more aggressive treatment algorithm for patients with intermediate-risk PE has been suggested due to mechanical thrombectomy's strong safety profile, effective thrombus removal, and low mortality rates.[2] In addition, upfront catheter-directed therapies have been considered for patients with intermediate-risk PE because of its long-term morbidity, such as post-PE syndrome.[3] Adding

meaningful endpoints to future studies and strengthening safety data will help clarify catheter-directed therapies' role in PE management. At present, operator dependence on clot retrieval is influenced by hemodynamics, particularly pulmonary artery (PA) pressures and cardiac index (CI), as well as improvements in the right ventricle/left ventricle (RV/LV) ratios.[4] As a potential parameter in pre- and post-mechanical thrombectomy for the treatment of intermediate-risk PE, pulmonary artery saturation monitoring (SvO_2) is discussed in this review.

THE NUANCES OF RIGHT VENTRICULAR DYSFUNCTION

Intermediate-risk PE patients are a vastly heterogeneous group often presenting with a wide range of unpredictable outcomes. This is partly due to the limitations of the prognostic tools currently used to stratify patients. The current evidence-based methods include prognostic scores (e.g., PE Severity Index [PESI] and Bova) that rely upon biomarkers and radiographic metrics (e.g., RV/LV ratio) that have been most examined in studies evaluating the efficacy of thrombolysis but have had limited exploration in the context of catheter-directed therapies.[5] In addition, the European Society of Cardiology (ESC) further divides intermediate-risk and intermediate-high-risk PE based on echocardiographic RV dysfunction (RVD) or PESI class (III/IV/V) and elevated troponin levels.[6] A subgroup of these patients may benefit from catheter-directed therapies upfront based on this distinction between intermediate-risk and intermediate-high risk. Recent research has also shown that a subset of patients with an intermediate-risk PE, who are typically normotensive, are actually in a state of subclinical shock. Specifically, Mina *et al*. sought to assess low CI (<2 $L/min^{-1}/m^{-2}$) in PE patients at intermediate-risk enrolled in the FLASH registry and also changes in CI during mechanical thrombectomy.[2] Risk stratification was performed using simplified PESI and Bova scores. Regardless of the risk stratification method used, 20% of patients with PE in each of the low-risk tiers had low CI before thrombectomy.[2] RVD has been shown to be a key factor in negative clinical outcomes after acute PE.[7] From a prospective cohort of 209 hemodynamically stable patients, Grifoni *et al*. observed RVD in 31% of cases.[8] In those with RVD, 10% developed PE-related shock and 5% died.[8] From the international cooperative pulmonary embolism registry (ICOPER) registry, 1035 normotensive patients who underwent echocardiography within 24 h of diagnosis of acute PE were found to have higher mortality in 405 (39%) patients with RV hypokinesis, with an overall 30-day mortality rate of 16.3% compared to 9.4% in those without hypokinesis.[9] Echocardiography findings indicate that RVD encompasses a wide spectrum of severity, including RV enlargement and hypokinesis, flattening of the interventricular septum, increased tricuspid regurgitation velocity, and reduced RV contractility measured by tricuspid annular plane systolic excursion (TAPSE).[10] Research on PE and other RV afterload diseases like pulmonary arterial hypertension (PAH) has focused on RV response to afterload rather than surrogates for RV strain (RV/LV ratio), defining RV-PA coupling as a relationship between RV contraction and afterload [Figure 1].[11] Impaired RV-PA coupling has been associated with higher mortality in patients with chronic heart disease.[12] The RV is very sensitive to acute afterload changes, with a limited capacity for compensatory enhancement of contractility. To maintain RV-PA coupling during acute PE, RV contractility must increase in response to increased afterload. Inadequate improvement of RV contractility resulted in RVD in porcine models.[13]

Echocardiographic findings would benefit from a quantitative scoring system, though it would be challenging given the inherent limitations of RV imaging in acute situations. Further stratification of patients with RVD has been achieved by incorporating additional echocardiographic parameters beyond the RV/LV ratio such as TAPSE and PA systolic pressure (PASP). A Doppler-based echocardiographic measure of RV-PA coupling has been also proposed for mortality risk stratification in cardiac intensive care unit patients. Jentzer *et al*. found a lower risk of mortality among 4259 patients who had higher tricuspid annular systolic velocity/RV systolic velocity pressure ratios in hospital (adjusted OR 0.7 per each 0.1-unit greater value) and among hospital survivors at 1 year after admission (adjusted hazard ratio [HR] 0.8 per each 0.1-unit higher value).[12] The echocardiographic ratio of tricuspid annular plane systolic excursion/systolic pulmonary artery pressure ratio (TAPSE/PASP) has also been found to predict short-term adverse outcomes in acute PE and has been validated as a non-invasive surrogate of RV-PA coupling.[14] There was a statistically significant association between TAPSE/PASP and 7-day composite outcomes of death or hemodynamic deterioration (odds ratio [OR] = 0.028, 95% CI 0.010–0.087; $P < 0.0001$).[14] In acute PE, TAPSE/PASP cutoff values of 0.4 were found to be most predictive of adverse outcomes.[14] In pulmonary arterial hypertension (PAH) patients, the registry to evaluate early and long-term PAH disease management (REVEAL) echocardiographic risk score (REVEAL-ECHO) risk score, which combines various RV parameters (RV chamber enlargement, reduced RV systolic function, tricuspid regurgitation severity, and pericardial effusion), has been used for prognosis.[15] El-Kersh *et al.* found that the additional echocardiographic parameters of RV function provided more information on longer-term survival.[15] Likewise, the TAPSE/PASP ratio is included in PAH risk stratification guidelines published by the European Respiratory Society and ESC in 2022.[16] Non-invasive parameters used to stratify patients with pulmonary

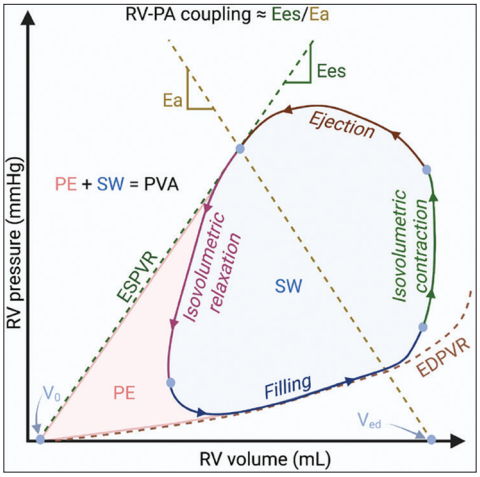

Figure 1: Right ventricle (RV) pressure-volume loop. Normally, end-systolic elastance (E_{ES}, ventricular contractility) increases to match effective arterial elastance (E_A, afterload). E_{ES}/E_A (RV-pulmonary artery [PA]) coupling is preserved. Diseased RV states such as acute pulmonary embolism increase afterload, E_{ES} cannot increase to match E_A (end-systolic pressure/SV), RV-PA unit becomes uncoupled. E_{ES}/E_A coupling is restored by catheter-directed interventions, RV ejection fraction, stroke volume/end-systolic volume, and diastolic function improves. Figure courtesy of Ro *et al.* with permission under the Creative Commons Attribution License (CC BY 4.0). (EDPVR: End diastolic pressure volume relationship, Ees: Right ventricular estance, SW: Stroke work, PE: Potential energy, PVA: pressure-volume area, ESPVR: end-systolic pressure–volume relationship.)

hypertension illustrate the complexity of RVD in acute PE, as current methods of risk stratification are limited.[17-19] Studies have shown that hemodynamic parameters that provide information on the complex interaction between afterload and RV adaptation/maladaptation are better predictors of clinical outcomes than anatomic parameters (RV/LV ratio) or afterload measurements (PA pressures). At present, mechanical thrombectomy catheters lack advanced functionality such as thermodilution channels, making it difficult to collect such data. The ready availability of PA SvO_2 measurement makes it an optimal surrogate with the consensus that balloon-directed right heart catheterization (RHC) is the gold standard of RV hemodynamic evaluation.

WHY MEASURING PA PRESSURES IS NOT ENOUGH

An elevated mean PA pressure (mPAP) >20 mmHg after catheter-directed mechanical thrombectomy has been associated with higher PE-attributable mortality.[20] However, limited information is provided by an isolated mPAP. Lyhne *et al.* found that RV afterload partially normalized

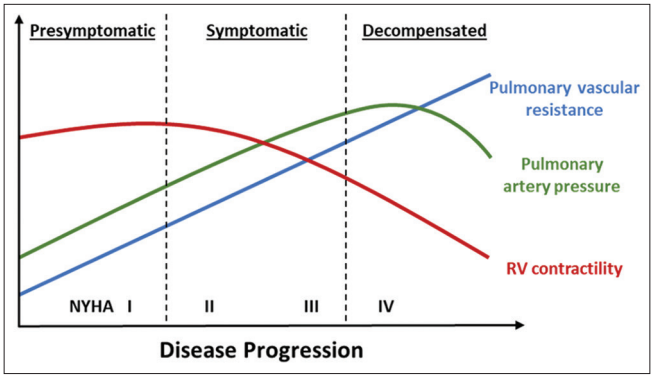

Figure 2: Progression of disease in patients with heart failure and pulmonary hypertension. When the right ventricle is unable to generate adequate pressure, the mean pulmonary artery pressure can decline and appear falsely better. Figure courtesy of Mariel Ma, MD; Department of Internal Medicine, University of Arizona, Phoenix, AZ. RV: Right ventricle, New York Heart Association (NYHA) functional class is a system for classifying heart failure and pulmonary hypertension. Class I indicates no symptoms, Class II includes mild dyspnea and slight limitations during ordinary activity, Class III is marked limitations in activity, and Class IV is severe limitations with symptoms even while at rest.

approximately 6 h after PE in porcine models despite sustained elevations in mPAP.[13] Following normalization of afterload, RV coupling, ejection fraction, and stroke volume improved. The mPAP reflects both pulmonary arterial compliance and pulmonary vascular resistance, as well as right ventricular elastance (RV contractility), PA elastance, and RV cardiac output [Figure 2]. The RV may become incapable of generating adequate PA pressure and flow in severe cases of PAH, as well as in intermediate-high-risk PE with low CI.[21] Consequently, a reduction in mPAP could be misleading because it does not signify improved hemodynamics. Thus, a patient with intermediate-risk PE in this scenario may have ongoing impaired RV-PA coupling if mechanical thrombectomy is prematurely stopped at this point. As a result, a reduction in mPAP or even pulmonary vascular resistance should be associated with a more functional and morphological RV. Various combinations of these variables may result in similar mPAP measurements but have vastly differing clinical outcomes. For example, an asymptomatic patient with mild RV afterload and normal RV elastance may have an identical mPAP value to a patient with severe RV afterload and poor RV elastance. In addition to these physiologic nuances, variable techniques in obtaining accurate leveling and zeroing can additionally introduce error. Based on a retrospective study of 981 patients with PAH undergoing serial RHC, Weatherald *et al.* found that stroke volume index (SVI) and right atrial pressure (RAP) were independently associated with death or lung transplantation.[22] A novel insight of the study was that lower SVI discriminated patients at a higher risk of death or transplantation, even among those who were low risk according to NYHA functional class I or II with CI

≥2.5 L/min^{-1}/m^{-2}. If only the CI is considered in risk assessment, rather than the value of SVI, a maladaptive right ventricular response might go undetected.

There are additionally specific implications unique to mechanical thrombectomy. The pulmonary arterial tree usually allows only for subtotal thrombectomy. Determination for adequate thrombectomy is user dependent and variable. The FLASH Registry, a large single-arm study evaluating mechanical thrombectomy with the FlowTriever System in intermediate-high-risk PE, did not specify a detailed treatment protocol, increasing the possibility of variation in procedures and hemodynamic measurements.[2] The EXTRACT-PE investigators also noted, terminating thrombectomy procedures ultimately depends on the operator's judgment and that can impact outcomes.[4] Pre- and post-mPAP measurement has become a commonly used way to assess whether mechanical thrombectomy is successful (or not). According to the previous RV-PA coupling concept, modest changes in afterload along with increased RV elastance would result in negligible changes in mPAP but significant improvements in RV compensation.

To guide treatment outcomes, we can also continue to seek an additional parameter. The right ventricular ejection fraction (RVEF) is known to be an independent predictor of survival in PAH. Ghio *et al*. found that a reduced RVEF predicted death regardless of PA pressures.[23] RVEF is usually determined by echocardiogram, but PA saturation (SvO_2) can serve as a surrogate. In the PAH population when determining pressure overload and all-cause mortality, RVEF (HR 2.43 [0.88–6.71] P = 0.086) had similar magnitudes to SvO_2 (HR 2.63 [0.76–9.11] P = 0.126) and mPAP (HR 2.71 [0.83–8.80] P = 0.097) compared with CI (HR 1.04 [0.34–3.18] P = 0.947). It is important to note that current invasive prognostic parameters in high afterload states such as PAH include mean RAP, SvO_2, CI, and SVI and do not include mPAP. As mentioned earlier, the SVI was prognostic even in patients with several low-risk parameters of pulmonary hypertension, such as CI, which represents a "purer" form of cardiac function without heart rate.[22]

REDUCTIONS IN RV/LV RATIO MAY NOT ALWAYS CORRELATE WITH MORTALITY

The RV/LV ratio refers to the comparison of the right to left ventricular intraluminal diameter on transverse sections of computed tomography pulmonary angiograms (CTPA).[19] It has commonly served as a surrogate marker for assessing RVD and is a primary endpoint in many studies that have evaluated various mechanical thrombectomy methods. However, studies have yielded conflicting results regarding its prognostic value. For instance, in a cohort of 411 patients with hemodynamically stable PE, an RV/LV ratio ≥0.9 was associated with increased death or clinical deterioration (HR 3.8, 95% CI 1.3–10.9, P = 0.007).[24] A subsequent large meta-analysis also evaluated the predictive value of several CTPA parameters, finding all-cause mortality was increased with an RV/LV ratio ≥1.0 (OR 2.5, 95% CI 1.8–3.4, $P \leq 0.0001$), bowing of the interventricular septum (OR 1.7, 95% CI 1.2–2.4, P = 0.0027), and contrast reflux into the inferior vena cava (OR 2.2, 95% CI, 1.5–3.2 $P \leq 0.0001$).[25] The predictive value of these parameters however applies to unselected PE patients and may not prognosticate those with very low-risk disease without RVD.[25] Therefore, the effect size of improved survival in intermediate-risk PE treated with advanced therapies is likely to be small, and its correlation with RV/LV ratio reduction is questionable. In such contexts, the correlation between RV/LV ratio reduction and improved survival remains uncertain.

PHYSIOLOGY AND TECHNICAL ASPECTS OF PA SATURATION (SvO_2)

PA saturation (also known as mixed venous oxygen, SvO_2) is an indicator of the average total body hemoglobin-bound oxygen content in blood after tissue oxygen extraction.[26] It therefore reflects the complex balance between tissue oxygen delivery and extraction. Direct Fick is the gold standard method for determining cardiac output [Figure 3]. Risk assessment in PAH is routinely done using a multidimensional stratification that includes CI either directly or as part of the calculator of pulmonary vascular resistance. Traditional methods to measure CI include thermodilution (TDCI), indirect Fick, and direct Fick (utilizing SvO_2). Based on RHCs performed on 75 PAH patients, Khirfan *et al.* found that direct Fick was more accurate in predicting outcomes compared to CI derived from TDCI.[27]

Obtaining a sample involves positioning a PA catheter within the main PA and obtaining mixed venous blood from the distal tip [Figure 4].[28] Waveform analysis and fluoroscopy are traditionally used to confirm catheter position. The normal range for SvO_2 is typically 60–80%. In the absence of

$$\text{Fick CO} = \frac{\text{Oxygen consumption}}{(\text{SaO2} - \text{SvO2}) \text{x Hgb x } 1.34 \text{ x } 10}$$

Figure 3: Direct Fick equation. An individual's cardiac output (CO) is determined by the difference in oxygenation saturation between arterial and venous blood (AVO_2 difference), as well as the rate of oxygen consumption at the time the arterial and venous blood samples are drawn. In most cases, oxygen consumption (VO_2) is estimated by validated equations rather than directly calculated (using the Dehmer equation for VO_2 estimation: 125*body surface area). (CO: cardiac output (L/min), oxygen consumption (mL/min), SaO_2: Arterial oxygen saturation, SvO_2: Pulmonary artery saturation.)

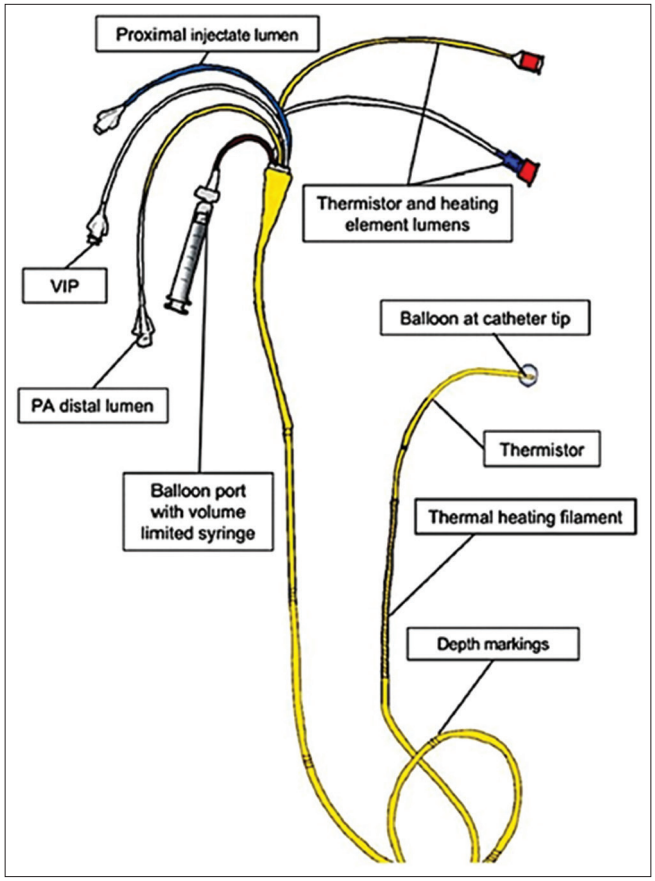

Figure 4: Pulmonary artery (PA) catheter. Obtaining a pulmonary artery saturation (SvO_2) involves positioning the pulmonary artery catheter within the main pulmonary artery and obtaining mixed venous blood from the distal tip (labeled above as PA distal lumen). Figure courtesy of Crowe *et al.* with permission under the Creative Commons Attribution license (CC BY-NC-ND 4.0). (VIP: Venous infusion port.)

left-to-right intracardiac shunts, a diminished SvO_2 signifies inadequate perfusion, often indicative of compromised cardiac output.[29] In contrast, an elevated SvO_2 may indicate effective oxygen delivery, but also inefficient oxygen consumption. Assuming stable tissue extraction, monitoring of SvO_2 provides pivotal information that summarizes the overall impact on oxygen delivery after an intervention such as mechanical thrombectomy for intermediate-risk PE. Clinicians can evaluate the impact of their therapy on ventriculoarterial coupling by combining this data with conventional measures of afterload (mPAP). The use of SvO_2 over historic measures of RV afterload/strain can better identify intermediate-high-risk PE subgroups that would benefit from mechanical thrombectomy.

LIMITATIONS OF PA SATURATION (SvO_2)

Supplemental oxygen is often needed by patients with intermediate or intermediate-high-risk PE. SvO_2 interpretation can be affected by fractional-inspired oxygen (FiO_2). Lin *et al.* measured the SvO_2 levels of 42 patients undergoing elective cardiac surgery. For patients requiring supplemental oxygen (higher-FiO_2 group >0.85), FiO_2 values increase SvO_2 values, and the calculated Fick CO has moderate Pearson correlations and similar linear regressions.[30] The extent to which supplemental oxygen (FiO_2) impacts SvO_2 and outcomes is not entirely clear in patients with acute PE undergoing catheter-directed interventions. A retrospective study found that an oxygen saturation of <50% in the pulmonary arteries (SvO_2) was associated with a higher death rate after catheter-directed intervention.[31] The average supplied FiO_2 for all patients, regardless of whether they were on mechanical ventilation was 34.4 ± 23.32%. According to the study's regression analysis, PA saturation was associated with mortality regardless of PESI score or type of endovascular intervention. Receiver operator curve testing indicated that PA saturation of 50% was inferior to PESI scores but superior to BOVA scores in predicting mortality after mechanical PE intervention, however; combined PA saturation + PESI outperformed PESI and PA saturation separately in predicting mortality.[31] A retrospective study found that intraoperative cardiac arrest occurred in 9 (6%) of 151 intermediate-to-high-risk PE patients undergoing mechanical thrombectomy. With veno-arterial extracorporeal membrane oxygenation (VA-ECMO) cannulation, four patients were successfully rescued. In the nine patients who underwent intraoperative cardiac arrest, several high-risk features were found: PASP ≥70 mm Hg (84% vs. 14%; $P \leq 0.001$), hypotension upon admission (systolic of 94 ± 14 mm Hg vs. 119 ± 23 mm Hg; $P = 0.004$), hypoxia (87 ± 6% vs. 92 ± 6%; $P = 0.023$), and recent surgery.[32] In intermediate-high-risk PE patients identified to be in subclinical shock (CI <2 $L/min^{-1}/m^{-2}$) presenting with high periprocedural arrest risk characteristics, it may be beneficial to obtain a pre-thrombectomy SvO_2 measurement, with values of <50% to consider VA-ECMO cannulation before thrombectomy.

CONCLUSION

The integration of SvO_2 into the current use of validated scoring algorithms in future prospective studies of catheter-directed mechanical thrombectomy may provide further insight and a more comprehensive understanding of the complex interactions of RV function and afterload. Trending cellular oxygen delivery indices, such as SvO_2, are a critical component of cardiac resuscitation and could be incorporated into the algorithm of PE risk assessment in patients undergoing catheter-directed interventions. However, the true physiologic value of SvO_2 is complicated, requiring an understanding of multiple aspects of the cardiovascular system working in combination. Using SvO_2 to make conclusions about hemodynamic variables such as cardiac output requires specific assumptions which may not be applicable to all patients. Thus,

routine measurement of SvO_2 post-thrombectomy without considering the clinical context and other hemodynamic measures may have limited application. It would be beneficial to include SvO_2 and consider CI as a method to identify patients at high risk for periprocedural cardiac arrest as well as guide and standardize mechanical thrombectomy.

Ethical approval

The Institutional Review Board approval is not required.

Declaration of patient consent

Patient's consent is not required as there are no patients in this study.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

Use of artificial intelligence (AI)-assisted technology for manuscript preparation

The authors confirm that there was no use of artificial intelligence (AI)-assisted technology for assisting in the writing or editing of the manuscript and no images were manipulated using AI.

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How to cite this article: Talon AF, Uppalapu S, Ma M, Khan AA, Sista RR, El-Kersh K, *et al.* Pulmonary artery saturation as a primary endpoint for mechanical thrombectomy. Am J Interv Radiol. 2024;8:12. doi: 10.25259/AJIR_26_2024