High-risk pulmonary embolism in a post-operative patient

Jordan Talan, Brian Reuland, Brian Kaufman and Anthony Andriotis

New York University Grossman School of Medicine, Department of Medicine, Division of Pulmonary, Critical Care, & Sleep Medicine, New York, NY, USA.

Corresponding author: Jordan Talan (jordan.talan@nyulangone.org)

Shareable abstract (@ERSpublications)
This article presents a simulated case of high-risk PE in a post-operative patient that is designed to teach key principles of diagnosis and management. The script is widely adaptable to many institutions and customisable to diverse learner populations. https://bit.ly/4eWC4qZ

Cite this article as: Talan J, Reuland B, Kaufman B, *et al*. High-risk pulmonary embolism in a post-operative patient. *Breathe* 2025; 21: 240101 [DOI: 10.1183/20734735.0101-2024].

Received: 10 Sept 2024
Accepted: 18 Nov 2024

Abstract

Despite a rapidly evolving role for advanced therapeutic interventions, the majority of patients with high-risk pulmonary embolism (PE) are still undertreated. Due to complex diagnosis and management, along with a high associated mortality rate, high-risk PE offers an excellent opportunity for simulation training. We present a simulated case of high-risk PE in a post-operative patient that is designed to teach key principles of diagnosis and management. The script is widely adaptable to many institutions and customisable to diverse learner populations.

Introduction

Pulmonary embolism (PE) is an occlusion of the pulmonary arteries, which most frequently occurs due to migration of a deep vein thrombosis (DVT) from the lower extremities through the right heart [1, 2]. The incidence of PE has increased since the late 1990s. The diagnosis can be challenging due to nonspecific symptoms such as fatigue, breathlessness, chest pain, dizziness or unexplained syncope [1, 3]. Approximately 5% of patients present with high-risk PE involving obstructive shock or cardiac arrest. These patients have a high mortality, estimated at ~44–65%, and they require immediate reperfusion and haemodynamic support [4–6]. Therefore, the presentation of undifferentiated shock must prompt diagnostic consideration of life-threatening PE. Due to the acuity of this presentation, trainees in critical care and respiratory medicine should be proficient with the workup and management of high-risk PE, including appropriate methods of haemodynamic support and options for revascularisation. In support of this educational goal, we describe the simulated presentation of a complex post-operative patient with undifferentiated shock due to massive PE.

Learning objectives

1) Evaluate a patient with acute onset of undifferentiated circulatory shock and diagnose obstructive shock due to high-risk PE.
2) Classify risk in acute PE and recognise high-risk PE.
3) Apply an understanding of the pathophysiology of high-risk PE by selecting appropriate interventions to stabilise haemodynamics.
4) Evaluate options for revascularisation of high-risk PE and manage the diagnosis in a simulated post-operative patient.

Required personnel

- Learner(s)
- Patient (mannequin)
- Intensive care unit (ICU) nurse (embedded participant)
- Faculty observer

TABLE 1 Initial presentation

Initial vital signs	Temperature 37.8°C, blood pressure 92/48 mmHg and decreasing progressively, heart rate 128 beats per min (sinus tachycardia with right bundle branch block), respiratory rate 28 breaths per min, S_{pO_2} 88% on room air
Overall appearance	*What do learners see when they first enter the room?* The patient, portrayed by a mannequin, is awake, alert and conversing normally. The patient has one 18-gauge antecubital peripheral *i.v.*, and is connected to the telemetry monitor with noninvasive blood pressure cuff cycling every 5 min.
Actors and roles in the room at the start of the case	*Who is present at the beginning and what is their role? Who may play them?* Primary learner: the primary learner may be in the role of a medical student, resident, fellow or other clinical care team member responding to the rapid response. The patient (mannequin): portrayed by a simulation staff member. Surgical ward nurse: portrayed by a simulation staff member. The nurse recently took over this patient assignment and is concerned about the syncopal event representing new instability. The nurse can give additional history if requested.
History of present illness (delivered by surgical ward nurse)	The patient is a 62-year-old male with a past medical history of hypertension and diabetes who was admitted to the hospital for a scheduled pancreaticoduodenectomy for pancreatic cancer. He tolerated the procedure well 3 days ago and was transferred from the SICU to the floor yesterday. The nurse now calls you to the bedside because the patient just experienced transient syncope while attempting to get out of bed.
Additional information (available if requested)	
Further hospital history	The patient had an uncomplicated perioperative course. The urinary catheter was removed on post-operative day 1 and the arterial line was also removed. The patient had been participating in physical therapy and was transferred to the floor yesterday. He was restarted on *s.c.* heparin for DVT prophylaxis and received his first dose yesterday.
Morning note documented by surgical team	No acute complaints, alert and oriented ×3, extremities warm with no oedema. Mild incisional tenderness. Remainder of the examination is unremarkable.
Laboratory results from this morning	Complete blood count: WBC 11.0×10^9 cells·L^{-1}, Hb 140 g·L^{-1}, haematocrit 41%, platelets 165×10^9 per L Basic metabolic panel: sodium 140 mmol·L^{-1}, potassium 4.0 mmol·L^{-1}, chloride 101 mmol·L^{-1}, bicarbonate 22 mmol·L^{-1}, BUN 11 mmol·L^{-1}, creatinine 90 µmol·L^{-1} Liver function testing: total protein 50 g·L^{-1}, albumin 35 g·L^{-1}, total bilirubin 17 µmol·L^{-1}, AST 30 units·L^{-1}, ALT 25 units·L^{-1}, alkaline phosphatase 70 units·L^{-1}
Interventions prior to scene	None
Past medical/surgical history	Hypertension Diabetes Pancreatic mass
Inpatient medications	Insulin sliding scale *s.c.* Heparin Acetaminophen Oxycodone (as needed, not taken today)
Allergies	None
Social history	Never smoker No alcohol use No recreational substances
Physical examination	
General	Awake, alert and oriented ×3, eyes open, conversing normally (mild dyspnoea at rest)
HEENT	Moderately elevated jugular venous pressure
Neck	Supple, full neck range of motion
Lungs	Tachypnoeic, breath sounds clear
Cardiovascular	Tachycardia with regular rhythm; no murmurs, rubs or gallops; no lower extremity oedema
Abdomen	Soft, nondistended, appropriate incisional tenderness
Neurological	Moving all extremities
Skin	Incisions clean, dry, intact; skin cool to touch
Genitourinary	Unremarkable
Psychiatric	Answers questions, slightly worried about his status but asymptomatic

S_{pO_2}: peripheral oxygen saturation; SICU: surgical intensive care unit; DVT: deep vein thrombosis; WBC: white blood cell count; Hb: haemoglobin; BUN: blood urea nitrogen; AST: aspartate aminotransferase; ALT: alanine transaminase; HEENT: head, eyes, ears, nose and throat.

Required equipment

- Laerdal SimMan 3G
- Intelligent Ultrasound Heartworks transthoracic/transoesophageal echocardiography simulator
- ICU bed
- ICU monitor

- Normal chest radiograph
- ECG with sinus tachycardia and right bundle branch block
- Syringes and labels for simulated medications
- *i.v.* pump and tubing
- Central venous catheter

Brief narrative case description

A 62-year-old male with a past medical history of hypertension and diabetes was admitted to the hospital for a scheduled pancreaticoduodenectomy for pancreatic cancer. He tolerates the procedure well, but develops acute high-risk PE on the surgical floor associated with obstructive shock.

Critical action checklist

- Performs focused history
- Requests additional history (hospital course and therapies received)
- Performs focused physical examination (mental status, tissue perfusion and focused cardiopulmonary examination)
- Performs point-of-care ultrasound to assess for aetiology of shock
- Considers differential diagnosis of shock and identifies obstructive shock due to PE
- Initiates vasopressor therapy with inotropic component
- Avoids large *i.v.* fluid boluses leading to right ventricle (RV) overload
- Avoids endotracheal intubation/positive pressure ventilation
- Consults PE response team (PERT team)
- Makes a plan for expedited revascularisation *via* thrombectomy

Learner preparation

The learner should be provided with the brief patient history from the ICU nurse upon entering the room (table 1).

ACT 1 Early shock (beginning of the scenario)

Change in case	Additional information
Possible interventions	Vital sign monitoring can be adjusted per leader's request
Requests additional history: • Disclose additional history (see table 1) • Review of systems otherwise positive for expected incisional pain, well managed • No previous hospitalisations or recent illness • Asks for weight: 60 kg • Asks for ECG: sinus tachycardia, right bundle branch block • Asks for prior ECG: normal sinus rhythm • Asks for last transthoracic echocardiogram: normal echocardiogram in preoperative evaluation	
Requests laboratory tests: nurse asks them to specify laboratory tests and sends (they will not be available until Act 2)	
Asks for fluids or vasopressors: nurse requests specifics of amount and type, proceed to Act 2	
Starts antibiotics: nurse asks which antibiotics to add, proceed to Act 2 (no additional credit for double Gram negative coverage)	
Starts oxygen: oxygen saturation increases to 90%, no further	
Critical actions	
Requests additional history • Requests prior hospital course • Asks for therapies received	
Performs focused physical examination • Examines for evidence of shock (mental status, lactate) • Examines for aetiology of shock (physical examination)	
Applies supplemental oxygen	

ACT 2 Obstructive shock (transition 5 min into the case or if interventions are given in Act 1)	
Change in case	**Additional information**
New vital signs	*i.v.* access: one 18 gauge forearm *i.v.*, unless changed in Act 1
Temperature 37.8°C, blood pressure (BP) gradually decreases to 78/40 mmHg, heart rate (HR) 138 beats per min (sinus tachycardia with right bundle branch block), respiratory rate 28 breaths per min, peripheral oxygen saturation (S_{pO_2}) 88% on room air	Monitoring: as in Act 1, unless changed
Physical examination changes	Blood gas will result and be given
General: lying in bed, eyes half-closed, mumbling incoherently, sleepy but arousable	
Neurological: moans and mumbles to verbal stimuli	
Genitourinary: no urinary output	
Blood gas results	
pH 7.28, carbon dioxide tension (P_{CO_2}) 4.7 kPa, oxygen tension (P_{O_2}) 7.9 kPa, lactate 0.61 mmol·L^{-1}	
If requested: troponin-I: 3 µg·L^{-1}, brain natriuretic peptide (BNP) 500 ng·L^{-1}	
Possible diagnostic interventions	
Asks for ultrasound: delay for 5 min while the ultrasound is being brought from the ICU, then allow use of the Heartworks Ultrasound set to PE with right heart strain	
Asks for chest radiograph: it will be delayed and not arrive during the case	
Asks for computed tomography (CT) scan: nurse will express concern that the patient is not stable to travel to radiology	
Asks for blood cultures: nurse will send cultures	
Asks for foley catheter: nurse will place foley	
Asks for central line: a triple lumen central line is placed in the right internal jugular vein by the fellow	
Asks for central venous pressure (CVP) or central venous oxygen saturation (S_{cVO_2}): following central line placement, CVP is 20 mmHg and S_{cVO_2} is 44%	
Asks for transoesophageal echocardiogram (TEE): Heartworks TEE set to saddle PE with right heart dilated and hypokinetic	
Possible therapeutic interventions	
Asks for antibiotics: nurse will hang specified antibiotics at specified dose	
Asks for fluids: nurse will hang specified fluids, systolic BP will decrease by 10 mmHg	
Asks for vasopressor: nurse will ask which pressor and titration parameters, patient will quickly titrate up to high-dose vasopressors and BP increase to 85/52 mmHg	
Asks for inotrope: systolic BP increases by 10 mmHg	
Asks for inhaled pulmonary vasodilators: oxygen saturation increases to 95%	
Asks for noninvasive positive pressure ventilation: HR increases by 10 beats per min, systolic BP drops by 5 mmHg	
Asks for intubation: nurse calls for anaesthesia, they will be delayed and not arrive during the case	
Asks for PERT team:	
Advanced learners: PERT not available	
Novice learners: PERT team advises against systemic thrombolysis in this post-operative patient, recommends calling Interventional Radiology	
Asks for heparin drip: nurse starts heparin	
Asks for systemic thrombolysis (tPA): surgeon has requested to avoid systemic thrombolysis due to recent major surgery	
Asks for surgical thrombectomy: surgery recommends calling for catheter-based procedure	
Asks for catheter-based thrombolysis/thrombectomy: Interventional Radiology will prepare the procedure room, proceed to Act 3	
Critical actions	
Considers differential diagnosis of shock • Interprets CVP and S_{cVO_2} from central line • Performs point-of-care ultrasound • Identifies obstructive shock due to PE	
Stabilises haemodynamics in massive PE • Administers vasopressors (avoids phenylephrine) • Withholds *i.v.* fluid resuscitation • Requests mechanical thrombectomy	

ACT 3 Conclusion (transition after mechanical thrombectomy is requested)

Change in case

Nurse announces that the transport team has arrived to take the patient to a hybrid operating room for a catheter thrombectomy with a backup plan for surgical thrombectomy. Begin the debrief.

Ideal scenario flow

The learner enters the room, where the nurse provides a brief history. The learner asks for current vital signs, additional history and additional laboratory tests including a lactate. The learner recognises new right bundle branch block, tachycardia and hypotension in a post-operative patient, and requests point-of-care ultrasound to help differentiate the shock process. While awaiting the ultrasound machine, the learner asks for a central venous catheter and recognises high CVP and low S_{cVO_2}. The learner starts an appropriate vasopressor to stabilise haemodynamics. Upon availability of the ultrasound machine, the learner identifies suggestive evidence for PE including new severe right ventricular dilation and hypokinesis with septal flattening. The learner considers multiple options for treatment in this high-risk post-operative PE, and decides to consult Interventional Radiology for catheter thrombectomy.

Anticipated management mistakes

1) Inability to differentiate obstructive shock: If the learner is unable to differentiate a shock process, the nurse will ask if the learner would like any data from a central line, or if the learner would like the ultrasound.
2) Inadequate resuscitation: If an inadequate attempt is made to stabilise haemodynamics, the nurse will express concern with worsening haemodynamics. For novice learners, the nurse can offer to call the pharmacy for medication or dosing recommendations.
3) Right ventricular volume overload: The learner may continue to give *i.v.* fluids, and in this case the nurse will call attention to the haemodynamics and ask if the learner would like a vasopressor. If the learner continues fluids despite lack of response, the nurse can express concern that the fluids are not helping.
4) Failure to plan thrombolysis: The learner may make no attempt to treat PE or initiate only a heparin drip. For novice learners, the nurse can suggest calling the PERT team for further guidance on treatment options.
5) Intubation as an early intervention: The learner may request intubation in an attempt to prevent respiratory failure. If requested, the nurse may ask if the learner is sure that this patient requires intubation.

Debriefing process

There are multiple methods of performing effective healthcare simulation debriefing, and with this case we vary our debriefing method based on the learners' experience level. For novice learners, this case can be challenging and often requires a facilitator-guided, within-event debriefing [7]. With this strategy, we allow the learners (often medical students, physician assistant students, nurse practitioner students or early postgraduate learners) to proceed with the simulation until they become "stuck", at which point the simulation is interrupted for short, highly focused debriefing points, which allow continuation of the case [7]. For more advanced learners, such as advanced residents or fellows, we use facilitator-guided, post-event debriefing, which is the most common and most studied method of simulation debriefing [7].

Regardless of the timing and facilitation, we employ "Debriefing With Good Judgement" to structure our debriefing conversations [7, 8]. This involves using advocacy inquiry to reveal each learner's cognitive frames, or the understanding that lead them to particular actions during the simulation. We then collaborate with the trainee to either reinforce or adjust frames as necessary [8].

Debriefing points

Here we describe our most frequently debriefed learning points from the more basic to the most complex. Notably, there is constructive alignment between the debriefing points and the above learning objectives [9]. Discussion is tailored to each individual learner based on both experience level and performance during the case.

Evaluate undifferentiated shock and diagnose PE

Shock represents cellular hypoxia from an imbalance of oxygen delivery and oxygen consumption [10, 11]. Circulatory shock can be diagnosed based on the presence of systemic hypotension associated with tissue hypoperfusion [11]. Supporting evidence can include skin findings (cool or cyanotic), neurological

findings (altered mental state, obtundation or confusion), decreased urine output, and lactic acidosis reflective of decreased oxygen delivery.

Shock is most commonly classified into four distinct, but not necessarily exclusive, pathophysiological mechanisms: hypovolaemic, cardiogenic, obstructive and distributive. While each piece of clinical data used to differentiate shock pathophysiology has its pitfalls and caveats, a combination of physical examination, laboratory values and point-of-care imaging can lead to a strong argument for obstructive shock in this case (table 2). The presence of a dilated and hypokinetic RV is supportive in the diagnosis of massive PE [11].

Risk-stratify PE and recognise life-threatening PE

PE is most commonly classified using schemes proposed by the American Heart Association (AHA) and European Society of Cardiology (ESC) [12, 13]. Low-risk PE does not meet higher-risk criteria. Submassive PE (by AHA criteria) includes patients with RV dysfunction without hypotension. Massive (AHA) or high-risk (ESC) PE requires sustained hypotension or vasopressor requirement. The patient in this scenario can be diagnosed with high-risk PE, which confers a mortality of ∼30% within 1 month. In patients with high-risk PE, the primary goal is to acutely reverse haemodynamic compromise. Although there is a role for therapeutic anticoagulation (*e.g.* heparin) to prevent additional thrombosis, patients with high-risk PE often require more aggressive therapies to more quickly eliminate the existing clot [13–15].

Understand PE pathophysiology and stabilise haemodynamics

In high-risk PE, obstruction of pulmonary arterial flow results in increased RV afterload. The result is displacement of the interventricular septum towards the left, with impaired left ventricle filling. If untreated, this causes decreased left ventricular preload, reduced cardiac output, and obstructive circulatory shock [16].

The stabilisation of haemodynamics in high-risk PE requires close attention to optimising RV preload. Although RV stroke volume is highly preload dependent under normal conditions, volume overload can cause increased interventricular dependence, worsening the state of circulatory shock [17]. Therefore, administration of *i.v.* fluids should be considered cautiously.

Stabilisation of perfusion pressures also commonly requires vasoactive medications. Systemic vasopressors such as norepinephrine increase coronary perfusion pressure while providing simultaneous β-adrenergic stimulation to increase RV contractility. Inotropes such as dobutamine can increase inotropy as well, but often necessitate concomitant systemic vasoconstriction. The beneficial effects of systemic vasoconstriction must be weighed against the potential for worsening vasoconstriction in the pulmonary circulation [16].

TABLE 2 Subtypes of shock

	Distributive	Hypovolaemic	Cardiogenic	Obstructive
Examples	Septic, anaphylactic, neurogenic, adrenal crisis	Dehydration, haemorrhage	Cardiomyopathy, arrhythmia, valvular disease	Pulmonary embolism, cardiac tamponade, tension pneumothorax
Physical examination	Warm skin	Cool skin, dry mucous membranes	Cool skin, leg oedema, jugular venous distention	Cool skin, absent lung sounds, distant heart sounds, jugular venous distention
CVP	Low until resuscitated	Low	High	High
S_{cvO_2}	High	Low	Low	Low
Point-of-care ultrasound	Infectious source	Collapsible inferior vena cava	Cardiomyopathy, noncollapsible inferior vena cava	Absence of lung sliding (or lung point), RV dilation and hypokinesis, septal shift, pericardial effusion with tamponade physiology, noncollapsible inferior vena cava

CVP: central venous pressure; S_{cvO_2}: central venous oxygen saturation; RV: right ventricle.

Some clinicians support the addition of vasopressin to other vasoactive medications because it can increase systemic vascular resistance while relatively sparing pulmonary vascular resistance; however, there is little clinical data to support this practice [18]. Inhaled pulmonary vasodilators (*e.g.* nitric oxide) may reduce hypoxic pulmonary vasoconstriction and improve ventilation–perfusion (V'/Q') mismatch without the detrimental decrease in systemic blood pressure associated with systemic vasodilators [16].

Intubation should generally be avoided if possible, due to increased RV afterload with positive pressure ventilation as well as systemic vasodilation from anaesthetic medications [16, 19]. Any degree of V'/Q' mismatch or shunt in the setting of PE must be carefully weighed against the potential for worsened obstructive shock and increased mortality associated with intubation. This simulation does not require endotracheal intubation, which would confer additional risk in this setting. Furthermore, the failure of supplemental oxygen to improve saturation in this patient reflects right-to-left intracardiac shunting, which may occur during high-risk PE through a patent foramen ovale [20]. Positive pressure ventilation may worsen the gradient associated with this shunt, possibly worsening hypoxaemia.

In the event of severe or refractory shock, venoarterial extracorporeal membrane oxygenation (VA-ECMO) can be considered for mechanical circulatory support because it bypasses the pulmonary arterial circulation. This strategy can serve as a bridge to recovery or further treatment, and it has advantages over ventricular assist devices (VADs) due to quicker and easier initiation of therapy [16].

Evaluate options for revascularisation in a post-operative patient

Recent guidelines recommend systemic thrombolysis for patients with high-risk PE or intermediate-risk PE with deterioration after starting anticoagulation due to more rapid improvement in haemodynamics [21]. Systemic thrombolysis (*e.g.* tissue plasminogen activator, tenecteplase) converts plasminogen to plasmin, actively promoting the hydrolysis of thrombus [16]. This therapy is best administered early, and the benefits must be carefully weighed against the risk of major haemorrhage. Absolute contraindications include: structural intracranial disease, previous intracranial haemorrhage, ischaemic stroke within 3 months, active bleeding, recent brain/spine surgery, recent head trauma with fracture or brain injury, and bleeding diathesis [13]. In this case, recent major surgery represents one of many relative contraindications, placing this patient at increased risk of major bleeding.

Given the risk associated with systemic thrombolysis, catheter-based interventions have gained increasing interest. Catheter-directed thrombolysis involves a lower dose of thrombolytic compared with systemic dosing, but does not eliminate bleeding risk. Catheter-based thrombectomy can rapidly remove thrombus, but may be associated with more procedural complications due to larger catheters. This strategy is more effective at removing proximal clots compared with distal. There is limited evidence to guide clinical decision-making around these catheter-based interventions, and outcomes are currently being investigated. Surgical embolectomy remains an alternative or rescue option [13].

The options for intervention in cases like this are complex, and patients with high-risk PE are generally undertreated. For this reason, PERT teams have been created, consisting of a multidisciplinary team of experts to assist the primary team in management decisions [13]. Clinical outcomes associated with PERTs require further study.

Discussion

Data from over 40 years of research on simulation-based medical education (SBME) demonstrate that SBME with deliberate practice is associated with a dose-related improvement in learner outcomes [22, 23]. Simulation offers a safe and controlled environment for learners to practice clinical management, and has been associated with superior learning and retention compared with traditional clinical teaching or no intervention [23–25]. Furthermore, SBME, especially when combined with mastery learning principles, can lead to better patient care and outcomes [24, 26]. In regards to PE specifically, there is a relative paucity of research on simulation outcomes, and studies vary in methodology and rigor. A simulated case of massive PE leading to cardiac arrest in an adolescent was associated with high learner-reported effectiveness and improved comfort among emergency medicine residents [27]. Among cardiologists and haematologists/oncologists, a virtual patient simulation, although not mannequin-based, was associated with an increase in correct clinical decisions [28]. Overall, outcomes from simulation education are complex and likely related to design choices, learner-related factors, faculty expertise, implementation strategies, integration into the overall curriculum, and multiple additional factors.

We have used this simulation case at our institution for ~10 years with excellent learner feedback. Learners find the case challenging due to the post-operative history, which provides a suspicion for all

aetiologies of shock. Learners also appreciate the opportunity to practice skills in diagnostic point-of-care ultrasound. Educators find the case effective as the medical complexity provides extensive opportunities for learning points. This case is universally adaptable by concentrating on different debriefing points for different learner levels. We have used it for the education of physician assistant students, medical students, residents in multiple graduate medical education programmes, and fellows in critical care.

Future directions should include studying the effects of this simulation intervention on self-reported confidence in the management of massive PE, checklist-based simulation assessment, and ultimately patient outcomes associated with simulation participants.

Conclusion

We describe the simulated presentation of a complex post-operative patient with undifferentiated shock due to massive PE. We hope this case can be used by other training programmes and simulation centres to achieve key learning objectives around this diagnosis.

Conflict of interest: There are no conflicts of interest to report.

References

1 Maughan BC, Jarman AF, Redmond A, *et al.* Pulmonary embolism. *BMJ* 2024; 384: e071662.
2 Freund Y, Cohen-Aubart F, Bloom B. Acute pulmonary embolism: a review. *JAMA* 2022; 328: 1336–1345.
3 Smith SB, Geske JB, Kathuria P, *et al.* Analysis of national trends in admissions for pulmonary embolism. *Chest* 2016; 150: 35–45.
4 Kahn SR, de Wit K. Pulmonary embolism. *N Engl J Med* 2022; 387: 45–57.
5 Agnelli G, Becattini C. Acute pulmonary embolism. *N Engl J Med* 2010; 363: 266–274.
6 Polaková E, Veselka J. Management of massive pulmonary embolism. *Int J Angiol* 2022; 31: 194–197.
7 Sawyer T, Eppich W, Brett-Fleegler M, *et al.* More than one way to debrief: a critical review of healthcare simulation debriefing methods. *Simul Healthc* 2016; 11: 209–217.
8 Rudolph JW, Simon R, Rivard P, *et al.* Debriefing with good judgment: combining rigorous feedback with genuine inquiry. *Anesthesiol Clin* 2007; 25: 361–376.
9 Gilic F, Dalgarno N, Simpson MTW. Applying constructive alignment and cognitive load in teaching: case study involving a foundational family medicine medical school course. *Can Fam Physician* 2022; 68: 308–310.
10 Simmons J, Ventetuolo CE. Cardiopulmonary monitoring of shock. *Curr Opin Crit Care* 2017; 23: 223–231.
11 Vincent JL, De Backer D. Circulatory shock. *N Engl J Med* 2013; 369: 1726–1734.
12 Russell C, Keshavamurthy S, Saha S. Classification and stratification of pulmonary embolisms. *Int J Angiol* 2022; 31: 162–165.
13 Giri J, Sista AK, Weinberg I. Interventional therapies for acute pulmonary embolism: current status and principles for the development of novel evidence: a scientific statement from the American Heart Association. *Circulation* 2019; 140: e774–e801.
14 Come PC. Echocardiographic evaluation of pulmonary embolism and its response to therapeutic interventions. *Chest* 1992; 101: Suppl., 151S–162S.
15 Konstantinides S, Tiede N, Geibel A, *et al.* Comparison of alteplase *versus* heparin for resolution of major pulmonary embolism. *Am J Cardiol* 1998; 82: 966–970.
16 Moorjani N, Price S. Massive pulmonary embolism. *Cardiol Clin* 2013; 31: 503–518.
17 Petit M, Vieillard-Baron A. Ventricular interdependence in critically ill patients: from physiology to bedside. *Front Physiol* 2023; 14: 1232340.
18 Sarkar J, Golden PJ, Kajiura LN, *et al.* Vasopressin decreases pulmonary-to-systemic vascular resistance ratio in a porcine model of severe hemorrhagic shock. *Shock* 2015; 43: 475–482.
19 Pérez-Nieto OR, Gómez-Oropeza I, Quintero-Leyra A, *et al.* Hemodynamic and respiratory support in pulmonary embolism: a narrative review. *Front Med (Lausanne)* 2023; 10: 1123793.
20 Goldhaber SZ, Elliott CG. Acute pulmonary embolism: part I: epidemiology, pathophysiology, and diagnosis. *Circulation* 2003; 108: 2726–2729.
21 Stevens SM, Woller SC, Kreuziger LB, *et al.* Antithrombotic therapy for VTE disease: second update of the CHEST Guideline and Expert Panel Report. *Chest* 2021; 160: e545–e608.
22 McGaghie WC, Issenberg SB, Petrusa ER, *et al.* A critical review of simulation-based medical education research: 2003–2009. *Med Educ* 2010; 44: 50–63.
23 Motola I, Devine LA, Chung HS, *et al.* Simulation in healthcare education: a best evidence practical guide. AMEE Guide No. 82. *Med Teach* 2013; 35: e1511–e1530.
24 Cook DA, Hatala R, Brydges R, *et al.* Technology-enhanced simulation for health professions education: a systematic review and meta-analysis. *JAMA* 2011; 306: 978–988.

25 Elendu C, Amaechi DC, Okatta AU, *et al.* The impact of simulation-based training in medical education: a review. *Medicine (Baltimore)* 2024; 103: e38813.

26 McGaghie WC, Draycott TJ, Dunn WF, *et al.* Evaluating the impact of simulation on translational patient outcomes. *Simul Healthc* 2011; 6: Suppl., S42–S47.

27 Myers M, Devlin C. Cardiac arrest in an adolescent with pulmonary embolism. *J Educ Teach Emerg Med* 2021; 6: S112–S137.

28 Lucero KS, Spyropoulos J, Blevins D, *et al.* Virtual patient simulation in continuing education: improving the use of guideline-directed care in venous thromboembolism treatment. *J Eur CME* 2020; 9: 1836865.