[16] Voci P, Testa G, Tritapepe L, Menichetti A, Caretta Q. Detection of false lumen perfusion at the beginning of cardiopulmonary bypass in patients undergoing repair of aortic dissection. Crit Care Med 2000;28:1841–6.
[17] Fusco DS, Shaw RK, Tranquilli M, Kopf GS, Elefteriades JA. Femoral cannulation is safe for type A dissection repair. Ann Thorac Surg 2004;78:1285–9 [discussion 1285-9].
[18] Hou XT, Sun YQ, Zhang HJ, Zheng SH, Liu YY, Wang JG. Femoral artery cannulation in Stanford type A aortic dissection operations. Asian Cardiovasc Thorac Ann 2006;14:35–7.
[19] Kamiya H, Kallenbach K, Halmer D, Ozsoz M, Ilg K, Lichtenberg A, et al. Comparison of ascending aorta versus femoral artery cannulation for acute aortic dissection type A. Circulation 2009;120:S282–6.
[20] Conzelmann LO, Weigang E, Mehlhorn U, Abugameh A, Hoffmann I, Blettner M, et al. Mortality in patients with acute aortic dissection type A: analysis of pre- and intraoperative risk factors from the German Registry for Acute Aortic Dissection Type A (GERAADA). Eur J Cardiothorac Surg 2016;49:e44–52.

Reply to: ECPR in acute aortic dissection - Really a no-go?

We thank the authors for their reply and interest to our case report on using transthoracic echocardiography to identify an acute aortic dissection during ECMO initiation [1].

The creation of an emergency department ECMO program is a complex process that relies on buy-in and agreement from multiple specialties including emergency medicine, cardiothoracic surgery, cardiology, and anesthesiology. This process requires predefined inclusion and exclusion criteria for initiation and termination agreed upon by all parties. As the authors state with regard to aortic dissection in ECPR, "no studies on feasibility, safety and efficacy of ECPR in this condition exist". While the authors report three previous case reports of aortic dissection in ECPR, with one favorable outcome, we do not think this constitutes a level of evidence that would merit change to protocols.

The authors acknowledge the risk of perfusing the false lumen, which they estimate to be low based on non-emergent femoral cannulation cases. We hesitate to extrapolate this limited data to patients that suffer cardiac arrests from an acute dissection and are actively undergoing CPR.

With regard to diagnosis accuracy of TEE for Type A aortic dissection, we refer to Alter et al., which is cited by the authors, which states that TEE has a "sensitivity of 90–98% that is equal to CT or magnetic resonance imaging (MRI)" [2]. We agree that there are potential artifacts that can mimic acute aortic dissection, however, we believe if using TEE, providers should have knowledge of these potential pitfalls and obtain views in multiple imaging planes to confirm diagnosis. We refer to our accompanying videos for multi-view confirmation.

We overall agree that the concept of using ECPR for aortic dissection patients is intriguing and may warrant further investigation. We do, however, hesitate a change to established protocols for a perceived benefit without supporting data, especially given the resource and time expense as well as risk of body fluid exposure and needle sticks to providers.

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References

[1] Kelly C, Ockerse P, Glotzbach JP, Jedick R, Carlberg M, Skaggs J, et al. Transesophageal echocardiography identification of aortic dissection during cardiac arrest and cessation of ECMO initiation. Am J Emerg Med 2019;37(6):1214.e5–6.
[2] Alter P, Herzum M, Maisch B. Echocardiographic findings mimicking type A aortic dissection. Herz 2006;31:153–5.

Pulmonary embolism in the differential diagnosis of right ventricular myocardial infarction

When right ventricular failure occurs as a result of right ventricular myocardial infarction (RVMI) [1], its manifestations can be closely simulated by right ventricular failure attributable to pulmonary embolism (PE) [2,3]. In the latter example of PE-related right ventricular failure a 35 year old previously healthy woman (a non-smoker) presented with severe abdominal pain and abdominal distension, but denied dyspnea or chest pain. Exploratory laparotomy revealed that she had a congested and very enlarged liver, and ascites as well. Further investigations included echocardiography, which disclosed a right ventricular systolic pressure of 77 mm Hg, and tricuspid regurgitation. These derangements were attributable to right ventricular failure resulting from massive PE [3]. According to Namana et al., RVMI most closely simulates PE when it presents with "chest pain, diaphoresis, nausea and vomiting with [the] hemodynamic triad:hypotension, jugular venous distension and clear lung fields" [2]. Dysrhythmias which occur in RVMI, such as atrial fibrillation and complete heart block [4], also occur in PE [5,6].

Hypoxemia is another manifestation of RVMI which can lead to a mistaken diagnosis of PE. The occurrence of RVMI-related hypoxemia was exemplified by a 70 year old man who presented with chest tightness and dyspnea. On examination his blood pressure was 100/60 mm Hg and jugular venous pressure was elevated. Oxygen saturation was 82% while he was breathing room air. The electrocardiogram disclosed ST segment elevation in leads V4-V6. Right sided precordial leads indicated 0.5 mm ST-segment elevation suggestive of RVMI. Transthoracic echocardiography showed infero-posterior wall akinesis. The right ventricle was dilated, with akinesis of the free and inferior wall. Furthermore, he had severely impaired right ventricular systolic function. Coronary angiography revealed total occlusion of the proximal segment of the right coronary artery. The persistence of hypoxemia (arterial partial pressure of oxygen amounting to 60 mm Hg), in spite of uptitration of inspired oxygen, prompted a search for coexistence of either PE or a right-to-left shunt. A computed pulmonary angiogram did not reveal PE. Transesophageal echocardiography (TEE) showed spontaneous constant flow from the right to the left atrium through a patent foramen ovale (PFO). This was a consequence of RVMI-related acute elevation in right atrial pressure. During his subsequent hospital stay hypoxemia gradually resolved. Repeat TEE, performed 6 weeks later, did not reveal any spontaneous right-to-left shunt through the PFO, presumably because right atrial pressure had reverted to normal levels [7]. In their account of the occurrence of a right-to-left shunt in a patient with inferior wall myocardial infarction, Albaghdadi et al. alluded to the occurrence of RVMI-related right-to-left shunting in eight cases reported in the literature [8]. They recommended that right-to-left shunting should be considered as the underlying cause of RVMI-related hypoxemia, after excluding more common causes such as pulmonary edema, underlying pulmonary disease, or pulmonary embolism [8].

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References

[1] Davis WT, Montrief T, Koyfman A, Long B. Dysrhythmias and heart failure complicating acute myocardial infarction: an emergency medicine review. Am J Emerg Med 2019 April 27. https://doi.org/10.1016/j.ajem.2019.04.047 [pii: S0735-6757(19)30272-4].
[2] Namana V, Gupta SS, Abbasi AA, Raheja H, Shani J, Hollander G. Right ventricular infarction. Cardiovasc Revasc Med 2018;19:41–50.
[3] Al-Mane N, Al-Mane F, Abdalla Z, McDonnell J. Acute surgical abdomen: an unusual presentation of pulmonary embolus. J Investig Med High Impact Case Rep 2014. https://doi.org/10.1177/2324709614542339hic.sagepub.com.
[4] Goldstein JA. Pathophysiology and management of right heart ischemia. JACC 2002;40:841–53.
[5] Bikdeli B, Ziki MD, Lip GYH. Pulmonary embolism and atrial fibrillation: two sides of the same coin? A systematic review. Semin Thromb Hemost 2017;43:849–63.
[6] Elias J, Kuniyoshi R, Moulin B, Cuhna F, Castro E, Nunes A, et al. Syncope and complete atrioventricular block related to pulmonary thromboembolism. Arq Bras Cardiol 2004;83:438–41.
[7] Makavos G, Cholevas NV, Rallidis LS. A reversible cause of hypoxaemia in a patient with acute right ventricular infarction. Hellenic J Cardiol 2019 [Article in press].
[8] Albaghdadi A, Teleb M, Porres-Agullar M, Porres-Munoz M, Marmol-Velez A. The dilemna of refractory hypoxemia after inferior wall myocardial infarction. Proc Baylor Univ Med Cent 2018;31:67–9.

Response: Pulmonary embolism and shunt in acute myocardial infarction

We thank the authors for these clinically important observations relating to the management of right ventricular (RV) failure in the ED and their discussion of several interesting cases. Pulmonary embolism (PE) should be considered in the differential diagnosis of a patient presenting to the ED with acute RV failure. Additional etiologies to consider include valvular heart disease, tamponade physiology, and cardiomyopathies [1]. Differentiation of PE and acute myocardial infarction (AMI) can be clinically challenging, as ST elevation in leads V1–V4 may be present in up to 5% of acute PE [2]. The time pressure to achieve early revascularization for AMI can lead to delays in recognizing PE [2]. Rarely, AMI and PE can present concomitantly due to paradoxical embolism from the PE across an atrial septal defect (ASD) or patent foramen ovale (PFO) causing AMI [3]. Early cardiology consultation for echocardiography and possible revascularization are critical for this patient population [4]. PE should remain on the differential diagnosis in patients with ECG changes suggestive of AMI, particularly for patients with severe hypoxemia without pulmonary edema or in those with clinical history suggestive of PE [3].

Hypoxemia should be addressed in acute RV failure to decrease RV afterload from hypoxic pulmonary vasoconstriction [1]. The authors point out that hypoxemia may be nonresponsive to supplemental oxygen due to shunt physiology from diastolic dysfunction secondary to RV infarction [5]. Increased pulmonary artery pressures and right atrial pressures may also result in the formation of a right-to-left shunt in the setting of an ASD with a baseline left-to-right shunt. Early revascularization for eligible patients is a priority in ED management [4]. Patients with refractory hypoxemia may require additional workup in the ICU setting to include transesophageal echocardiography with agitated saline contrast to evaluate for PFO [5].

Declaration of Competing Interest

None.

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References

[1] Lahm T, McCaslin CA, Wozniak TC, et al. Medical and surgical treatment of acute right ventricular failure. J Am Coll Cardiol 2010;56(18):1435–46. https://doi.org/10.1016/j.jacc.2010.05.046.
[2] Omar HR. ST-segment elevation in V1–V4 in acute pulmonary embolism: a case presentation and review of literature. Eur Hear J Acute Cardiovasc Care 2016;5(8):579–86. https://doi.org/10.1177/2048872615604273.
[3] Alkhalil M, Cahill TJ, Boardman H, Choudhury RP. Concomitant pulmonary embolism and myocardial infarction due to paradoxical embolism across a patent foramen ovale: a case report. Eur Hear J-Case Reports 2017;1(2). https://doi.org/10.1093/ehjcr/ytx010.
[4] Davis WT, Montrief T, Koyfman A, Long B. Dysrhythmias and heart failure complicating acute myocardial infarction: an emergency medicine review. Am J Emerg Med April 2019. https://doi.org/10.1016/j.ajem.2019.04.047.
[5] Albaghdadi A, Teleb M, Porres-Aguilar M, Porres-Munoz M, Marmol-Velez A. The dilemma of refractory hypoxemia after inferior wall myocardial infarction. Proc (Bayl Univ Med Cent) 2018;31(1):67–9. https://doi.org/10.1080/08998280.2017.1401347.

Understanding the benefits of early high-flow nasal cannula therapy for adults with acute hypoxemic respiratory failure in the ED

To the Editor:

We have read with great interest the recent study published by Macé et al. [1], which described the impact of early high-flow nasal cannula oxygen therapy in adults presenting with acute hypoxemic respiratory failure in the ED; most notably observing faster recovery or regression of respiratory failure with HFNC.

However, we believe this observation necessitates a more precise analysis of the improvement in oxygenation, and the unique factors associated with regression, to better understand the faster recovery of respiratory failure described in this study.

The study population consisted mainly of patients with community-acquired pneumonia with similar PaCO2 values that did not differ in the