Pulmonary embolism (PE) is a cause of cardiac arrest that carries a high mortality rate [1, 2]. In one study, PE was found to be a major missed diagnosis in 84% of autopsies and in another, PE along with ischemic bowel were the major missed diagnosis in 89% of cases [3-6]. Given the high mortality rate associated with this diagnosis, it is imperative that there is a quick, efficient diagnostic tool that can be utilized during cardiopulmonary resuscitation (CPR) efforts. Bedside ultrasonography (US) has become a routine part of the practice of emergency medicine. This tool can be utilized to diagnose pulmonary embolism as a cause of cardiac arrest leading to the ability to effectively treat this fatal disease [7-10]. Treatment of PE in cardiac arrest can consist of thrombolysis, anticoagulation, or thrombectomy. Thrombolysis has been successful in the setting of cardiac arrest, but there has not yet been a randomized controlled trial to determine which treatment has the most significant effect on morbidity and mortality [11-20].

Case report

A 66-year-old male with recently diagnosed stage IV gastric cancer and diabetes was transported to the emergency department with the complaint of acute shortness of breath. EMS found the patient in respiratory distress. His initial rhythm was sinus. He then went into pulseless electrical activity (PEA) arrest in route to the ED. He had return of spontaneous circulation (ROSC) after one round of advanced cardiovascular life support (ACLS). Upon arrival to the emergency department, he was again in full arrest and ROSC achieved shortly after. Bedside transthoracic echocardiography was performed by the Emergency Medicine resident and attending, revealing a dilated right ventricle with a large thrombus visualized within the right ventricle. Figure 1 shows the parasternal long cardiac ultrasound view. The decision at that time was made to give the patient tPA. He was transported to the ICU and was noted to be alert and following commands the following morning. He was ultimately discharged within a week of arrival neurologically intact.

Massive PE and acute myocardial infarction account for almost two thirds of all out of hospital cardiac arrests of no apparent cause [21, 22]. In those with cardiac arrest, PEA can be found in as many as 63% of cases of PE [7, 23]. During massive PE, there is a dramatically diminished return of blood to the left ventricle resulting in marked reduction of cardiac output. However, the electrical conduction of the heart is not immediately reduced, resulting in PEA as the presenting rhythm [24].
The primary cause of death in acute PE is right ventricular (RV) failure. When 25-30% of the pulmonary vascular bed becomes occluded by clot, pulmonary arterial pressures begin to rise in an effort to compensate for the decreased blood flow [25]. This decrease in blood flow leads to further increased pulmonary arterial pressure because of hypoxia induced vasoconstriction in the pulmonary circulation. This increased pressure results in RV dilation and increased RV wall tension. However, the right ventricle is a thin-walled structure and is not conditioned normally to compensate for pressures over 40 mmHg [26]. The increased right ventricular wall tension results in prolonged RV contraction time that can lead to leftward bowing of the interventricular septum as well as a right bundle branch block, which is often seen on echocardiogram and electrocardiogram, respectively, in acute PE [12]. The prolonged RV contraction time ultimately leads to decreased left ventricular (LV) filling and thus decreased cardiac output [27].

The diagnostic test of choice for diagnosing PE is multidetector computer tomographic pulmonary angiography (MDCT-PA) [28-31]. MDCT-PA improved imaging techniques have increased the ability to diagnosis segmental and sub-segmental emboli and has led to a better estimation of the overall thrombus burden. These improvements have led to improved risk stratification for patients, thus allowing better to determine the most appropriate treatment options [32]. However, in the hemodynamically unstable patient, this is not an option. In our patient, knowing that he had metastatic gastric cancer heightened our suspicion of thromboembolic disease, but without ultrasound we would not have had any way of making a concrete diagnosis given his hemodynamic instability. Echocardiography, particularly in the unstable patient, can be utilized to diagnose PE [33-35]. Ultrasonographic findings suggestive of PE include: RV wall hypokinesis, RV dilation, RV apical wall sparing, right atrial enlargement, increased RV/ LV ratio, pulmonary arterial hypertension, tricuspid regurgitation, underfilled hyperdynamic LV, and right heart thrombus [12, 33, 35-39]. In our patient, we began our bedside echocardiography by utilizing the parasternal long view. We visualized a dilated right ventricle. The finding of a dilated right ventricle alone on transthoracic echocardiography (TTE) is fairly specific for PE (87%-98%) though not very sensitive for PE (31-72%) [40-43]. RV hypokinesis can be found in up to 92% of individuals with acute, large PE [44, 45]. McConnell's sign (regional wall motion abnormalities sparing the right ventricular apex), if present, have a 94% specificity for a PE [38]. Although, this was further questioned in a study looking at findings of PE, as well as RV infarction. They found that up to two thirds of individuals with RV infarction may have apical sparing [46]. On further scanning, we were able to visualize a mobile thrombus in the right ventricle.

Right heart thrombus (RHT) is not commonly seen in PE and can be found in 4-23% of cases [8, 17, 47-51]. The mortality rate in these instances is very high, up to 100% without treatment and 45% with treatment [20, 47, 51]. While there are many causes of RHT, the vast majority (up to 90%) are caused by deep venous thrombus [52]. The finding of right heart thrombus is diagnostic of PE and justifies reperfusion treatment [11, 20, 50, 53-55]. We were able to visualize a thrombus in the right ventricle during our bedside echocardiogram, thus allowing us to appropriately target our therapy towards the now definitely diagnosed cause of PEA cardiac arrest.

Therapy choices for RHT published in case reports have included anticoagulation with heparin, systemic thrombolysis, surgical embolectomy, and percutaneous embolectomy [18, 56-58]. These therapies have shown varied rates of success and mortality and there is no current consensus amongst physicians as to which therapy is best. In massive PE, thrombolysis is often the accepted method of treatment [59-62]. In a retrospective case series that evaluated 177 cases of right heart thromboembolism, the mortality post thrombolysis was significantly lower than with surgery or anticoagulation alone (11.3% vs 23.8% and 28.6% respectively) [20]. In another study, individuals with right heart thrombus treated with heparin alone had a worse prognosis [54]. The most feared complication of thrombolysis is bleeding. In one study, the overall risk of hemorrhage with the use of thrombolytics as reported as 6-20% [63]. Thrombolysis was risky in our patient with metastatic cancer, but he was also a poor surgical candidate and we decided this therapy was worth the risk given the potential benefits. The fact that our patient was able to walk out of the hospital neurologically intact, supports the potential benefits. The evidence that thrombolysis can be a successful therapy. Our case further supports the need for additional studies, particularly randomized control trials, which would provide guidance in future treatment of RHT.

Discussion

We present a case of metastatic cancer causing PE that lead to PEA arrest. Bedside transthoracic echocardiography was employed in the work-up of this patient during CPR and right heart thrombus was visualized in real time. Given the clinical instability of the patient, the decision was made to treat with thrombolysis. The use of bedside ultrasonography is instrumental in diagnosing critical medical problems and in this case it helped to provide a favorable patient outcome. The use of thrombolytics also contributed to the rapid improvement in the overall clinical

Figure 1 Parasternal long cardiac ultrasound view. This PSL cardiac view shows a dilated right ventricle concerning for right heart strain. The left ventricle is collapsed.
course. Our case report is in agreement with previous case reports of patients that have survived after both the diagnosis by ultrasound of right heart thrombus and treatment with thrombolyis [50, 64-67]. This case further supports the needs for a randomized controlled trial to better determine the best treatment option for right heart thrombus during cardiac arrest [14].

Conclusion
Use of bedside echocardiography in the emergency department allowed for the timely diagnosis of massive pulmonary embolism in this patient. Routine use of thrombolytics is not a part of ACLS protocol. Without the aid of this advanced technology, this patient would not have received the treatment he needed to survive his cardiac arrest as he was too unstable for MDCT-PA. This case is evidence of the importance of use of bedside ultrasound in the diagnosis of pulmonary embolism in unstable emergency medicine patients.

Conflicts of interest
Authors declare no conflicts of interest.

References
[1] Doerge HC, Schoendube FA, Looser H, Walter M, Messmer BJ. Pulmonary embolometry: Review of a 15-year experience and role in the age of thrombolytic therapy. Eur J Cardiothorac Surg.1996; 10(11):952–957.
[2] Schmid C, Zietlow S, Wagner TO, Laas J, Bort HS. Fulminant pulmonary embolism: symptoms, diagnostics, operative technique, and results. Ann Thorac Surg. 1991; 52(5):1102–1105.
[3] Hauch O, Jørgensen LN, Khattar SC, Teglbjærg CS, Wiblin AB, et al. Fatal pulmonary embolism associated with surgery. An autopsy study . Acta Chir Scand. 1990; 156(11-12):747–749.
[4] Silfström T. Cause of death in unsuccessful prehospital resuscitation. J Intern Med.1991; 229(4):331–335.
[5] Bedell SE, Fulton EJ. Unexpected findings and complications at autopsy after cardiopulmonary resuscitation (CPR). Arch Intern Med. 1986; 146(9):1725–1728.
[6] Karwinski B, Svendsen E. Comparison of clinical and postmortem diagnosis of pulmonary embolism. J Clin Pathol. 1989; 42(2):135–139.
[7] Kürkçüyan I, Meron G, Sterz F, Sterz F, Janata K, et al. Pulmonary embolism: Results of a multicenter registry. J Am Coll Cardiol. 1996; 33(2):138–139.
[8] Chung-Esaki H, Knight R, Noble J, Wang R, Coralic Z. Detection of acute pulmonary embolism by bedside ultrasound in a patient presenting in PEA arrest: A case report. Case Rep Emerg Med. 2012; 2012:794019.
[9] Casazza F, Bongarzoni A, Centonze F, Morpurgo M. Prevalence and prognostic significance of right-sided cardiac mobile thrombi in acute massive pulmonary embolism. Am J Cardiol. 1997; 79(10):1433–1435.
[10] Künkler R, Weisman DS, Khan I. Mobile thrombi in three chambers of the heart. J Community Hosp Inter Med Perspect. 2013; 3(3-4):1–3.
[11] Jammal M, Milano P, Cardenas R, Mailhot T, Mandavia D, et al. The diagnosis of right heart thrombus by focused cardiac ultrasound in a critically ill patient in compensated shock. Crit Ultrasound J. 2015; 7:6.
[12] Rose PS, Punjabi NM, Pearse DB. Treatment of right heart thromboemboli. Chest. 2002; 121(3):806–814.
[13] Vanbrabant P, Dhondt E, Billen P, Sabbe M. Aetiologie of unsuccessful prehospital witnessed cardiac arrest of unclear origin. Eur J Emerg Med. 2006; 13(3):144–147.
[14] Bhattacharjee N, Vonggourou H, Krames S. Acute myocardial infarction and massive pulmonary embolus presenting as cardiac arrest: Initial rhythm as a diagnostic clue. Case Rep Emerg Med. 2013; 2013:343918.
[15] Comess KA, DeRook FA, Russell ML, Tognazzi-Evans TA, Beach KW. The incidence of pulmonary embolism in unexplained sudden cardiac arrest with pulseless electrical activity. Am J Med. 2000; 109(5):351–356.
[16] Ewy GA. Defining electromechanical dissociation. Ann Emerg Med. 1984; 13(9 Pt 2):830–832.
[17] McIntyre KM, Sasaahara AA. The hemodynamic response to pulmonary embolism in patients without prior pulmonary disease. Am J Cardiol. 1991; 78(3):288–294.
[18] Manthous JC, McLaughlin V. Acute right ventricular failure in the setting of acute pulmonary embolism or chronic pulmonary hypertension: A detailed review of the pathophysiology, diagnosis, and management. Curr Cardiol Rep. 2008; 4(1):49–59.
[19] Konstantinides SV, Torbicki A, Agnelli G, Danchin N, Fitzmaurice D, et al. 2014 ESC guidelines on the diagnosis and management of acute pulmonary embolism. Eur Heart J. 2014; 35(43):3033–3036.
[20] Mos IC, Klok FA, Kroft LJ, de Roos A, Huisman MV. Imaging tests in the diagnosis of pulmonary embolism. Semin Respir Crit Care Med. 2012; 33(2):138–143.
[21] van der Hulle T, Dronkers CE, de Roos A, Huisman MV. Recent developments in the diagnosis and treatment of pulmonary embolism. J Intern Med. 2016; 279(1):16–29.
[22] Galanau JP, Blanchet-Devery A, Peronod G, Quéret I. Pour le Collège des enseignants de médecine vasculaire. Management of pulmonary embolism: A 2015 update. J Mal Vasc. 2016; 41(1):51–62.
[23] Devraj A, Sayer C, Sheard S, Grubbnic S, Nair A, et al. Diagnostic acute pulmonary embolism with computed tomography: Imaging update. J Thorac Imaging. 2015; 30(3):176–179.
[24] Hoang JK, Lee WK, Hennessy OF. Multidetector CT pulmonary angiography features of pulmonary embolus. J Med Imaging Radiat Oncol. 2008; 52(4):307–317.
[25] Guidelines on diagnosis and management of acute pulmonary embolism. Task Force on pulmonary embolism, European Society of Cardiology. Eur Heart J. 2000; 21(16):1301–1336.
[26] Goldhaber SZ, Visani L. Acute pulmonary embolism: symptoms, diagnostics, operative technique, and results. Cardiology. 1996; 87(2):169–174.
[27] Mansencal N, Dubourg O. Free-floating thrombus in the right heart and pulmonary embolism. Int J Cardiol. 2006; 112(2):e33–34.
[28] Devaraj A, Sayer C, Sheard S, Grubbnic S, Nair A, et al. Regional right ventricular dysfunction detected by echocardiography among patients with acute pulmonary embolism: A systematic review of the pathophysiology, diagnosis, and management. Curr Cardiol Rep. 2008; 10(11):952–957.
[29] van der Hulle T, Dronkers CE, Klok FA, Kroft LJ, de Roos A, Huisman MV. Imaging tests in the diagnosis of pulmonary embolism. Semin Respir Crit Care Med. 2012; 33(2):138–143.
[30] van der Hulle T, Dronkers CE, de Roos A, Huisman MV. Recent developments in the diagnosis and treatment of pulmonary embolism. J Intern Med. 2016; 279(1):16–29.
[31] Galanau JP, Blanchet-Devery A, Peronod G, Quéret I. Pour le Collège des enseignants de médecine vasculaire. Management of pulmonary embolism: A 2015 update. J Mal Vasc. 2016; 41(1):51–62.
[32] Devraj A, Sayer C, Sheard S, Grubbnic S, Nair A, et al. Diagnostic acute pulmonary embolism with computed tomography: Imaging update. J Thorac Imaging. 2015; 30(3):176–179.
[33] Hoang JK, Lee WK, Hennessy OF. Multidetector CT pulmonary angiography features of pulmonary embolus. J Med Imaging Radiat Oncol. 2008; 52(4):307–317.
[34] Guidelines on diagnosis and management of acute pulmonary embolism. Task Force on pulmonary embolism, European Society of Cardiology. Eur Heart J. 2000; 21(16):1301–1336.
[35] Goldhaber SZ, Visani L, De Rosa M. Acute pulmonary embolism: clinical outcomes in the International Cooperative Pulmonary Embolism Registry (ICOPER). Lancet. 1999; 353(9162):1386–1389.
[36] Stawicki SP, Seamon MJ, Kim PK, Meredith DM, Chovanes J, et al. Transthoracic echocardiography for pulmonary embolism in the ICU: Finding the “right” findings. J Am Coll Surg. 2008; 206(1):42–47.
[37] Kucher N, Rossi E, De Rosa M, Goldhaber SZ. Prognostic role of echocardiography among patients with acute pulmonary embolism and a systolic arterial pressure of 90 mmHg or higher. Arch Intern Med. 2005; 165(15):1777–1781.
[38] Sanchez O, Trinquart L, Colombet I, Durieux P, Huisman MV, et al. Prognostic value of right ventricular dysfunction in patients with haemodynamically stable pulmonary embolism: A systematic review. Eur Heart J. 2008; 29(12):1569–1577.
[39] McConnell MV, Solomon SD, Rayan ME, Come PC, Goldhaber SZ, et al. Regional right ventricular dysfunction detected by echocardiography in acute pulmonary embolism. Am J Cardiol. 1996; 78(4):469–473.
[40] Dahhan T, Alenezi F, Samad Z, Rajagopal S. Echocardiography in the risk assessment of acute pulmonary embolism. Semin Respir Crit Care Med. 2017; 38(1):18–28.
[41] Nazerian P, Vanni S, Volpicielli G, Gigli C, Zanobetti M, et al. Accuracy of point-of-care multigorgan ultrasonography for the diagnosis of pulmonary embolism. Chest. 2014; 145(5):950–957.
[41] Miniat M, Monti S, Pratali L, Di Ricco G, Marini C, et al. Value of transthoracic echocardiography in the diagnosis of pulmonary embolism: Results of a prospective study in unselected patients. Am J Med. 2001; 110(7):528–535.

[42] Jackson RE, Rudoni RR, Hauser AM, Pascual RG, Hussey ME. Prospective evaluation of two-dimensional transthoracic echocardiography in emergency department patients with suspected pulmonary embolism. Acad Emerg Med. 2000; 7(9):994–998.

[43] Bova C, Greco F, Misuraca G, Serafini O, Crocco F, et al. Diagnostic utility of echocardiography in patients with suspected pulmonary embolism. Am J Emerg Med. 2003; 21(3):180–183.

[44] Mookadam F, Jiamsripong P, Goel R, Warsame TA, Emani UR, et al. Critical appraisal on the utility of echocardiography in the management of acute pulmonary embolism. Cardiol Rev. 2010; 18(1):29–37.

[45] Ribeiro A, Juhlin-Dannfelt A, Brodin LA, Holmgren A, Jorfeldt L. Pulmonary embolism: relation between the degree of right ventricle overload and the extent of perfusion defects. Am Heart J. 1998; 135(5 Pt 1):868–874.

[46] Casazza F, Bongarzoni A, Capozi A, Agostoni O. Regional right ventricular dysfunction in acute pulmonary embolism and right ventricular infarction. Eur J Echocardiogr. 2005; 6(1):11–14.

[47] Chartier L, Béra J, Delomez M, Asseman P, Beregi JP, et al. Free-floating thrombi in the right heart: diagnosis, management, and prognostic indexes in 38 consecutive patients. Circulation. 1999; 99(21):2779–2783.

[48] Huang SL, Chien CH, Chang YC. A floating thrombus of the right ventricle in severe massive pulmonary embolism. Am J Emerg Med. 2008; 26(9):1071.e1–2.

[49] Panduranga P, Mukhaini M, Saleem M, Al-Delamie T, Zachariah S, et al. Mobile right heart thrombus with pulmonary embolism in a patient with polycythemia rubra vera and splanchic vein thrombosis. Heart Views. 2010; 11(1):16–20.

[50] Greco F, Bisignani G, Serafini O, Guzzo D, Stingone A, et al. Successful treatment of right heart thromboemboli with IV recombinant tissue-type plasminogen activator during continuous echocardiographic monitoring: A case series report. Chest. 1999; 116(2):538–535.

[51] Ferrari E, Benhamou M, Berthier F, Baudouy M. Mobile thrombi of the right heart in pulmonary embolism: delayed disappearance after thrombolytic treatment. Chest. 2005; 127(3):1051–1053.

[52] Matzdorff AC, Green D. Deep vein thrombosis and pulmonary embolism: prevention, diagnosis, and treatment. Geriatrics. 1992; 47(8):54–56.

[53] Kucher N, Luder CM, Dörnhöfer T, Windecker S, Meier B, et al. Novel management strategy for patients with suspected pulmonary embolism. Eur Heart J. 2003; 24(4):366–376.

[54] Dalen JE, Alpert JS, Hirsh J. Thrombolytic therapy for pulmonary embolism: Is it effective? Is it safe? When is it indicated? Arch Intern Med. 1997; 157(22):2550–2556.

[55] Willy LA, Krichman A, Tapson VF. Thrombolytic therapy for venous thromboembolism. Utilization by practicing pulmonologists. Arch Intern Med. 1994; 154(14):1601–1604.

[56] Davies MG, El-Sayed H. Current status of clot removal for acute pulmonary embolism. Ann Vasc Surg. 1997; 10(2):142–145.

[57] Saura D, García-Puente J, Oliva MJ, et al. Treatment of right heart thromboemboli: The need of a randomized multicentre trial. Int J Cardiol. 2009; 134(3):419–420.

[58] Soga T, Sato Y, Matsumoto N, Tachibana E, Watanabe K, et al. Right atrial thrombi: percutaneous mechanical thrombectomy. Cardiovasc Intervent Radiol. 1997; 20(2):142–145.

[59] Harris T, Meek S. When should we thrombolise patients with pulmonary embolism? A systematic review of the literature. Emerg Med J. 2005; 22(11):766–771.