

Massive Pulmonary Embolism after Prolonged Spinal Fusion Surgery and the Golden Hour

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Abstract

Massive pulmonary embolism in the perioperative period almost inevitably leads to death if a delay in diagnosis and treatment occurs. The time window in which treatment should be initiated in order to reduce morbidity and mortality is very short; therefore a so-called golden hour for pulmonary embolism exists. Preoperatively, the role of ultrasound in the diagnosis of this potential life threatening condition can be crucial. The present case report describes the role of transesophageal echocardiography after spinal fusion surgery in getting a timely diagnosis and treatment of massive perioperative pulmonary embolism.

Keywords: Pulmonary embolism; Transesophageal echocardiography; Major surgery

Introduction

Pulmonary embolism after spinal surgery is not uncommon [1-3]. Apart from stasis and the general hypercoagulable state during surgery [4,5], specific problems arise during orthopedic surgery. The use of a stem or rod in long bones, but also the use of spinal implants in degenerative lumbar spinal disorders may result in fat and bone marrow micro emboli [2]. These can be obstructive themselves, or grow into large emboli by additional blood thrombi. Mortality rates after PE depend on the clinical condition at presentation but may be as high as 52% when patients present with an initial systolic blood pressure < 90 mmHg. Mortality rate increases to 65% when patients need cardiopulmonary resuscitation [6]. Accordingly, massive PE is defined as acute PE with sustained hypotension (systolic blood pressure < 90 mmHg) for at least 15 min or requiring inotropic support [6].

Case Description

A 57-year old woman, weighing 62 kg, ASA physical status 3, was scheduled for elective revision of spinal fusion surgery. Her medical history revealed a 15 pack-year smoking history, hypertension and coronary artery disease, for which a stent was implanted 10 years prior to the date of surgery. Four years after initial placement, a new stent was placed, as restenosis had occurred. At time of surgery there were no signs of coronary artery disease or valvular pathology and recent cardiac evaluation had revealed excellent functional capacity. Her daily medications were continued up to the day of surgery, including aspirin.

General anesthesia was induced with IV propofol (200 mg), sufentanil (40 µg) and rocuronium (50 mg) and was maintained with sevoflurane. Pre-emptive tranexamic acid (2 g) was given to reduce postoperative bleeding. For access of the surgical area, the patient was turned from supine to prone position. For access to the surgical area and to reduce lordosis, the patient was positioned on pelvic pillows.

Spinal fusion was uneventfully completed after 6 hours. Blood loss of 1600 ml was covered with one unit of red blood cells, additionally, 913 ml of cell saver blood was returned to the patient. At the time of wound closure Hb was 7.9 g/dl, BP 103/60 mmHg, and pulse 100 BPM and oxygen saturation 100%. Hereafter, the patient was returned to a supine position and taken to the recovery room while she was kept intubated. Upon arrival at the recovery room, the pulse rose to 122 BPM, oxygen saturation decreased to 93% and capnography showed a steady end tidal CO₂ (EtCO₂) of 30-40 mmHg. BP was maintained around 90/55 mmHg with boluses of phenylephrine and continuously rapid infusion of Ringer's acetate solution. Considering the patient's medical history, there was concern about cardiac ischemia, thus metoprolol 3 mg and 2 mg was given to slow down the heart rate. At that time blood gas analysis revealed a pH of 7.3, pO₂ of 265 mmHg, pCO₂ of 50 mmHg, BE -1.2, and a lactate of 1.2 mmol/L. Suddenly the systolic blood pressure decreased to 20 mmHg and cardiopulmonary resuscitation by means of chest compressions was initiated, followed by noradrenaline 0.5 mg i.v. and Ringer's acetate solution infusion. EtCO₂ dropped to 13 mmHg and subsequently arterial blood gas analysis worsened; pH 7.14, pO₂ 235 mmHg, pCO₂ 58 mmHg, BE -10.1 lactate 6.8 mmol/L.

Figure 1 shows the preoperative and 3 serial 12-lead ECGs made over a ten minutes period during the acute postoperative event in the recovery room. During the event ECGs revealed sinus tachycardia and diffuse ST-segment depression in combination with progressive axis

deviation towards the right. In addition to the development of an S wave in lead I and a Q wave in lead III with an incomplete right bundle branch block (RBTB) developed, suggestive of acute right ventricular strain.

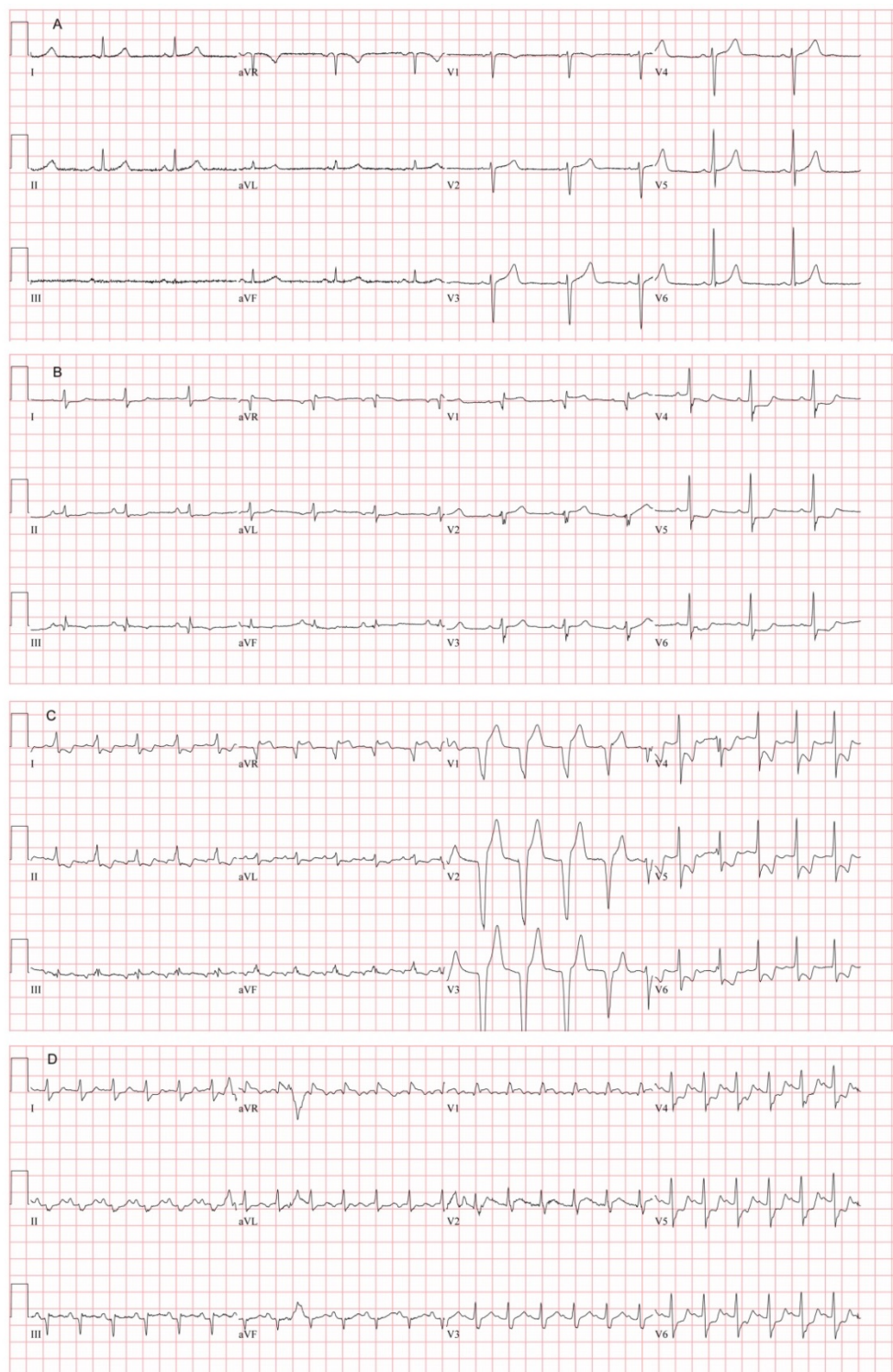
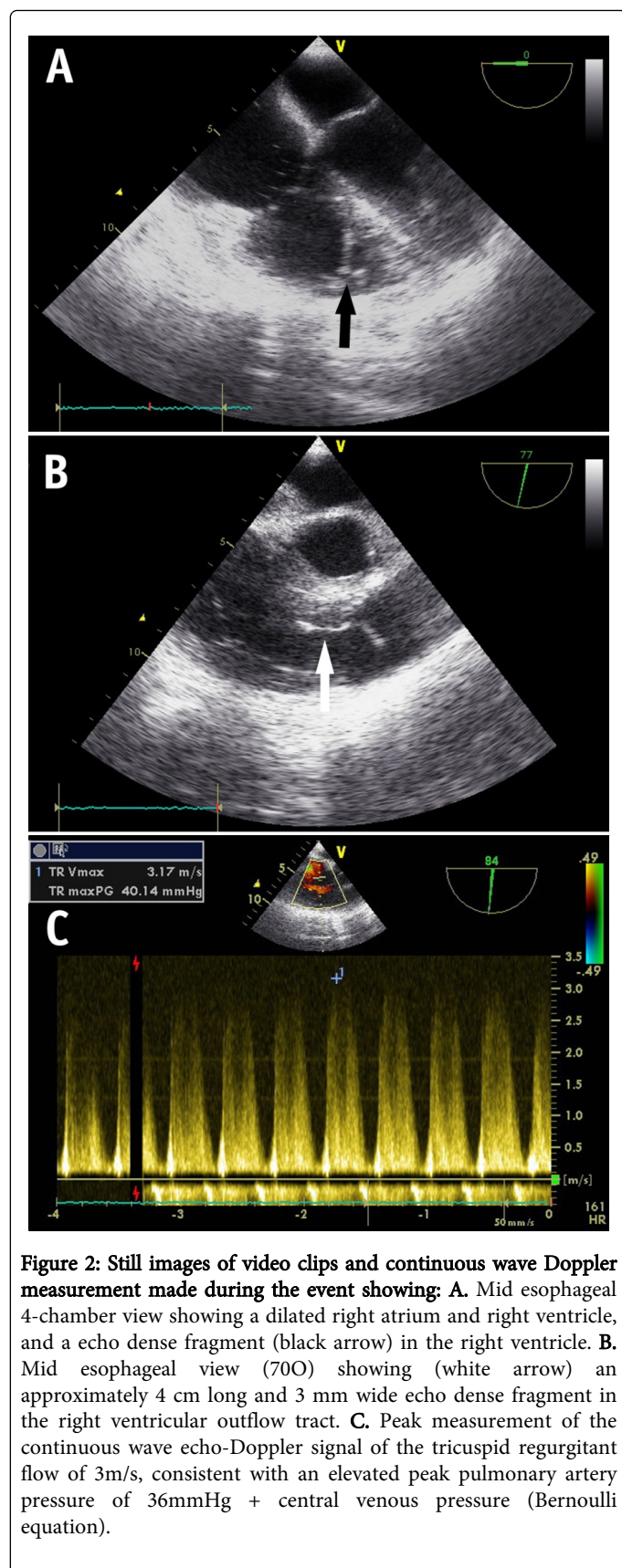


Figure 1: ECG recordings. A: preoperative, B-D during the event over a ten minutes period. ECGs showed sinus tachycardia and diffuse ST-segment depression in combination with progressive axis deviation towards the right. In addition to the development of an S wave in lead I and a Q wave in lead III with an incomplete right bundle branch block (RBTB) developed, suggestive of acute right ventricular strain.



Within 15 min after arrival at the recovery room, transesophageal echocardiography (TEE) was conducted this showed a well contracting non dilated left ventricle, a dilated right ventricle, significant tricuspid valve insufficiency, and bulging of the dilated right atrium into the left atrium. In the right ventricle a mobile, echo dense fragment attached to the ventricular wall was visualized (Figure 2, Videos 1 and 2 (supplementary file)). The maximum tricuspid regurgitant flow velocity was 3 m/s, consistent with an elevated peak pulmonary artery pressure of 36 mmHg + central venous pressure (Bernoulli equation). Taken together, ultrasound findings (signs of increased right ventricular pressures, a moving fragment in the right ventricle), the increased EtCO₂ to arterial pCO₂ gap and signs of pan-ischemia on the ECG (an S wave in lead I, a Q wave in lead III and a newly developed incomplete RBTB) were strongly suggestive of massive pulmonary embolism. The patient was administered continuous fluid infusion and noradrenaline until blood pressures remained stable and chest compressions were no longer needed; she was then quickly transported to the interventional radiology room. Angiography showed bilateral large thrombi, occupying large and small vessels of the left and right pulmonary circulation leaving minimal flow to peripheral pulmonary circulation (Figure 3). Approximately 90 minutes after recovery room admission, successful thrombolysis by fragmentation with a guidewire and local application of actilyse 20 mg was performed.

After thrombolysis, her vital signs were stable, ECG recordings normalized and the patient was transferred to the intensive care unit (ICU) where she received standard care according to protocol, including induced mild hypothermia therapy to protect brain function and a continuous heparin infusion for prevention of recurrent thromboembolic events. On the second postoperative day the patient woke up with mild cognitive abnormalities, which recovered within 4 days. As the patient continued to use heparin after her near fatal pulmonary embolism, she underwent several repeat procedures as a result of bleeding complications of the surgical area in the three months after the initial procedure.

Discussion

Our case is exemplary for the value of TEE in getting a quick diagnosis and treatment of massive pulmonary embolism. Early diagnosis in the direct postoperative period without echocardiography is difficult by the fact that cardiovascular collapse may be caused by postoperative bleeding, myocardial infarction, anaphylaxes or ventilatory insufficiency. Although ECG changes may be indicative of pulmonary embolism, ECG changes might easily be overlooked or misinterpreted by the overwhelming signs of cardiac ischemia especially in patients with known cardiovascular disease. Indeed, serial ECG changes in our patient showed signs of ischemia as well as signs consistent with massive pulmonary embolism. Therefore valuable time may be lost, delaying the prompt transportation of the patient to the intervention radiology department for thrombolysis or (in case of absolute contraindications for thrombolysis) catheter embolectomy or fragmentation of proximal pulmonary arterial clots [7].

Of all fatal cases of PE, approximately two-thirds will die within the first hour after presentation [8]. The so-called “golden hour” after massive PE is directly related to the underlying pathophysiologic mechanism. Thromboemboli increase the pulmonary vascular resistance, thereby acutely increasing the afterload of the right

ventricle. Hereafter, three possible scenarios occur: 1) sudden death based on electromechanical dissociation, 2) hypotension and shock due to acute right ventricular failure, superimposed with left ventricular failure due to diastolic dysfunction caused by the bulging right ventricle, eventually leading to quick death, 3) initial survival due

to systemic activation of the sympathetic system, resulting in inotropic and chronotropic stimulation. However, the sequel of these events can easily accumulate in a vicious circle, which has to be interrupted as early as possible if survival is to be the outcome [7-10]. Therefore early diagnosis is extremely important.

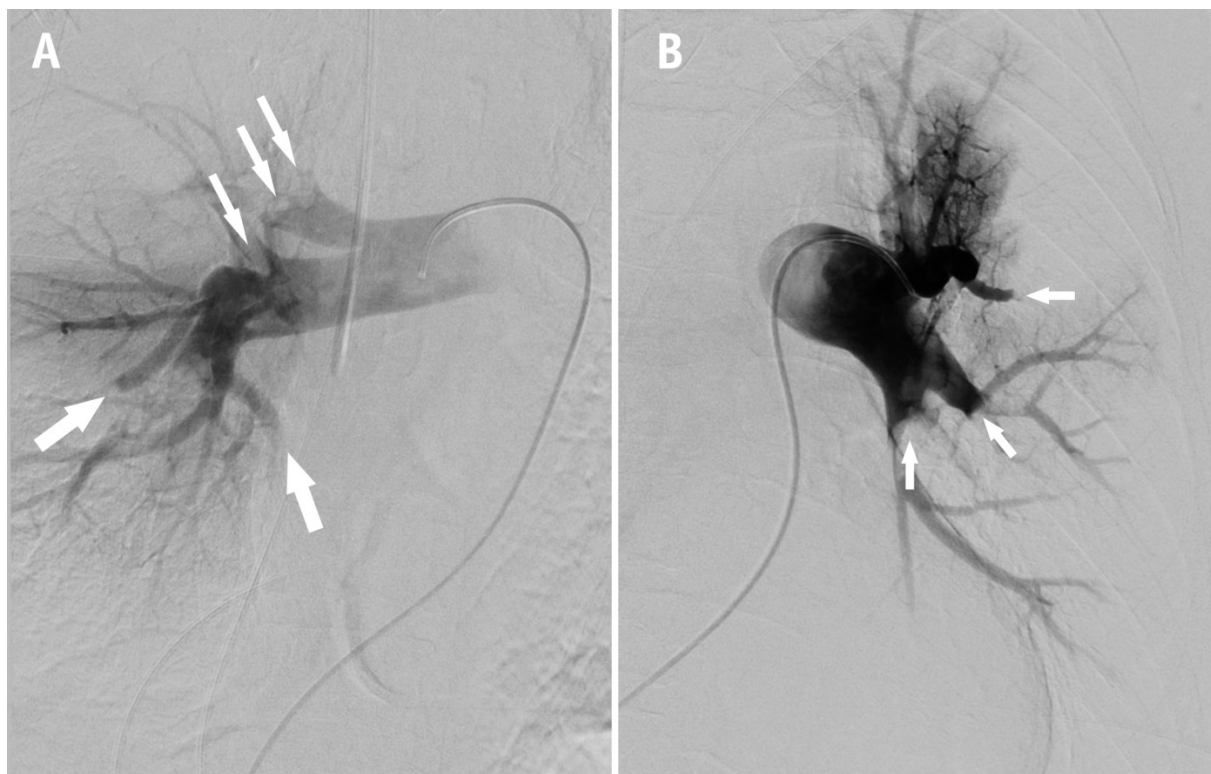


Figure 3: Angiography of right (A) and left (B) pulmonary artery showing (white arrows) occluded arteries and diminished flow to the periphery, indicating large thrombi bilaterally.

In order to start effective treatment within the golden hour of massive PE the European Society of Cardiology is very clear about the preferred diagnostic step when immediate access to CT is not available: echocardiography is to be performed [11]. Furthermore, the use of TEE in situations of unexplained hemodynamic disturbances, suspected valvular disease or thromboembolic problems in unstable patients is considered a category I indication in the American Society of Anesthesiologists guidelines [12]. The use of perioperative echocardiography was proven very useful in many cases, and excellent earlier reports have been written [13,14]. Abnormal echocardiographic findings that can be found in patients with acute massive PE are related to acute right ventricular failure with concomitant phenomena such as right ventricular dilation with hypo kinesis, leftward septal shift, tricuspid regurgitation increased right sided filling pressures and right atrial enlargement [15]. In 85 patients with RV dysfunction of any cause including 13 patients with proven PE, the presence of right ventricular dysfunction with sparing of the apex had a sensitivity of 77% and a specificity of 94% [16] for PE (McConnell's sign).

Apart from the echocardiographic changes indicative of acute right ventricular failure we were able to visualize a moving fragment in the right ventricle that appeared to be a thrombus. Indeed, direct visualization of thrombi by TEE can be done [17]; a sensitivity of 80% and a specificity of 100% have been reported [18]. Difficulty to directly

visualize thrombi and thereby lower sensitivity is related to the often-distal location of the thrombi in the pulmonary vascular tree, and the location of the air-filled left main bronchus, which can obscure the vision of left main pulmonary artery located thrombi. Additionally, patients with massive PE are often hemodynamically unstable and require continuous resuscitation efforts, which can result in sub-optimal conditions of performing TEE [19].

Our case shows the challenges the anesthesiologist is faced with considering the diagnosis and subsequent quick initiation of treatment for thromboembolic disease and massive PE in particular. Perioperatively TEE proved to be of great value for early diagnosis to interrupt the deathly vicious circle within the golden hour of massive PE.

References

1. Schizas C, Neumayer F, Kosmopoulos V (2008) incidence and management of pulmonary embolism following spinal surgery occurring while under chemical thromboprophylaxis. *Eur Spine J* 17: 970-974.
2. Takahashi S, Kitagawa H, Ishii T (2003) Intraoperative pulmonary embolism during spinal. Instrumentation surgery *The Journal of bone and joint surgery*.

3. Takai N, Takasugi Y, Kajikawa R, Takase T, Yamamoto Y, et al. (2014) Fatal pulmonary embolism following spinal surgery in a patient with permanent inferior vena cava filter placement. *J Anesth* 28: 606-609.
4. White H, Zhou H, Romano S (2003) Incidence of symptomatic venous thromboembolism after different elective or urgent surgical procedures. *Thrombosis and Haemostasis* 90: 446-455.
5. Rogers SO, Kilaru RK, Hosokawa P, Henderson WG, Zinner MJ, et al. (2007). Multivariable predictors of postoperative venous thromboembolic events after general and vascular surgery: results from the patient safety in surgery study. *J Am Coll Surg* 204: 1211-1221.
6. Jaff MR, McMurtry MS, Archer SL, Cushman M, Goldenberg N, et al. (2011) American Heart Association Council on Arteriosclerosis, Thrombosis and Vascular Biology Management of massive and submassive pulmonary embolism, iliofemoral deep vein thrombosis, and chronic thromboembolic pulmonary hypertension: a scientific statement from the American Heart Association. *Circulation* 123: 1788-1830.
7. Torbicki A, Perrier A, Konstantinides S, Agnelli G, Galiè N, et al (2008) ESC Committee for Practice Guidelines (CPG). Guidelines on the diagnosis and management of acute pulmonary embolism: the Task Force for the Diagnosis and Management of Acute Pulmonary Embolism of the European Society of Cardiology (ESC). *Eur Heart J* 29: 2276-2315.
8. Wood KE (2002). Major pulmonary embolism: review of a pathophysiologic approach to the golden hour of hemodynamically significant pulmonary embolism. *Chest* 121: 877-905.
9. Hall D (2013) Perioperative pulmonary embolism: detection, treatment, and outcomes. *Am J Ther* 20: 67-72.
10. Tapson VF (2008) Acute pulmonary embolism. *N Engl J Med* 358: 1037-1052.
11. Konstantinides SV, Torbicki A, Agnelli G, Danchin N, Fitzmaurice D, et al (2014) ESC Guidelines on the diagnosis and management of acute pulmonary embolism: The Task Force for the Diagnosis and Management of Acute Pulmonary Embolism of the European Society of Cardiology (ESC). *Eur Heart J* 35: 3033-3069.
12. (1996) Practice guidelines for perioperative transesophageal echocardiography. A report by the American Society of Anesthesiologists and the Society of Cardiovascular Anesthesiologists Task Force on Transesophageal Echocardiography. *Anesthesiology* 4: 986-1006.
13. Shillcutt SK, Markin MW, Montzingo CR, Brakke TR (2012) Use of rapid "rescue" perioperative echocardiography to improve outcomes after hemodynamic instability in noncardiac surgical patients. *J Cardiothorac Vasc Anesth* 3: 362-370.
14. Memsoudis S, Rosenberger P, Löffler M, Eltzhig HK, Mizuguchi A, (2006) Usefulness of transesophageal echocardiography during intraoperative cardiac arrest. *Anesth Analg* 102: 1653-1657.
15. Goldhaber SZ (2002) Echocardiography in the management of pulmonary embolism. *Ann Intern Med* 136: 691-700.
16. McConnell MV, Solomon SD, Rayan ME, Come PC, Goldhaber SZ, et al (1996) Regional right ventricular dysfunction detected by echocardiography in acute pulmonary embolism. *Am J Cardiol* 78: 469-473.
17. Rosenberger P, Shernan SK, Mihaljevic T, Eltzhig HK (2004). Transesophageal echocardiography for detecting extrapulmonary thrombi during pulmonary embolectomy. *Ann Thorac Surg* 78: 862-866.
18. Pruszczyk P, Torbicki A, Pachó R, Chlebus M, Kuch-Wocial A, et al (1997) Noninvasive diagnosis of suspected severe pulmonary embolism: transesophageal echocardiography vs spiral CT. *Chest* 112: 722-728.
19. Rosenberger P, Shernan SK, Body SC, Eltzhig HK (2004) Utility of Intraoperative Transesophageal Echocardiography for Diagnosis of Pulmonary Embolism *Anesthesia & Analgesia* 99: 12-16.