GKDA Derg 20(1):49-54, 2014
doi:10.5222/GKDAD.2014.049

Olgu Sunumu

Anaesthetic Management of The Patient Undergoing Cardiopulmonary Bypass Graft Surgery After Pneumonectomy

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SUMMARY

The aim of this case report is to discuss the recommendations for preoperative evaluation, the technical difficulties experienced in the perioperative period and the ventilation methods used to minimize pulmonary injury in a post-pneumonectomy patient undergoing coronary artery bypass grafting surgery.

Key words: *anaesthetic management, cardiac surgery, pneumonectomy*

ÖZET

Pnömonektomi Sonrası Kardiyopulmoner Baypas Cerrahisi Geçiren Hastada Anestezi Yönetimi

Bu olgu sunumunun amacı, pnömonektomi sonrası koroner arter baypas greftleme cerrahisi geçiren hastada, preoperatif değerlendirme önerilerini, perioperatif dönemde yaşanan teknik zorlukları ve pulmoner hasarı azaltmak için kullanılan ventilasyon yöntemlerini tartışmaktır.

Anahtar kelimeler: *anestezi yönetimi, kardiyak cerrahi, pnömonektomi*

INTRODUCTION

Most anaesthesiologists have limited experience in caring for patients undergoing cardiopulmonary bypass surgery who have undergone prior pneumonectomy. To date, reports summarizing only 21 cases have been published [1,2]. After pneumonectomy, the respiratory and cardiovascular systems adapt to the absence of one lung and various compensatory mechanisms which result in the development of restrictive lung disease in the remaining lung [3]. Right ventricular (RV) morphology also adapts itself to the absence of one lung, and with time, moderate tricuspid valve insufficiency and significantly higher pulmonary artery systolic pressures are seen [4,5]. Normal anatomic relationships -of the thorax are altered after pneumonectomy - mediastinal structures and thoracic organs shift toward the empty hemithorax, and fill this area as a compensatory mechanism. Deviation of the trachea and rotation of the heart and great vessels may lead to difficulties in intubation, central venous access, surgical cannulation, and surgical access to the coronary vessels [1,2].

Acute lung injury (ALI) is the most important pulmonary complication occurring in post-pneumonectomy patients undergoing cardiopulmonary bypass (CPB) [1]. Perioperative anaesthesia management is by necessity different in these patients in terms of mode of ventilation, lung protective strategies, and fluid management. The aim of this case report is to give details regarding preoperative technical difficulties we experienced, ventilation modes we used to minimize pulmonary injury, and recommendations for preoperative evaluation of post-pneumonectomy patients who will undergo coronary artery bypass graft (CABG) surgery.

Alındığı tarih: 24.12.2013
Kabul tarihi: 18.02.2014
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CASE REPORT

A 65-year-old man who had had a right pneumonectomy due to squamous cell carcinoma at age 43 and thyroidectomy at age 60 was admitted to the hospital with symptoms of unstable angina 10 days prior to our consultation. Cardiac catheterization revealed triple vessel disease with 50% stenosis of the left main coronary artery (LMCA), 80% stenosis of the proximal and distal segments of the left anterior descending coronary artery (LAD), and 40% stenosis of the middle segment of the right coronary artery (RCA). Left ventricular ejection fraction was 55 percent.

We examined the patient after he was scheduled for coronary bypass grafting. The patient had no sequelae or complaints related to his prior pneumonectomy. His history did not reveal any evidence of valvular disease, atrial fibrillation, major arrhythmias, or heart surgery. His medications included ACE inhibitors and beta blockers. He had an 86 pack-year smoking history, but had stopped smoking 12 years ago. He denied coughing or dyspnea. On physical examination, he had no inspiratory or expiratory stridor. His PA chest X-ray is given in Figure 1. His thoracic CT scan showed an enlarged left hemithorax and lung, deviation of the trachea and left main bronchus to the right, non-compression of the left upper and lower lobe bronchi, and a small right hemithorax which was partially occupied by mediastinal structures. The right internal jugular vein appeared to lie in a position which would allow for routine insertion of a central catheter. Although the quality of echocardiography was suboptimal due to overexpansion of the remaining lung, serofibrothorax and shifted heart, transthoracic echocardiography found his ejection fraction to be 55% without significant valvular regurgitation. The pulmonary function test showed a restrictive pattern: FEV1 was 2.5 L (76% of predicted), FVC was 3.6 L (80% of predicted), and FEV1/FVC was 75% of the predicted values.. Preoperative arterial blood gas analysis showed an increase in alveolar-arterial oxygen gradient (Table 1). One week prior to the operation, the patient began daily respiratory physiotherapy exercises in order to optimize the health of the remaining lung.

In the operating room, the patient was monitored routinely. We prepared the skin and readied the equipment so as to place a triple lumen 7 F central venous catheter (Braun®, Melsungen, Germany) by Seldinger technique in the right internal jugular vein (on the side of the prior pneumonectomy). Puncture of the internal jugular vein (IJV) was successful, but the guide wire could only be advanced 10 cm before resistance was felt. Realizing that the IJV was probably more out of place (due to the ipsilateral pneumonectomy) than we had predicted from the CT scan, we thought it best not to make further attempts at guide wire insertion at this site. Therefore, a central venous catheter was inserted through the right femoral vein.

Anaesthesia was induced with IV fentanyl and etomidate, then the patient was intubated with IV rocuronium. Anaesthesia was maintained with fentanyl and sevorane inhalation. Ventilation was managed by pressure-controlled ventilation (Datex-Ohmeda Aestiva S/5 Avance ventilator and anesthesia monitor, GE Healthcare, Madison, USA), as a component of our lung protective strategy (Table 1). Prednisone was given before (IV 8 mg kg^{-1}) and during CPB (IV 8 mg kg^{-1}). After the induction, the mean arterial pressure fell (70 mm Hg to 45 mm Hg) and haemodynamic status was maintained with the help of inotropic (dobutamine; 5 $\mu g.kg^{-1}min^{-1}$) and vasoconstrictor (noradrenaline 0.15 $\mu g.kg^{-1}min^{-1}$) agents.

When the chest was opened, the heart and aorta were seen to be shifted towards the right thorax and rotated to the right, while the apex of the heart was just under the sternum. Left internal mammary artery (LIMA) was harvested with difficulty because of adhesions related to the prior pneumonectomy operation, but adequate conduit length was achieved to reach the LAD. The right atrium could not be cannulated from the surgeon's side because of the extreme degree of heart rotation. Thus, the operating table was tilted down towards the patient's left side and the surgeon performed the operation from the patient's left side so that he could successfully cannulate the atrium. CPB was done with the patient under moderate (32°C) systemic hypothermia. Cardiac arrest was achieved with antegrade administration of cardioplegic solutionn. Topical cooling was not used in order to avoid the risk of phrenic nerve damage. The left internal mammarian artery was used for left anterior decending coronary bypass (LIMA-LAD) and saphenous vein was used for circumflex coronary bypass (Ao-Cx bypass).

Table 1. Preoperative and intraoperative ventilatory modes in our post-pneumonectomy patient and resulting blood gas parameters.

	Preop at room air	Before CPB PCV: 12 cmH_2O PEEP: 5 cmH_2O PIP: 18 cmH_2O TV: 450 ml RR: 12	During CPB Endotracheal tube left open to air extra-corporal oxygenation)	During weaning PCV: 14 cm H_2O PEEP: 5 cmH_2O PIP: 21 cmH_2O TV: 450 ml RR: 12	1 hr after weaning PCV: 15 cm H_2O PEEP: 5 cmH_2O PIP: 20 cmH_2O TV: 450 ml RR: 15
FiO_2 (mmHg)	0.21	0.5	0.5	0.5	0.5
PaO_2 (mmHg)	88.9	199.4	235.6	74.2	91.1
$PaCO_2$ (mmHg)	39.8	21.8	35.5	49.8*	50.3*
SaO_2 (%)	99.8	99.2	99.4	92.6	95.6
PAO_2 (mmHg)	103	199.4	235.6	91*	91.1*
A-aO_2 (mmHg)	13.9	0	0	16.8*	0
PaO_2/FiO_2	227	398	344	148*	182.2*
Hb (g/dL)	13.5	10.2	7.7	11.1	11.9
Hct (%)	40.2	19.3	21.4	32.4	35.5
pH	7.43	7.44	7.41	7.37	7.30
BE (mmol/L)	1.9	1.3	-2.3	-1	-2.1
HCO_2 (mmol/L)	25.8	20.7	22.5	23.3	22.4
Osm (mOsm/L)	269	281.3	268.3	269	268.5

** Increased alveolar-arterial oxygen gradient, acute lung injury and hypercapnia.*

His TV was estimated according to predicted body weight (73 kg , 6 mL kg^{-1}). He was 181 cm length.
Abbreviations: FiO_2; inspired oxygen concentration, PaO_2 and $PaCO_2$; the partial pressure of oxygen and corbon dioxide in arterial blood, SaO_2; the saturation level of oxygen in hemoglobin, A-aO_2 Alveolar–arterial oxygen gradient, Hb; hemoglobin, Hct; hematocrit, pH; BE; base exes, HCO_3; bicarbonate, Osm; osmolality, Pressure controlled ventilation (PCV), PEEP; positive end expiratory pressure, PIP; peak inspiratory pressure, TV; tidal volume, RR; respiratory rate.

During CPB, the endotracheal tube was left open to air because mediastinal shift of the lung did not allow for ventilation maneuvers such as CPAP during CPB. Before weaning from CPB, inflation of the lungs to a peak airway pressure of 40 cm H_2O for 40 seconds was performed, and after weaning from CPB, 5 cmH2O of PEEP was applied. Blood gas analyses showed signs of acute lung injury and increased alveolar-arterial oxygen gradient at the time of weaning from CPB (Table 1). However, one hour after arrival to the ICU, gas exchange of lung started to return to normal (Hamilton Medical Raphael Xtc ventilator, Bonaduz, Switzerland) (Table 2). These changes in gas exchange were due to shunting in lung during weaning from CPB. During weaning from CPB, systolic blood pressure decreased to 70 mmHg, while his CVP (measured through femoral vein) remained at 6 mmHg. Cardiac dysfunction was unable to be evaluated using a pulmonary artery catheter or TEE (our department have not yet had a TEE). By directly observing the heart and vital signs, we managed the patient's systolic and diastolic pressures by giving dobutamine (5 μg $kg^{-1}min^{-1}$), noradrenaline (0.1 μg $kg^{-1}min^{-1}$) and nitroglycerine (0.1-0.3 μg $kg^{-1}min^{-1}$). While receiving combination of these infusions, the patient was successfully weaned from CPB. All infusions were stopped within four hours after arrival to the ICU. CPB and cross-clamping times were 80 and 45 minutes, respectively. Operation time was 330 min.

After the patient came to the ICU, we continued to use lung protective strategies, giving low tidal volumes and then 5 cm H2O PEEP. His TV was estimated according to the predicted body weight (73 kg, 6 mL kg^{-1}). He was 181 cm tall. In order to avoid overdistension, lower PEEP level of 5 cmH_2O was preferred as recommended. Pressure control mode was used. PaO_2/FiO_2 ratios were followed in an attempt to detect ALI/ARDS. Alveolar-arterial oxygen gradients were followed to detect the development of shunting during and after CPB (Table 2).

The patient did not experience hemodynamic instability or common post-CABG complications such as hypoxemia or atrial fibrillation. The patient was extubated 8 hours after the end of surgery using standard weaning methods. Standard protocol included synchronized intermittent mandatory ventilation (SIMV+PC adjusted to $PaCO_2$ 38-45 mmHg), pressured supported (PS) at level of 10 cm H_2O and then 5 cm H_2O and extubation respectively [6].

Perioperative fluid therapy was given based on central

Table 2. Ventilatory modes and resulting blood gas parameters in the early postoperative period.

	After 1 hour in the ICU	After 4 hours in the ICU	After 6 hours in the ICU	After 11 hours in the ICU (3 hours after extub.)
	PCV:14 cmH_2O PEEP: 5 cmH_2O PIP: 19 cmH_2O TV: 450 ml RR: 14	SIMV + PC: 12 cmH_2O PEEP: 5 cmH_2O PIP: 17 cmH_2O TV: 450 ml RR: 15 (total)	PSV: 10 cmH_2O PEEP: 5 cmH_2O PIP: 15 cmH_2O TV: 400 ml RR: 15 (spont.)	Mask O_2 at 4 L/min RR: 15 (spont.)
FiO_2 (mmHg)	0.5	0.4	0.4	0.3
PaO_2 (mmHg)	164.5	139.2	128	103.8
$PaCO_2$ (mmHg)	43.4	41.6	37	44.8
SaO_2 (%)	99.4	99.2	99.1	99.3
PAO_2 (mmHg)	276	212	218	142
A- aO_2 (mmHg)	131*	73*	100*	39*
PaO_2/FiO_2	330	348	320	346
Hb (g/dL)	9.2	9.8	10.4	10.4
Hct (%)	26.8	28.9	30.7	30.8
pH	7.39	7.43	7.48	7.43
BE (mmol/L)	0.5	2.9	3.7	3.9
HCO_3 (mmol/L)	24.7	26.7	27.4	28.8
Osm (mOsm/L)	272.9	271.5	269.3	270.8

** Increased alveolar-arterial oxygen gradient.*

Abbreviations: PCV; pressure controlled ventilation, SIMV + PC; Synchronised Intermittent Mandatory Ventilation - Pressure Controlled, CPAP; continue positive airway pressure, PEEP; positive end expiratory pressure, RR; respiratory rate

venous pressure monitoring, direct observation of the heart (full vs empty), and monitoring of urine output. In these ways, we avoided fluid overload and minimized the risk of pulmonary edema [7]. Fluid therapy was provided with 1800 mL of a balanced electrolyte solution (Isolyte S®, Eczacıbaşı-Baxter, Istanbul, Turkey) and 500 mL of hydroxyethyl starch solution (HES Steril®, 200/ 0.5, Fresenius Kabi, Bad Homburg, Germany). Intraoperative urine volume was 1600 mL. The volume of urine was 950 ml during CPB, and pump balance was –150 mL. Two units of packed RBCs were transfused to maintain a target hemoglobin level of 10 mg/dL. One unit of FFP was transfused in the post-CPB period. No blood or blood products were given in the ICU. Mediastinal drainage was 350 mL on the first postoperative day. He was discharged from the hospital on postoperative 8th day.

DISCUSSION

Total resection of one lung leads to loss of approximately 45 to 55% of the pulmonary tissue (3). Although respiratory and cardiovascular systems adapt to the patient's present condition who had only one lung, compensatory mechanisms eventually result in restrictive lung disease (3,4). In one study which followed patients for up to four years after pneumonectomy, right ventricular (RV) morphology and function slowly altered to result in RV overload, moderate tricuspid valve insufficiency, and high pulmonary artery pressures [4]. In our patient, cardiac function and valvular tissues were found to be normal on transthoracic echocardiography. This unexpected finding might have been due to the excellent physical condition of the patient (an ex-commando who regularly exercised).

Catheterization of the IJV has certain risks in post-pneumonectomy patients due to its abnormal course. Due to the relatively normal course of our patient's IJV on thoracic CT, we attempted to carefully place a right IJV line but we were unsuccessful. In the light of our experience, we now place a femoral line for central venous catheterization rather than an IJV line in post-pneumonectomy patients, regardless of the IJV's course on thoracic CT. Other method to monitor right and left heart functions intraoperatively was TEE. This technique allows also us to estimate pulmonary artery systolic pressure, the right ventricle diastolic diameter, free wall thickness and the tricuspid valve morphology, and finally the regurgitation jet speed [4,5].

ALI is the most important pulmonary complication (25%) that occurs in post-pneumonectomy patients undergoing CABG, resulting in death in up to 50% of

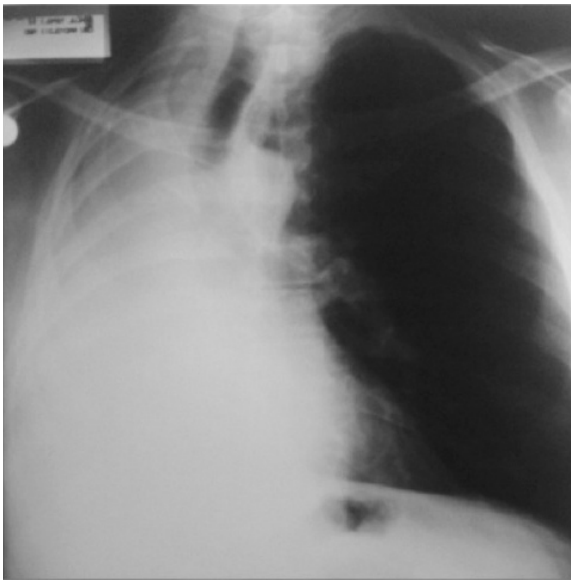

Figure 1. Chest X-ray showing a shift of the trachea and mediastinum to the right, hyperinflation of the left lung, the opacity of the right hemithorax and the patency of tracheal laminar air.

the cases[1,8]. Restrictive pulmonary disease, common in post-pneumonectomy patients, is also a risk factor for the development of ALI [9]. Protective ventilatory strategies may prevent or reduce ALI [8]. The protective ventilatory strategies we used in our patient during the perioperative period were based on general principles for preventing ALI [8,10-13]. The results of previous studies indicate that main risk factors associated with ALI are large tidal volumes, prior restrictive lung disease, and blood product transfusion [10,13]. Our current understanding of OLV-associated ALI supports the following strategies to minimize lung injury: maintain FiO_2 as low as possible, use PEEP routinely with lower tidal volumes, apply frequent recruitment manoeuvres to avoid atelectasis, and employ lower ventilatory pressures throughout the use of PCV [10,14,15].

Although the incidence of ARDS after CPB is low (2%), it carries a 50% mortality risk [16]. Many strategies to limit lung injury during CPB have been reported including medications (steroids, aprotinin) to reduce pro-inflammatory cytokine release, biocompatible CPB circuits and ultrafiltration to reduce levels of inflammatory substances, and protective ventilation strategies [17]. Many studies have investigated the effect of vital capacity manoeuvers, CPAP, and PEEP during CPB on PaO_2, $AaDO_2$ and PaO_2/FiO_2 [13,18,19]. To date, there is no convincing evidence that any of these interventions has a significant impact on clinical outcomes. In our case, mediastinal shift of the lung did not allow for ventilation maneuvers such as CPAP during CPB. We applied recruitment (40 cm H_2O for 40 seconds) before weaning from CPB, and 5 cmH_2O of PEEP during ventilation after weaning from CPB. The oxygenation recovered to normal level 1 hour following CPB in the ICU, but shunting continued to decrease in ICU after 11 hours of ICU stay.

Correct fluid management in post-pneumonectomy patients is essential. Excessive perioperative fluid infusions may cause pulmonary edema in the remaining lung with increased pulmonary arterial pressure, pulmonary vasoconstriction, and hyperinflation [7,20]. Postoperative right heart failure occurs frequently in patients given more than 2000 mL of intraoperative fluid [7]. Thus, to prevent development of postoperative right-sided heart failure in post-pneumonectomy patients, intraoperative fluid balance should be restricted as much as possible [7]. Because of expected anatomic difficulties, we recommend central venous catheterization for fluid administration to be performed through the femoral vein instead of the internal jugular vein. Monitoring filling pressures, i.e. central venous pressure and pulmonary artery occlusion pressure were the "standard" methods used for the assessment of volume status. However, assessment of volume responsiveness with echocardiography can be categorized as being very safe because of its minimal invasiveness. TEE evaluates the volume status by calculating the stroke volume index, tmitral E/A ratio, left ventricular CO, and the superior vena cava index [21].

In conclusion, anatomic and functional changes in post-pneumonectomy patients pose challenges during perioperative management. Although we experienced no airway difficulties in our patient, central venous access through the right IJV was not possible. Central venous monitoring might be done successfully without catheterization by using TEE intraoperatively. Lung-protective ventilation, and fluid restriction should be performed to reduce the incidence of ALI/ARDS in post-pneumonectomy patients undergoing CABG.

It was presented at the 17th Congress of Cardio-Vascular-Thoracic Anaesthesia and Intensive Care Society.

REFERENCES

1. **Stoller JK, Blackstone E, Pettersson G, et al.** Coronary artery bypass graft and/or valvular operations following prior pneumonectomy: report of four new patients and review of the literature. *Chest* 2007;132:295-301. http://dx.doi.org/10.1378/chest.06-2545
2. **Zhao BQ, Chen RK, Song JP.** Coronary artery bypass grafting after pneumonectomy. *Tex Heart Inst J* 2008;35:470-471.
3. **Hsia CCV, Johnson RL Jr.** Further examination of alveolar septal adaptation to left pneumonectomy in the adult lung. *Respiratory Physiology & Neurobiology* 2006;151:167-177. http://dx.doi.org/10.1016/j.resp.2006.01.013
4. **Venuta F, Sciomer S, Andreetti C, et al.** Long-term Doppler echocardiographic evaluation of the right heart after major lung resections. *Eur J Cardiothorac Surg* 2007;32:787-790. http://dx.doi.org/10.1016/j.ejcts.2007.07.033
5. **Foroulisa CN, Kotoulasa CS, Kakouros S et al.** Study on the late effect of pneumonectomy on right heart pressures using Doppler echocardiography. *Eur J Cardiothorac Surg* 2004;26:508-514. http://dx.doi.org/10.1016/j.ejcts.2004.05.036
6. **Petter AH, Chioléro RL, Cassina T, Chassot PG, Müller XM, Revelly JP.** Automatic "respirator/weaning" with adaptive support ventilation: the effect on duration of endotracheal ıntubation and patient management. *Anesth Analg* 2003;97:1743-1750. http://dx.doi.org/10.1213/01.ANE.0000086728.36285.BE
7. **Suehiro K, Okutani R, Ogawa S.** Anesthetic considerations in 65 patients undergoing unilateral pneumonectomy: problems related to fluid therapy and hemodynamic control. *J Clin Anesth* 2010;22:41-44. http://dx.doi.org/10.1016/j.jclinane.2009.02.013
8. **Kilpatrick B and Slinger P.** Lung protective strategies in anaesthesia. *Br J Anaesth* 2010;105:108-116. http://dx.doi.org/10.1093/bja/aeq299
9. **Beck-Schimmer B and Schimmer RC.** Perioperative tidal volume and intraoperative open lung strategy in healthy lungs:where are we going? *Best Pract Res Clin Anaesthesiol* 2010;24:199-210. http://dx.doi.org/10.1016/j.bpa.2010.02.005
10. **Licker M, Diaper J, Villiger Y, et al.** Impact of intraoperative lung protective interventions in patients undergoing lung cancer surgery. *Crit Care* 2009;13:R41. http://dx.doi.org/10.1186/cc7762
11. **Vlaar AP, Cornet AD, Hofstra JJ, et al.** The effect of blood transfusion on pulmonary permeability in cardiac surgery patients: a prospective multicenter cohort study. *Transfusion* 2012;52:82-90. http://dx.doi.org/10.1111/j.1537-2995.2011.03231.x
12. **Alsara A, Warner DO, Li G, et al.** Derivation and validation of automated electronic search strategies to identify pertinent risk factors for postoperative acute lung injury. *Mayo Clin Proc* 2011;86:382-388. http://dx.doi.org/10.4065/mcp.2010.0802
13. **Murphy GS, Szokol JW, Curran RD, et al.** Influence of a vital capacity maneuver on pulmonary gas exchange after cardiopulmonary bypass. *J Cardiothorac and Vasc Anesth* 2001;15:336-340. http://dx.doi.org/10.1053/jcan.2001.23287
14. **Mols G, Priebe HJ, Guttmann J.** Alveolar recruitment in acute lung injury. *Br J Anaesth* 2006;96:156-166. http://dx.doi.org/10.1093/bja/aei299
15. **Della Rocca G and Coccia C.** Acute lung injury in thoracic surgery. *Curr Opin Anaesthesiol* 2013;26:40-46. http://dx.doi.org/10.1097/ACO.0b013e32835c4ea2
16. **Ng CS, Wan S, Yim AP, et al.** Pulmonary dysfunction after cardiac surgery. *Chest* 2002;121:1269-1277. http://dx.doi.org/10.1378/chest.121.4.1269
17. **Apostolakis EE, Koletsis EN, Baikoussis NG, et al.** Strategies to prevent intraoperative lung injury during cardiopulmonary bypass. *J Cardiothorac Surg* 2010;5:1. http://dx.doi.org/10.1186/1749-8090-5-1
18. **Schreiber JU, Lancé MD, de Korte M, et al.** The Effect of different lung-protective strategies in patients during cardiopulmonary bypass: A meta-analysis and semiquantitative review of randomized trials. *J Cardiothorac and Vasc Anesth* 2012;26:448-454. http://dx.doi.org/10.1053/j.jvca.2012.01.034
19. **Zupancich E, Paparella D, Turani F, et al.** Mechanical ventilation affects inflammatory mediators in patients undergoing cardiopulmonary bypass for cardiac surgery: a randomized clinical trial. *J Thorac Cardiovasc Surg* 2005;130:378-383. http://dx.doi.org/10.1016/j.jtcvs.2004.11.061
20. **Soll C, Hahnloser D, Frauenfelder T, et al.** The postpneumonectomy syndrome: clinical presentation and treatment. *Eur J Cardiothorac Surg* 2009;35:319-324. http://dx.doi.org/10.1016/j.ejcts.2008.07.070
21. **Gouveia V, Marcelino P, Reuter DA.** The role of transesophageal echocardiography in the intraoperative period. *Current Cardiology Reviews* 2011;7:184-196. http://dx.doi.org/10.2174/157340311798220511