

# Neighbours affect each other; pulmonary affection after cardiac surgery

Reham M. Elkolaly<sup>a</sup>, Mohab Sabry<sup>b</sup>, Mohamed Abo-Elnasr<sup>b</sup>, Amr Arafat<sup>b</sup>

**Introduction** Lungs and heart are in close relation all the time; they affect each other. Pulmonary complications due to cardiac surgeries may alter the surgery outcome and patient survival.

**Aim** The aim of this study was to report the most frequent respiratory complications after cardiac surgeries.

**Materials and methods** The study included 22 patients (group 1) who underwent coronary artery bypass grafting and 56 patients (group 2) who underwent valve replacement surgery. Preoperative and postoperative investigations such as chest radiology, spirometric lung functions and PaO<sub>2</sub>/FiO<sub>2</sub> were performed. Pleural effusion, pneumothorax, acute respiratory distress syndrome, pneumonia, atelectasis, wound infection or sternal dehiscence were reported.

**Results** Pleural effusion, pneumothorax, acute respiratory distress syndrome, pneumonia, atelectasis, and sternal wound infection in group 1 were found among 81.81, 4.55, 9.10, 31.82, 86.36 and 18.18% of the included patients,

respectively, whereas in group 2 they were found among 33.93, 0, 0, 35.71, 64.29 and 10.71%, respectively.

**Conclusion** Complications after cardiac surgeries are common and precautions must be taken to decrease them and to improve outcome.

*Egypt J Bronchol* 2018 12:240–246

© 2018 Egyptian Journal of Bronchology

*Egyptian Journal of Bronchology* 2018 12:240–246

**Keywords:** cardiac surgery, pleural effusion, pneumothorax, pulmonary complication

<sup>a</sup>Department of Chest Diseases, Faculty of Medicine, <sup>b</sup>Department of Cardiothoracic Surgery, Tanta University, Tanta, Egypt

Correspondence to Reham M. Elkolaly, MD, Department of Chest Diseases, Faculty of Medicine, Tanta University, Tanta, 31111, Egypt.

Tel: 01227650268;

e-mail: r.elkolaly@gmail.com

**Received** 30 June 2017 **Accepted** 8 November 2017

## Introduction

During embryological development, lung, pleura, diaphragm and heart are in close relation all the time, and thus it is logical to find that any surgical intervention in one of them can easily affect the others.

At present, open-heart surgery is one of the common interventions that are met every day in hospitals and its outcome and complications became well known to physicians.

Cardiothoracic surgeries necessitate good patient preparation, efficient surgeon and close excellent follow-up; all these are to avoid or at least to decrease complications [1]. The surgeon sometimes faces many complications of cardiac surgery that may alter the surgery outcome and even patient survival and quality of life later on.

The pathogeneses of these complications are not fully understood, but many surgery-related factors contribute to these complications, such as general anaesthesia, median sternotomy, cardiopulmonary bypass (CPB) [2], the injurious effect of mechanical ventilation [3], systemic inflammatory mediators release and capillary wall injury [4]. Many mechanisms explain the effects of these surgeries on other organs than the heart, including lung and pleura.

Atelectasis is a common finding after cardiac surgery, which may be due to mucociliary affection and reflex bronchoconstriction, that leads to distant secretion stagnation and small airway obstruction [5]. Atelectasis may occur during CPB, as a result of slow patient breathing at low lung volumes and deficient lung perfusion [6].

Another complication that is frequently met after cardiac surgery is pulmonary infection, which not only affects the lung as an organ but contributes to the surgery outcome and patient survival. Postoperative pulmonary infection has different degrees of severity ranging from mild cough to respiratory failure necessitating invasive ventilation [1].

Diaphragmatic dysfunction is not a serious complication except in patients with underlying pulmonary problem; it may pass smoothly or may affect the patient leading to frequent pulmonary infections or even respiratory failure [7].

Hypoxaemia is one finding that the surgeon meets during or commonly after the cardiac surgery. It can be managed easily in most cases and usually is reversible,

---

This is an open access journal, and articles are distributed under the terms of the Creative Commons Attribution-NonCommercial-ShareAlike 4.0 License, which allows others to remix, tweak, and build upon the work non-commercially, as long as appropriate credit is given and the new creations are licensed under the identical terms.

but in some cases it persists and needs more intervention to overcome. It may occur owing to decreased lung compliance, impaired lung surfactant production or even respiratory centre depression because of anaesthetic drugs and also muscle relaxant use [8].

Other than the previously mentioned complications, pulmonary embolism, acute respiratory distress syndrome (ARDS), cardiogenic pulmonary oedema and pneumothorax [1,7] may occur, and most of these are manageable but some not.

## Aim

The aim of this study was to find out the incidence of some respiratory outcomes and complications after open cardiac surgeries.

## Materials and methods

This prospective study was conducted in the Cardiothoracic Department with collaboration of Chest Department in Tanta University hospitals through a period of 14 months duration.

The study included 78 patients who were classified into two groups according to the type of surgery that was planned to be done:

- (1) Group 1: this group included 22 patients in whom coronary artery bypass grafting (CABG) surgery was performed.
- (2) Group 2: this group included 56 patients in whom valve replacement surgery was performed.

All studied patients were subjected to the following:

- (1) Thorough history taking.
- (2) Complete clinical examination.
- (3) Preoperative laboratory investigations: e.g. liver and kidney functions.
- (4) ECG, echocardiogram (EF% ejection fraction).
- (5) Preoperative and postoperative chest radiography (computed tomography if needed).
- (6) Preoperative and 2 weeks postoperative spirometric lung functions: FEV<sub>1</sub> (volume that has been exhaled at the end of the first second of forced expiration), FVC (volume of air that can be forcibly exhaled from the lungs after taking the deepest breath), and FEV<sub>1</sub>% (proportion of a person's FEV<sub>1</sub> to FVC).
- (7) Preoperative and postoperative PaO<sub>2</sub> (arterial partial pressure of oxygen)/FiO<sub>2</sub> (fraction or percentage of inspired oxygen).

- (8) Coronary angiography for those having ischaemic cardiac disease or valvular heart lesions (if needed).

The following data were recorded:

- (1) PaO<sub>2</sub>/FiO<sub>2</sub> (after 1/2 h, 1 h and after 4 h).
- (2) Coronary bypass time (CBT), ischaemic time, pleura opening and side of chest tube insertion.
- (3) Duration of mechanical ventilation, duration of hospital and ICU stay.
- (4) Presence of pleural effusion, pneumothorax, ARDS, pneumonia (culture and sensitivity), atelectasis, wound infection or sternal dehiscence.

## Statistical analysis

Data were analysed using SigmaStat, statistical analysis package (version 4.00, relaunched as SigmaStat version 4.00 on 1 February 2016). Quantitative data were presented as mean±SD. The threshold for the level of significance is fixed at the 5% level, and a *P* value less than 0.05 was considered significant.

## Results

A total of 78 patients were included in this study; there were 43 (55.13%) male and 35 (44.87%) female patients, and their age ranged between 16 and 69 years old (37.89±14.98) (Table 1). They were classified into two groups according to the type of indicated surgery:

- (1) Group 1: this group included 22 patients indicated for CABG surgery; their age ranged between 40 and 69 years (54.091±8.082).
- (2) Group 2: this group included 56 patients indicated for valve replacement surgery; their age ranged between 14 and 54 years (31.518±11.947).

EF% in group 1 was 57.82±6.68, whereas in group 2 it was 60.82±10.49.

**Table 1 Demographic data and medical history of patients in groups 1 and 2**

	Group 1 (22 patients) [n (%)]	Group 2 (56 patients) [n (%)]	Total (78 patients) [n (%)]
Male	18 (81.82)	25 (44.64)	43 (55.13)
Female	4 (18.18)	31 (55.36)	35 (44.87)
COPD	6 (27.3)	5 (8.93)	11 (14.10)
Smokers	11 (50)	6 (10.71)	16 (20.51)
Hypertension	12 (54.55)	2 (3.57)	14 (17.95)
DM	9 (40.91)	0 (0)	9 (11.54)

COPD, chronic obstructive pulmonary disease; DM, diabetes mellitus.

History of smoking, chronic obstructive pulmonary disease, hypertension and diabetes mellitus in both groups was reported in Table 1.

Preoperative and postoperative spirometric data (FEV<sub>1</sub>, FVC and FEV<sub>1</sub>%) were measured for patients in group 1 and group 2; these parameters showed a significant decrease in their postoperative values in group 2 ( $P < 0.001$  for FVC, 0.017 for FEV<sub>1</sub> and 0.002 for FEV<sub>1</sub>%, but their values did not change significantly in group 1;  $P$  value was 0.117 for FVC, 0.083 for FEV<sub>1</sub> and 0.355 for FEV<sub>1</sub>%; Table 2).

PaO<sub>2</sub> and its relation to FiO<sub>2</sub> were measured in preoperative and postoperative periods (30 min, 1 h and 4 h postoperatively) in both groups, and they showed a significant decrease after 1/2 h and after 1 h postoperatively when compared with preoperative records, but after 4 h their values began to improve but did not reach the preoperative values (Table 3).

Incidence of pleural effusion, pneumothorax, ARDS, pneumonia, atelectasis and sternal wound infection were reported in both groups and in all patients (Table 4).

In group 1, the causative organism for pneumonia was mainly Gram negative in five (22.73%) cases and gram positive organisms in two (9.1%) cases. However, in group 2, Gram negative organisms causing pneumonia was in 10 (17.86%) cases, Gram positive organisms in six (10.71%) cases, and mixed infection in four (7.14%) cases. As a result, the total isolated causative organism was Gram negative in 15/78 (19.23%), Gram positive in 8/78 cases (10.26%) and mixed infection in 4/78 cases (5.13%).

Pleural effusion site was either right, left or bilateral. In group 1, six cases were right, 15 cases were left and four cases were bilateral. In group 2, 13 cases were right, 13 cases were left and seven cases were bilateral (Table 5).

In patients complicated with atelectasis, left lower lobe was the most affected one in group 1, whereas right lower lobe was mostly affected in group 2 (Table 6).

Duration of ischaemic time, CBT, postoperative mechanical ventilation, hospital and ICU stay in both groups were reported in Table 7.

Death rate in both groups was low – one case in each group; uncontrollable bleeding peptic ulcer was the

**Table 2 Spirometric data in preoperative and postoperative time in both groups**

	Group 1				Group 2			
	Preoperative	Postoperative	<i>t</i>	<i>P</i>	Preoperative	Postoperative	<i>t</i>	<i>P</i>
FVC	3.21±0.68	2.864±0.752	1.60	0.117	3.70±0.78	3.18±0.83	3.42	<0.001
FEV <sub>1</sub>	2.59±0.56	2.25±0.69	1.78	0.083	2.95±0.66	2.64±0.68	2.42	0.017
FEV <sub>1</sub> %	80.67±2.74	78.16±12.26	0.94	0.355	79.58±3.80	83.54±8.44	-3.20	0.002

FEV<sub>1</sub>, exhaled volume at the end of the first second of forced expiration after deepest inspiration; FEV<sub>1</sub>%, proportion of a person's FEV<sub>1</sub> to FVC; FVC, exhaled volume maximally after deepest inspiration.

**Table 3 PaO<sub>2</sub>/FiO<sub>2</sub> in preoperative time and postoperative time (1/2 h, 1 h and 4 h postoperatively) in groups 1 and 2**

	Group 1	<i>F</i>	<i>P</i>	Group 2	<i>F</i>	<i>P</i>
PaO <sub>2</sub> /FiO <sub>2</sub> (preoperative)	393.41±46.91	4.75	0.004	419.34±45.91	4.81	0.003
PaO <sub>2</sub> /FiO <sub>2</sub> (1/2 h postoperative)	323.41±101.36			382.98±83.52		
PaO <sub>2</sub> /FiO <sub>2</sub> (1 h postoperative)	308.00±98.01			371.86±57.22		
PaO <sub>2</sub> /FiO <sub>2</sub> (4 h postoperative)	311.46±87.79			391.68±82.39		

FiO<sub>2</sub>, fraction or percentage of inspired oxygen; PaO<sub>2</sub>, arterial partial pressure of oxygen.

**Table 4 Pulmonary complications after cardiac surgeries in groups 1 and 2**

	Group 1 (22 patients) [n (%)]	Group 2 (56 patients) [n (%)]	Total (78 patients) [n (%)]
Pleural effusion	18 (81.81)	19 (33.93)	37 (47.44)
Pneumothorax	1 (4.55)	0 (0)	1 (1.28)
ARDS	2 (9.10)	0 (0)	2 (2.56)
Pneumonia	7 (31.82)	20 (35.71)	27 (34.62)
Atelectasis	19 (86.36)	36 (64.29)	55 (70.51)
Sternal wound infection	4 (18.18)	6 (10.71)	10 (12.82)

ARDS, acute respiratory distress syndrome.

cause in group 1 (4.55%); and heart failure in group 2 (1.79%) with a total death rate of 2.56%.

## Discussion

Cardiac surgeries are indicated for many causes; sometimes, there are contraindications as a result of patient conditions or comorbidities, but in other situations some complications may occur to influence the patient general status and also affect the surgery outcome. This study aimed to clarify some side effects related to cardiac surgeries on lung and pleura.

In the present study, 78 patients – 43 (55.13%) male and 35 (44.87%) female – were admitted to hospital for cardiac surgery, either valvular replacement (56 cases) or coronary artery bypass (22 cases).

PaO<sub>2</sub>/FiO<sub>2</sub> changed significantly in group 1 as regards preoperatively and 1/2 h postoperatively (393.41±46.91 and 323.41±101.36, respectively) and it also changed significantly in group 2 (419.34±45.91 and 382.98±83.52, respectively). However, 1 and 4 h postoperatively they did not show significant changes in both groups.

FEV<sub>1</sub>, FVC and FEV% did not change significantly in group 1 when preoperative values were compared with postoperative ones after 2 weeks (they changed by

22–35%), but preoperative and postoperative values changed significantly in group 2 (35–60%). These changes were nearly equal to those obtained by Nicholson *et al.* [9]; FVC and FEV<sub>1</sub> values were reduced by 40–50% on the first-to-third postoperative days after CABG surgery, and also were mostly relevant to those obtained by Urell *et al.* [10]; 50 and 40% reduction in lung functions and lung volumes, respectively, were reported on the second day after open-heart surgery.

Other authors reported decreased vital capacity up to 60% at the second postoperative days, and most lung volumes remained lower than preoperative values up to 4 months after cardiac surgeries [11–13].

As regards postoperative complications in the present study, 81.81% of cases had pleural effusions in group 1 (six cases had right, 15 had left and four had bilateral effusion). In group 2, 33.93% of cases had pleural effusions (13 cases had right, 13 had left and seven had bilateral effusion). This high incidence may be explained by intraoperative pleural injury, iatrogenic cardiac hypothermia during surgery, lung atelectasis or even bleeding.

In agreement with the present study, Vargas *et al.* [14] found that after second day of CABG 57% of cases developed right-sided pleural effusion that decreased by the seventh day, whereas 67% of studied cases developed left-sided pleural effusion. Another study by Light *et al.* [15] reported that only 29 of 3707 developed left-sided pleural effusion after CABG surgeries and this is a very low incidence; this may be because of their high number of studied cases in comparison with the limited number that highlighted the high incidence of effusion.

Labidi *et al.* [16] studied the prevalence of pleural effusion after either valve replacement or bypass graft surgery over a period of 2 years; they recorded that 6.6% of patients had effusion (mainly in females) and 59.4% of these effusion cases were exudative, whereas 50% was haemorrhagic. The incidence of pleural effusion after heart surgeries is not low [17]. It may occur owing to many causes such as atelectasis, pneumonia, pleural injury or pulmonary oedema [18]. Nikas *et al.* [19] added that pleural effusion may occur also after internal mammary artery use as a graft for bypass either owing to pleurotomy or pleural lymphatic interruption.

Atelectasis in the present study was found among 86.36% of patients in group 1 and 64.29% in group

**Table 5 Pleural effusion incidence in groups 1 and 2**

	Group 1 (22 patients) [n (%)]	Group 2 (56 patients) [n (%)]	Total (78 patients) [n (%)]
Right side	6 (27.27)	13 (23.21)	19 (24.36)
Left side	15 (68.18)	13 (23.21)	28 (35.9)
Bilateral	4 (18.18)	7 (12.5)	11 (14.1)

**Table 6 Atelectasis incidence in groups 1 and 2**

	Group 1 (22 patients) [n (%)]	Group 2 (56 patients) [n (%)]	Total (78 patients) [n (%)]
Right lobe	2 (9.1)	18 (32.14)	20 (25.64)
Left lobe	11 (50)	10 (17.86)	21 (26.92)
Bilateral	6 (27.27)	9 (16.07)	15 (19.23)

**Table 7 Machine patients' dependence parameters and admission duration in groups 1 and 2**

	Group 1	Group 2
Coronary bypass time	100.82±32.67	82.732±24.742
Ischaemic time	77.91±28.15	62.589±21.390
Mechanical ventilation duration	13.55±12.98	7.04±5.40
Hospital stay duration	14.23±4.63	11.75±1.95
ICU stay duration	4.27±3.06	3.11±1.04



2 and diagnosed clinically and radiologically. These high percentages may be due to low lung ventilation (at residual volume) especially in CABG surgery, and when lung began to ventilate again at acceptable tidal volumes some areas of the lung did not react well and remained atelectatic.

The results of the present study were nearly equivalent to those obtained by Brooks-Brunn [6] who reported a high atelectasis incidence (70% of cases) in their study. In another relevant study conducted by Al-Qubati *et al.* [20] to study postcardiac surgeries complications, atelectasis was 1.11%, and it was of low incidence compared with the previous ones.

Atelectasis had more than hypothesis to occur; lobe compression by the surgeon in a trial to reach the posterior surface of the heart or during dissecting the internal mammary artery [21], another one is breathing at a low tidal volume and rate or even breathing cessation at CPB time [22]. The use of hypothermic solution during cardiac surgery was postulated to decrease surfactant production, which may play a role in lung atelectasis [23].

Pleural effusion (if present) plus pain intolerance, shallow breathing and intolerable cough effort all participate in the pathogenesis of postoperative atelectasis [1].

Pneumonia as a complication after cardiac surgery in the present study revealed a high incidence: 31.82% in group 1 and 35.71% in group 2 (total was 34.62% in all patients). The most common isolated causative organisms were gram negative bacteria in both groups. This incidence was the highest when compared with other relative studies by Tamayo *et al.* [24], Ailawadi *et al.* [25], and El Solh *et al.* [26]. This may be explained by insufficient use of antibiotics, high prevalence of resistant organisms and long intraoperative tissue hypoxaemia. As one of the developing countries, we still have a high incidence of pulmonary infections when compared with international rates.

Pneumonia is still the most important infection that patient and physician face after open-heart surgeries and still endangers patient life if not prevented or well-treated early [25].

In a similar study of 162 patients who underwent either CABG or valve replacement therapy, Topal and Eren [27] stated a lower incidence of pneumonia in 21 (13%) patients without significant difference between their two studied groups.

Although heart surgery itself is considered the main trigger of pneumonia, other causes may participate in pathogenesis of pneumonia, such as old age of the patient [28], unwise use of antimicrobial agents [29], inefficient haemostasis, hypovolaemia and long mechanical ventilation [30].

Ailawadi *et al.* [25] studied 5158 after cardiac surgeries for pneumonia and its relation to patient's age, haemoglobin level, previous obstructive lung diseases and corticosteroid usage, and found that the incidence of pneumonia in these patients was not high (2.4%) inspite of many risk factors that their patients were exposed to either preoperatively or intraoperatively. El Solh *et al.* [26] studied 73 patients as regards nosocomial pneumonia after cardiac surgeries and they found only 8.3% of patients developed pneumonia after the third day of surgery.

In the present study, pneumothorax was found only in one (4.55%) case in group 1. It is not a frequent complication after cardiac surgery, which has universal low incidence of 0.7–1.7% [31], but this prevalence may increase if the patient was mechanically ventilated for a long period or the physician used high positive end expiratory pressure [32].

This result was in coincidence with that of Jensen and Yang [33] who reported pneumothorax in 23 (7.3%) patients in their series of 315 patients after CABG surgeries and also with that of a study by Cingoz *et al.* [34]; they studied 531 patients with ventricular or atrial septal defect repair and they reported pneumothorax in seven patients.

In addition, Urschel *et al.* [35] reported 1.4% of pneumothoraces (21 of 1463 patients) in those who underwent cardiac surgeries, especially patients with a history of obstructive lung diseases; they also reported four patients with life-threatening pneumothoraces and required further interventions other than chest tubes, and these results support those obtained by others and those of the recent study. Pneumothorax in cardiac surgery may be due to direct lung injury intraoperatively or during chest closure; it may also be injured during nearby vascular cannulation [31].

In the present study, ARDS incidence was 9.10 and 2.56% in group 1 and group 2, respectively.

This was in agreement with that of Al-Qubati *et al.* [20] that included 179 patients; developed in three cases of coronary revascularization group while in the

valve replacement group; ARDS developed in one case. In a cohort study by Gajic *et al.* [36] to prevail the incidence of ARDS in hospitalised patients who were admitted because of different causes, they reported ARDS in 55 patients out of 541 (10.17%) with cardiac surgeries as a predisposing factor for lung injury.

These results were lower than those obtained by Kogan *et al.* [37], who studied incidence, risk factors and death rate after cardiac surgeries; they enrolled 6069 patients in their study. They reported that a low incidence of 37 (0.61%) patients developed ARDS with a mortality rate of 40.5%. This may be hypothesised by long time of CPB and low lung ventilation and perfusion.

ARDS is not commonly seen in cardiac surgery patients, but if it happens it has a high mortality rate. There is more than one hypothesis for its pathogenesis to occur: CPB that decreased pulmonary circulation and ventilation in addition to systemic inflammatory cascade and increased vascular permeability, which all facilitate lung injury [38]. In addition, surgical transient tissue ischaemia and reperfusion leads to the release of reactive oxygen radicals that injure the lung too [39]. Some patients need excessive blood transfusion during surgery, and it also can lead to lung injury and ARDS [40].

The limitations of the present study are the short follow-up period for patients, complete lung functions was not fully performed (and needed to be compared in other detailed studies), perioperative risk factors for complications should be put into consideration and also the study was observational in nature, which restricted modellization of the results.

## Conclusion

Lung and pleura are affected during and after cardiac surgeries either at the physiologic level or as side effects.

Complications after cardiac surgeries are not rare and have many risk factors either related to surgery type, medical patient condition or postoperative manoeuvres.

Careful opening and closure of the thoracic cavity to avoid pleural and lung tear, shortening of cardiopulmonary bypass time, avoiding unnecessary excess blood transfusion, decreasing duration of ICU and hospital stay and good choice of antibiotics, all must be put into consideration to decrease lung affection.

It is difficult to prevent all complications, but at least trials must be done to decrease them and to get patients better and improve quality of their lives.

## Acknowledgements

Reham M. Elkolaly contributed to follow-up of cases with collaboration with cardiothoracic surgeons and performing pulmonary functions; Mohab Sabry selected the cases included in the study, performed the cardiac surgeries, and was involved in patient follow-up; Mohamed Abo-Elnasr assisted the cardiac surgeries and was involved in patient follow-up; Amr Arafat assisted the cardiac surgeries and was involved in patient follow-up.

## Financial support and sponsorship

Nil.

## Conflicts of interest

There are no conflicts of interest.

## References

- 1 Weissman C. Pulmonary complications after cardiac surgery. *Semin Cardiothorac Vasc Anesth* 2004; **8**:185–211.
- 2 Taggart DP. Respiratory dysfunction after cardiac surgery: effects of avoiding cardiopulmonary bypass and the use of bilateral internal mammary arteries. *Eur J Cardiothorac Surg* 2000; **18**:31–37.
- 3 Dreyfuss D, Saumon G. Ventilator-induced lung injury: lessons from experimental studies. *Am J Respir Crit Care Med* 1998; **157**:294–323.
- 4 Badenes R, Lozano A, Belda FJ. Postoperative pulmonary dysfunction and mechanical ventilation in cardiac surgery. *Crit Care Res Pract* 2015; **2015**:420513.
- 5 Matthay MA, Wiener-Kronish JP. Respiratory management after cardiac surgery. *Chest* 1989; **95**:424–434.
- 6 Brooks-Brunn JA. Postoperative atelectasis and pneumonia. *Heart Lung* 1995; **24**:94–115.
- 7 Maciej R, Robert S, Kustrzycki W. Postoperative pulmonary complications after conventional coronary artery bypass grafting. *Clin Exp Med Lett* 2008; **49**:1–3.
- 8 Ramos R, Salem B, de Pawlikowski MP. The efficiency of pneumatic compression stocking in pervention of pulmonary embolism after cardiac surgery. *Chest* 1996; **109**:82–85.
- 9 Nicholson DJ, Kowalski SE, Hamilton GA, Meyers MP, Serrette C, Duke PC. Postoperative pulmonary function in coronary artery bypass graft surgery patients undergoing early tracheal extubation: a comparison between short-term mechanical ventilation and early extubation. *J Cardiothorac Vasc Anesth* 2002; **16**:27–31.
- 10 Urell C, Westerdahl E, Hedenstrom H, Janson C, Emtner M. Lung function before and two days after open-heart surgery. *Crit Care Res Pract* 2012; **2012**:291628.
- 11 Matte P, Jacquet L, Van Dyck M, Goenen M. Effects of conventional physiotherapy, continuous positive airway pressure and non-invasive ventilatory support with bilevel positive airway pressure after coronary artery bypass grafting. *Acta Anaesthesiol Scand* 2000; **44**:75–81.
- 12 Shenkman Z, Shir Y, Weiss YG, Bleiberg B, Gross D. The effects of cardiac surgery on early and late pulmonary functions. *Acta Anaesthesiol Scand* 1997; **41**:1193–1199.
- 13 Westerdahl E, Lindmark B, Bryngelsson I, Tenling A. Pulmonary function 4 months after coronary artery bypass graft surgery. *Respir Med* 2003; **97**:317–322.
- 14 Vargas FS, Uezumi KK, Janete FB, Terra-Filho M, Hueb W, Cukier A, *et al.* Acute pleuropulmonary complications detected by computed tomography following myocardial revascularization. *Rev Hosp Clin Fac Med Sao Paulo* 2002; **57**:135–142.

- 15 Light RW, Rogers JT, Cheng D, Rodriguez RM. Large pleural effusions occurring after coronary artery bypass grafting. Cardiovascular Surgery Associates, PC. *Ann Intern Med* 1999; **130**:891–896.
- 16 Labidi M, Baillet R, Dionne B, Lacasse Y, Maltais F, Boulet LP. Pleural effusions following cardiac surgery: prevalence, risk factors, and clinical features. *Chest* 2009; **136**:1604–1611.
- 17 Light RW, Rogers JT, Moyers JP, Lee YC, Rodriguez RM, Alford WC Jr, et al. Prevalence and clinical course of pleural effusions at 30 days after coronary artery and cardiac surgery. *Am J Respir Crit Care Med* 2002; **166**:1567–1571.
- 18 Ali IM, Lau P, Kinley CE, Sanalla A. Opening the pleura during internal mammary artery harvesting: advantages and disadvantages. *Can J Surg* 1996; **39**:42–45.
- 19 Nikas DJ, Ramadan FM, Elefteriades JA. Topical hypothermia: ineffective and deleterious as adjunct to cardioplegia for myocardial protection. *Ann Thorac Surg* 1998; **65**:28–31.
- 20 Al-Qubati FAA, Damag A, Noman T. Incidence and outcome of pulmonary complications after open cardiac surgery, Thowra Hospital, Cardiac center, Sana'a, Yemen. *Egypt J Chest Dis Tuberc* 2013; **62**:775–780.
- 21 Magnusson L, Zemgulis V, Tenling A, Wemlund J, Tyden H, Thelin S, et al. Use of a vital capacity maneuver to prevent atelectasis after cardiopulmonary bypass: an experimental study. *Anesthesiology* 1998; **88**:134–142.
- 22 Duggan M, Kavanagh BP. Pulmonary atelectasis: a pathogenic perioperative entity. *Anesthesiology* 2005; **102**:838–854.
- 23 Rachwalik M, Skalik R, Kustrzycki W. Postoperative pulmonary complications after conventional coronary artery bypass grafting. *Clin Exp Med Lett* 2008; **49**:1–3.
- 24 Tamayo E, Alvarez FJ, Martinez-Rafael B, Bustamante J, Bermejo-Martin JF, Fierro I, et al. Ventilator-associated pneumonia is an important risk factor for mortality after major cardiac surgery. *J Crit Care* 2012; **27**:18–25.
- 25 Ailawadi G, Chang HL, O'Gara PT, O'Sullivan K, Woo YJ, DeRose JJ Jr, et al. Pneumonia after cardiac surgery: Experience of the National Institutes of Health/Canadian Institutes of Health Research Cardiothoracic Surgical Trials Network. *J Thorac Cardiovasc Surg* 2017; **153**:1384–91e3.
- 26 El Solh AA, Bhora M, Pineda L, Dhillon R. Nosocomial pneumonia in elderly patients following cardiac surgery. *Respir Med* 2006; **100**:729–736.
- 27 Topal AE, Eren MN. Risk factors for the development of pneumonia post cardiac surgery. *Cardiovasc J Afr* 2012; **23**:212–215.
- 28 Hortal J, Giannella M, Perez MJ, Barrio JM, Desco M, Bouza E, et al. Incidence and risk factors for ventilator-associated pneumonia after major heart surgery. *Intensive Care Med* 2009; **35**:1518–1525.
- 29 Kinlin LM, Kirchner C, Zhang H, Daley J, Fisman DN. Derivation and validation of a clinical prediction rule for nosocomial pneumonia after coronary artery bypass graft surgery. *Clin Infect Dis* 2010; **50**:493–501.
- 30 Morrow BM, Argent AC, Jeena PM, Green RJ. Guideline for the diagnosis, prevention and treatment of paediatric ventilator-associated pneumonia. *S Afr Med J* 2009; **99**:255–267.
- 31 Douglas JM, Spaniol S. Prevention of postoperative pneumothorax in patients undergoing cardiac surgery. *Am J Surg* 2002; **183**:551–553.
- 32 Schuller D, Morrow LE. Pulmonary complications after coronary revascularization. *Curr Opin Cardiol* 2000; **15**:309–315.
- 33 Jensen L, Yang L. Risk factors for postoperative pulmonary complications in coronary artery bypass graft surgery patients. *Eur J Cardiovasc Nurs* 2007; **6**:241–246.
- 34 Cingoz F, Güler A, Bahin MA, Tatar H. The incidence of post-operative spontaneous pneumothorax after isolated ASD and VSD repair in adults; a single-centered retrospective study. *Anatolian J Clin Investig* 2009; **3**: 120–122.
- 35 Urschel JD, Parrott JC, Horan TA, Unruh HW. Pneumothorax complicating cardiac surgery. *J Cardiovasc Surg (Torino)* 1992; **33**:492–495.
- 36 Gajic O, Dabbagh O, Park PK, Adesanya A, Chang SY, Hou P, et al. Early identification of patients at risk of acute lung injury: evaluation of lung injury prediction score in a multicenter cohort study. *Am J Respir Crit Care Med* 2011; **183**:462–470.
- 37 Kogan A, Preisman S, Levin S, Raanani E, Sternik L. Adult respiratory distress syndrome following cardiac surgery. *J Card Surg* 2014; **29**:41–46.
- 38 Verheij J, van Lingen A, Raijmakers PG, Rijnsburger ER, Veerman DP, Wisselink W, et al. Effect of fluid loading with saline or colloids on pulmonary permeability, oedema and lung injury score after cardiac and major vascular surgery. *Br J Anaesth* 2006; **96**:21–30.
- 39 Fink MP. Role of reactive oxygen and nitrogen species in acute respiratory distress syndrome. *Curr Opin Crit Care* 2002; **8**:6–11.
- 40 Vlaar AP, Hofstra JJ, Determann RM, Veelo DP, Paulus F, Kulik W, et al. The incidence, risk factors, and outcome of transfusion-related acute lung injury in a cohort of cardiac surgery patients: a prospective nested case-control study. *Blood* 2011; **117**:4218–4225.