# Continuous positive airway pressure does not improve lung function after cardiac surgery

## *[La ventilation en pression positive continue n'améliore pas la fonction pulmonaire après la cardiochirurgie]*

Ece Altmay MD,* Pelin Karaca MD,* Nurgül Yurtseven MD,* Vedat Özkul MD,* Tamer Aksoy MD,* Azmi Özler MD,† Sevim Canik MD*

**Purpose:** Despite the well-documented impairment of pulmonary function after cardiopulmonary bypass, effective precautions and ideal management strategies for this problem are still under debate. This study aimed to evaluate the effects of continuous positive airway pressure (CPAP) applied during cardiopulmonary bypass on respiratory and hemodynamic variables.

**Methods:** In this randomized, prospective, controlled trial, 120 male patients, aged 45 to 70 yr undergoing first-time elective bypass surgery, were randomly assigned to receive either 10 cm $H_2O$ of CPAP (Group I; $n = 60$) during cardiopulmonary bypass, or serve as control (Group II; $n = 60$), where the patient's lungs were vented to atmosphere during the bypass period.

**Results:** Alveolar-arterial oxygen partial pressure difference and shunt fraction were significantly higher in the control group compared with the CPAP group after cardiopulmonary bypass ($T_2$) and after closure of sternum ($T_3$), ($P < 0.05$). No differences between groups with respect to hemodynamic variables were observed at any time. Postoperative pulmonary function variables were lower in both groups compared to baseline values.

**Conclusions:** Continuous positive airway pressure administered during cardiopulmonary bypass decreased shunt fraction and alveolar-arterial oxygen partial pressure difference during surgery, but had no sustained effect on either variable postoperatively. We conclude that, in patients with normal preoperative pulmonary function, application of 10 cm $H_2O$ CPAP does not improve lung function after cardiac surgery.

***Objectif** : Malgré les connaissances acquises sur l'atteinte de la fonction pulmonaire après la circulation extracorporelle, les précautions efficaces et le traitement idéal touchant ce problème font toujours l'objet de débats. Nous voulions évaluer les effets d'une ventilation en pression positive continue (CPAP pour «continuous positive airway pressure») pendant la circulation extracorporelle sur les variables respiratoires et hémodynamiques.*

***Méthode** : Dans notre étude randomisée, prospective et contrôlée, 120 hommes de 45 à 70 ans devant subir un premier pontage électif, ont reçu soit 10 cm $H_2O$ de CPAP (Groupe I ; $n = 60$) pendant la circulation extracorporelle, soit ont servi de témoins (Groupe II ; $n = 60$) et les poumons ont été ventilés à la pression atmosphérique pendant le pontage.*

***Résultats** : La différence alvéolaire-artérielle de pression partielle en oxygène et la fraction de shunt ont été significativement plus élevées chez les témoins que chez les patients sous CPAP après la circulation extracorporelle ($T_2$) et après la fermeture du sternum ($T_3$), ($P < 0,05$). Les variables hémodynamiques ont toujours été similaires dans les deux groupes. Dans les deux groupes aussi, les variables de la fonction pulmonaire postopératoire étaient plus basses que les valeurs de départ.*

***Conclusion** : La ventilation à pression positive continue pendant la circulation extracorporelle a réduit la fraction de shunt et la différence alvéolaire-artérielle de pression partielle en oxygène pendant l'opération, mais n'a pas eu d'effet postopératoire prolongé sur chacune des variables. Donc, chez les patients dont la fonction pulmonaire préopératoire est normale, une CPAP de 10 cm $H_2O$ n'améliore pas la fonction pulmonaire après la cardiochirurgie.*

From the Department of Anesthesiology and Reanimation,* and the Department of Thoracic and Cardiovascular Surgery,† Dr. Siyami Ersek Thoracic and Cardiovascular Surgery Center, Ïstanbul, Turkey.

*Address correspondence to*: Dr. Pelin Karaca, Postane Mah. Rauf Orbay Cad. Bilginoglu Sitesi, NO: 3 Tuzla / Istanbul, Turkey. Phone: +90-262-6785090; Fax: +90-262-6540055; E-mail: pelus72@yahoo.com

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DESPITE improvements in cardiopulmonary bypass (CPB) techniques and postoperative intensive care, impaired pulmonary function is a well-documented complication of CPB, resulting in increased morbidity and mortality. The complication of 'postbypass lung' occurs with an incidence from 2%–64% of cardiac surgeries involving CPB,[1,2] and is characterized by an increased alveolar-arterial oxygen partial pressure difference ($P_{(A-a)}$ $DO_2$), atelectasis, and increased extravascular lung water (EVLW).[3,4] Absent or reduced blood flow through the lungs during CPB, surgical trauma, intravascular microaggregates, and leucocyte activation are the main causes of 'postbypass lung'.

Several strategies have been evaluated to reduce the incidence and severity of pulmonary dysfunction seen following CPB including repeated vital capacity maneuvers, intermittent ventilation, and continuous airway pressure (CPAP) during CPB. Although in some reports the use of CPAP during CPB was found to improve pulmonary function, the ideal management for the non-functioning lung during CPB remains controversial.[4,5]

The aim of this study was to determine whether CPAP at 10 cm $H_2O$ during CPB, would decrease intrapulmonary shunt fraction and improve postoperative pulmonary function in patients undergoing elective coronary artery bypass graft surgery.

## Methods

After the study protocol had been approved by the local Ethics Committee, written informed consent was obtained from 120 ASA physical status II–III male patients, aged 45 to 70 yr undergoing elective coronary artery bypass graft surgery. Included were patients with preserved left ventricular function (ejection fraction > 40%), and with normal preoperative respiratory function [forced vital capacity (FVC ) > 60% of predicted, and forced expiratory volume at one second ($FEV_1$)/FVC > 60%]. Patients with valvular disease or a history of chronic obstructive lung disease were excluded.

Premedication consisted of diazepam 0.15 $mg \cdot kg^{-1}$ *po* the evening prior to surgery and midazolam 0.07 $mg \cdot kg^{-1}$ *im* and scopolamine 0.01 $mg \cdot kg^{-1}$ *im* one hour preoperatively. A pulmonary artery catheter was inserted into the right internal jugular vein of all patients to evaluate hemodynamics. In addition, continuous electrocardiography, invasive blood pressure (radial artery, non-dominant side), end-tidal carbon dioxide and oxyhemoglobin saturation were monitored throughout surgery. Anesthesia was induced with fentanyl 20 $\mu g \cdot kg^{-1}$ *iv* and propofol 2 $mg \cdot kg^{-1}$ *iv*. Muscle relaxation was provided with pancuronium 0.1 $mg \cdot kg^{-1}$ *iv*. Anesthesia was maintained with fentanyl 0.3–1.0 $\mu g \cdot kg^{-1} \cdot min^{-1}$, propofol 1 $mg \cdot kg^{-1} \cdot hr^{-1}$, and isoflurane (0.4–1.0% end-tidal concentration) until initiation of CPB. During CPB, fentanyl was infused at 0.1 $\mu g \cdot kg^{-1} \cdot min^{-1}$, and propofol was infused at 0.5 $mg \cdot kg^{-1} \cdot hr^{-1}$. After completion of CPB, fentanyl and propofol infusions were increased to individualized pre-bypass rates. Intermittent positive pressure ventilation with 10 $mL \cdot kg^{-1}$ tidal volume at 12 $breaths \cdot min^{-1}$, and 100% oxygen ($F_IO_2$ = 1.0) was administered before and after completion of CPB. Vasodilators were not administered during any part of the study.

After initiation of CPB, mechanical ventilation was discontinued in all patients. In Group I (CPAP group; $n$ = 60) lung inflation was maintained by delivery of an oxygen-air mixture administered at 2 $L \cdot min^{-1}$ ($F_IO_2$:0.25) with CPAP. Continuous pulmonary airway pressure was established via a circle system with airway pressure maintained at 10 cm $H_2O$ by adjusting the ventilator pop-off valve. The actual pressure in the circuit was monitored with an external manometer. In Group II (control group; $n$ = 60) the ventilator was disconnected from the breathing circuit, and the patient's lungs were vented to atmosphere.

Cardiopulmonary bypass was performed using a roller pump (Sarns 9000, Ann Arbor, MI, USA) and membrane oxygenator (Jostra Quadrox +VHK 4200, Hirrlingen, Germany). Priming was performed with 1500 mL Ringer's solution, 1 $mg \cdot kg^{-1}$ heparin, $2 \times 10^6$ kallikrein inactivation units (KIU) aprotinin, 150 mL mannitol, 50 mEq $NaHCO_3$. Non pulsatile–flow 2.4 $L \cdot min^{-1} \cdot m^2$ and mild hypothermia (rectal temperature of 28°C) were maintained. During cardiac arrest antegrade and retrograde administration of cold (4°C) hyperkalemic (20 $mEq \cdot L^{-1}$) blood cardioplegia was administered. Anticoagulation was achieved with bovine heparin (400 $IU \cdot kg^{-1}$) and monitored to achieve activated clotting time (ACT) levels of 480 sec. At the end of CPB heparin was neutralized with 1.3 mg of protamine for every 100 U of total heparin dosage. Additional protamine was administered to restore ACT to pre-bypass levels when necessary. All procedures were performed by the same surgeon using the same technique for myocardial protection and coronary revascularization. Proximal anastomoses were performed after aortic cross clamp release during the revascularization period. The criteria for separation from CPB were as follows: core body temperature of 37°C, stable rhythm (preferentially sinus) with adequate heart rate, and acceptable arterial blood gas values pH > 7.30, K < 5.5 $mEq \cdot L^{-1}$, hematocrit > 25%.

All patients were managed in the surgical intensive care unit (SICU) postoperatively and mechanical ven-

tilation was continued until the following conditions were present for at least two hours: 1) adequate level of consciousness, 2) tidal volume ($V_T$) > 5 mL·kg$^{-1}$, 3) respiratory rate < 25 breaths·min$^{-1}$, 4) arterial pH > 7.30, 5) an arterial partial pressure of oxygen ($P_aO_2$) > 80 mmHg with $F_IO_2$ = 0.4, 6) an arterial partial pressure of carbon dioxide ($P_aCO_2$) < 45 mmHg, 7) hemodynamic stability, and 8) chest tube drainage < 50 mL·hr$^{-1}$.

Arterial and mixed venous blood gas analysis and hemodynamic measurements were performed at the following times:

$T_0$: Before induction of anesthesia, on room air,
$T_1$: After induction of anesthesia immediately before surgical incision at $F_IO_2$ = 1.0,
$T_2$: 20 min after termination of CPB at $F_IO_2$ = 1.0,
$T_3$: Immediately after closure of sternum at $F_IO_2$ = 1.0,
$T_4$: Postoperative fourth hour in the SICU at $F_IO_2$ = 0.4, and
$T_5$: After tracheal extubation, when breathing nasal oxygen (2 L·min$^{-1}$)

Arterial and mixed venous blood-gas tensions were measured using the α-stat method. The following hemodynamic variables were recorded: heart rate (HR), mean arterial pressure (MAP), mean pulmonary artery pressure, pulmonary capillary wedge pressure (PCWP), and central venous pressure. The following were calculated using standard formulae: cardiac index (CI), systemic vascular resistance index (SVRI), pulmonary vascular resistance index (PVRI), $P_{(A-a)}DO_2$, and shunt fraction ($Q_S/Q_T$).

All subjects underwent pulmonary function tests (PFT) before surgery and on the first, third, and fifth days following surgery using an automatic spirometer (Minato Autospiro AS-500, Minato Medical Science Co. Ltd., Osaka, Japan). Forced expiratory volume at one second, FVC, vital capacity (VC), maximal voluntary ventilation (MVV), forced expiratory flow between 25% and 75% of FVC [forced expiratory flow between 25% and 75% of FVC ($FEF_{25-75}$)], and peak expiratory flow rate (PEFR) were recorded. Samples for arterial blood gas analysis were drawn at the time of each assessment of pulmonary function.

Sample size was calculated as follows: a 5% increase in shunt fraction was considered clinically significant with a standard deviation of 10. Fifty-nine patients in each group were required to achieve 80% statistical power with an α = 0.05. The SPSS (Statistical Package for Social Sciences for Windows version 10.0 Chicago, IL, USA ) was used for all statistical analyses. Data were compared using analysis of variance for repeated measures (ANOVA) and the Mann-Whitney U test was applied whenever ANOVA was significant. Within group comparisons were performed with one-way analysis of variance. Data are expressed as mean ± SD. A $P$ value < 0.05 was considered to indicate statistical significance.

TABLE I Patient characteristics and operative details

| | *Group I (n = 60)* | *Group II (n = 60)* |
|---|---|---|
| Age (yr) | 57.8 ± 8.6 | 56.2 ± 7.6 |
| Height (cm) | 176.7 ± 6.7 | 172.4 ± 7.8 |
| Weight (kg) | 75.9 ± 8.1 | 73.6 ± 8.6 |
| BSA (kg·m$^{-2}$) | 1.9 ± 0.1 | 1.9 ± 0.2 |
| Hypertension (*n*) | 15 (75%) | 16 (80%) |
| Diabetus mellitus (*n*) | 8 (40 %) | 7 (35%) |
| Smoker (*n*) | 15 (75%) | 16 (80%) |
| Cross-clamp time (min) | 63.8 ± 26.4 | 56.2 ± 17.4 |
| CPB time (min) | 99.6 ± 28.2 | 93.8 ± 23.7 |
| Duration of operation (min) | 225.8 ± 22.7 | 231.5 ± 16.2 |
| Number of grafted vessels | 3.1 ±0.7 | 3.0 ± 0.7 |
| Use of internal mammary artery (*n*) | 19 (95%) | 20 (100%) |
| Postoperative fluid balance (mL) | +804.2 ± 410.2 | +1035.7 ± 403.08 |
| Length of hospital stay (days) | 6.3 ± 0.6 | 6.3 ± 0.7 |
| Chest tube drainage | 817.5 ±79.4 | 820.5 ± 118 |

Data are expressed as mean ± SD. BSA = body surface area; CPB = cardiopulmonary bypass.

## Results

Groups were comparable with respect to demographic and intraoperative characteristics (Table I). There were no between-group differences with respect to HR, MAP, PCWP or CI (Table II). After induction of anesthesia SVRI decreased in both groups ($P$ < 0.05), (Table III). Whereas PVRI was significantly higher at the fourth postoperative hour and after tracheal extubation compared to baseline ($P$ < 0.05), MAP and PCWP remained unchanged in both groups.

The $P_{(A-a)}DO_2$ and shunt fraction values are presented in Table IV. The $P_{(A-a)}DO_2$ was greater in the control group compared with the CPAP group after CPB ($T_2$) and after closure of sternum ($T_3$), ($P$ < 0.05). Shunt fraction was also greater in the control group compared with the CPAP group at corresponding measurement periods ($P$ < 0.05). No differences were observed between groups with respect to $P_aO_2$, $P_aCO_2$ and $S_aO_2$ values measured on first, third, and fifth postoperative days ($P$ > 0.05), (Table V).

Preoperative and postoperative values for lung volumes and expiratory flow rates are presented in Table VI. Although VC, $FEV_1$, FVC, $FEF_{25-75}$, PEFR and

TABLE II Hemodynamic variables

| | *Group* | $T_0$ | $T_1$ | $T_2$ | $T_3$ | $T_4$ | $T_5$ |
|---|---|---|---|---|---|---|---|
| HR ($min^{-1}$) | I | 73.4 ± 22.4 | 77.8 ± 19.6 | 75.5 ± 21.7 | 79.3 ± 22.8 | 74.9 ± 24.5 | 78.5 ± 23.1 |
| | II | 75.2 ± 17.0 | 79.7 ± 19.3 | 77.1 ± 22.3 | 78.2 ± 18.2 | 76.7 ± 23.6 | 79.2 ± 28.4 |
| MAP (mmHg) | I | 76.9 ± 19.2 | 74.6 ± 16.3 | 75.2 ± 18.7 | 78.2 ± 21.6 | 79 ± 25.6 | 79.9 ± 27.3 |
| | II | 77.4 ± 22.5 | 72.4 ± 19.2 | 76.3 ± 21.2 | 77.9 ± 25.4 | 78.6 ± 29.1 | 77.2 ± 22.8 |
| MPAP (mmHg) | I | 18.2 ± 7.2 | 17.9 ± 9.6 | 19.1 ± 8.3 | 20.2 ± 9.8 | 19.6 ± 8.4 | 20.0 ± 6.7 |
| | II | 18.3 ± 8.3 | 17.6 ± 9.5 | 17.9 ± 9.1 | 18.8 ± 7.6 | 20.1 ± 8.6 | 19.8 ± 7.8 |
| CVP (mmHg) | I | 7.7 ± 4.8 | 7.9 ± 2.1 | 8.2 ± 4.9 | 7.5 ± 4.2 | 7.9 ± 4.8 | 7.3 ± 5.2 |
| | II | 7.9 ± 3.9 | 7.2 ± 3.1 | 8.1 ± 2.6 | 7.3 ± 5.4 | 7.4 ± 3.9 | 8.2 ± 3.1 |
| PCWP (mmHg) | I | 9.8 ± 6.2 | 9.6 ± 5.7 | 10.3 ± 4.1 | 10.1 ± 5.2 | 9.4 ± 5.1 | 9.8 ± 5.8 |
| | II | 9.1 ± 6.7 | 8.8 ± 6.2 | 9.8 ± 5.3 | 9.0 ± 5.4 | 9.5 ± 4.7 | 9.6 ± 5.5 |
| CI ($L{\cdot}min^{-1}{\cdot}m^{-2}$ ) | I | 4.1 ± 0.9 | 4.2 ± 0.8 | 3.9 ± 0.7 | 3.8 ± 0.6 | 4.0 ± 0.8 | 3.8 ± 0.8 |
| | II | 3.9 ± 0.8 | 4.0 ± 0.6 | 4.0 ± 0.7 | 3.9 ± 0.7 | 3.9 ± 0.8 | 3.9 ± 0.7 |

Data are expressed as mean ± SD. HR = heart rate; MAP = mean arterial pressure; MPAP = mean pulmonary artery pressure; CI = cardiac index; CVP = central venous pressure; PCWP = pulmonary capillary wedge pressure (PCWP; units mmHg). For abbreviations $T_0$ to $T_5$ refer to text.

TABLE III Systemic and pulmonary vascular resistance indices

| | *Group* | $T_0$ | $T_1$ | $T_2$ | $T_3$ | $T_4$ | $T_5$ |
|---|---|---|---|---|---|---|---|
| SVRI | I | 1532 ± 472 | 1278* ± 453 | 1432 ± 467 | 1428 ± 418 | 1654 ± 640 | 1659 ± 562 |
| | II | 1602 ± 536 | 1329* ± 478 | 1398 ± 436 | 1541 ± 527 | 1478 ± 412 | 1582 ± 512 |
| PVRI | I | 168 ± 58 | 157 ± 68 | 169 ± 74 | 171 ± 72 | 192* ± 69 | 188* ± 54 |
| | II | 164 ± 65 | 159 ± 55 | 174 ± 69 | 169 ± 65 | 189* ± 65 | 191* ± 64 |

Data are expressed as mean ± SD. PVRI = pulmonary vascular resistance index ($dyne.sec^{-1}{\cdot}m^{-2}{\cdot}cm^{-5}$); SVRI = systemic vascular resistance index ($dyne{\cdot}sec^{-1}{\cdot}m^{-2}{\cdot}cm^{-5}$), $*P < 0.05$ *vs* baseline. For abbreviations $T_0$ to $T_5$ refer to text.

TABLE IV Alveolar-arterial oxygen partial pressure differences and shunt fraction

| | *Group* | $T_0$ | $T_1$ | $T_2$ | $T_3$ | $T_4$ | $T_5$ |
|---|---|---|---|---|---|---|---|
| $P_{(A\text{-}a)}\,DO_2$ (mmHg) | I | 19.8 ± 11.4 | 192.6 ± 98.7 | 223.4 ± 46.5 | 232.4 ± 55.2 | 96.3 ± 36.2 | 99.7 ± 46.5 |
| | II | 20.2 ± 12.2 | 202.5 ± 96.7 | 240.2* ± 58.4 | 262.4* ± 50.6 | 97.4 ± 29.8 | 96.7 ± 42.8 |
| $Q_S/Q_T$ (%) | I | 13.5 ± 5.0 | 15.2 ± 6.8 | 18.8 ± 8.9 | 20.6 ± 10.8 | 6.8 ± 4.8 | 10.6 ± 5.6 |
| | II | 14.6 ± 7.0 | 16.3 ± 8.4 | 23.4* ± 11.2 | 23.9* ± 11.2 | 7.5 ± 6.1 | 9.8 ± 6.1 |

Data are expressed as mean ± SD. Alveolar-arterial oxygen partial pressure difference ($P_{(A\text{-}a)}\,DO_2$) shunt fraction ($Q_S/Q_T$). $*P < 0.05$ *vs* Group I. For abbreviations $T_0$ to $T_5$ refer to text.

MVV decreased significantly ($P < 0.05$) after surgery compared to baseline values, there were no significant differences between groups at corresponding times ($P > 0.05$). Hyperinflation of the lungs due to added CPAP did not compromise surgical exposure in Group I patients. No patient experienced difficulty emerging from bypass or required inotropic support. All patients were discharged to the ward from the SICU on the first postoperative day. There were no perioperative myocardial infarcts or deaths.

## Discussion

Cardiopulmonary bypass is well known to trigger an inflammatory process, and has adverse effects on end-organ function. The etiology of impaired pulmonary function after open heart surgery is multifactorial, including effects of the anesthetic technique, extracorporeal oxygenation technique, and metabolic changes. In particular, CPB activates leucocytes and inflammatory pathways, resulting in an alteration in capillary permeability.[6–9] Activation of inflammatory pathways, such as the complement system and arachidonic acid metabolism, damage endothelial integrity and cause accumulation of interstitial fluid.[10,11]

Various techniques have been evaluated to decrease the incidence and severity of pulmonary impairment following CPB. One measure is static inflation of the lungs (CPAP) during CPB. Loeckinger *et al.*[5] showed that the amount of intrapulmonary shunt was significantly larger in a control group after thoracic closure and four hours after CPB, whereas the magnitude of shunt in their CPAP (10 cm $H_2O$) group remained

TABLE V Arterial blood gas values

| | *Preoperative* | | *Postoperative day 1* | | *Postoperative day 3* | | *Postoperative day 5* | |
|---|---|---|---|---|---|---|---|---|
| | *Group I* | *Group II* | *Group I* | *Group II* | *Group I* | *Group II* | *Group I* | *Group II* |
| $P_aO_2$ (mmHg) | 85.3 ± 17.2 | 88.6 ± 16.9 | 86.7 ± 15 | 84.8 ± 17.6 | 83.6 ± 14 | 84.2 ± 17.8 | 81.2 ± 13.9 | 82.8 ± 14.2 |
| $P_aCO_2$ (mmHg) | 36.4 ± 7.2 | 35.8 ± 6.3 | 35.1 ± 4.3 | 35.8 ± 4.2 | 34.6 ± 5.2 | 34.9 ± 5.0 | 36.7 ± 4.6 | 36.3 ± 5.1 |
| $S_aO_2$ (%) | 97.8 ± 0.9 | 97 ± 1.0 | 96.8 ± 1.1 | 96.7 ± 1.2 | 96.1 ± 1.5 | 96.3 ± 1.3 | 96.9 ± 1.3 | 96.4 ± 1.1 |

Data are expressed as mean ± SD.

TABLE VI Pre- and postoperative pulmonary function

| | *Preoperative* | | *Postoperative day 1* | | *Postoperative day 3* | | *Postoperative day 5* | |
|---|---|---|---|---|---|---|---|---|
| *Group* | *I* | *II* | *I* | *II* | *I* | *II* | *I* | *II* |
| VC (L) | 3.1 ± 0.8 | 2.9 ± 0.7 | 1.2* ± 0.4 | 1.1* ± 0.5 | 1.4* ±0.3 | 1.4* ± 0.4 | 1.8* ± 0.5 | 1.7* ± 0.4 |
| $FEV_1$ ($L \cdot sec^{-1}$) | 2.4 ± 0.6 | 2.5 ± 0.6 | 1.2* ± 0.4 | 1.1* ± 0.4 | 1.3* ± 0.4 | 1.2* ± 0.4 | 1.4* ± 0.5 | 1.4* ± 0.6 |
| FVC (L) | 2.7 ± 0.7 | 2.8 ± 0.8 | 1.1* ± 0.3 | 1.2* ± 0.4 | 1.4* ± 0.4 | 1.3* ± 0.5 | 1.7* ± 0.5 | 1.8* ± 0.4 |
| FEF25-75% ($L \cdot sec^{-1}$) | 3.1 ± 0.7 | 3.0 ± 0.9 | 1.6* ± 0.6 | 1.6* ± 0.6 | 1.7* ± 0.6 | 1.7* ± 0.5 | 1.8* ± 0.8 | 1.9* ± 0.8 |
| PEFR ($L \cdot sec^{-1}$) | 4.3 ± 1.4 | 4.2 ± 1.7 | 2.4* ± 1.0 | 2.5* ± 0.9 | 2.7* ± 0.8 | 2.8* ± 0.7 | 3.4* ± 1.1 | 3.2* ± 1.0 |
| MVV ($L \cdot min^{-1}$) | 68.8 ± 18.8 | 68.2 ± 19.1 | 42.4* ± 17.7 | 45.2* ± 18.1 | 45.9* ± 18.1 | 47.1* ± 15.5 | 50.6* ± 15.2 | 52.3* ± 14.8 |

Data are expressed as mean ± SD. *$P < 0.05$ *vs* baseline. VC = vital capacity; $FEV_1$ = forced expiratory volume in first second; FVC = forced vital capacity; FEF 25–75% = forced expiratory flow between 25% and 75% of FVC; PEFR = peak expiratory flow rate; MVV = maximal voluntary ventilation.

unchanged.[5] Ishikawa also reported that positive end-expiratory pressure (PEEP) with a pressure of 5 cm $H_2O$ initiated immediately after pleurotomy, prevents oxygen impairment and atelectasis after extracorporeal circulation.[12] However, the duration of the positive effects of PEEP was not documented in that study.

In the current investigation, we examined the effects of CPAP at 10 cm $H_2O$ on gas exchange and respiratory parameters during and after cardiac surgery. Although we found a lower shunt fraction and $P_{(A\text{-}a)}DO_2$ in the CPAP Group I at $T_2$ (20 min after the termination of CPB ) and $T_3$ (after thoracic closure), like Loeckinger and Ishikawa, the difference between groups did not extend to the postoperative period. In this respect our results are similar to those of Berry *et al.*[4] who reported significantly lower $P_{(A\text{-}a)}DO_2$ values in patients receiving 5 cm $H_2O$ CPAP at 30 min after CPB, but not at four hours and eight hours after CPB.

Although many studies have evaluated the use of CPAP to minimize pulmonary dysfunction after CPB, the results are conflicting. The technique was reported as an effective treatment in some studies,[5,12,13] however others reported either no difference, or a short-lived difference between patients who received CPAP and those who did not.[4,14,15] In addition, there is no consensus regarding the optimal level of CPAP. Magnusson *et al.*[14] reported that CPAP with 5 cm $H_2O$ applied during CPB did not prevent postoperative atelectasis and gas exchange impairment in pigs. Moreover, the maneuver was associated with a decrease in cardiac output compared to baseline.[14] Boldt *et al.*,[3] on the other hand, reported that EVLW and $Q_S/Q_T$ increased after bypass regardless of the CPAP pressure, and changes in $P_aO_2$ correlated significantly with changes in EVLW. These investigators showed that static inflation with a moderate level of PEEP (5 cm $H_2O$) at an $F_IO_2$ of 0.21 decreased EVLW and improved $P_aO_2$. Taking these studies into consideration, we applied a CPAP level of 10 cm $H_2O$ in the present study. Magnusson *et al.*[13] demonstrated that a vital capacity maneuver (inflating the lungs to 40 cm $H_2O$ before termination of CPB) is effective in preventing post-CPB atelectasis and gas-exchange

impairment. Furthermore, they also showed that $P_aO_2$, and shunt fraction are well correlated with atelectasis.[16] It is now well-known that intrapulmonary shunt increases 20–25% after CPB.[17,18] Westerdahl *et al.*[19] reported that atelectasis persists up to the fourth postoperative day following cardiac surgery and deep-breathing exercises significantly decrease atelectasis.

Previous reports have shown significant deterioration in pulmonary function after CPB. Vargas *et al.*[20] analyzed the relationship between pleural changes and PFT in patients who received saphenous vein graft (SVG group) alone or in combination with internal mammary artery (IMA group) grafting. These authors concluded that additional thoracic trauma in patients having IMA graft is associated with a larger decrement in pulmonary function. Shapira *et al.*[21] reported reductions of 19%-33% in all lung volumes, with the exception of residual volume and expiratory flow rates, which decreased from 33% to 37% in healthy males undergoing elective coronary artery bypass grafting. We also observed a significant decrease in PFT values after surgery, while hemodynamic variables remained similar in the control and CPAP groups.

In summary, CPAP administered during CPB decreases shunt fraction and alveolar-arterial oxygen partial pressure difference during surgery, but has no sustained effect on either variable postoperatively. We conclude that, in patients with normal preoprative pulmonary function, application of 10 cm $H_2O$ CPAP does not improve lung function after cardiac surgery.

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