Does Bilevel Positive Airway Pressure Improve Outcome of Acute Respiratory Failure after Open-heart Surgery?

Abstract
Background: Respiratory failure is of concern in the postoperative period after cardiac surgeries. Invasive ventilation (intermittent positive pressure ventilation [IPPV]) carries the risks and complications of intubation and mechanical ventilation (MV). Aims: Noninvasive positive pressure ventilation (NIPPV) is an alternative method and as effective as IPPV in treating insufficiency of respiration with less complications and minimal effects on respiratory and hemodynamic parameters next to open-heart surgery. Design: This is a prospective, randomized and controlled study.

Materials and Methods: Forty-four patients scheduled for cardiac surgery were divided into two equal groups: Group I (IPPV) and Group II (NIPPV). Heart rate (HR), mean arterial pressure (MAP), respiratory rate (RR), oxygen saturation (SpO₂), arterial blood gas, weaning time, reintubation, tracheotomy rate, MV time, postoperative hospital stay, and ventilator-associated pneumonia during the period of hospital stay were recorded. Results: There was a statistically significant difference in HR between groups with higher in Group I at 30 and 60 min and at 12 and 24 h. According to MAP, it started to increase significantly at hypoxemia, 15 min, 30 min, 4 h, 12 h, and at 24 h which was higher in Group I also. RR, PaO₂, and PaCO₂ showed significant higher in Group II at 15, 30, and 60 min and 4 h. According to pH, there was a significant difference between groups at 15, 30, and 60 min and at 4, 12, and 24 h postoperatively. SpO₂ showed higher significant values in Group I at 15 and 30 min and at 12 h postoperatively. Duration of postoperative supportive ventilation was higher in Group I than that of Group II with statistically significant difference. Complications were statistically insignificant between Group I and Group II. Conclusion: Our study showed superiority of invasive over noninvasive mode of ventilator support. However, NIPPV (bilevel positive airway pressure) was proved to be a safe method.

Keywords: Acute, failure, open heart, respiratory

Introduction
Respiratory complications after cardiac surgeries are one of the most important concerns for cardiac surgeons and anesthesiologists. These postoperative events may range from a minor respiratory impairment to acute respiratory distress syndrome (ARDS). Atelectasis is one of the most common causes of these complications that can lead to hypoxia and pneumonia. Each of these complications increases the incidence of morbidity and mortality. The main inducing factors for atelectasis are as follows: general anesthesia, cardiopulmonary bypass (CPB), and discontinued blood perfusion of the lungs and gas-exchange impairment during the surgery. Complications of intubation and mechanical ventilation (MV) include barotrauma, nosocomial pneumonia, sinusitis, and psychological problems. Use of invasive ventilation may cause loss of airway-protecting mechanisms, more sedative use, and airway trauma, all of which may increase the risk of ventilator-associated pneumonia (VAP), hospital stay, morbidity, and mortality.

Application of noninvasive positive pressure ventilation (NIPPV) at different levels of inspiration and expiration using face or nose masks has reduced the necessity of endotracheal intubation. In recent years, noninvasive ventilation (NIV), by reopening atelectasis, can prevent postoperative pneumonia. NIV can also offer beneficial effects on the cardiovascular function by lowering left ventricle after load and improving cardiac output, also mild reduction of the cardiac function due to NIV has been reported. A strict monitoring is required in patients with labile cardiac function.

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Although NIPPV has been successfully used for various kinds of acute respiratory failure (ARF), there are only limited studies regarding the application of NIPPV to treat postoperative respiratory failure after cardiac surgery. Therefore, we conducted this study to evaluate the efficacy and safety of NIPPV in the treatment of ARF in this aforesaid group.

Materials and Methods

This study was carried out for 1-year duration in cardiothoracic surgery department. A total of 96 patients were enrolled. Of these 96 patients, 52 were excluded. The remaining 44 eligible ones were randomized, allocated to intervention, and scheduled for cardiac surgery. The Ethical Committee approval was obtained and informed written consent was taken from all the patients included in this study, after explanation of the procedure, and all patients were identified by coded numbers to maintain privacy. Research results were only used for scientific purposes.

Patients’ exclusion criteria included cardiac or respiratory arrest, loss of consciousness, any condition causing abdominal distention, severe other organ dysfunctions such as refractory hypotension, severe arrhythmia, undrained pneumothorax or pneumomediastinum, refusal for the use of NIPPV, psychomotor agitation requiring sedation, need of immediate endotracheal intubation (ETI) for excessive airway secretions, hyperthermia (T > 38°C) or hypothermia (T < 35°C), hemodynamic instability, use of intra-aortic balloon counterpulsation, and urine output <0.5 ml/kg/h.

All patients underwent standard monitoring, general anesthesia as per the institutional protocol, and were operated with a midline incision and CPB. After the end of the surgery and Intensive Care Unit (ICU) transfer, patients were assessed continuously and all patients who developed respiratory failure and fulfilled all the criteria of new-onset ARF after initial weaning in ICU (ARF was defined by arterial partial O₂ pressure [PaO₂] ≤60 mmHg after oxygen therapy through nasal prong or PaO₂/fraction of inspired O₂ [PaO₂/FiO₂] ≤200 mmHg after oxygen therapy through a venturi mask with or without respiratory acidosis [pH < 7.35 with arterial partial CO₂ pressure (PaCO₂) >45 mmHg], and respiratory rate [RR] >25 breaths/min) were randomly divided into two groups.

Group I (intermittent positive pressure ventilation)

Patients were intubated by rapid sequence induction by intravenous injection of fentanyl 1–2 µg/kg, thiopental 3 mg/kg, and succinylcholine 1.5 mg/kg; intubation was done with cuffed endotracheal tube (internal diameter from 7 to 7.5 mm) and MV was started on synchronized intermittent mandatory ventilation mode.

Initially, the ventilator was set to provide a tidal volume of 6–8 ml/kg, RR of 12–15 breaths/min, and FiO₂ of 1.0; the head of the bed was elevated at an angle of 45° to minimize the risk of aspiration. Positive end-expiratory pressure level was set at 5 cmH₂O initially, and then it was increased in increments of 2–3 cmH₂O up to 10 cmH₂O, until the FiO₂ requirement was 0.6 or less and pressure support at 12 cmH₂O. Sedation was given with propofol infusion (1–1.5 mg/kg/h) or by fentanyl infusion (1–2 µg/kg/h). None of the patients received a muscle relaxant after the intubating dose. All the patients were weaned and extubated, once found acceptable by decreasing and stopping sedation, reducing the level of pressure support and decreasing the ventilator rate and fulfilled criteria of being awake, cooperative, with no residual neuromuscular blockade, partial pressure of oxygen (PaO₂) >80 mmHg (FiO₂ = 0.4), partial pressure of carbon dioxide (PaCO₂) <45 mmHg, stable hemodynamic and metabolic parameters, and bleeding <100 ml/h.

Group II (noninvasive positive pressure ventilation)

Patients in this group received bilevel positive airway pressure (BiPAP). It was administered using the BiPAP spontaneous/triggered mode through a properly fitted face mask, with low risk of air leaks, and the head of the bed was raised to 30°–45° to minimize the risk of aspiration. Moreover, utilizing a disposable spacer was possible to reduce the pressure on the bridge of the patient’s nose and to reduce dead space. The mask was secured with head straps to avoid an excessively tight fit (patients were not sedated in this group).

The initial inspiratory positive airway pressure (IPAP) was set at 12 cmH₂O, and the initial expiratory positive airway pressure (EPAP) was set at 5 cmH₂O. Backup respiratory frequency was set at 12 breaths/min according to the clinical efficacy and patient’s tolerance. IPAP and EPAP were raised by 2–3 cmH₂O every 5–10 min, but the IPAP/EPAP was not exceeding 25/10 cmH₂O. FiO₂ was adjusted to maintain a pulse oxygen saturation (SpO₂) of more than 92% and in chronic obstructive pulmonary disease patients up to 92%.

All patients continued to receive BiPAP except for coughing, eating, and talking until their condition improved. Then, BiPAP was administered intermittently and the IPAP/EPAP was decreased gradually. When the support pressure level (IPAP-EPAP) reached to ≤5 cmH₂O, a weaning trial was done. When there are no signs of dyspnea at rest, Glasgow Coma Scale = 15; RR ≤ 25 breaths/min, pH ≥ 7.35, PaO₂ ≥ 60 mmHg, and SpO₂ > 90% at room air or FiO₂ ≤ 40% with venturi mask during spontaneous breathing and sustained for 48 h, NIPPV was considered successful. When there was failure to meet the above-mentioned criteria, BiPAP was resumed until meeting the criteria or need of ETI.
Measurements of parameters

1. Vital signs such as heart rate (HR), mean arterial pressure (MAP), RR, and Sp\textsubscript{O}\textsubscript{2}.
2. Arterial blood gases (pH, Pa\textsubscript{CO}\textsubscript{2}, and Pa\textsubscript{O}\textsubscript{2}) were recorded immediately before management or at 15 min, 30 min, 1 h, 4 h, 12 h, and finally after 24 h.
3. Weaning time, reintubation rate, and tracheotomy rate.
4. MV time, postoperative hospital stay, and VAP during the period of hospital stay.

Statistical analysis

Qualitative variables were expressed as frequency and percentage, and quantitative variables were expressed as mean ± standard deviation when the data were in a normal distribution or expressed as median (P25, P75) when there was skewed distribution (MV time, ICU stay, and postoperative hospital stay).

For normal distribution and equal variance qualitative data, the two-sample Student’s t-test and paired Student’s t-test were used to determine differences between and within the groups, respectively. For the nonparametric variables, Mann–Whitney test was used for variables that were not normally distributed. Qualitative variables were tested using Chi-square or Fisher’s exact test.

To identify the risk factors for NIPPV failure, univariate analysis was used to compare patients demographic and baseline characteristic data in the NIPPV success and failure subgroup patients, and then multivariate logistic regression analysis was used to identify risk factors for NIPPV failure.

$P < 0.05$ was considered statistically significant.

Sample size was calculated to be 40 on the following considerations: 95% confidence level, 80% power of the study, equal size of both studied groups, and the estimated outcome to range from 50 to 80 between the studied groups.

Results

Twenty-two patients were included in each group according to previously mentioned inclusion criteria.

Demographic data were comparable with insignificant difference in age, sex, and body mass index. Furthermore, patients’ comorbidities and duration and type of the surgery in the two groups were statistically insignificant [Table 1].

There was statistically significant difference in HR between the two groups with higher rate in Group I at 30 and 60 min and at 12 and 24 h. According to MAP, it started to increase significantly at hypoxemia, 15 min, 30 min, 4 h, 12 h, and at 24 h which was higher in Group I also ($P < 0.05$). RR, Pa\textsubscript{O}\textsubscript{2}, and Pa\textsubscript{CO}\textsubscript{2} showed significant higher rate in Group II at 15, 30, and 60 min and 4 h. According to pH, there was significant difference between the two groups at 15, 30, and 60 min and at 4, 12, and 24 h postoperatively.

### Table 1: Demographic data

|                        | Group I ($n=22$) | Group II ($n=22$) | Test of significance | $P$ |
|------------------------|------------------|-------------------|----------------------|-----|
| Age (years)            | 62.0±10.3        | 61.0±12.2         | $F=0.294$            | 0.770 |
| Sex (%)                |                  |                   |                      |     |
| Male                   | 12               | 13                | $\chi^2=0.093$       | 0.760 |
| Female                 | 10               | 9                 |                      |     |
| BMI (kg/m$^2$)         | 25.3±4.6         | 24.4±3.5          | $F=0.730$            | 0.469 |
| COPD                   | 1                | 3                 | $\chi^2=0.275$       | 0.672 |
| HTN                    | 13               | 11                | $\chi^2=0.092$       | 0.762 |
| DM                     | 7                | 9                 | $\chi^2=0.098$       | 0.754 |
| Liver disease (Child A)| 1                | 2                 | $\chi^2=0.125$       | 0.821 |
| Smoking                | 10               | 11                | $\chi^2=0.091$       | 0.763 |
| Duration of surgery (min) | 227±51           | 224±49            | $F=0.199$            | 0.843 |
| Cross clamp time (min) | 55.25±25.6       | 60.6±13.18        | $F=0.253$            | 0.46  |
| Valve surgery          | 15               | 17                | $\chi^2=0.570$       | 0.752 |
| CABG                   | 7                | 5                 | $\chi^2=0.115$       | 0.735 |

BMI: Body mass index, COPD: Chronic obstructive pulmonary disease, HTN: Hypotension, DM: Diabetes mellitus, CABG: Coronary artery bypass graft.

Sp\textsubscript{O}\textsubscript{2} showed higher significant values in Group I at 15 and 30 min and at 12 h postoperatively [Table 2].

Duration of postoperative supportive ventilation was higher in Group I (7.9 ± 0.921 h) than that of Group II (5.56 ± 0.995 h) with statistically significant difference at $P = 0.112$. Complications and ICU and postoperative hospital stay in days among both groups, Group I (IPPV) and Group II (BiPAP), were statistically insignificant [Tables 3-6].

Discussion

In the present study, we have compared the efficacy of NIV (BiPAP) with the conventional invasive ventilation.

In regard to changes in HR and MAP between the two groups, tachycardia occurred in the two groups at the onset of hypoxemia which is accompanied by sympathetic stimulation. Once intervention was done, HR started to decline in Group II while it continued to increase in Group I which may be attributed to add-on stress by the in situ endotracheal tube. After cessation of supportive ventilation, tachycardia in Group I also declined to preoperative levels. Furthermore, significant statistical difference occurred between the two groups in the MAP after hypoxemia showing higher MAP values in invasive ventilation group relative to BiPAP group. The RR relatively increased in Group II as compared to Group I significantly which may be due to sedatives used in Group I.

Sa\textsubscript{O}\textsubscript{2} reached the accepted saturations after 30 min and satisfactory levels at 1 h after hypoxemia with significant statistical difference between the two groups in the degree...
Table 2: Heart rate, mean arterial pressure, respiratory rate, PaO₂, PaCO₂, pH, and SpO₂

|                  | Group I (n=22)       | Group II (n=22)      | P       |
|------------------|----------------------|----------------------|---------|
| **HR**           |                      |                      |         |
| Baseline         | 83.86±6.76           | 82.77±11.78          | 0.7083  |
| Hypoxemia        | 103.31±3.59          | 104.59±7.79          | 0.4903  |
| 15 min           | 108.68±6.33          | 94.63±8.07           | 0.0001* |
| 30 min           | 96.27±6.06           | 88.7±6.43            | 0.0066* |
| 60 min           | 85.45±5.95           | 82.04±5.67           | 0.4561  |
| 4 h              | 81.95±6.00           | 80.9±5.93            | 0.5309  |
| 12 h             | 82.76±6.9            | 78.2±6.02            | 0.0235  |
| 24 h             | 83.80±6.93           | 78.5±6.17            | 0.0115  |
| **MAP**          |                      |                      |         |
| Baseline         | 88.54±9.35           | 83.09±10.05          | 0.0694  |
| Hypoxemia        | 103.68±11.57         | 89.59±12.35          | 0.0002* |
| 15 min           | 101.63±9.26          | 85.54±8.62           | 0.0001* |
| 30 min           | 99.36±9.26           | 84.77±9.06           | 0.0001* |
| 60 min           | 97.72±8.18           | 78.59±7.90           | 0.0001* |
| 4 h              | 89.55±8.16           | 78.72±7.07           | 0.0001* |
| 12 h             | 88.55±8.15           | 79.33±6.08           | 0.0001* |
| 24 h             | 88.15±7.6            | 80.38±5.21           | 0.0001* |
| **RR**           |                      |                      |         |
| Baseline         | 23.6±2.72            | 22.3±2.88            | 0.1264  |
| Hypoxemia        | 39.3±2.66            | 39.04±1.55           | 0.6808  |
| 15 min           | 33.09±2.13           | 36.54±1.58           | 0.0001* |
| 30 min           | 27.04±1.63           | 31.27±1.60           | 0.0001* |
| 60 min           | 23.8±1.11            | 26.9±1.99            | 0.0003* |
| 4 h              | 24.27±0.91           | 21.7±2.83            | 0.0001* |
| 12 h             | 19.00±1.87           | 18.9±1.89            | 0.7674  |
| 24 h             | 20.42±2.78           | 19.13±1.76           | 0.0707  |
| **PaO₂**         |                      |                      |         |
| Baseline         | 88.59±4.63           | 86.54±5.06           | 0.170   |
| Hypoxemia        | 52.88±3.46           | 52.95±1.66           | 0.928   |
| 15 min           | 63.4±3.79            | 60.27±2.27           | 0.0019* |
| 30 min           | 78.90±6.76           | 68.5±3.31            | 0.0001* |
| 60 min           | 152.40±5.11          | 143.04±4.79          | 0.0001* |
| 4 h              | 189.81±18.97         | 166.75±48.00         | 0.0001* |
| 12 h             | 87.19±4.31           | 86.05±24.85          | 0.290   |
| 24 h             | 89.38±4.23           | 87.95±25.36          | 0.164   |
| **PaCO₂**        |                      |                      |         |
| Baseline         | 41.59±1.96           | 42.5±2.25            | 0.142   |
| Hypoxemia        | 62.59±3.33           | 62.5±2.25            | 0.959   |
| 15 min           | 50.27±2.15           | 51.68±3.15           | 0.091*  |
| 30 min           | 43.68±2.22           | 46.5±2.44            | 0.0002* |
| 60 min           | 40.95±1.55           | 44.6±2.06            | 0.0001* |
| 4 h              | 40.09±2.52           | 42.11±2.72           | 0.0174* |
| 12 h             | 41.38±1.96           | 42.00±2.72           | 0.380   |
| 24 h             | 40.75±2.05           | 41.7±3.06            | 0.234   |
| **pH**           |                      |                      |         |
| Baseline         | 7.42±0.02            | 7.40±0.02            | 0.027   |
| Hypoxemia        | 7.25±0.019           | 7.26±0.016           | 0.1003  |
| 15 min           | 7.30±0.012           | 7.28±0.017           | 0.0006* |
| 30 min           | 7.32±0.01            | 7.30±0.016           | 0.0001* |
| 60 min           | 7.37±0.019           | 7.33±0.017           | 0.0001* |
| 4 h              | 7.40±0.018           | 7.35±0.03            | 0.0001* |
| 12 h             | 7.42±0.017           | 7.35±0.02            | 0.0001* |

Contd...
of correction of SpO₂ at 15 min, 30 min, and 12 h after hypoxemia. PaO₂ reached accepted tensions after 15 min in Group I and 30 min in Group II with satisfactory tensions at 2 h after hypoxemia in the two groups. This gradual improvement can be explained by the gradual recruitment of the collapsed alveoli. The superiority of invasive ventilation over NIPPV in improving oxygenation can be explained by the absence of tidal volume leakage that occurs in NIPPV making better pulmonary inflation and lung recruitment as well as full dependence of NIPPV on patients’ respiratory effort which is impaired by surgical pain in poststernotomy. The decrease in PaO₂ after discontinuation of supportive ventilation can be explained by recollapse of some alveolar units. Similarly, effectiveness of ventilation was significantly better in invasive ventilation group as compared to NIPPV group when the carbon dioxide levels and pH were compared probably due to better air exchange and complete recruitment of the lungs in ventilated group. Our results of NIPPV were somewhat similar to what were reported by Guang-Fa et al.[8] The complications, ICU and postoperative hospital stay days were more in ventilated patients as compared to those who had been managed with NIPPV but the difference did not reach any statistical significance.

From more than 300,000 patients who undergo cardiac surgery every year in the United States, as many as 20% will have ARDS,[9,10] however, in our cardiac surgery center and in a 1-year study, out of 96 patients, 44 needed respiratory support either invasive or noninvasive, it was a high (45%) incidence of respiratory insufficiency after cardiac surgery, and we believed that the causes may be due to longer bypass times, the use of hypothermic circulatory arrest, and requirement for more blood products which are now under control.

This study had some limitations related to location and starting time of the study: it was started at the early stage of respiratory insufficiency and performed in the ICU, due to the inherent better availability of the required equipment, better monitoring, better staffing, and better experience and knowledge. The early use of the NIPPV (BIPAP) postoperative is cost-effective, but available data are limited. We conclude that though NIPPV (BIPAP) is considered an effective and safe method in the postoperative period after cardiac surgeries, but still there seems to be a superiority of invasive ventilation over it and further larger and multicentric studies are still needed to better define the best protocols to improve efficacy in early development of ARF postcardiac surgery.

Table 2: Contd...

|        | Group I (n=22) | Group II (n=22) | P      |
|--------|---------------|----------------|--------|
| 24 h   | 7.42±0.012    | 7.36±0.03      | 0.0001*|
| SpO₂   |               |                |        |
| Baseline | 97.00±1.167  | 96.95±1.26     | 0.902  |
| Hypoxemia | 83.95±2.495 | 83.00±1.53     | 0.134  |
| 15 min  | 91.09±1.90    | 89.36±2.496    | 0.0135*|
| 30 min  | 95.40±1.94    | 93.54±2.791    | 0.0139*|
| 60 min  | 98.7±0.86     | 98.13±1.35     | 0.092  |
| 4 h     | 99.36±0.710   | 99.05±0.80     | 0.118  |
| 12 h    | 96.90±1.13    | 95.45±1.43     | 0.0063*|
| 24 h    | 96.81±1.26    | 95.80±1.12     | 0.058  |

Data are represented as mean±SD. *Statistically significant at P<0.05. HR: Heart rate, MAP: Mean arterial pressure, RR: Respiratory rate, SD: Standard deviation

Table 3: Duration and type of the surgery

|        | Group I (n=22) | Group II (n=22) | Test of significance | P     |
|--------|---------------|----------------|----------------------|-------|
|        |               |                |                      |       |
| Duration of surgery (min) | 227±51         | 224±49         | F=0.199              | 0.843 |
| Cross clamp time (min)    | 55.25±25.6     | 60.6±13.18     | F=0.253              | 0.46  |
| Valve surgery             | 15             | 17             | χ²=0.570             | 0.752 |
| CABG                     | 7              | 5              | χ²=0.115             | 0.735 |

F: F-test (ANOVA). Data are represented as mean±SD. CABG: Coronary artery bypass graft, SD: Standard deviation

Table 4: Complications

|        | Group I (n=22) | Group II (n=22) | P  |
|--------|---------------|----------------|----|
| Facial erythema | -             | 2              | 0.488 |
| Aspiration    | -             | -              | 1   |
| Ventilator associated pneumonia | 2              | 1              | 0.654 |
| Postoperative pneumonia | 1             | -              | 0.745 |
| Reintubation  | 1             | 2              | 0.625 |
| Tracheotomy   | 1             | 1              | 1   |
| Inhospital mortality | 2             | -              | 0.488 |
| Stroke        | 1             | -              | 0.745 |
### Table 5: Duration of supportive ventilation

| Number | Group I | Group II |
|--------|---------|----------|
| 1      | 28      | 26       |
| 2      | 29      | 27       |
| 3      | 30      | 27       |
| 4      | 27      | 25       |
| 5      | 29      | 26       |
| 6      | 31      | --       |
| 7      | 33      | 29       |
| 8      | 29      | 26       |
| 9      | 28      | 25       |
| 10     | -       | 36       |
| 11     | 36      | 27       |
| 12     | -       | 25       |
| 13     | 28      | 27       |
| 14     | 36      | 36       |
| 15     | 35      | 32       |
| 16     | 37      | 32       |
| 17     | 36      | -        |
| 18     | 38      | 35       |
| 19     | 39      | 37       |
| 20     | 38      | 35       |
| 21     | -       | 35       |
| 22     | 36      | 34       |
| Mean±SD| 7.9±0.92| 5.5±0.99 |

*P value <0.05. Comparison between the two groups in duration of supportive ventilation (h). SD: Standard deviation

### Table 6: Intensive Care Unit and postoperative hospital stay days

| Variables                        | Group I | Group II |
|----------------------------------|---------|----------|
| ICU stay days                    | 6       | 5        |
| Postoperative hospital stay days | 8       | 6        |

ICU: Intensive Care Unit

### Conclusion

We conclude that though NIPPV (BIPAP) is considered an effective and safe method in the postoperative period after cardiac surgeries, but still there seems to be a superiority of invasive ventilation over it and further larger and multicentric studies are still needed to better define the best protocols to improve efficacy in early development of ARF postcardiac surgery.

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Nil.

### Conflicts of interest

There are no conflicts of interest.

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