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Effects of Noninvasive Positive-Pressure Ventilation with Different Interfaces in Patients with Hypoxemia after Surgery for Stanford Type A Aortic Dissection

Authors' Contribution:
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Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
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Background: Hypoxemia is a severe perioperative complication that can substantially increase intensive care unit and hospital stay and mortality. The aim of this study was to determine the effects of non-invasive positive-pressure ventilation (NIPPV) in patients with hypoxemia after surgery for Stanford type A aortic dissection, and to compare the effects of helmet and mask NIPPV.





Material/Methods: We recruited 40 patients who developed hypoxemia within 24 h after extubation after surgery for Stanford type A aortic dissection in the Beijing Anzhen Hospital. The patients were randomly divided into the helmet and mask NIPPV groups. The primary endpoints were blood oxygenation levels at 1 and 6 h after initiation and at the end of the treatment. The secondary endpoint was patient outcome, including mortality; incidence of pulmonary atelectasis, pneumonia, re-intubation, and sepsis; and length of ICU and hospital stays.

Results: NIPPV improved oxygenation in both groups. Compared with pretreatment levels, the oxygenation index ($\text{PaO}_2/\text{FiO}_2$), PaO_2 , PaCO_2 , and respiratory rate (RR) improved in the initial (0–1 h), maintenance (1–6 h), and end stages of the treatment ($P < 0.05$). Compared with mask ventilation, helmet ventilation better improved pH, PaO_2 , SpO_2 , $\text{PaO}_2/\text{FiO}_2$, and decreased PaCO_2 in the 3 stages ($P < 0.05$). The incidence of major complications, including flatulence, intolerance, and facial pressure sores, was significantly lower with helmet ventilation.

Conclusions: NIPPV effectively improved oxygenation and reduced PaCO_2 in patients who developed hypoxemia soon after extubation following surgery for Stanford type A aortic dissection. Compared with mask NIPPV, helmet NIPPV more rapidly increased PaO_2 and reduced PaCO_2 , increased patient tolerance and comfort, and reduced complications.

MeSH Keywords: Anoxia • Aortic Diseases • Continuous Positive Airway Pressure • Head Protective Devices

Full-text PDF: <http://www.medscimonit.com/abstract/index/idArt/893956>

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Background

Due to age, preoperative complications from chronic cardiopulmonary function impairment, intraoperative anesthesia, surgical trauma, cardiopulmonary bypass, surgical incision pain, and secondary infection, cardiac surgery patients may suffer from respiratory function impairment caused by complications of alveolar collapse, acute lung injury, pulmonary edema, heart failure, pulmonary thromboembolism, or infection. Hypoxemia is a common but severe perioperative complication following cardiac surgery, and can substantially increase intensive care unit (ICU) and hospital stay and mortality. Perioperative hypoxemia has been associated with heart dysfunction atelectasis, obesity, smoking, activation of the fibrinolytic system, and excessive inflammatory response [1]. The incidence of pulmonary complications such as atelectasis in patients with perioperative hypoxemia is approximately 54–92% [2,3]. Increased secretion of inflammatory factors can also result in an abnormal ventilation/perfusion ratio. The incidence of hypoxemia can be as high as 51.6% in patients with type A aortic dissection [2]. In these cases prolonged hypoxemia may appear during the perioperative period, in particular during the postoperative period, because of hematological impairment induced by the stress response, release of inflammatory factors, destruction of lung capillary bed, interstitial lung exudate, intraoperative deep hypothermic circulatory arrest, massive blood loss, and blood transfusion.

High incidence and prolonged duration of hypoxemia may result in difficult weaning from mechanical ventilation or post-weaning re-intubation and even eventual tracheotomy, which increases the incidence of trauma and infection and prolongs the intensive care unit and hospital stays, resulting in increased economic burden and a waste of medical resources. Applying positive end-expiratory pressure (PEEP) or positive-pressure ventilation during mechanical ventilation can prevent pulmonary atelectasis, reduce the severity of inflammatory pulmonary effusion, and improve cardiac function to a certain extent. However, non-invasive positive-pressure ventilation (NIPPV) is the only method that can produce the above outcomes after mechanical ventilation [4]. There are 2 methods of NIPPV: continuous positive airway pressure (CPAP) with a constant positive pressure throughout the cycle, and bi-level positive airway pressure (BIPAP) in which the ventilator delivers different levels of pressure during inspiration and expiration.

The most commonly used interfaces for NIPPV include oral-nasal masks, nasal masks, rhinobyon, and hoods. Oral-nasal masks are universally applied in clinical practice. However, these masks are not very comfortable, and many patients cannot tolerate the masks well. Furthermore, the relative high occurrence of air leakage and skin lesions at the nose induced by long-term use of these masks result in frequent treatment

interruptions or even treatment discontinuation. In contrast, helmet NIPPV is associated with good tolerance, low incidence of complications, and similar ventilation effects as those of the oral-nasal mask, and is therefore becoming increasingly popular. However, very few studies in China, especially those pertaining to cardiac surgery or surgery of the great vessels, have focused on helmet NIPPV. We hypothesized that helmet NIPPV would provide more benefit to patients who developed hypoxemia after undergoing surgery for Stanford type A aortic dissection than mask NIPPV. Therefore, in the present study, the effects of helmet NIPPV and mask NIPPV were investigated and compared.

Material and Methods

Patients

The present study was approved by the ethics committee of Beijing Anzhen Hospital (ID: 2013014), and written informed consent was obtained from the patients or their family.

We recruited 40 patients who developed hypoxemia within 24 h extubation after undergoing surgery for Stanford type A aortic dissection in the Department of Cardiac Surgery, Beijing Anzhen Hospital, and were consecutively enrolled between November 2013 and July 2014.

The inclusion and exclusion criteria were based on those used in previous studies [5,6]. The inclusion criteria were patients who: (1) underwent surgery for Stanford type A aortic dissection, (2) were conscious (Glasgow score ≥ 13), normal cough reflex, and not requiring sedatives, (3) had normal muscle strength within 24 h after extubation in the ICU, (4) had acute respiratory failure as indicated by tachypnea, respiratory rate (RR) > 30 breaths/min, respiratory muscle fatigue, and $\text{PaO}_2 < 60$ mmHg, $\text{PaO}_2/\text{FiO}_2 \leq 200$ mmHg, $\text{PaCO}_2 > 50$ mmHg, or $\text{pH} < 7.35$ after oxygen therapy; (5) had dyspnea and paradoxical breathing; (6) had no active bleeding in the mediastinum or pericardium, and volume of chest drainage ≤ 100 mL/h; (7) with stable circulation, without high dose vasoactive drugs (epinephrine $\leq 0.05 \mu\text{g}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$, dopamine $\leq 5 \mu\text{g}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$), and without severe arrhythmia; (8) urine $\geq 0.5 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{h}^{-1}$ and hemoglobin ≥ 90 g/L; and (9) had no contradictions to NIPPV.

The exclusion criteria were patients with one or more of the following: (1) unstable hemodynamics (systolic arterial pressure < 80 mmHg or electrocardiogram suggestive of myocardial ischemia or severe arrhythmia); (2) severe cardiac insufficiency (ejection fraction $< 25\%$) or cardiorespiratory arrest; (3) weak or absent spontaneous respiration and disturbed consciousness; (4) high risk of aspiration, inability for oropharyngeal and upper respiratory tract secretions to be removed, with

sticky sputum and ineffective expectoration; (5) requirement of secondary mechanical ventilation after extubation and before NIPPV, or severe chronic obstructive pulmonary disease (COPD; forced expiratory volume in 1 s <50% after oxygen therapy); (6) heart and/or lung transplantation; (7) dysfunction of other organs (gastrointestinal perforation/hemorrhage, or severe cerebrovascular diseases); (8) undrained hemopneumothorax or mediastinal emphysema; (9) neck or facial trauma, burns, or bleeding or obstruction of the upper airway; (10) recent facial, upper airway, or gastrointestinal surgery; (11) irritation, extreme nervousness, or refusal to undergo NIPPV; (12) severe infective or toxic symptoms; and (13) refusal to sign the informed consent form.

Study design

Of the 40 patients, 25 were men, and 15 were women. The mean age of the patients was 54.1 ± 8.7 years. The patients were randomly divided into two groups, by a computer generated random number table, one of which received non-invasive NIPPV through a helmet and the other received this through a mask. The obvious differences in the NIPPV interfaces meant that there was no ability to use a blinding method. The operative methods were as follows: ascending aorta replacement + Sun procedure, 16 patients; Bentall procedure + Sun procedure, 14 patients; ascending aorta replacement, 6 patients; ascending aorta replacement + partial aortic arch replacement, 3 patients; and Bentall procedure + Sun procedure + mitral valve replacement + coronary artery bypass grafting, 1 patient.

Treatment method

First, a test for spontaneous breathing was performed in each eligible patient. In brief, the inspired oxygen concentration (FiO_2) was adjusted to 50% using a Venturi mask and maintained at this level for 15 min. Then patients with a $PaO_2/FiO_2 > 200$ stopped ventilation treatment, while patients with a $PaO_2/FiO_2 \leq 200$ were randomly divided into two groups, namely, the helmet and mask groups. Patients in the helmet group were treated with helmet CPAP (Intersurgical S.p.A., Italy). Their FiO_2 was adjusted to 40–50%, and PEEP was adjusted to 8–10 cm H_2O in order to maintain pulse oxygen saturation (SpO_2) >95%. For patients in the mask group, the Philips V60 non-invasive ventilator was used for BIPAP (initial parameters: inspiration pressure [IPAP], 10–20 cm H_2O ; expiration pressure [EPAP], 0–4 cm H_2O ; FiO_2 , 60–100%; inspiration: expiration, 1:1.5 to 1:2; and time for pressure increase, 0.5–1 s). All these parameters were adjusted gradually according to the clinical outcomes and patient tolerance. Blood gas analysis, assessment of vital signs were performed before treatment, 1 and 6 h after treatment initiation, and at the end of the treatment, and hepatorenal function and LVEF tests were performed before and after treatment. In addition, tests for spontaneous

respiration were repeated every 6 h. Conscious patients who had a $PaO_2/FiO_2 > 200$, IPAP-EPAP ≤ 5 cm H_2O , $FiO_2 \leq 45\%$, RR <25 breaths/min, no dyspnea, and a good cough reflex could stop NIPPV treatment. In patients with a $PaO_2/FiO_2 \leq 200$, NIPPV treatment was continued. Except during expectoration, speech, eating, and oral care, the patients were kept on NIPPV during the initial stage. After the acute respiratory failure had been abated and the SpO_2 had been maintained at a satisfactory level, the parameters of both the helmet and Philips V60 ventilators were gradually adjusted, and the NIPPV time was reduced for NIPPV weaning. In patients with no substantial improvement in hypoxemia during the treatment, the underlying causes were identified as soon as possible, while in patients with indications for re-intubation, the treatment was discontinued, re-intubation and mechanical ventilation performed [5,6].

Indications for intubation

Intubation was performed in patients with at least one of the major indications or two of the secondary indications. The major indications were: (1) weak or absent respiration; (2) unconsciousness, epilepsy, or coma (Glasgow score ≤ 8); (3) severe dysphoria; (4) cardiogenic shock; (5) unstable hemodynamics; (6) requirement of secondary surgical intervention; and (7) no improvement in oxygenation and inability to tolerate NIPPV. The secondary criteria included: (1) RR >35 breaths/min; (2) pH <7.30; (3) $PaO_2 < 45$ mmHg (which could not be alleviated by FiO_2 increase) or $PaO_2/FiO_2 \leq 140$; (4) mental deterioration; and (5) weak cough that was ineffective at removing airway secretions [6,7].

Data collection

The primary endpoints of the present study were change in blood oxygenation levels at three time points from baseline. Evaluation of whether oxygenation levels improved was performed at 1 (initial stage) and 6 h (maintenance stage) after the initiation of the treatment and at the end of the treatment (end stage). The secondary endpoint was patient outcome, which included mortality; incidence of pulmonary atelectasis, pneumonia, re-intubation, and sepsis; shortening of ICU and hospital stays.

Observations for adverse events lasted until the end of NIPPV. However, they were not graded in severity due to the lack of large-scale observation and uniform standards. Only the presence/absence was observed since these adverse effects, once they have occurred, affect the efficacy and persistence of the treatment.

Data, including age, sex, body mass index (BMI), body surface area (BSA), comorbidities (hypertension, coronary artery disease, diabetes, COPD, and preoperative renal dysfunction), smoking, preoperative left ventricular ejection fraction (LVEF),

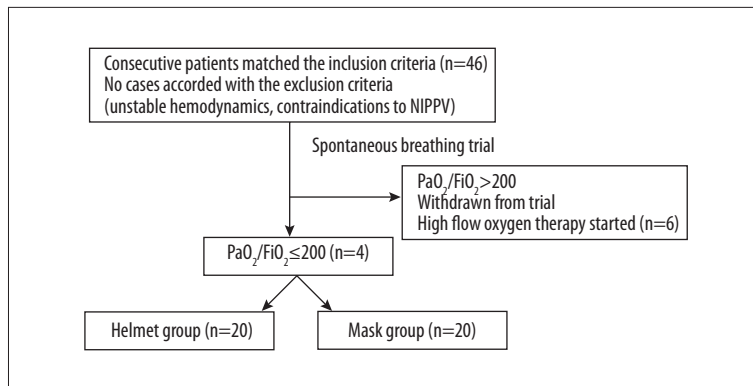


Figure 1. Flow chart showing the patient enrollment and group selection.

surgical method including cerebral perfusion time, preoperative European System for Cardiac Operative Risk Evaluation (EuroSCORE), postoperative Acute Physiology and Chronic Health Evaluation II (APACHE II) score, and causes of hypoxemia, were collected. Blood gas analysis results, pH, PaO₂, PaCO₂, SpO₂, lactate (Lac), PaO₂/FiO₂, vital signs, and circulatory parameters (temperature, heart rate, RR, mean arterial pressure [MAP]) before (baseline), at 1 (initial stage) and 6 h (maintenance stage) during treatment, and at the end of the treatment (end stage) were calculated in each group and compared between the two groups. In addition, LVEF, hepatorenal function (alanine transaminase [ALT] and aspartate transaminase [AST]), and procalcitonin (PCT) levels before and at the end of the treatment were recorded, and the changes in these parameters were noted. The incidence of pulmonary and non-invasive ventilation-related complications, including pulmonary atelectasis, pulmonary infection, hydrothorax, ventilator-associated pneumonia (VAP), acute pulmonary injury, respiratory muscle weakness, flatulence, aspiration, intolerance, and facial pressure sore, were compared between the two groups. Re-intubation, tracheotomy, treatment failure, NIPPV time, ICU stay, hospital stay, ICU mortality, and in-hospital mortality were also compared between the two groups.

Statistical analysis

SPSS 19.0 for Windows (SPSS Inc., Chicago, IL, USA) was used for statistical analysis. Quantitative data with a normal distribution were expressed as mean and standard deviation, while quantitative data with a non-normal distributions (including NIPPV time, ICU stay, and hospital stay) were expressed as median and interquartile range (IQR; P25 and P75). In case of quantitative data with a normal distribution and equal variance, the independent-samples *t*-test was used for comparisons between groups, and the paired *t*-test was used for comparisons before and after treatment within each group. The rank-sum test was used to compare data with non-normal distribution and unequal variance. The chi-square test and Fisher exact test were used to compare qualitative data. *P*<0.05 was considered statistically significant.

Results

In total, 40 patients (25 men and 15 women) developed hypoxemia within 24 h extubation after undergoing surgical treatment for Stanford type A aortic dissection in the ICU of the Department of Cardiac Surgery, Beijing Anzhen Hospital, between November 2013 and July 2014. These patients were randomly divided into two groups to receive helmet or mask NIPPV treatment. The patient enrollment and allocation into groups is shown in Figure 1.

General characteristics

No statistically significant differences in age, sex, BMI, BSA, comorbidities, smoking, preoperative LVEF, surgical methods, preoperative EuroSCORE, postoperative APACHE II score, and causes of hypoxemia were found between the two groups (*P*>0.05; Table 1).

Primary endpoints: blood oxygenation changes from baseline

Blood gas analysis results are shown in Table 2: In the helmet group, blood gas analysis data, including pH, PaO₂, PaCO₂, SpO₂, and PaO₂/FiO₂, significantly improved in the initial, maintenance, and end stages (*P*<0.05). Lac levels significantly improved in the maintenance and end stages (*P*<0.05). In the mask group, PaO₂, SpO₂, and PaO₂/FiO₂ significantly improved in all three treatment stages (*P*<0.05), while pH and PaCO₂ were not improved significantly until the end stage (*P*<0.05). Lac levels significantly improved in only the initial and end stages (*P*<0.05).

The pretreatment blood gas analysis results did not significantly differ between the two groups (*P*>0.05). However, the improvements in pH, PaO₂, SpO₂, and PaO₂/FiO₂ in the initial, maintenance, and end stages were significantly greater in the helmet group than in the mask group (*P*<0.05), suggesting that helmet NIPPV rapidly improved oxygenation. The decrease in PaCO₂ in the helmet group in the maintenance and end stages was significantly greater than that in the mask group (*P*<0.05).

Table 1. General characteristics of the patients in the two groups.

Characteristic	Helmet group (n=20)	Mask group (n=20)	P-value
Age (years)	52.7±8.9	55.5±8.6	0.325
Gender/Men	13 (65)	12 (60)	0.744
BMI (kg/m ²)	27.0±3.6	27.5±2.3	0.615
BSA (m ²)	1.9±0.2	1.9±0.2	0.458
Hypertension	14 (70)	15 (75)	1.000
CAD	7 (35)	5 (25)	0.731
Diabetes	3 (15)	3 (15)	1.000
COPD	1 (5)	4 (20)	0.342
Preoperative renal dysfunction	1 (5)	1 (5)	1.000
Smoking	9 (45)	10 (50)	1.000
Preoperative LVEF (%)	57.3±8.0	60.0±6.8	0.330
Surgical method			
Ascending aorta replacement + Sun procedure	7 (35)	9 (45)	0.519
Bentall procedure + Sun procedure	8 (40)	6 (30)	0.507
Ascending aorta replacement	2 (10)	4 (20)	0.661
Ascending aorta replacement + partial aortic arch replacement	2 (10)	1 (5)	1.000
Bentall procedure + Sun procedure + MVR + CABG	1 (5)	0	1.000
CBP duration (min)	153.0±5.1	156.5±5.4	0.776
Arrest duration (min)	65.0±7.2	58.0±5.9	0.513
Preoperative EuroSCORE	7.2±2.1	6.6±1.8	0.342
Postoperative APACHE II score	8.6±2.0	8.8±2.1	0.762
Causes of hypoxemia			
Pulmonary atelectasis	7 (35)	5 (25)	0.490
Pulmonary infection	2 (10)	1 (5)	1.000
Acute lung injury	5 (25)	8 (40)	0.311
Cardiogenic pulmonary edema	1 (5)	0	1.000
COPD	5 (25)	8 (40)	0.311

Data is presented as mean ± standard deviation or n (%). BMI – body mass index; BSA – body surface area; CAD – coronary artery disease; COPD – chronic obstructive pulmonary disease; LVEF – left ventricular ejection fraction; MVR – mitral valve replacement; CABG – coronary artery bypass graft; CBP – cardiopulmonary bypass; EuroSCORE – European System for Cardiac Operative Risk Evaluation; APACHE II – Acute Physiology and Chronic Health Evaluation II.

In contrast, the change in Lac levels did not significantly differ between the two groups ($P>0.05$).

Secondary endpoint: patient outcomes

Vital signs and circulatory parameters: In the helmet group,

Table 2. Changes in blood related parameters after NIPPV treatment.

Parameter	Helmet group (n=20)	P-value (Pairwise t test, compared with those before NIPPV)	Mask group (n=20)	P-value (Pairwise t test, compared with those before NIPPV)	P-value (Two independent sample t-test)
pH					
Baseline	7.38±0.07		7.36±0.04		0.384
Initial NIPPV stage	7.41±0.05	0.001	7.36±0.03	1.000	0.003
Maintenance NIPPV stage	7.43±0.04	<0.001	7.37±0.05	0.461	0
End of NIPPV	7.43±0.04	<0.001	7.38±0.05	<0.001	0.001
PaO₂ (mmHg)					
Baseline	70.6±9.6		72.3±8.2		0.552
Initial NIPPV stage	100.7±22.4	<0.001	84.0±6.2	<0.001	0.003
Maintenance NIPPV stage	126.9±37.1	<0.001	94.5±12.9	<0.001	0.001
End of NIPPV	151.1±38.2	<0.001	113.8±33.9	<0.001	0.002
PaCO₂ (mmHg)					
Baseline	38.7±5.8		39.1±2.9		0.75
Initial NIPPV stage	37.1±4.7	0.002	39.0±2.3	0.626	0.122
Maintenance NIPPV stage	35.1±4.0	<0.001	38.4±2.8	0.118	0.004
End of NIPPV	34.2±4.0	<0.001	37.5±3.6	0.001	0.009
SpO₂ (%)					
Baseline	91.8±3.0		90.8±2.0		0.194
Initial NIPPV stage	97.5±2.8	<0.001	93.6±2.4	<0.001	0
Maintenance NIPPV stage	98.9±1.4	<0.001	95.9±3.5	<0.001	0.001
End of NIPPV	99.4±0.7	<0.001	96.6±4.3	<0.001	0.007
Lac (mmol/L)					
Baseline	2.5±1.1		2.1±0.7		0.225
Initial NIPPV stage	2.4±1.3	0.561	2.0±0.8	0.040	0.208
Maintenance NIPPV stage	1.9±1.0	<0.001	1.9±0.8	0.067	0.987
End of NIPPV	1.6±1.0	<0.001	1.7±0.9	0.022	0.757
PaO₂/FIO₂					
Baseline	140.9±19.2		144.5±16.3		0.525
Initial NIPPV stage	194.7±31.6	<0.001	170±21.6	<0.001	0.006
Maintenance NIPPV stage	245.4±74.1	<0.001	181.2±48.5	0.001	0.002
End of NIPPV	298.3±76.7	<0.001	215.7±63.9	<0.001	0.001
Temperature (°C)					
Baseline	37.7±0.6		37.3±0.5		0.052
Initial NIPPV stage	37.6±0.6	0.028	37.5±0.8	0.305	0.826
Maintenance NIPPV stage	37.2±0.6	0.001	37.2±0.5	0.117	0.847
End of NIPPV	37.0±0.6	<0.001	37.3±0.7	0.679	0.125
Heart rate (beats/min)					
Baseline	93.5±13.7		93.1±11.1		0.91
Initial NIPPV stage	90.1±10.7	0.057	93.3±11.7	0.891	0.373
Maintenance NIPPV stage	81.0±12.7	<0.001	97.5±13.8	0.188	0
End of NIPPV	78.6±13.4	<0.001	92.1±16.9	0.808	0.008

Table 2 continued. Changes in blood related parameters after NIPPV treatment.

Parameter	Helmet group (n=20)	P-value (Pairwise t test, compared with those before NIPPV)	Mask group (n=20)	P-value (Pairwise t test, compared with those before NIPPV)	P-value (Two independent sample t-test)
RR (breaths/min)					
Baseline	25.4±6.5		26.5±5.5		0.55
Initial NIPPV stage	23.7±5.2	0.035	25.8±5.4	0.160	0.228
Maintenance NIPPV stage	19.3±4.9	<0.001	24.3±6.6	0.036	0.01
End of NIPPV	18.6±5.2	<0.001	22.1±6.3	<0.001	0.065
MAP (mmHg)					
Baseline	90.2±13.8		86.9±9.3		0.383
Initial NIPPV stage	87.4±13.2	0.081	86.8±9.4	0.957	0.877
Maintenance NIPPV stage	81.5±9.9	0.001	86.1±11.6	0.661	0.19
End of NIPPV	78.9±9.1	<0.001	84.2±10.6	0.198	0.094

Data is presented as mean ± standard deviation. NIPPV – non-invasive positive pressure ventilation; PaO₂ – partial pressure of oxygen in the blood; FiO₂ – fraction of inspired oxygen; MAP – mean arterial pressure.

temperature and RR significantly improved in all three treatment stages ($P<0.05$), while heart rate and MAP significantly improved in only the maintenance and end stages ($P<0.05$). In the mask group, the improvements in temperature, heart rate, and MAP in the three treatment stages were not statistically significant ($P>0.05$), but RR significantly improved in the maintenance and end stages ($P<0.05$).

The pre- and post-treatment temperature and MAP did not significantly differ between the two groups ($P>0.05$). Heart rate did not significantly differ between the two groups before the treatment ($P>0.05$); however, the decrease in heart rate was significantly greater in the helmet group than in the mask group in the maintenance and end stages ($P<0.05$). The pretreatment RR did not significantly differ between the two groups ($P>0.05$); however, the decrease in RR was significantly greater in the helmet group than in the mask group in the maintenance stage ($P<0.05$), but the difference was comparable in the end stage ($P>0.05$).

LVEF: LVEF significantly increased after treatment in the helmet group ($P<0.05$) but not in the mask group ($P>0.05$). Before the treatment, cardiac function was significantly poorer in the helmet group than in the mask group ($P<0.05$); however, LVEF did not significantly differ between the two groups at the end of the treatment ($P>0.05$). As the mean post-treatment LVEF value was significantly higher than the pretreatment value ($P<0.05$), cardiac function significantly improved in the helmet group.

Hepatorenal function and PCT level: In the helmet group, no significant differences were found between the pre- and

post-treatment ALT, AST, creatinine, and PCT levels ($P>0.05$). In the mask group, ALT, AST, and creatinine levels significantly increased after treatment ($P<0.05$), while the PCT level was not significantly changed ($P>0.05$), suggesting a deterioration in hepatorenal function (Table 2).

The incidence of re-intubation, tracheotomy, and treatment failure did not significantly differ between the two groups ($P>0.05$). NIPPV time was significantly shorter in the helmet group than in the mask group ($P<0.05$), but ICU stay, hospital stay, ICU mortality, and in-hospital mortality did not significantly differ between the two groups ($P>0.05$; Table 3).

Adverse events

The incidence of flatulence, intolerance, and facial pressure sores significantly differed between the two groups ($P<0.05$), while the incidence rates of pulmonary atelectasis, pulmonary infection, pleural effusion, VAP, acute lung injury, respiratory muscle weakness, and aspiration were similar in the two groups ($P>0.05$) (Table 4).

Discussion

The aim of this study was to evaluate the use of NIPPV for patients with hypoxemia after surgery for Stanford type A aortic dissection, and to compare the use of helmet and mask interfaces for NIPPV delivery. The results show that NIPPV effectively improved oxygenation and reduced PaCO₂ in the patients and that had helmet NIPPV more rapidly increased PaO₂ and

Table 3. Comparison of patient outcomes between the two groups.

	Helmet group (n=20)	P-value (Pairwise t test, compared with those before NIPPV)	Mask group (n = 20)	P-value Pairwise t test, compared with those before NIPPV)	P-value (Two independent sample t-test)
LVEF (%)					
Baseline	51.2±5.3		54.7±4.8		0.035
End of NIPPV	57.4±5.2	<0.001	55.6±4.7	0.323	0.247
ALT (U/L)					
Baseline	44.0±8.5		50.4±5.4		0.493
End of NIPPV	40.0±6.3	0.315	66.4±8.1	0.013	0.109
AST (U/L)					
Baseline	49.3±5.2		53.5±6.7		0.309
End of NIPPV	42.8±5.3	0.108	78.3±10.6	0.018	<0.001
Creatinine (µmol/L)					
Baseline	108.2±15.0		92.7±6.5		0.313
End of NIPPV	108.9±20.7	0.908	127.1±9.4	0.003	0.673
PCT (ng/mL)					
Baseline	0.12±0.03		0.13±0.02		0.117
End of NIPPV	0.09±0.04	0.323	0.11±0.02	0.525	0.323
NIPPV time (h)	6.0 (6.0,12.0)		12.0 (6.0,93.0)		0.002
ICU stay (h)	95.5 (44.0, 338.0)		100.5 (49.0, 288.0)		0.620
Hospital stay (d)	18.0 (12.0, 28.0)		20.0 (14.0, 29.0)		0.201
ICU mortality	1 (5)		2 (10)		1.000
In-hospital mortality	2 (10)		2 (10)		1.000

Data are presented as mean ± standard deviation, n (%), M (Q₁). NIPPV – non-invasive positive pressure ventilation; LVEF – left ventricular ejection fraction; ALT – alanine aminotransferase; AST – aspartate aminotransferase; ICU – intensive care unit.

reduced PaCO₂, increased patient tolerance and comfort, and reduced complications compared to mask delivery.

Refractory hypoxemia is a common complication following cardiothoracic surgery. The incidence of hypoxemia following cardiothoracic surgery with cardiopulmonary bypass is 18.17% [8], but it increases to 51.6% in patients who undergo surgery for Stanford type A aortic dissection [9]. Re-intubation and invasive mechanical ventilation are generally required in patients with hypoxemia [10]. The establishment of an artificial airway can increase VAP risk, hospital stay, hospitalization expenses, and mortality (the latter to about 25–50%) [11,12]. Hypoxemia following surgery for Stanford type A aortic dissection is associated with several factors, including poor cardiac function, pulmonary atelectasis, obesity, and inflammatory response [1]. In

such patients, the use of positive-pressure ventilation or PEEP can prevent pulmonary atelectasis, reduce inflammatory pulmonary effusion, and improve cardiac function to a certain extent. CPAP has even been shown to decrease C-reactive protein a marker of inflammation [13]. After extubation, however, the above objectives can only be achieved through NIPPV, a type of NIPPV [4]. During NIPPV, a certain degree of PEEP and inspiratory pressure is provided in patients with spontaneous respiration, in order to improve ventilation, prevent alveolar collapse, increase functional residual capacity and oxygenation, and reduce breathing effort. In addition, NIPPV can simultaneously reduce cardiac preload and afterload. Cardiac preload is decreased because the increased pressure in the thoracic cavity reduces venous return, while cardiac afterload is decreased because the increased pressure in the thoracic cavity reduces

Table 4. Adverse events.

Parameter	Helmet group (n=20)	Mask group (n=20)	P-value
Flatulence	0	7 (35)	0.008
Intolerance	2 (10)	9 (45)	0.031
Facial pressure sores	0	5 (25)	0.047
Pulmonary atelectasis	2 (10)	5 (25)	0.407
Pulmonary infection	1 (5)	5 (25)	0.182
Pleural effusion	1 (5)	1 (5)	1.000
VAP	0	3 (15)	0.231
Acute lung injury	0	3 (15)	0.231
Respiratory muscle weakness	2 (10)	0	0.487
Aspiration	0	2 (10)	0.487
Re-intubation	2 (10)	6 (30)	0.235
Tracheotomy	2 (10)	4 (20)	0.661
Treatment failure	1 (5)	6 (30)	0.091

Data are presented as number (%). VAP – ventilator-associated pneumonia.

transmural pressure [14]. As most patients who undergo cardiac surgery have some degree of cardiac dysfunction, NIPPV, which affects respiration and left ventricular load simultaneously, can improve both cardiac output/visceral perfusion and oxygenation without patient–ventilator synchrony, and can thus best benefit patients. Hoffmann et al. [15] investigated the effects of NIPPV on hemodynamics, and found that NIPPV effectively improved the cardiac index in stable patients who had undergone cardiac surgery.

In the present study, we found that in both the helmet and mask groups, NIPPV significantly improved oxygenation. In both groups, PaO₂/FiO₂ significantly improved in the initial, maintenance, and end stages, as compared with the values before treatment ($P<0.01$, helmet group; $P<0.05$, mask group). PaO₂ also significantly increased after treatment ($P<0.01$, both groups). PaCO₂ significantly decreased (helmet group: $P<0.01$, initial stage; $P<0.05$, maintenance and end stages; mask group: $P<0.05$, end stage). Lac levels significantly decreased (helmet group: $P<0.01$, maintenance and end stages; mask group: $P<0.05$, initial and end stages). RR significantly reduced (helmet group: $P<0.05$, initial stage; $P<0.01$, maintenance and end stages; mask group: $P<0.05$, maintenance stage; $P<0.01$, end stage). Hepatorenal function did not significantly change in the helmet group ($P>0.05$), but ALT, AST, and creatinine levels significantly increased after treatment in the mask group ($P<0.05$). The findings of the present study show that NIPPV effectively improved oxygenation, increased PaO₂, and decreased

PaCO₂ and RR. Lac levels also significantly reduced after improvement in oxygenation. However, hepatorenal function was not significantly altered in the helmet group and was actually worsened in the mask group. This may be attributable to several factors, including administration of drugs, an imbalance of intake and excretion, and oxygen debt repayment during ICU stay. Experience of using NIPPV in patients with respiratory failure after cardiac surgery is limited, and reports of its use in patients who undergo surgery for Stanford type A aortic dissection are even rarer. De Santo et al. [7] used with NIPPV (bi-level positive airway pressure; BiPAP) to treat 43 patients with acute respiratory failure after extubation following cardiac surgery, and the success rate was as high as 74.4%, while the mortality was 14%. Boeken et al. [16] retrospectively analyzed the effects of three methods of treating acute respiratory failure following extubation after cardiac surgery: immediate intubation, CPAP, and BiPAP. They found that the rates of re-intubation were 25.8% and 22.2% in the CPAP and BiPAP groups, respectively, while the mortality was 8.8%, 4.2%, and 5.6% in the immediate intubation, CPAP, and BiPAP groups, respectively, suggesting that NIPPV (either CPAP or BiPAP) effectively improved oxygenation and patient outcomes. Zhu et al. [17] found that the selective use of NIPPV in patients with acute respiratory failure following cardiac surgery effectively improved oxygenation, reduced RR, stabilized vital signs, and decreased re-intubation rates. The results of the present study are consistent with previous findings, suggesting that NIPPV (including CPAP) is safe and effective in patients with

hypoxemia or respiratory failure following cardiac surgery (including surgery for Stanford type A aortic dissection).

Another focus of the present study is the interfaces of NIPPV. Navalesi et al. [18] investigated the importance of the interfaces of non-invasive ventilation, and found that an appropriate interface is more important than the ventilation mode to ensure good results of non-invasive ventilation. The effects of the oral-nasal mask are better than those of other interfaces; however, the use of this mask is not very comfortable, many patients cannot tolerate this device well, and the device is associated with a relatively high occurrence of air leakage [18,19]. Furthermore, skin lesions at the nose induced by long-term use of this device [20] can result in frequent treatment interruptions and even treatment discontinuation. In contrast, the use of nasal mask and rhinobyon is substantially more comfortable, but the ventilation effects are not as good as those of the oral-nasal mask. In recent years, non-invasive helmet ventilation has been widely used in clinical practice because it shows good patient tolerance, low incidence of complications, and excellent ventilation effects. However, very few studies have investigated the effects of non-invasive helmet ventilation in patients who have undergone cardiac surgery, including surgery for Stanford type A aortic dissection.

In the present study, the effects of two different NIPPV interfaces were compared, and the results showed that helmet ventilation effectively improved oxygenation. Furthermore, the improvements in pH, PaO₂, SpO₂, and PaO₂/FiO₂ in the initial, maintenance, and end stages were more pronounced in the helmet group than in the mask group ($P<0.05$). There was a significantly greater decrease in PaCO₂ in the maintenance and end stages in the helmet group than in the mask group ($P<0.05$). RR was significantly slower in the helmet group at 6 h after the treatment than in the mask group ($P<0.05$), but the RR in both groups at the end of treatment was comparable ($P>0.05$). The decrease in heart rate at 6 h after treatment and at the end of the treatment was significantly greater in the helmet group than in the mask group ($P<0.05$). The mean LVEF increased after treatment in the helmet group ($P<0.05$), suggesting cardiac function improvement. The incidence of complications, including flatulence, intolerance, and facial pressure sore, was significantly lower in the helmet group than in the mask group ($P<0.05$), but the incidence of re-intubation, tracheotomy, and treatment failure did not significantly differ between the two groups ($P>0.05$). NIPPV time was significantly shorter in the helmet group than in the mask group ($P<0.05$), but ICU stay, hospital stay, ICU mortality, and in-hospital mortality did not significantly differ between the two groups ($P>0.05$). These findings showed that helmet ventilation could improve oxygenation, reduce PaCO₂, decrease RR and heart rate, and improve left ventricular function to a greater extent than could mask ventilation. In addition, helmet ventilation

was associated with greater comfort, lower complication rates, and higher patient compliance, which resulted in fewer treatment interruptions and therefore, a shorter overall treatment time. We conclude that the use of helmet ventilation produces good results within a short time. Giorgio et al. [6] compared the effects of helmet and mask CPAP in patients with respiratory failure following abdominal surgery, and found that both helmet and mask CPAP effectively improved PaO₂/FiO₂ and reduced RR. However, the rate of re-intubation was significantly lower in the helmet group (20%) than in the mask group (48%; $P<0.036$), and the incidence of complications (e.g., intolerance, air leakage, and VAP) was also significantly lower in the helmet group (16%) than in the mask group (76%; $P<0.03$). This suggests that helmet CPAP is associated with better tolerability and a lower incidence of complications. Barbagallo et al. [21] administered preventive helmet CPAP treatment to patients who had undergone pulmonary lobectomy, and found that compared with mask CPAP, helmet CPAP effectively improved PaO₂/FiO₂ (366 ± 106 mmHg vs. 259 ± 60 mmHg, $P=0.004$) and reduced hospital stay (7 ± 4 d vs. 8 ± 13 d, $P=0.042$). In addition, the tolerance and safety profile of helmet CPAP treatment were better than those of mask CPAP, suggesting that helmet CPAP can be used to treat hypoxemia following multiple surgeries. The findings of the present study showed that helmet NIPPV could rapidly improve oxygenation, reduce complications, decrease gas leakage, and improve patient comfort, which are consistent with previous findings. However, due to differences in surgical methods, study design, and disease severity, no significant differences in the re-intubation rate and hospital stay were found in the present study. Further studies are needed to validate our findings.

The safety profile (including treatment failure and mortality) is another issue that needs to be explored prior to the clinical application of NIPPV for treating hypoxemia after surgery. In the present study, in the mask group, 6 patients experienced treatment failure due to frequent interruptions, which were caused by intolerance (5 patients, 25%) and re-intubation due to pulmonary infection (1 patient, 5%). In contrast, treatment failure occurred in only 1 patient (5%) in the helmet group (due to death caused by subarachnoid hemorrhage). This death may have been caused by a cerebrovascular malformation, which is commonly found in patients with aortic dissection. However, an autopsy was not performed, and thus, the exact cause was not identified. Helmet NIPPV was well tolerated by the other patients, and the gas flow could be adjusted if slight stuffiness was reported. Two patients (10%) died in each group. There were two ICU deaths in the mask group, due to infectious shock (one patient) and infection caused by vomiting and aspiration following flatulence (one patient). The deaths in the helmet group were caused by pulmonary infection followed by multiple organ failure after transfer from the ICU to the general ward (one patient), and subarachnoid hemorrhage (one

patient; although this patient had good respiration while on helmet NIPPV). During mask ventilation, careful observation and treatment can resolve complications, such as flatulence, pressure sore, aspiration, VAP, and pulmonary infection. An artificial airway should be established, and mechanical ventilation should be used in patients who require intubation due to poor respiratory improvement on NIPPV or due to complications.

This study has some limitations. The study population was quite small and limited by being undertaken in a single center; in future the population size might be increased by including patients after other cardiac surgical procedures and including multiple centers. We did not perform a calculation of the required sample size before the study; instead we included all eligible consecutive patients within the study period. A larger sample size would be expected to add more weight of evidence for these results. The mask and helmet delivery systems also differed in the type of NIPPV they provided with the mask using BIPAP and the helmet CPAP. These differences might have biased the results as these different NIPPV methods provide different degrees of benefit to patients [15]. It has also been shown that different ventilators provide different performance and triggering workload when BIPAP ventilators were tested in a laboratory [22], therefore, these differences should be considered alongside these results.

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Conclusions

In summary, the findings of the present study show that NIPPV can be effectively used to improve oxygenation in patients with hypoxemia following surgery for Stanford type A aortic dissection. Compared with mask ventilation, helmet ventilation results in better tolerance, fewer complications, and better ventilation, and thus, is associated with greater patient compliance. Further multicenter, randomized, controlled, prospective, studies with large sample sizes are required to clarify the effects of NIPPV and the factors influencing NIPPV application with different interfaces in patients who have undergone surgery for Stanford type A aortic dissection.

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Statement

The authors declare no competing interests.