Study the Efficacy of Using Non Invasive Positive Pressure Ventilation as a Prophylactic Modality against Post-Extubation Respiratory Failure in Patients with Cardiogenic Pulmonary Edema

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Abstract: Background: Post-extubation respiratory failure is a common event after discontinuation of mechanical ventilation. The incidence of reintubation is relatively high, being about 6- 23% among patients undergoing, mechanical ventilation for more than 48 hours within 48-72 hours of endotracheal removal, so it is important to identify those patients at risk of post extubation respiratory failure. **Aim of the Work:** The present study aimed to assess the efficacy of early application of non invasive positive pressure ventilation in preventing post-extubation respiratory failure in patients with ACPE. **Patients and Methods:** The present study was done in Critical Care Medicine Department of Alexandria main University Hospital, it was carried out on 32 adult patients of both sexes who were presented with acute cardiogenic pulmonary edema, required mechanical ventilation with endotracheal intubation for a certain time duration till resolution of pulmonary edema, correction of potentially life threatening acid-base and blood gases abnormalities, stabilization of hemodynamic parameters and improvement of level of consciousness. Informed consent was taken from every patient included in the study or from one of his/her relatives. Demographic data, full medical history, complete clinical examination, arterial blood gases measurements, laboratory investigations, chest radiography, central venous pressure measurement, and 12 lead electrocardiography were done on admission of the studied patients. In addition to measurements of some weaning parameters before extubation to ensure successful weaning trial. Patients then were randomized into two groups: Group A (control group): This group included 16 patients who were disconnected from the ventilator; breathed spontaneously through a T-tube circuit for at least 2 hours, and supplied with humidified O_2 ill arterial oxygen saturation equal to or above 90 % as measured by pulse oximetry was achieved. Group B (NIPPV group): This group included 16 patients who were disconnected from the ventilator; extubated, and immediately ventilated with *non* invasive positive pressure ventilation via oronasal mask. The pulse, respiratory rate, blood pressure, arterial blood gases, alveolar oxygen tension, shunt fraction, oxygen extraction ratio, and central venous pressure were measured for both groups every 4 hours for 48 hours with continuous electrocardiographic monitoring and the efficacy was recorded as the number of patients successfully weaned. **Results**: Weaning was considered successful if spontaneous breathing is sustained for more than 48 hrs after discontinuation of mechanical ventilation. There was statistically significant difference between the two studied groups as regards the mean pulse rate, mean respiratory rate, mean systolic and diastolic blood pressure through out the study. There was statistically significant difference between the two studied groups as regards the mean $Pa0_2$, mean alveolar oxygen tension, mean shunt fraction and mean central venous pressure throughout the study. There was no statistically significant difference between the two studied groups as regards the mean pH, mean $PaCO_2$, mean serum HCO_3, and mean oxygen extraction ratio throughout the study. There was no statistically significant difference between the two studied groups as regards the causes of weaning failure, weaning time, duration from extubation to reintubation, length of ICU stay, mortality rate, and incidence and types of complications throughout the study. There was statistically significant difference between the two studied groups as regards success of weaning as there were 15 patients successfully weaned in group B in comparison to 11 patients in group A. So, early application of noninvasive positive pressure mechanical ventilation in the present study was more efficient than the standard medical therapy to prevent post-extubation respiratory failure in the selected patients.
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1. Introduction

Cardiogenic pulmonary edema (CPE) is defined as development of pulmonary edema as a consequence of increased capillary hydrostatic pressure secondary to elevated pulmonary venous pressure [(1)]. CPE is characterized by the rapid transudation of excess fluid in the lungs secondary to increased pulmonary artery wedge pressure. This

occurs in the absence of a primary change in the permeability or integrity of the endothelial and epithelial layers of the pulmonary capillaries [2] . CPE can occur as a result of left ventricular failure secondary to systolic or diastolic dysfunction, mitral valvular disease, volume overload or pulmonary venous outflow obstruction (e.g. mitral stenosis or left atrial myxoma). The most common cause of cardiogenic pulmonary edema is left ventricular dysfunction [3] .

The salient clinical features of cardiogenic pulmonary edema are breathlessness, tachypnea, and signs of increased sympathetic activity such as tachycardia , hypertension and diaphoresis. Hypotension is uncommon but may occur if pulmonary edema results from a large myocardial infarction. Central cyanosis may be observed if there is profound arterial hypoxemia; more often, cyanosis is peripheral and results from intense cutaneous vasoconstriction and a decreased cardiac output. Early, there may be wheezing caused by airway edema; later, diffuse coarse rales are heard.[3]

Initial management of patients with CPE should address the ABCs of resuscitation that is: airway, breathing, and circulation. Humidified oxygen should be administered to all patients to keep oxygen saturation more than 90%. Medical therapy of CPE focuses on three main goals including reduction of pulmonary venous return (preload reduction), reduction of systemic vascular resistance (after load reduction) and inotropic support [1] . While the majority of patients respond to conventional therapy, some patients who remain hypoxic despite supplemental oxygenation and patients who have severe respiratory distress require ventilatory support. Traditionally, this has been provided via endotracheal intubation and invasive mechanical ventilation [4-7] .

While mechanical ventilation through an endotracheal tube is a well established, accepted and life-saving procedure for patients with acute respiratory failure [8,9] , it exposes the patient to a variety of complications resulting from: the intubation procedure, during the course of ventilation or after extubation [10] . A prolonged attempt at intubation may result, infrequently but dangerously, in cardiac arrest and generalized seizures [11] . Placement and maintenance of endotracheal tube increases patient's discomfort and stress, and often necessitates administration of sedative agents [12,13] . Injury to the pharynx, larynx and trachea can occur at the points of contact between the mucosa and the tube or cuff, resulting in ulceration, edema, and hemorrhage with potential long-term complications such as airway stenosis [14] . In addition to local damage, the endotracheal tube (ETT) places the patient at significant risk of developing nosocomial infections, mainly sinusitis and ventilator-associated pneumonia [15] . Decrease in cardiac output, barotrauma and increase in work of breathing (WQB) due to the added dead space of the ETT are additional problems of invasive mechanical ventilation [16] .

Premature discontinuation of mechanical ventilation can contribute to unsuccessful extubation, requiring reintubation [17]. Reintubation which occurs in 6 to 23% of cases within 48 h after planned extubation [18-20], potentially induces harm with associated airway trauma, gastric aspiration, cardiovascular compromise, and hypoxia [21] . The pathophysiology of respiratory failure after extubation includes upper airway obstruction, inadequate cough, excess respiratory secretions, encephalopathy, and cardiac dysfunction [22-24] .

In addition to an accurate prediction of extubation outcome, strategies for preventing the development of respiratory failure after extubation and subsequent reintubation are needed [25] . Several ventilatory modalities like non invasive positive pressure ventilation (NIPPV) have been proposed to facilitate the recovery from mechanical ventilation and removal of ETT as early as possible [26] .

NIPPV is the delivery of assisted mechanical ventilation without the need for an invasive artificial airway such as an endotracheal tube [27] . It is a safe and effective means of improving gas exchange in patients with many forms of Acute Respiratory Failure. NIPPV is flexible and can be applied both continuously and intermittently, allows speech and swallowing and is accepted well by patients [28] . It improves alveolar ventilation, respiratory rate, tidal volume and work of breathing. In addition, it decreases morbidity, mortality, the need for invasive mechanical ventilation, hospital length of stay, and the incidence of nosocomial infections, compared to invasive mechanical ventilation [29,30] .

Few complications are associated with NIPPV. The most common problem is local damage to facial tissue because of the pressure effects of the mask and straps. Mild gastric distension may occur but is not significant. Eye irritation and sinus pain or congestion may also occur Barotrauma is uncommon. Modest air leaks at the facial seal are common but do not decrease the benefit patients receive from NIPPV. Adverse hemodynamic effects resulting from NIPPV are unusual, although preload reduction and hypotension may occur [31,32] .

The aim of this work was to study the efficacy of using non invasive positive pressure ventilation as a prophylactic modality against post-extubation respiratory failure in patients with cardiogenic pulmonary edema.

2. Patients

The present study was carried out on 32 adult

patients of both sexes who were admitted to the Critical Care Department in the Alexandria Main University Hospital, having acute cardiogenic pulmonary edema requiring invasive mechanical ventilation.

After correction of potentially life threatening acid-base and blood gases abnormalities, stabilization of hemodynamics and improvement of level of consciousness, patients was randomized into two groups:

Group A (control group): This group included 16 patients who was disconnected from the ventilator; breath spontaneously through a T-tube circuit, O_2 was added to achieve an arterial oxygen saturation equal to or above 90 % as measured by pulse oximetry.

Group B (NIPPV group): This group included 16 patients who were extubated and ventilated via non invasive positive pressure ventilation.

Methods

Every patient was initially subjected to the following assessment:

1. Demographic data: as regard name, age and sex.
2. Full medical history: Present complaint, Past medical and Drug history.
3. Complete clinical examination: Vital signs, General examination, Chest examination, Heart examination and Abdominal examination.
4. Arterial blood gas measurements to asses for acid base abnormalities, impaired oxygenation and/or ventilation.
5. 12 lead electrocardiography to find out: left ventricular hypertrophy, arrhythmia, myocardial ischemia or infarction.
6. Laboratory studies including: complete blood count, serum Na, serum K, BUN, creatinine, CkMB and troponin.
7. Chest radiography.
8. Central venous pressure measurement.

Indotracheal intubation and mechanical ventilation was required based on the following criteria[33]: SaO_2 <90 % despite high levels of supplemental oxygen, Respiratory rate >25, Hypoventilation and Hymodynamic instability.

Initiation of endotracheal intubation was done through a standard protocol [34] .

Protocol for Initiation of invasive mechanical ventilation[35]:

- Mode: synchronized intermittent mandatory ventilation (SIMV).
- Respiratory Rate: 12-14 breaths/minute.
- Tidal volume: 6 -8 ml/kg.
- FiO2: starting with 100% 02 then adjusted according to ABG.
- Flow pattern: decelerating.
- Pressure support: starting with 20 cm 1-120 adjusted according to patient tolerance.
- PEEP is added in increments of 2-5 cm until the desired effect on Pa02 without lowering blood pressure, reducing cardiac output, or increasing the plateau pressure on the ventilator.
- The initial setting was adjusted according to AB gases and clinical condition of the patient.

Mechanical ventilation was accompanied with standardized pharmacological treatment with the following[1,36]: (according to etiology)

Preload Reducers:

Furosemide and Nitroglycerin.

Afterload Reducers: Captopril, Nitroprusside and Morphine sulphate.

Inotropic agents: Dobutamine and Dopamine.

Patients were assessed every half an hour in the first 2 hours then every 2 hours as regard AB Cases (PaO_2, $PaCO_2$, PH, HCO_3), Vital signs, Level of consciousness and Central venous pressure readings.

If the following criteria were present, the patient is considered ready for weaning and discontinuation of mechanical ventilation[37-39]: Reversal of underlying cause of respiratory failure, No new myocardial ischemia, Adequate mentation (arousable, GCS> 13, no continuous sedative infusions), Adequate oxygenation (PaO_2/FiO_2>200, PaO_2> 60 mmHg on FiO_2 <0.4 and PEEP<5cmH_2O), Stable cardiovascular system (HR < 140 beats/mm and SBP between 80 and 160 mmHg), pH ≥ 7.25, No significant respiratory acidosis ($PaCO_2$ <45), No significant electrolyte abnormalities,

Temperature <38°C, Hemoglobin level> 10 g/dl, Adequate cough reflex, Negative inspiratory force (NIF) <- 25 cm H20 and Rapid shallow breathing index (RSBI) < 105 or <130 in elderly patients.

Reevaluation was done to exclude from the study the following patients[40,41]:

- Patients with facial burn or trauma.
- Recent facial, upper airway or upper gastrointestinal tract surgery.
- Patients with excessive agitation or inadequate mention (GCS<8).
- Patients unable to protect their airways.
- Patients with inadequate cough reflex.
- Excessive amount of respiratory secretions.
- Patents unable to cooperate with the application of NIPPV.
- Patients with frequent vomiting or active upper gastrointestinal bleeding.

Group A (control group):

Protocol for initiation of spontaneous breathing trial with a T-piece[37]:

- Spontaneous breathing trials were conducted early in the morning, when the patient is fully rested and there is a full compliment of staff available.
- Patient should be awake and co-operative.
- Patient is placed in the upright or semi-upright position
- The procedure was explained to the patient.
- Cuff leak was checked by deflating the cuff and occluding the endotracheal tube
- Regular suctioning of the tube.

Extubation could be considered if the patient breathes on his own for at least 2 hours without distress.

This rapid weaning trial was considered a failure when patient needs reintubation within 48 hrs or do not tolerate spontaneous breathing and requires reconnection to mechanical ventilation.

Criteria of poor Clinical tolerance to a spontaneous breathing trial[42-44]:

- Agitation, depressed mental status or diaphoresis.
- RR> 35 breaths/mm or increased by 50 % or more of baseline.
- Heart rate> 140 beats/mm or increased by 20% or more of baseline.
- EGG changes (frequent ectopy, ST changes and conduction defects).
- Systolic blood pressure (SBP) lower than—80-mmHg-or greater than 160 mmHg.
- PaO_2 <60 mmHg and/or pH < 7.35

Weaning was considered successful if spontaneous breathing was sustained for more than 48 hours after extubation.

Group B (NIPPV group):

Protocol for Initiation of NIPPV[31,40]:

1. The head of the bed was positioned at 45°.
2. The correct size of mask was chosen.
3. Mode of Ventilation was initiated at continuous positive airway pressure (CPAP) with pressure support (PS).
4. CPAP was initiated at 0 cm H20 and pressure support at 10 cm H2O.
5. The mask was held gently on the patient's face until the patient is comfortable and in full synchrony with the ventilator.
6. The mask was secured with head straps with avoidance of tight fit.
7. CPAP was increased in increments of 2-3 cm H2O until FiO_2 is less than 0.6.
8. Pressure support was increased 10-20 cm 1-120 to achieve maximal exhaled tidal volume> 7 ml/kg.
9. Adequate ventilatory support was assured by an improvement in dyspnea, decreased respiratory rate, and achievement of desired tidal volume and good comfort for the patient.
10. Oxygen supplementation was achieved to maintain SaO_2> 90%.
11. Ventilator alarms and backup apnea parameters were adjusted.
12. Monitoring with oximetry, and adjustment of ventilator settings after obtaining ABG results.

A trial of weaning was performed once a stable setting is achieved. Clinical stability was defined as an improvement in oxygenation (paO2 > 60 mm Hg or SaO_2 > 90 % with FiO2 <0.5), a respiratory rate *<25* breaths/mm or a reduction of at least 25% of baseline combined with the presence of a normal breathing pattern, and a heart rate <110 beats/min.[45]

Weaning failure was considered if reintubation is necessary within 48 h after discontinuation of mechanical ventilation.

Criteria to terminate NIPSV and convert patients to invasive mechanical ventilation through endotracheal intubation: [40,41,46,47]

- Deterioration of conscious level.
- Intolerance or failure of coordination with NIPPV.
- Failure to alleviate symptoms.
- Mask intolerance due to pain, discomfort or claustrophobia.
- Inability to improve gas exchange.
- Hemodynamic instability.
- Evidence of cardiac ischemia or ventricular dysrhythmia.
- Need for urgent endotracheal intubation to manage secretions or protect airways.

Weaning from MV was considered successful when spontaneous breathing is sustained and patients remained clinically stable after discontinuation of ventilation for more than 48 hrs.

Measurements:

The following measurements were done for both groups every 4 hours for 48 hours:

- Vital signs: Blood pressure and respiratory rate
- Arterial Blood Gases: PaO_2, PH and HGO_3
- Alveolar Oxygen tension:

$P_AO_2 = [(PB — 47) FiO_2] — (PaCo_2 /RQ)$

- PB is the barometric pressure = 760 mmHg.
- FiO_2 is the fraction of oxygen in the inspired gas.

- $PaCo_2$ is the arterial carbon dioxide tension in mmHg.
- RQ is the respiratory quotient = 0.8
- 47 mmHg is the partial pressure of water vapour.

- Shunt fraction:

$(Q_S/Q_T) = (CcO_2 — CaO_2) / (CcO_2 — CvO_2)$

  - CcO_2 is the pulmonary capillary oxygen content [CcO_2 = hemoglobin in g/dl x 1.39 + 0.003 x PAO_2]
  - Ca O_2 is the arterial Oxygen content [CaO_2 hemoglobin in g/dl x 1.39 x arterial oxygen saturation (SaO_2) +0.003 x PaO_2]
  - CvO_2 is the mixed venous oxygen content [CvO_2 hemoglobin in g/dl x 1.39 x mixed venous oxygen saturation (SvO_2) + 0.003 x mixed venous oxygen tension (PvO_2)]

- Oxygen extraction ratio: Oxygen extraction ratio = $(CaO_2 — CvO_2) / CaO_2$
- Central venous pressure.

Comparison between both groups was made regarding the following:

- Efficacy: the fraction of patients successfully weaned.
- Weaning time: the time from having acceptable weaning parameters to completion of successful weaning. [48]
- Incidence of respiratory failure after extubation: the time from extubation to reintubation.
- The duration of ICU stay.
- ICU mortality.
- Complications related to endotracheal mechanical ventilation.

3. Results

Demographic data:

There was no statistically significant difference between the two studied groups as regards age and sex (Table 1)

Past medical history:

There was no statistically significant difference between the two studied groups as regards past medical history of Hypertension, Ischemic Heart Disease (IHD), Diabetes Mellitus (DM), Chronic Obstructive Pulmonary Disease (COPD) and Renal Impairment (Table 1) .

Cause of development of ACPE:

There was no statistically significant difference between the two studied groups as regards LV systolic dysfunction, Dysrrhythmia and Hypertension. (Table 1)

Laboratory investigations:

There was no statistically significant difference between the two studied groups as regards Hemoglobin concentration, White blood cells count, Blood urea level, Serum creatinine level, Serum sodium level (Na+), Serum potassium level (K+), Serum Ck-MB level and Serum Troponin T level (Table 2) .

Conscious level at time of admission:

There was no statistically significant difference between the two studied groups as regards conscious level at time of admission (p=0.399). (Table 3)

Duration of invasive MV:

There was no statistically significant difference between the two studied groups as regards duration of invasive mechanical ventilation (p=0.l08). (Table 3)

Vital signs:

- **Systolic blood pressure:**

There was no statistically significant difference between the two studied groups as regards the systolic blood pressure on admission (p>0.05) and before MV discontinuation (p>0.05).

There was no statistically significant difference between the two studied groups as regards the systolic blood pressure at the start of the study (p>0.05) while there was statistically significance difference as regards the systolic blood pressure after 48 hours (P<0.0l). (Table 4)

- **Diastolic blood pressure:**

There was no statistically significant difference between the two studied groups as regards the diastolic blood pressure at the start of the study (p=0.15) while there was statistically significance difference as regards the diastolic blood pressure after 48 hours (p=0.032). (Table 5)

- **Heart rate:**

There was no statistically significant difference between the two studied groups as regards the heart rate at the start of the study (P>0.05) while there was statistically significance difference as regards the heart rate after 48 hours (p=0.0125). (Table 6)

- **Respiratory rate:**

There was no statistically significant difference between the two studied groups as regards the respiratory rate at the start of the study (p>0.05) while there was statistically significance difference as regards the respiratory rate after 48 hours (p=0.012). (Table 7).

Arterial blood gases:

- **Arterial oxygen tension ($Pa0_2$):**

In group A; the mean PaO_2 on admission was 47.4+5.69 mmHg while before MV discontinuation it was 98.1±11.6 mmHg with statistically significant difference (p=0.0001).

In group B; the mean $Pa0_2$ on admission was 5 1.7+10.78 mmHg while before MV discontinuation it was 102.0+13.5 mmHg with statistically significant difference

(p=0.0001).

There was no statistically significant difference between the two studied groups as regards the $Pa0_2$ at the start of the study (p>0.05) while there was statistically significance difference as regards the $Pa0_2$ after 48 hours (P<0.001). (Table 8)

- **Arterial carbon dioxide tension ($PaCO_2$):**

There was no statistically significant difference between the two studied groups as regards the $PaCO_2$ at the start of the study (p>0.05) or after 48 hours (p>0.05). (Table 9)

Central venous pressure (CVP):

In group A; the mean CVP on admission was 20.75 +1.22 mmHg while before MV discontinuation it was 13.88±2.03 mmHg with statistically significant difference (p=0.01).

In group B; the mean CVP on admission was 19.21±2.01 mmHg while before MV discontinuation it was 12.5±1.85 mmHg with statistically significant difference (p=0.0l).

There was no statistically significant difference between the two studied groups as regards the CVP at the start of the study (p>0.05) while there was statistically significance difference as regards the CVP after 48 hours (p=0.01). (Table 12)

Alveolar oxygen tension (PAO2):

In group A; the mean PAO_2 at the start of the study was 23 3.20+3.43 mmHg while at the end of the study it was 149.3 1±2.13 mmHg with statistically significant difference (P=0.0l).

In group B; the mean PAO_2 at the start of the study was 23 5.29±4.52 mmHg while at the end of the study it was 176±3.52 mmHg with statistically significant difference (P=0.0l).

There was no statistically significant difference between the two studied groups as regards the PAO_2 at the start of the study (p>0.05) while there was statistically significance difference as regards the PAO_2 after 48 hours (p=0.021). (Table 13)

Shunt fraction (QS/QT):

In group A; the mean QS/QT at the start of the study was 0.201±0.005 mmHg while at the end of the study it was 0.340+0.027 mmHg with statistically significant difference (p=0.033).

In group B; the mean QS/QT at the start of the study was 0.214+0.005 mmHg while at the end of the study it was 0.254±0.025 mmHg with no statistically significant difference (p>0.05).

There was no statistically significant difference between the two studied groups as regards the QS/QT at the start of the study (p>0.05) while there was statistically significance difference as regards the QS/QT after 48 hours (p=0.048). (Table 14)

Oxygen extraction ratio:

In group A; the mean 02 extraction ratio at the start of the study was 0.25±1.32 mmHg while at the end of the study it was 0.29±1.63 mmHg with no statistically significant difference (p>0.05).

In group B; the mean O_2 extraction ratio at the start of the study was 0.24+1.63 mmHg while at the end of the study it was 0.24±1.58 mmHg with no statistically significant difference (p>0.05).

There was no statistically significant difference between the two studied groups as regards the O_2 extraction ratio at the start of the study (p>0.05) or after 48 hours (p>0.05). (Table 15*)*

Efficacy:

In group A; there were 11 patients successfully weaned while in group B there were 15 patients successfully weaned with statistically significant difference as regards the efficacy between the two groups (p=0.039). (Table 16)

Incidence of respiratory failure after extubation:

In group A; there were 5 patients had post-extubation respiratory failure, while in group B; there was only one patient had weaning failure with statistically significant difference as regards the incidence of respiratory failure after extubation between the two groups. (Table 16)

Causes of weaning failure:

In group A; from the five patients who had weaning failure, four of them had hypoxemia, 3 patients had hemodynamic instability, 1 patient had dysrrhythmia, and 2 patients had retained secretions.

In group B; the only patient who had weaning failure suffered from hypoxemia, and hemodynamic instability.

There was no statistically significant difference between the two studied groups as regards the number of patients failed because of hypoxemia (p=0.l44), hemodynamic instability (p=0.285), dysrrhythmia (P=0.309), and retained secretions (p=0.309). (Table 16)

- **Weaning time:**

In group A; in successfully weaned patients; the time from having acceptable weaning parameters to the completion of successful weaning ranged from 2.0 tol2.0 hours with a mean of 4.65±3.65 hours, while in group B; the weaning time ranged from 3.0 to 10.0 hours with a mean of 4.2 1+3.98 hours, there was no statistically significant difference as regards the weaning time between the two groups (p=0.322). (Table 17)

- **Duration from extubation to reintubatjon:**

In group A; in patients who had weaning failure; the duration from extubation till reintubation again ranged from 3.0 to 7.0 hours with a mean of 4.75+2.4 hours, while in group B; the duration in the only failed case was 6.0 hours, there was no statistically significant difference as regards the duration from extubation to reintubation between the two groups (Table 17) .

- **Duration of ICU stay:**

In group A; the duration of ICU stay ranged from 3.0 to 12.0 days with a mean of 4.6±2.3 days, while in group B; the duration of ICU stay ranged from 2.0 to 9.0 days with a mean of 4.5+2.2 days, there was no statistically significant difference as regards the duration of ICU stay between the two groups (p=0.5 12). (Table 17)

- **Incidence of ICU mortality:**

In group A; the number of ICU mortality was 3 patients, while in group B; the number of ICU mortality was 2 patients.

There was no statistically significant difference between the two studied groups as regards ICU mortality (P=0.298). (Table 18)

- **Incidence and types of complications related to endotracheal intubation and mechanical ventilation:**

In group A; there were 4 patients had complications related to ETT and MV, from them 2 patients had laryngitis accompanied with hoarseness of voice, 1 patient had laryngeal edema with associated stridor, and 3 patients had ventilator associated pneumonia.

While in group B; there were 2 patients had complications related to ETT and MV, from them 1 patient had laryngeal edema, and the other had ventilator associated pneumonia.

There was no statistically significant difference between the two studied groups as regards the incidence of complications related to ETT and MV (p=0.365). Also there was no statistically significant difference between the two studied groups as regards the incidence of laryngitris (p=0.144), laryngeal edema (p=l.00), and chest infection (p=0.285). (Table 18)

Table (1) Comparison between the two studied groups regarding demographic data, past medical history, and cause of ACPE

	Group A (No.= 16)		Group B (No.= 16)		P
Age (years)					
Range	45-81		54-75		
Mean	59.9		61.1		0.465
S.D.	8.9		6.3		
Sex	**No.**	**%**	**No.**	**%**	
Male	11	68.75	9	56.25	0.24
Female	5	31.25	7	43.75	
Past medical history	**No.**	**%**	**No.**	**%**	
HTN	8	50.0	9	56.25	0.52
IHD	9	56.25	11	68.75	0.44
DM	8	50.0	10	62.5	0.211
COPD	3	18.75	2	12.5	0.185
Renal impairment	4	25.0	4	25.0	0.99
Cause of ACPE	**No.**	**%**	**No.**	**%**	
LV systolic dysfunction	6	37.5	8	50.0	0.215
Dysrrhythmia	2	12.5	2	12.5	1.00
Hypertension	8	50.0	6	37.5	0.215

*P is significant if P ≤ 0.05

Table (2): Comparison between the two studied groups regarding laboratory investigations on admission

	Group A (No.= 16)		Group B (No.= 16)		P
Hb (g/dl)					
Range	9.2-15		9-14.6		0.378
Mean ±S.D	11.8±1.8		12.0±1.8		
WBCs (/mm^3)					
Range	4.6-15.3		4.6-18.3		0.177
Mean ±S.D	9.4±2.9		10.4±3.5		
Blood urea (mg/dl)					
Range	20-160		20-216		0.236
Mean±S.D	56.8±35.2		62.5±42.65		
Serum Cr (mg/dl)					
Range	0.9-3.3		0.5-4.3		0.47
Mean ±S.D	1.9±0.7		1.9±1.1		
Serum Na (mEq/L)					
Range	122-153		113-147		0.259
Mean ±S.D	134.7±9.0		132.6±8.9		
Serum K (mEq/L)					
Range	2.4-5.9		2.7-5.9		0.441
Mean ±S.D	4.2±0.9		4.1±0.8		
Ck-MB (mEq/L)					
Range	13-25		11-23		0.286
Mean ±S.D	16.9±3.6		16.1±3.9		
Serum Troponin T	No.	%	No.	%	1.00
Negative	16	100	16	100	

*P is significant if P ≤ 0.05

Table (3): Comparison between the two studied groups regarding conscious level at time of admission and duration of invasive MV

	Group A (No.= 16)		Group B (No.= 16)		P
	No.	%	No.	%	
Conscious level Disturbed conscious level	9	56.25	8	50	0.399
Duration of invasive MV (hours) Range Mean ±S.D	4.6-15.3 9.4±2.9		4.6-18.3 10.4±3.5		0.108

*P is significant if P ≤ 0.05

Table (4): Comparison between the two studied groups regarding systolic blood pressure at different periods of follow up

Time	Group A	P1	Group B	P1	P2
On Admission	173.7±21.49		164.42±15.9		>0.05
Before MV discontinuation	130.7±21.94	0.001*	131.7±20.6	0.01*	>0.05
30 min after extubation	128.9±22.2		125.8±22.3		>0.05
4h after extubation	127.0±18.3	>0.05	123.0±19.8	>0.05	>0.05
8h after extubation	128.0±19.4	>0.05	122.1±20.3	>0.05	>0.05
12h after extubation	129.9±24.15	>0.05	120.7±21.5	>0.05	0.036*
16h after extubation	133.2±37.1	>0.05	122.5±22.3	>0.05	0.01*
20h after extubation	132.5±14.6	>0.05	123.1±24.3	>0.05	0.01*
24h after extubation	137.0±15.7	0.042*	121.3±20.4	>0.05	0.01*
28h after extubation	139.0±22.3	0.032*	123.5±21.5	>0.05	0.01*
32h after extubation	142.5±20.5	0.01*	122.5±18.9	>0.05	0.01*
36h after extubation	144.0±19.8	0.01*	124.8±19.5	>0.05	0.01*
40h after extubation	145.0±20.3	0.01*	126.4±16.8	>0.05	0.01*
44h after extubation	143.3±19.8	0.01*	125.5±17.9	>0.05	0.01*
48 h after extubation	144.7±20.3	0.01*	126.0±16.8	>0.05	0.01*

P1 comparison between interval times and baseline. P2 comparison between the two studied groups.
*P is significant if P ≤ 0.05

Table (5): Comparison between the two studied groups regarding diastolic blood pressure at different periods of follow up.

Time	Group A	P1	Group B	P1	P2
On Admission	80.0±11.09		76.7±8.98		0.12
Before MV discontinuation	65.7±12.3	0.002*	65.0±10.21	0.021*	0.456
30 min after extubation	70.0±10.6		68.7±9.82		0.15
4h after extubation	72.5±11.85	>0.05	69.0±8.95	>0.05	0.025*
8h after extubation	74.2±12.3	>0.05	71.0±10.33	>0.05	>0.05
12h after extubation	77.7±9.85	0.042*	70.7±9.78	>0.05	0.033*
16h after extubation	76.5±10.65	0.040*	73.6±9.78	0.045*	>0.05
20h after extubation	79.7±11.33	0.036*	74.5±8.65	0.032*	0.042*
24h after extubation	81.0±8.98	0.002*	73.1±9.14	0.004*	0.048*
28h after extubation	82.0±11.25	0.001*	72.1±10.32	>0.05	0.01*
32h after extubation	81.5±10.6	0.002*	70.4±11.25	>0.05	0.012*
36h after extubation	81.0±10.7	0.003*	73.4±10.6	0.042*	0.036*
40h after extubation	83.0±11.3	0.001*	72.7±8.95	>0.05	0.012*
44h after extubation	82.3±9.65	0.001*	72.0±7.96	>0.05	0.01*
48 h after extubation	83.2±9.78	0.001*	71.7±8.65	>0.05	0.032*

P1 comparison between interval times and baseline. P2 comparison between the two studied groups.
*P is significant if P ≤ 0.05

Table (6): Comparison between the two studied groups regarding heart rate at different periods of follow up.

Time	Group A	P1	Group B	P1	P2
On Admission	137.1±15.3		129.5±12.9		>0.05
Before MV discontinuation	89.7±12.35	0.001*	88.8±10.1	0.001*	>0.05
30 min after extubation	87.7±12.35		85.8±10.1		>0.05
4h after extubation	89.8±11.5	> 0.05	87.1±9.65	>0.05	>0.05
8h after extubation	88.7±10.9	> 0.05	85.3±10.3	>0.05	>0.05
12h after extubation	89.0±9.78	> 0.05	85.9±9.85	>0.05	>0.05
16h after extubation	90.6±9.6	> 0.05	85.8±8.99	>0.05	>0.05
20h after extubation	92.7±10.32	> 0.05	83.9±6.98	>0.05	0.001*
24h after extubation	92.0±11.1	> 0.05	84.1±10.31	>0.05	>0.05
28h after extubation	93.8±10.08	0.045*	85.5±9.85	>0.05	>0.05
32h after extubation	95.5±6.85	0.0021*	85.7±10.33	>0.05	>0.05
36h after extubation	97.0±5.98	0.001*	82.5±9.85	>0.05	>0.05
40h after extubation	96.2±6.01	0.001*	83.2±8.09	>0.05	0.01*
44h after extubation	99.7±6.11	0.001*	85.2±7.96	>0.05	0.01*
48 h after extubation	100.7±7.96	0.0001*	84.7±9.25	>0.05	0.0125

P1 comparison between interval times and baseline. P2 comparison between the two studied groups. *P is significant if P ≤ 0.05

Table (7): Comparison between the two studied groups regarding respiratory rate at different periods of follow up.

Time	Group A	P1	Group B	P1	P2
On Admission	38.3±3.51		35.6±4.01		0.098
Before MV discontinuation	18.03±3.89	0.001*	19.3±2.85	0.001*	>0.05
30 min after extubation	19.4±2.89		16.0±2.33		>0.05
4h after extubation	18.1±2.19	> 0.05	17.6±2.08	>0.05	>0.05
8h after extubation	17.4±1.95	> 0.05	16.9±1.96	>0.05	>0.05
12h after extubation	18.3±1.76	> 0.05	15.4±3.01	>0.05	>0.05
16h after extubation	18.3±2.03	> 0.05	15.3±1.85	>0.05	>0.05
20h after extubation	19.2±1.99	> 0.05	14.8±2.74	>0.05	0.035*
24h after extubation	18.4±2.07	> 0.05	15.8±2.33	>0.05	>0.05
28h after extubation	17.6±1.69	> 0.05	13.1±1.98	0.042*	>0.05
32h after extubation	16.4±1.67	> 0.05	14.3±3.07	>0.05	>0.05
36h after extubation	15.6±1.98	> 0.05	13.3±1.96	0.040*	>0.05
40h after extubation	17.3±2.11	> 0.05	14.5±2.11	>0.05	0.025*
44h after extubation	18.0±2.07	> 0.05	14.8±2.07	>0.05	0.01*
48 h after extubation	19.0±1.85	> 0.05	14.1±1.89	>0.05	0.012*

P1 comparison between interval times and baseline. P2 comparison between the two studied groups.
*P is significant if P ≤ 0.05

Table (8): Comparison between the two studied groups regarding PaO_2 at different periods of follow up.

Time	Group A	P1	Group B	P1	P2
On Admission	47.4±5.69		51.7±10.78		>0.05
Before MV discontinuation	98.1±11.6	0.0001*	102.0±13.5	0.0001*	>0.05
30 min after extubation	87.9±10.5		90.4±11.69		>0.05
4h after extubation	85.4±10.7	>0.05	92.7±12.7	>0.05	>0.05
8h after extubation	87.2±9.86	>0.05	91.3±8.98	>0.05	>0.05
12h after extubation	86.0±8.69	>0.05	92.3±7.52	>0.05	>0.05
16h after extubation	84.2±10.4	>0.05	90.5±8.03	>0.05	>0.05
20h after extubation	82.3±8.96	>0.05	90.7±8.33	>0.05	0.042*
24h after extubation	83.0±9.68	>0.05	91.3±9.01	>0.05	>0.05
28h after extubation	81.6±10.2	0.013*	90.6±10.33	>0.05	0.032*
32h after extubation	79.4±12.3	0.01*	89.8±8.79	>0.05	0.02*
36h after extubation	80.0±11.8	0.02*	88.8±9.65	>0.05	0.048*
40h after extubation	80.9±10.7	0.02*	89.6±10.23	>0.05	0.047*
44h after extubation	79.0±9.68	0.001*	88.2±9.85	>0.05	0.02*
48 h after extubation	78.5±8.65	0.001*	88.1±10.2	>0.05	0.001*

P1 comparison between interval times and baseline. P2 comparison between the two studied groups. *P is significant if P ≤ 0.05

Table (9): Comparison between the two studied groups regarding $PaCO_2$ at different periods of follow up.

Time	Group A	P1	Group B	P1	P2
On Admission	44.93±9.25		42.1±10.3		>0.05
Before MV discontinuation	39.42±6.78	0.01*	36.5±9.98	0.01*	>0.05
30 min after extubation	38.9±7.22		39.1±8.54		>0.05
4h after extubation	38.6±6.25	>0.05	40.0±6.89	>0.05	>0.05
8h after extubation	38.8±5.89	>0.05	40.8±6.99	>0.05	>0.05
12h after extubation	38.7±6.32	>0.05	40.1±5.68	>0.05	>0.05
16h after extubation	38.3±7.58	>0.05	40.4±10.3	>0.05	>0.05
20h after extubation	38.8±8.22	>0.05	40.2±6.32	>0.05	>0.05
24h after extubation	39.4±6.98	>0.05	40.2±7.65	>0.05	>0.05
28h after extubation	39.1±8.25	>0.05	40.6±8.65	>0.05	>0.05
32h after extubation	39.7±8.31	>0.05	40.1±8.01	>0.05	>0.05
36h after extubation	40.5±9.65	>0.05	40.2±7.65	>0.05	>0.05
40h after extubation	40.4±7.85	>0.05	40.9±6.58	>0.05	>0.05
44h after extubation	41.0±7.89	>0.05	40.6±7.01	>0.05	>0.05
48 h after extubation	41.5±8.31	>0.05	40.6±6.21	>0.05	>0.05

P1 comparison between interval times and baseline. P2 comparison between the two studied groups. *P is significant if $P \leq 0.05$

Table (10): Comparison between the two studied groups regarding CVP at different periods of follow up

Time	Group A	P1	Group B	P1	P2
On Admission	20.7±1.22		19.21±2.01		>0.05
Before MV discontinuation	13.88±2.03	0.01*	12.5±1.85	0.01*	>0.05
30min after extubation	14.5±1.78		12.9±2.03		>0.05
4h after extubation	14.6±1.16	>0.05	10.6±1.74	>0.041*	>0.01*
8h after extubation	13.2±2.01	>0.05	11.1±2.11	>0.049*	>0.01*
12h after extubation	14.6±1.78	>0.05	10.4±2.09	>0.04*	>0.01*
16h after extubation	14.3±1.66	>0.05	10.5±1.85	>0.042*	>0.01*
20 h after extubation	14.5±1.85	>0.05	9.6±1.65	>0.01*	>0.01*
24h after extubation	14.0±2.01	>0.05	9.9±1.74	>0.01*	>0.01*
28h after extubation	14.1±2.33	>0.05	9.7±1.33	>0.01*	>0.01*
32h after extubation	13.8±2.04	>0.05	9.6±2.08	>0.01*	>0.01*
36h after extubation	14.1±1.75	>0.05	8.8±1.76	>0.01*	>0.01*
40h after extubation	14.3±1.95	>0.05	9.8±1.65	>0.01*	>0.01*
44h after extubation	13.5±2.01	>0.05	9.2±1.09	>0.01*	>0.01*
48h after extubation	13.8±1.98	>0.05	9.9±2.01	>0.01*	>0.01*

P1 comparison between interval times and base line P2 comparison between the studied groups * P is significant if P<0.05

Table (11):Comparison between the two studied groups regarding alveolar O2 tension at different periods of follow up

Time	Group A	P1	Group B	P1	P2
30min after extubation	233.20±3.43		0.214±0.005		>0.05
4h after extubation	231.51±2.86	0.01*	0.216±0.0036	>0.05	>0.05
8h after extubation	219.46±2.65		0.19±0.001		>0.05
12h after extubation	207.47±3.72	>0.05	0.226±0.0025	>0.05	>0.05
16h after extubation	198.44±1.66	>0.05	0.220±0.008	>0.05	>0.05
20 h after extubation	185.47±4.17	>0.42	0.223±0.013	>0.05	>0.033*
24h after extubation	178.39±3.31	>0.31*	0.231±0.022	>0.05	>0.034*
28h after extubation	176.40±2.11	>0.01*	0.232±0.013	>0.05	>0.027*
32h after extubation	164.29±3.21	>0.01*	0.237±0.052	>0.042*	>0.033*
36h after extubation	162.36±4.19	>0.01*	0.240±0.027	>0.032*	>0.021*
40h after extubation	158.31±2.14	>0.01*	0.247±0.013	>0.022*	>0.016*
44h after extubation	150.60±3.09	>0.01*	0.252±0.015	>0.031*	>0.028*
48h after extubation	149.31±2.13	>0.01*	0.254±0.025	>0.01*	>0.021*

P1 comparison between interval times and base line P2 comparison between the studied groups * P is significant if P<0.05

Table (12):Comparison between the two studied groups regarding QS/QT at different periods of follow up

Time	Group A	P1	Group B	P1	P2
30min after extubation	0.201±0.005		0214±0.005		>0.05
4h after extubation	0.206±0.013	>0.05	0.216±0.0036	>0.05	>0.05
8h after extubation	0.229±0.006		0.219±0.001	>0.05	>0.05
12h after extubation	0.235±0.012	>0.05	0.226±0.0025	>0.05	>0.05
16h after extubation	0.243±0.018	>0.05	0.220±0.008	>0.05	>0.05
20 h after extubation	0.258±0.006	>0.05	0.223±0.013	>0.05	>0.05
24h after extubation	0.253±0.007	>0.05	0.231±0.0222	>0.05	>0.05
28h after extubation	0.260±0.005	>0.05	0.232±0.013	>0.05	>0.05
32h after extubation	0.282±0.006	>0.05	0.237±0.052	>0.05	>0.05
36h after extubation	0.307±0.007	>0.042*	0.240±0.027	>0.05	>0.05
40h after extubation	0.260±0.013	>0.036*	0.247±0.013	>0.05	>0.05
44h after extubation	0.282±0.016	>0.038*	0.252±0.015	>0.05	>0.049*
48h after extubation	0.307±0.0027	>0.033*	0.254±0.025	>0.05	>0.048*

P1 comparison between interval times and base line P2 comparison between the studied groups * P is significant if P<0.05

Table (13):Comparison between the two studied groups regarding O_2 extraction at different periods of follow up

Time	Group A	P1	Group B	P1	P2
30min after extubation	0.25±1.32		0.24±1.63		>0.05
4h after extubation	0.26±0.76	>0.05	0.23±1.75	>0.05	>0.05
8h after extubation	0.27±1.02	>0.05	0.23±1.65	>0.05	>0.05
12h after extubation	0.26±1.67	>0.05	0.23±1.74	>0.05	>0.05
16h after extubation	0.26±1.49	>0.05	0.24±1.65	>0.05	>0.05
20 h after extubation	0.27±1.83	>0.05	0.24±1.04	>0.05	>0.05
24h after extubation	0.26±0.89	>0.05	0.23±1.36	>0.05	>0.05
28h after extubation	0.27±1.16	>0.05	0.24±1.85	>0.05	>0.05
32h after extubation	0.27±1.36	>0.05	0.24±1.62	>0.05	>0.05
36h after extubation	0.28±1.04	>0.05	0.25±1.43	>0.05	>0.05
40h after extubation	0.28±1.21	>0.05	0.24±1.65	>0.05	>0.05
44h after extubation	0.29±1.07	>0.05	0.24±1.04	>0.05	>0.05
48h after extubation	0.29±1.63	>0.05	0.24±1.58	>0.05	>0.05

P1 comparison between interval times and base line P2comparison between the studied groups *P is significant if P<0.05

Table (14): Comparison between the two studied groups regarding the efficacy and causes of weaning failure.

	Group A (No.=16)		Group B (No.=16)		P
Efficacy	**No.**	**%**	**No.**	**%**	0.039*
Successful weaning	11	68.75	15	93.75	
Incidence of respiratory failure	**No.**	**%**	**No.**	**%**	
Weaning failure	5	31.25	1	6.25	
Causes of weaning failure	**No.**	**%**	**No.**	**%**	
Hypoxemia	4	25	1	6.25	0.144
Hemodynamic instability	3	18.75	1	6.25	0.285
Dysrrhythmia	1	6.25	0	0	0.309
Retaied secretions	1	6.25	0	0	0.309

* P is significant if P≤0.05

Table (15): Comparison between the two studied groups regarding the weaning time, the duration form extubation to reintubation, and the duration of ICU stay

	Group A	Group A	t	P
Weaning time (hours)				
Range	2.0-12.0	3.0-10.0	0.98	0.322
Mean	4.65	4.21		
S.D	3.65	3.98		
Duration from extubation to reintubation (hours)				
Range	3.0-7.0	6.0	-	-
Mean	4.75			
S.D	2.4			
Duration of ICU stay (days)				
Range	3.0-12.0	2.0-9.0	0.68	0.512
Mean	4.6	4.5		
S.D	2.3	2.2		

Table (16):Comparison between the two studied groups regarding incidence of ICU mortality incidence and types of complications .

	Group A (No.=16)		Group B (No.=16)		P
ICU mortality	**No.**	**%**	**No.**	**%**	0.039*
	2	12.5	3	18.8	
Incidence of Complication	**No.**	**%**	**No.**	**%**	
	4	25.0	2	12.5	0.365
Types of complication	**No.**	**%**	**No.**	**%**	
Laryngitis	2	12.5	0	0	0.144
Laryngeal edema	1	6.25	1	6.25	1.00
Chest infection	3	18.8	1	6.25	0.285

4. Discussion

The use of noninvasive positive-pressure ventilation in acute respiratory failure to avoid the need for endotracheal intubation was first reported in the late 1980s by **Meduri *et al*.** [51]. The apparent successful application of this form of ventilation has led to more intensive scrutiny of this technology in randomized controlled trials. It appears that successful avoidance of endotracheal intubation through the addition of NIPPV may depend - on the population studied [52] .

NIPPV has also been used to decrease the duration of mechanical ventilation for patients who require endotracheal intubation. For these patients, NIPPV has been applied in 1 of 3 ways: (1) as an adjunct to weaning patients from mechanical ventilation by early extubation directly to NIPPV,[53-54] (2) as a routine application of NIPPV to all patients or a selected group of higher-risk patients who were extubated at the time they fulfilled standard extubation criteria,[49] or (3) as an application of NIPPV only to patients who develop respiratory distress after having been extubated according to standard criteria [55] .

The idea of using noninvasive positive pressure ventilation to manage patients with post-extubation respiratory distress came from several trials demonstrating efficacy of noninvasive positive pressure ventilation in postoperative respiratory failure [56,57] . In a prospective study of 72 patients whose acute respiratory failure after upper abdominal surgery was treated with NIPPy, intubation was not required in 48 patients [58] .

In the present study, early application of NJPPV immediately after extubation was more efficient than standard medical therapy alone in prevention of post-extubation respiratory failure in the selected patients.

In agreement with **Nava *et al*.** [50] who described the benefit of noninvasive ventilation in preventing post-extubation respiratory failure in a controlled trial included 97 patients considered at risk of developing post-extubation respiratory failure; the patients were randomly assigned to receive conventional medical therapy with or without noninvasive mechanical ventilation. Patients were considered at risk if they had hypercapnia, congestive heart failure, ineffective cough, excessive tracheobronchial secretions, more than one failed weaning trial, more than one co-morbidity, and/or upper airway obstruction. This study reported that the noninvasive ventilated group had also a lower rate of reintubation.

The same was reported by **Ferrer *et al*.** [59] who studied the role of noninvasive ventilation in patients with persistent weaning failure and reported in their study that the early use of noninvasive ventilation helps to prevent respiratory failure after extubation among patients at increased risk.

In agreement with **Girault *et al*.** [54] showed in their study that noninvasive ventilation prevents the occurrence of post-extubation respiratory distress and permits earlier removal of the endotracheal tube, without increasing the risk of weaning failure and they reported that noninvasive ventilation should be considered as a new and useful systematic approach to weaning in patients with acute on top of chronic respiratory failure who are difficult to wean.

Chiang *et al*. [60] in their study evaluated the use of noninvasive positive pressure ventilation via nasal mask in patients with respiratory distress after extubation, they reported that NIPPV may be considered as an alternative to endotracheal reintuhation in selected extubated patients with respiratory distress who require no immediate reintubation.

Burns *et al*. [61] also reported that the use of noninvasive ventilation to facilitate weaning during persistent weaning failure in mechanically ventilated patients, with predominantly chronic obstructive lung disease, is associated with promising evidence of net clinical benefit and lower incidence of post-extubation respiratory failure.

The same was reported by **Nava *et al*.** [53] who studied the role of noninvasive ventilation in the weaning of patients with respiratory failure due to obstructive pulmonary diseases and they reported that Patients randomized to receive NJPPV had a shorter duration of ventilatory support, a shorter weaning time, a shorter length of ICU stay, less incidence of pneumonia, less incidence of reintubation, and improved survival compared with those undergoing a

conventional weaning from mechanical ventilation.

Girault *et al*. [54] conducted a similar study on a more heterogeneous population (not all with COPD) and after a variable period of conventional mechanical ventilation. They found a decreased duration of conventional mechanical ventilation among patients randomized to early extubation to NIPPV but no difference in other outcomes.

In agreement with **El Solh *et al*.** [62] who studied the efficacy of noninvasive ventilation for prevention of post-extubation respiratory failure in obese patients, 62 consecutive severely obese patients were assigned to NIV via nasal mask immediately-post-extubation and compared with 62 historically matched controls who were treated with conventional therapy. They reported that the institution of NIV resulted in 16 % absolute risk reduction in the rate of respiratory failure. There was a significant difference in the lengths of ICU and hospital stay between the two groups. Subgroup analysis of hypercapnic patients showed reduced hospital mortality in the NIV group compared with the control group. In conclusion, the study revealed that NIV may be effective in averting respiratory failure in severely obese patients when applies during the first 48h post-extubation. Also, in selected patients with chronic hypercarbia, early application of NIV may confer a survival benefit

On the other hand, **Keenan *et al*.** [63] showed in a heterogeneous group of 81 patients who developed respiratory distress during the first 48 hours after extubation that treatment with noninvasive ventilation didn't improve the outcome compared with the standard medical therapy. The criteria for defining post-extubation failure were not based on determination of arterial blood gases but mainly on the clinical signs such as an increased- - respiratory rate or the presence of accessory muscle recruitmeut and abdominal paradox. This study was a relatively small, single-center trial that evaluated the rate of reintubation as a primary end point. The extent to which its results can be generalized has been questioned.

Nava *et al*. [50] showed in their study that the need for reintubation was associated with a higher risk of ICU mortality and the use of noninvasive ventilation immediately after extubation in high risk patients resulted in a reduction of the risk of ICU mortality.

On the other hand, the main finding of the study carried out by **Esteban** and colleagues[64] is that noninvasive ventilation did not reduce mortality or the need for reintubation among patients receiving mechanical ventilation who had respiratory failure after extubation. The mortality rate tended to be higher among the patients assigned to noninvasive ventilation than among those assigned to standard medical therapy, and the interval from the development of respiratory failure to reintubation was significantly longer with noninvasive ventilation than with standard therapy.

Again, one randomized, controlled trial examining the use of noninvasive positive-pressure ventilation in patients with respiratory failure after extubation, **Keenan** and colleagues[63]enrolled 81 patients in a single-center study and did not find differences between patients assigned to noninvasive ventilation and those assigned to standard therapy in the rate of death either in the intensive care unit or in the hospital overall.

In the present study, from the 16 patients included in the control group; there were 11 patients successfully weaned. Among the five patients who suffered post-extubation respiratory failure; four of them suffered significant hypoxernia, three patients had hemodynamic instability, one patient had dysrrhythmia in the form rapid atrial fibrillation with multiple premature ventricular contractions, and 2 patients had obviously retained secretions. On the other side; in the NIPPV group; there were 15 patients successfully weaned. The only patient who had weaning failure suffered from hypoxemia, and hemodynamic instability.

In general, few complications are associated with NIPPV. The most common problem is local damage to facial tissue because of the pressure effects of the mask and straps. Mild gastric distension may occur but is not significant. Eye irritation and sinus pain or congestion may also occur. Barotrauma is uncommon. Modest air leaks at the facial seal are common but do not decrease the benefit patients receive from NJPPV. Adverse hemodynamic effects resulting from NIPPV are unusual, although preload reduction and hypotension may occur.[65]

In the present study; among patients in the control group, there were 4 patients had complications related to ETT and MV, from them two patients had laryngitis accompanied with hoarseness of voice, one patient had laryngeal edema with associated stridor, and three patients had ventilator associated pneumonia. While in NIPPV group; there was only one patient suffered laryngeal edema and another one had ventilator associated pneumonia. There was no statistically significant difference between the two studied groups as regards the incidence of complications related to UTT and MV.

These findings are in agreement with **Skyba *et al.*** [66] who studied blood pressure-and heart rate variability in response to noninvasive ventilation and they demonstrated significant reductions in systolic and diastolic blood pressures and heart rate.

The hemodynamic effects of noninvasive ventilation was also studied by **Naughton *et al.*** [67] and they revealed that the decrease in the systolic and diastolic blood pressure was due to improvement of cardiac index and reduction in left ventricular transmural pressure as

continuous positive airway pressure abolished the impact of large negative swings in pleural pressure on the left ventricular performance. They finally concluded that the failing heart is sensitive to minimal changes in the after load. Moreover, the reduction in the heart rate in patients with congestive heart failure could potentially improve subendocardial perfusion and allow for better left ventricular diastolic filling.

Chadda *et al.* [68] also reported in their study that noninvasive ventilation reduced the mean transmural right and left atrial flung pressuresrrhis study also demonstrates that noninvasive ventilation produced a reduction in right and left ventricular preload, which suggests an improvement in cardiac performance.

On the other hand, **Leech *et al.*** [69] and **Montner *et al.*** [70] who reported no significant changes in heart rate in their studied patients while receiving noninvasive mask ventilation of up to 15 cmH_2o pressure associated with related fall in stroke volume and cardiac output in the first study, and no significant homodynamic changes in the second one. The absence of compensatory increase in heart rate as stroke volume and cardiac output fell was attributed to that noninvasive mask ventilation may have triggered parasympathetic reflexes preventing an increase in heart-rate.

In the present study the determination of baseline clinical factors such as heart rate and respiratory rate at the time of initiation and through out the study predicted the likelihood of failure or success of noninvasive ventilation as the heart rate and respiratory rate increased significantly in the failed cases while these parameters were maintained in the normal range in the successfully weaned patients.

These findings are in agreement with **Singh *et al.*** [71] and **Nava *et al.*** [50] who showed that there was significant improvement in heart rate and respiratory rate within 1st hr after initiation of noninvasive ventilation in success group of patients and these parameters continues to improve even after few hours of noninvasive ventilation treatment. They reported that determination of baseline clinical factors such as heart rate and respiratory rate at the time of initiation and after a short period, can predict the likelihood of success or failure of noninvasive ventilation. As a result, delay in intubation can be avoided which itself is associated with significant mortality.

In the present study the mean arterial oxygen tension PaO_2 decreased significantly through out the study in the patients received the standard medical therapy after their extubation and it did not change significantly in the patients received noninvasive ventilation immediately after their extubation.

In agreement with **Rasanen *et al.*** [72] and **Nava *et al.*** [50] who reported in their study significant improvement in PaO_2 after initiation of noninvasive ventilation. They also reported significant difference between mask CPAP treated group and the control group in which patients were treated medically plus supplemental oxygen therapy through conventional mask with the same fractional oxygen concentration.

On the contrary to the present study, **Gay *et al.*** [73] and **Ferrer *et al.*** [59] reported significant decrease in arterial carbon dioxide tension with the use of and they postulated that positive inspiratory pressure during noninvasive ventilation helps to generate the elevated transpulmonary pressures necessary for lung inflation at higher functional residual capacity and to overcome the inspiratory threshold imposed by auto positive end expiratory pressure (auto PEEP). Alternatively, noninvasive ventilation may recruit expiratory muscles to defend end expiratory lung volume. Both of these actions unload the inspiratory muscles, which are mechanically disadvantaged when the lungs are hyperinflated.

The disagreement between our results and results of the previously mentioned studies may be attributed to that **Ferrer *et al.*** [59] and **Gay *et al.*** [73] studied mainly patients with acute exacerbation of COPD with hypercapnic respiratory failure, those patients get the maximum benefit from NIPPV in reducing effort of breathing and improving gas exchange. However only five patients in our selected population were COPD, also the main problem in our patients was impaired oxygenation rather than compromised ventilation. The relatively healthier lungs of our patients might offer a defense against post extubation ventilatory impairment even without the application of NIPPV.

In the present study noninvasive mechanical ventilations maintains a normal or slightly changed alveolar O_2 tension and shunt fraction throughout the study for the patients who received it immediately after their extubation while these parameters changed significantly in the group of patients received the standard medical therapy. However this significant change between the two studied groups was not clearly identified as regard oxygen extraction ratio.

In agreement with **Burns *et al.*** [61] who showed in their study a significant reduction in shunt fraction and alveolar arterial oxygen gradient with continuous improvement when serial incremental CPAP therapy was applied gradually.

Ferrer *et al.* [74] reported that noninvasive ventilation improved pulmonary gas exchange and shunt fraction and oxygen extraction ratio by increasing alveolar ventilation with significant reduction in respiratory rate and increase tidal volume and minute ventilation and increase in PaO_2 and decrease in $PaCO_2$.

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References

1. Ali A Sovari, Abraham G Kocheril, Amal Mattu, Sat Sharma, Ari M Perkins, Michael E Zevitz. Cardiogenic Pulmonary Edema. eMedicine; 2006; 94: 1625-33
2. Gropper MA, Wiener-Kronish J, Hashimoto S. Acute cardiogenic pulmonary edema. Clin Chest Med.; 1994; 15: 501.
3. Gerald W Staton. Gardiogenic Pulmonary Edema. ACP Medicine Online. 2002.
4. Meduri GU, Turner RE, Abou-Shala N, Wunderink R, Tolley E. Noninvasive positive pressure ventilation via face mask. First line intervention in patients with acute hypercapnic and hypoxemic respiratory failure. Chest; 1996;109: 179-93.
5. Wysocki M, Tric L, Wolff MA, Millet H, Herman B. Noninvasive pressure support ventilation in patients with acute respiratory failure. A randomized comparison with conventional therapy. Chest;.1995; 107: 761-8.
6. Wysocki M, Tric L, Wolff MA, Gertner J, Millet H, Herman B. Noninvasive pressure support ventilation in patients with acute respiratory failure. Chest; 1993; l03:907-13.
7. Kramer N, Meyer TJ, Meharg J, Cece RD, Hill NS. Randomized prospective trial of noninvasive positive pressure ventilation in acute respiratory failure. Am J Respir Grit Care Med; 1995; 151: 1799-806.
8. Meduri GU. Noninvasive ventilation. Physiological Basis of Ventilatory Support. Edited by Marini J, Slustky A. Series on Lung Biology in Health and Disease. Marcel Dekker, mc: New York 1998.
9. Nourdine K, Combes P, Carton MJ, Beuret P, Cannamela A Ducreux JC. Does noninvasive ventilation reduce the JCU nosocomial infection risk. A prospective clinical survey. Intens Care Med. 1999; 25: 567-73.
10. Pingleton S. Complications of acute respiratory failure. Am Rev Respir Dis. 1988; 137: 1463-93.
11. Rashkin MC, Davis T. Acute complications of endotracheal intubation. Chest. 1986; 89: 165-7.
12. Torros A, Aznar R, Gatell JM. Incidence, risk and prognosis factors of nosocomial pneumonia in mechanically ventilated patients. Amer Rev Respir Dis 1990; 142:523-8.
13. Burns HP, Dayal VS, Scott A, Van Nostran ANP, Bryce DP. Laryngotracheal trauma, observation on its pathogenesis and its prevention following prolonged TO intubation in the adult. Laryngoscope; 1979; 89: 1316-25.
14. Heffner JE, Miller KS, Sahn SA. Tracheostomy in the intensive care unit. Part 2. Complications. Chest; 1986; 90: 430-6.
15. Deutschman CS, Wilton P, Sinow J, Dibbell D, Konstantinides FN, Cerra FB. Pananasal sinusitis associated with nasotracheal intubation, a frequently unrecognized and treatable source of sepsis. Grit Cane Med. 1986; 14: 11-114.
16. Shapiro M, Wilson RK, Casar G, Bloom K, Teague RB. Work of breathing through different sized endotracheal tubes. Grit Care Med. 1986; 14: 1028-31.
17. Maclntyre NR, Cook DJ, Ely EW. Evidence-based guidelines for weaning and discontinuing ventilatory support. Chest; 120(6 suppl); 2001; 375S-95S.
18. Epstein SK, Giubotaru RL, Wong J. Effect of failed extubation on the outcome of mechanical ventilation. Chest; 1997; 112: 186-92.
19. Epstein SK. Decision to extubate. Intensive Care Med; 2002; 28: 535-46.
20. Esteban A, Alia I, Tobin MJ, Gil A, Gordo F, Blanch L. Effect of spontaneous breathing trial duration on outcome of attempts to discontinue mechanical ventilation. Am J Respir Grit Care Med; 1999; 159: 512-8.
21. Esteban Al, Alia J, Ibanez, Benito, Tobin MJ. Modes of mechanical ventilation and weaning; a national survey of Spanish hospitals. Chest. 2000; 19-94; 106: 1188-93.
22. Khamiees M, Raju P, Degirolamo A, Amoateng-Adjepong Y, Manthous CA. Predictors of extubation outcome in patients who have successfully completed a spontaneous breathing trial. Chest 2001; 120: 1262-70.
23. Salam A, Tilluckdharry L, Amoateng-Adjepong Y, Manthous CA. Neurologic status, cough, secretions and extubation outcomes. Intensive Care Med 2004; 30: 1334-9.
24. Smina M, Salam A, Khamiees M, Gada P, Amoateng-Adjepong Y, Manthous GA. Cough peak flows and extubation outcomes. Chest 2003; 124: 262-8.
25. Ely EW, Meade M, Haponik EF. Mechanical ventilator weaning protocols driven by non physician health-care professionals; evidence-based clinical practice guidelines. Chest. 2001; 120 (6 suppl): 454S-63S.
26. Hall JB, Schmidt GA, Wood LDH. Principle of critical care. Mc. Grow Hill New yourk 1998: 125-37.
27. Meduri GU. Noninvasive ventilation. In Physiological Basis of Ventilatory support. Edited by Marini J, Slustky A. Series on Lung Biology in Health and Disease. Marcel Dekker, mc: New York, 1998: 92 1-98.
28. Antonelli M, Conti G, Rocco M. A comparison of noninvasive positive-pressure ventilation and conventional mechanical ventilation in patients with acute respiratory failure. N Engl J Med 1998; 339: 429-35.
29. Mehta 5, Hill NS. Noninvasive ventilation. Am J Respir Grit Care Med 2001; 163:540.
30. Liesching T, Kwok H, Hill NS. Acute applications of noninvasive positive pressure ventilation. Chest 2003; 124:699.
31. Sat Sharma. Non invasive ventilation. eMedicine. Last Updated: May 15, 2006.
32. Joao C Winck, Luis F Azevedo, Altamiro Costa-Pereira , Massimo Antonelli, Jeremy C Wyatt. Efficacy and safety of non-invasive ventilation in the treatment of acute cardiogenic pulmonary edema; a systematic review and meta-analysis. Critical Care 2006, 10:R69 doi: 10.1186/cc4905.
33. Rusterholtz T, Kemp FJ, Berton C, Gayol S, Tournoud C, Zaehringer M, et al. Noninvasive pressure support ventilation with face mask in patients with acute cardiogenic pulmonary edema. Intensive Care Med 1999; 25: 21-8
34. Charles E Saunders, Mary T Ho (eds): Current Emergency Diagnosis and Treatment. Fourth edition, 1992. Barash PG , Cullen BF, Stoelting RK[eds]: Clinical Anesthesia. Philadelphia, JB Lippincott, 1992. Davison JK, Eckardt III WF, Perese DA [eds]: Clinical Anesthesia Procedures of the Massachusetts General Hospital. Little Brown, 1993. Duke J, Rosemberg SG [eds]: Anesthesia Secrets. Hanley & Belfus, 1996.
35. Allon Amitia. Emergency Medicine, Guidelines for ventilator setting. eMedicine Last Updated: March 16, 2006.
36. Shamai Grossman, David FM Brown. Congestive Heart Failure and Pulmonary Edema. eMedicine, Last Updated: May, 11, 2006.
37. Maclntyre NR, Cook DJ, Ely EW, Epstein 8K, Fink JB, Heffner JE, etal. Evidence-based guidelines for weaning and discontinuing ventilatory support; a collective task force facilitated by the American College of Chest Physicians; the American Association for Respiratory Care, and the American College of Critical Care Medicine. Chest 2001; 120 (6 Suppl): 375S-95S.
38. British Journal of Anaesthesia 92 (6): 793±9 (2004). DOI: 10.1093/bja/aehl39 Advance Access publication April 30, 2004. Evaluation of simple criteria to predict successful weaning from mechanical ventilation in intensive care patients.
39. American Association for Respiratory Care (AARC). Removal of the endotracheal tube. 2007 revision & update. Respir Care 2007 Jan; 52 (1): 81-93.
40. S Baudouin, S Blumenthal, B Cooper, C Davidson, A Davison, M Elliott, et al. British Thoracic Society Guidelines, Non-invasive ventilation in acute respiratory failure. British Thoracic Society Standards of Care Committee. Thorax 2002; 57:192-211.
41. Massimo Antonelli and Giorgio Conti. Noninvasive positive

pressure ventilation as treatment for acute respiratory failure in critically ill patients. Critr Care 2000; 4: 15-22
42. Brochard LA, Rauss S, Benito G Conti J Mancebo N Rekik A, et al. Comparison of three methods of gradual withdrawal from ventilatory support during weaning from mechanical ventilation. Am. J. Respir. Grit. Care Med; 150: 896-903.
43. R Fernandez, S Benito, R Tomas, D Caniedo, S Macias, J Blanco, and the Spanish Lung Failure Collaborative Group. 1995. A comparison of four methods of weaning patients from mechanical ventilation: N. Engl. Med; 332: 345-50
44. R Fernandez, J F Solsona, I Valverdu, S Macias, J Allegue, J Blanco, etal. Extubation outcome after spontaneous breathing trials with T-tube or pressure support ventilation. Am. J. Respir. Grit. Care Med; *156: 459—65.*
45. Kramer N, Meyer TJ, Meharg J, Cece RD, Hill NS. Randomized, prospective trial of noninvasive positive pressure ventilation in acute respiratory failure. Am J Respir Grit Care Med 1995; 151: 1799-806.
46. Meduri G U, Abou—shala N, Fox RC. Non invasive face mask mechanical ventilation in patients with acute hypercapnic respiratory failure. Chest 1991; 100: 445-54.
47. Ferrer M, Esquinas A Arancibia F, Bauer TT, GonzalezG~ Carrillo A, et al. Noninvasive ventilation during persistent weaning failure; a randomized controlled trial. Am J Respir Grit Care Med 2003; 168: 70-6.
48. Steven D Nathan, Abraham M Ishaaya, Spenser K Goener. Prediction of minimal pressure support during weaning fro ventilation. Chest 1993: 103: 1215-9.
49. Jiang JS, Kao SJ, Wang SN. Effect of early application of biphasic positive airway pressure on the outcome of extubation in ventilator weaning. Respirology. 1999 Jun;4(2):161-5.
50. Nava S, Gregoretti C,—Fanfulla- -F, Squadrone- E Grassi M, Gariucci A, et al. Noninvasive ventilation to prevent respiratory failure after extubation in high-risk patients. Grit Care Med. 2005 Nov; 33(1 l):2465-70.
51. Meduri GU, Conoscenti CC, Menashe P, Nair S. Noninvasjve face mask ventilation in patients with acute respiratory failure. Chest. 1989 Apr; 95(4):865-70.
52. Keenan SP. Noninvasive positive pressure- in acute respiratory failure. - JAMA. 2000; 284: 2376-8.
53. Nava S, Ambrosino N, Clini F, et al. Noninvasive mechanical ventilation in the weaning of patients with respiratory failure due to chronic obstructive pulmonary disease: a randomized, controlled trial. Ann Intern Med. 1998:128:721-8.
54. Girault C, Daudenthun I, Chevron V, Tamion F, Leroy J, Bonmarchand G. Noninvasive ventilation as a systematic extubation and weaning technique in acute-on-chronic respiratory failure: a prospective, randomized controlled study. Am J Respir Grit Care Med. 1999;160:86-92.
55. Wysocki M, Tric L, Wolff MA, Gertner J, Millet H, Herman B. Noninvasive pressure support ventilation in patients with acute respiratory failure. Chest. 1993 Mar; 103(3):907-13.
56. Epstein SK, Vuong V. Lack of influence of gender on outcomes of mechanically ventilated medical ICU patients. Chest. 1999 Sep; 116(3):732-9.
57. Kollef MH. Acute respiratory failure: a gender-based outcomes analysis. J Gend Specif Med. 1998-Dec; 1(3):24-30.
58. Kollef MH. Do age and gender influence outcome from mechanical ventilation Heart -Lung. 1993 Sep-Oct; 22(5):442-9.
59. Ferrer M, Esquinas A, Arancibia F, Bauer TT, Gonzalez G, Carrillo A. Noninvasive ventilation during persistent weaning failure; a randomized controlled trial. Am J Respir Grit Care Med 2003; 168: 70-6.
60. Chiang AA, Lee KG. Use of noninvasive positive pressure ventilation via nasal mask in patients with respiratory distress after extubation. Zhonghua Yi Xue Za Zhi (Taipei). 1995 Aug; 56(2):94- 101.
61. Burns KE, Adhikari NK, Meade MO. Noninvasive positive pressure ventilation as a weaning strategy for intubated adults with respiratory failure. Cochrane Database Syst Rev. 2003(4):CD004127.
62. El Solh, A. Aquilina, L. Pineda, V. Dhanvantri, B. Grant and P. Bouquin. Noninvasive ventilation for prevention of post-extubation respiratory failure in obese patients. Eur Respir J. 2006: 28:588-95.
63. Keenan SP, Powers C, McCormack DG, Block G. Noninvasive positive pressure ventilation for postextubation respiratory distress: a randomized controlled trial. Jama. 2002 Jun 26; 287(24):32I8-44.
64. Esteban A, Alia I, Gordo F, et al. Extubation outcome after spontaneous breathing trials with T-tube or pressure support ventilation. Am J Respir. Grit Care Med.1997; 156:459-65.
65. Sat Sharma. Non invasive ventilation. eMedicine. Last Updated: May 15, 2006.
66. Skyba P, Joppa P, Orolin M, Tkacova R. Blood pressure and heart rate variability response to noninvasive ventilation in patients with exacerbations of chronic obstructive pulmonary disease. Physiol Res. 2006 Dec 19.
67. Naughton MT, Rahman MA, Hara K, Eloras JS, Bradley TD. Effect of continuous positive airway pressure on intrathoracic and left ventricular transmural pressures in patients with congestive heart failure. Circulation. 1995 Mar 15; 91(6): l725-31.
68. Chadda K, Annane D, Hart N, Gajdos P, Raphael JG, Lofaso F. Cardiac and respiratory effects of continuous positive airway pressure and noninvasive ventilation in acute cardiac pulmonary edema. Grit Care Med. 2002 Nov; 30(11): 2457-61.
69. Leech JA, Ascah KJ. Hemodynamic effects of nasal CPAP examined by Doppler echocardiography. Chest. 1991 Feb; 99(2):323 -6.
70. Montner PK, Greene FR, Murata GH, Stark DM, Timms M, Chick TW. Hemodynamic effects of nasal and face mask continuous positive airway pressure. Am J Respir Grit Care Med. 1994 Jun; 149(6):1614-8.
71. Singh VK, Khanna P, Rao BK, Sharma SC, Gupta R. Outcome predictors for non-invasive positive pressure ventilation in acute respiratory failure. J Assoc Physicians India. 2006 May; 54:361-5.
72. Rasanen J, Heikkila J, Downs J, Nikki P. Vaisanen I, Viitanen A. Continuous positive airway pressure by face mask in acute cardiogenic pulmonary edema. Am J Gardiol.1985 Feb 1; 55(4):296-300.
73. Gay PC, Rodarte JR, Hubmayr RD. The effects of positive expiratory pressure on isovolume flow and dynamic hyperinflation in patients receiving mechanical ventilation. Am Rev Respir Dis. 1989 Mar;139(31621-6.
74. Ferrer M, Vaiencia M, Nicolas JM, Bernadich O, Badia JR, Torres A. Early noninvasive ventilation averts extubation faihireln in patients at risk: a ransomized trial. Am J Respir Crit Care Med. 2006 Jan15; 173(2):164-70.

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