Noninvasive face mask ventilation has been used successfully in patients with paralytic respiratory failure. This study evaluated whether noninvasive face mask ventilation can be used for patients with acute respiratory failure due to intrinsic lung disease. Six patients with hypercapnia and four with hypoxemic acute respiratory failure met clinical and objective criteria for mechanical ventilation, which was delivered with pressure control and pressure support via a tightly strapped, clear face mask. No patient terminated the study because of inability to deliver adequate ventilation or to improve oxygen exchange; three eventually required endotracheal intubation. The mask was generally well tolerated. All patients had a nasogastric tube placed on suction, and none vomited or aspirated. The mean duration of treatment was 33 h (range, 3 to 88). The physiologic response was considered similar to that which would have been achieved with conventionally delivered ventilation. Noninvasive face mask ventilation may have a role in managing respiratory failure. (Chest 1989; 95:865-70)

Noninvasive mechanical ventilation is used successfully in patients with acute or chronic paralytic respiratory failure. Many patients with only moderate respiratory muscle weakness during the daytime develop severe respiratory failure at night and require ventilatory support. In recent years, noninvasive positive-pressure ventilation via the mouth or nose has partially replaced tracheostomy or the use of negative-pressure ventilation for this group of patients. Noninvasive positive-pressure ventilation with a face mask or mouth seal is effective in supporting ventilation for years; however, it can be associated with complications such as asphyxiation, dehydration of the oropharynx, or bite deformities from the mouth seal.1,2 Nose intermittent ventilation administered via a nasal continuous positive-airway pressure (CPAP) mask and a fitted foam rubber piece have been equally effective and better tolerated in patients with neuromuscular disease, severe kyphoscoliosis, or central hypoventilation.3-11

We initiated this study to evaluate whether noninvasive ventilation via a face mask can be used for

Material and Method

Patients with acute hypoxic or hypercapnic respiratory failure nonselectively entered the study from April 1987 to June 1988 if they met clinical and physiologic parameters indicating the need for mechanical ventilation. The protocol was approved by the Institutional Review Board.

Diagnostic criteria for hypercapnic respiratory failure were severe difficulty in breathing as expressed by the patient, hypercapnia, acute respiratory acidosis with a respiratory rate more than 30 breaths/min, and signs of increased respiratory work such as intercostal and suprasternal retraction. Intubation was considered for those patients with COPD only after they failed conventional aggressive treatment with inhaled and systemic bronchodilators and IV steroids.

Diagnostic criteria for hypoxic respiratory failure included difficulty in breathing spontaneously as expressed by the patient, respiratory rate more than 30 breaths/min, PaO2:F1O2 less than 200, and signs of increased work of breathing, such as use of accessory muscles of respiration or Pco2 retention.

Criteria for excluding patients from the study included hemodynamic and ECG instability (patients with pulmonary edema without hypotension were included), need for endotracheal intubation to protect the airways or to manage respiratory secretions, and inability to properly fit the face mask. Criteria for leaving the study included patient's request, hemodynamic or ECG instability, need for intubation to protect the airways or manage secretions, inability to improve alveolar ventilation or oxygen exchange, and failure to improve mental status of patients who were lethargic from CO2 retention or agitated from hypoxemia before initiating noninvasive face mask ventilation.

Each patient used one of two masks: the Snugger anesthesia and respiratory mask (No. 8887, Hospitak Inc) incorporated a large, high-compliance, low-pressure inflatable cuff for facial sealing, and the Downs CPAP mask (9000, Vital Signs Corp) allowed for a larger mask surface area. A nasogastric tube was inserted before initiating face mask ventilation and placed on suction.
Table 1—Diagnosis, Outcome, and Baseline Blood Gas Values of 10 Patients with Acute Respiratory Failure*

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Diagnosis</th>
<th>Age, yr</th>
<th>Sex</th>
<th>Outcome</th>
<th>Survival</th>
<th>H</th>
<th>pH</th>
<th>Pco₂, mm Hg</th>
<th>Po₂, mm Hg</th>
<th>FiO₂</th>
<th>PEEP, cm H₂O</th>
<th>RR, breaths/min</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>COPD/RTI</td>
<td>58</td>
<td>F</td>
<td>Excellent</td>
<td>S</td>
<td>43</td>
<td>7.10</td>
<td>83</td>
<td>114</td>
<td>0.28</td>
<td>0</td>
<td>36</td>
</tr>
<tr>
<td>2</td>
<td>COPD</td>
<td>78</td>
<td>F</td>
<td>Seizure/Intub.</td>
<td>S</td>
<td>3</td>
<td>7.25</td>
<td>84</td>
<td>60</td>
<td>0.35</td>
<td>0</td>
<td>44</td>
</tr>
<tr>
<td>3</td>
<td>COPD/CHF</td>
<td>70</td>
<td>M</td>
<td>Refused to continue</td>
<td>NS</td>
<td>88</td>
<td>7.21</td>
<td>93</td>
<td>46</td>
<td>0.32</td>
<td>0</td>
<td>28</td>
</tr>
<tr>
<td>4</td>
<td>COPD/RTI</td>
<td>68</td>
<td>M</td>
<td>ML/Died</td>
<td>NS</td>
<td>16</td>
<td>7.22</td>
<td>92</td>
<td>47</td>
<td>0.24</td>
<td>0</td>
<td>13</td>
</tr>
<tr>
<td>5</td>
<td>COPD/RTI</td>
<td>84</td>
<td>F</td>
<td>Excellent</td>
<td>S</td>
<td>29</td>
<td>7.39</td>
<td>59</td>
<td>66</td>
<td>0.28</td>
<td>0</td>
<td>40</td>
</tr>
<tr>
<td>6</td>
<td>COPD/RTI/OD</td>
<td>62</td>
<td>F</td>
<td>Excellent</td>
<td>S</td>
<td>7</td>
<td>7.25</td>
<td>59</td>
<td>59</td>
<td>0.35</td>
<td>0</td>
<td>40</td>
</tr>
<tr>
<td>7</td>
<td>ARDS/Sepsis</td>
<td>32</td>
<td>F</td>
<td>Excellent</td>
<td>S</td>
<td>71</td>
<td>7.33</td>
<td>52</td>
<td>87</td>
<td>0.70</td>
<td>18</td>
<td>50</td>
</tr>
<tr>
<td>8</td>
<td>ARDS/AIDS-PCP</td>
<td>40</td>
<td>F</td>
<td>Excellent</td>
<td>S</td>
<td>48</td>
<td>7.42</td>
<td>42</td>
<td>74</td>
<td>0.90</td>
<td>10</td>
<td>40</td>
</tr>
<tr>
<td>9</td>
<td>Cardiac edema</td>
<td>48</td>
<td>F</td>
<td>Not tolerated, intubated</td>
<td>S</td>
<td>20</td>
<td>7.34</td>
<td>34</td>
<td>51</td>
<td>0.50</td>
<td>0</td>
<td>40</td>
</tr>
<tr>
<td>10</td>
<td>Cardiac edema</td>
<td>77</td>
<td>M</td>
<td>Excellent</td>
<td>S</td>
<td>4</td>
<td>7.25</td>
<td>59</td>
<td>73</td>
<td>1.00</td>
<td>0</td>
<td>34</td>
</tr>
</tbody>
</table>

*H = Duration of face mask ventilation; PEEP = positive end-expiratory pressure; RR = respiratory rate; RTI = respiratory tract infection; OD = overdose; PCP = Pneumocystis carinii pneumonia; S = survivor; NS = nonsurvivor.

All patients received mechanical ventilation using a Servo 900C Ventilator (Siemens-Elema Corporation). During mechanical ventilation, all lung volumes, including exhaled minute ventilation (Ve) and exhaled tidal volume (Vt) were measured using the standard Servo flow monitoring system. Blood gases were measured with a Radiometer ABL 30 Acid-Base Analyzer (Radiometer Corp). Measurements were made following daily calibration of the acid-base analyzer using standard testing solutions. Patients received continuous ECG and oximetric monitoring.

The following data were collected: use of the accessory muscles of respiration, presence of paradoxical abdominal motion, exhaled volumes, vital signs, baseline pulmonary function testing, duration of face mask ventilation, causes of exit from the study, and possible complications.

RESULTS

Ten patients with acute respiratory failure entered the study (Table 1). Six had COPD with acute ventilatory failure, and four had refractory hypoxemia of different etiologies. Two patients who met the entrance criteria were unable to participate in the study because of the inability to find a mask that properly fit their facial contour.

Hypercapnic Respiratory Failure

Six patients had acute exacerbation of severe COPD with a mean baseline FEV₁ of 577 ml. Pulmonary function test results for the four women and two men are shown in Table 2. Results for patients 1 and 3 were obtained 10 years before this admission. The mean age was 70 (range, 58 to 84 years). The cause of the acute decompensation was believed to be a respiratory tract infection in patients 1, 4, and 5; congestive heart failure in patient 3; a combination of bronchitis and narcotic overdose in patient 6; and not known in patient 2.

All patients were in severe respiratory distress, four were using the accessory muscles of respiration, and all but one (patient 4) were tachypneic, with a mean respiratory rate of 34 breaths/min. All patients failed to improve despite aggressive treatment with systemic and inhaled bronchodilators and IV steroids. Three patients (2, 4, and 6) were lethargic but became alert and responsive soon after beginning ventilation.

The arterial blood gas drawn before initiating face mask ventilation showed a pH ranging from 7.10 to 7.39, with a mean of 7.23, and Pco₂ ranging from 58 to 93 mm Hg, with a mean of 82.5. Based on their clinical or physiologic status, all would have required intubation and mechanical ventilation. The single patient with a normal pH (patient 5) was in severe distress, and it was thought that decompensation was imminent. Two patients (3 and 6) expressed the will not to be intubated.

Pressure control ranging from 20 to 30 cm H₂O delivered a TV from 340 to 790 ml with a VE between

Table 2—Pulmonary Function Testing in Patients with COPD*

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>FVC, ml (%)</th>
<th>FEV₁, ml (%)</th>
<th>FEV₁/FVC, (%)</th>
<th>Dsb, ml/min/mm Hg (%)</th>
<th>RV/TLC, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>1†</td>
<td>1,700 (52)</td>
<td>900 (36)</td>
<td>53</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>2</td>
<td>945 (40)</td>
<td>591 (37)</td>
<td>62</td>
<td>NA</td>
<td>65</td>
</tr>
<tr>
<td>3†</td>
<td>1,031 (23)</td>
<td>486 (14)</td>
<td>47</td>
<td>9.8 (32)</td>
<td>75</td>
</tr>
<tr>
<td>4</td>
<td>1,592 (38)‡</td>
<td>694 (24)</td>
<td>44</td>
<td>9.3 (32)</td>
<td>68</td>
</tr>
<tr>
<td>5</td>
<td>760 (30)</td>
<td>430 (23)</td>
<td>56</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>6</td>
<td>1,074 (38)</td>
<td>752 (36)</td>
<td>70</td>
<td>10.3 (41)</td>
<td>63</td>
</tr>
</tbody>
</table>

*NA = not available.
†PFT obtained 10 years before this admission.
‡15% increase in FVC following inhaled bronchodilator.

Face Mask Ventilation in Patients with ARF (Medurl et al)
6.3 and 9.1. Acceptance of the face mask was excellent, and immediate improvement in the sensation of dyspnea was recorded in four patients.

Clinical and physiologic improvements were seen shortly after beginning face mask ventilation (eg, a mean fall in respiratory rate of 18 breaths/min). An arterial blood gas determination within 1 h of initiating face masks ventilation showed a mean fall in Pco2 of 18 mm Hg (Fig 1). By the sixth hour, the Pco2 had fallen by 24 mm Hg and pH had risen by 0.145. Face mask ventilation lasted from 3 to 88 h (mean, 31 h). The mask was removed for brief periods of rest for the patients to receive nebulizer treatment with a beta-agonist agent, to drink water, to expectorate secretions, or to speak.

Patients 1, 5, and 6 had an excellent response and an uncomplicated course. The death of two patients (3 and 4) was not due to a failure of face mask ventilation. Patient 3, a 70-year-old man with severe COPD, was a chronic CO2 retainer who received oxygen therapy at home and had a history of congestive heart failure. After successful resuscitation at home following cardiopulmonary arrest, he was admitted intubated. The following day the patient self-extubated and expressed the will not to be reintubated. Twenty-four hours later, he developed severe respiratory difficulties and acidosis. For four days he tolerated face mask ventilation well and showed clinical and physiologic improvement. On day 5, after several unsuccessful weaning trials, he decided not to continue the therapy; 15 hours later he died.

Patient 4, a 68-year-old man with severe COPD, was receiving nocturnal oxygen at home and had a history of coronary artery disease. He was admitted with an acute exacerbation and theophylline intoxication (µg/ml level 51.5). Two days after entering the hospital, he developed severe respiratory difficulties and lethargy. Arterial pH fell from 7.36 to 7.22 (respiratory rate, 13 breaths/min). He responded quickly and well to face mask ventilation with clinical and physiologic improvement. Sixteen hours later he developed acute bradycardia and electromechanical dissociation. He was intubated but failed to respond to resuscitation efforts. An arterial blood gas analysis 30 min before the arrest showed a pH of 7.29, Pco2 of 66 mm Hg, and Po2 of 69 mm Hg. Vital signs and oximetry results before the event were satisfactory. (Postmortem study limited to the heart showed a myocardial infarction.)

One other patient (2) required intubation after she developed generalized seizure, which is thought to be secondary to hyponatremia and acute respiratory alkalois in patients with a previous history of seizure. Arterial pH rose from 7.25 to 7.50 with face mask ventilation and did not correct itself despite a decrease in ventilatory rate.

**Hypoxemic Respiratory Failure**

The second group consisted of four patients (patients 7 to 10) with acute hypoxemic respiratory failure, two with adult respiratory distress syndrome (ARDS) and two with cardiogenic pulmonary edema. Patient 7 had noncardiogenic pulmonary edema from sepsis. She was neutropenic and thrombocytopenic 10 days following chemotherapy for Wilms’ tumor; the source of infection was not identified. Patient 8 had AIDS and developed noncardiogenic pulmonary edema following an open lung biopsy examination that diagnosed the presence of *Pneumocystis carinii* pneumonia. Patients 9 and 10 had acute cardiogenic pulmonary edema. Myocardial infarction was suspected in one and diagnosed in the other.

Their mean age was 40 years (range, 32 to 77), and the female-to-male ratio was 3:1. Three patients were in severe respiratory distress; patient 10 was described as in moderate distress. All four patients were tachy-
pneic, with a mean respiratory rate of 40 breaths/min (range, 34 to 50). The decision to begin mechanical ventilation in each patient was based on the following information:

Patient 7 had ventilatory fatigue with respiratory acidosis (PCO₂, 52 mm Hg; respiratory rate, 50 breaths/ min) and acute change in mental status (agitation and disorientation) while receiving CPAP of 18 cm H₂O by face mask.

Patient 8 had refractory hypoxemia (PO₂, 74 mm Hg on 90 percent FiO₂ and CPAP 10 cm H₂O) and clinical signs of imminent ventilatory decompensation with rising PCO₂.

Patient 9 had a marked increase in an effort to breathe soon after extubation and two days following cardiac arrest.

Patient 10 had resistant hypoxemia (PO₂, 73 mm Hg on 100 percent FiO₂ by nonbreathing face mask) and acute ventilatory decompensation (pH, 7.25; PCO₂ 50 mm Hg; respiratory rate, 34 breaths/min, using the accessory muscles of respiration).

The initial ventilatory settings in this group are given in Table 3. Patient 8 initially experienced a moderate degree of anxiety from having a tight-fitting mask; the anxiety slowly improved. Patient 9 tolerated the mask well initially but after 20 h became uncomfortable and was finally intubated, despite adequate oxygen tension (PO₂, 122 mm Hg on 40 percent FiO₂). This was the only patient who required intubation because of an inability to tolerate the face mask.

The mean PaO₂/FiO₂ before initiating face mask ventilation was 95 mm Hg and increased to 192 mm Hg at the first hour and to 261 mm Hg after 6 h, with a mean increase of 97 and 167 mm Hg, respectively (Fig 2). The mean duration of face mask ventilation was 36 h (range, 4 to 71). After initiating face mask ventilation, the respiratory rate halved in the subgroup with cardiogenic edema, from a mean of 38 to 19.

In the subgroup with ARDS, the respiratory rate did not change, which may have resulted from our inability to deliver a higher tidal volume or from intrinsic lung disease. Significant oxygen desaturation was seen in these two patients if the mask was removed for even a brief period, while they were receiving high positive end-expiratory pressure (PEEP).

The only observed complication was the development of the nasal bridge abrasion at the site of mask application in patients 7 and 8. The abrasion healed spontaneously in a few days.

**DISCUSSION**

Mechanical ventilation is a lifesaving support measure for patients with respiratory failure. Its primary purposes are to achieve adequate alveolar ventilation and to improve oxygen exchange. Traditionally, inserting an endotracheal tube is needed to deliver the mechanical tidal breath. Complications can result from the intubation procedure, either while the tube is in place or after extubation.¹¹,¹²

**PPO₂/FiO₂ in Four Patients With Hypoxemic Respiratory Failure**

![Figure 2. Change in PO₂/FiO₂ over time in the group with hypoxemic respiratory failure.](image-url)
Placement of a tube in the oral cavity and the inability of patients to verbalize are associated with a great degree of discomfort and anxiety that can necessitate the use of sedatives or paralyzing agents to control them. In recent years, new noninvasive modalities have been developed to improve alveolar ventilation and oxygenation in patients with respiratory failure. Patients with respiratory failure from neuromuscular disorders and without significant intrinsic lung disease have been successfully ventilated noninvasively through a face mask\(^1\) or a nose mask\(^2-10\) thereby avoiding or postponing for years the need for tracheostomy.

CPAP applied noninvasively through a face or nose mask has been used successfully to correct refractory hypoxemia in patients with cardiogenic\(12\) and noncardiogenic pulmonary edema.\(14,16\) Retention of CO\(_2\) has occasionally been a limiting factor for this technique, however.

Ten patients nonselectively entered this study. Six had severe COPD with acute hypercapnic respiratory failure and four had hypoxic respiratory failure from cardiogenic and noncardiogenic pulmonary edema. All patients met objective or clinical criteria indicating the need for mechanical ventilation. Face mask ventilation rapidly ameliorated dyspnea and reduced respiratory rates.

The use of a tight-fitting mask was well tolerated; only one patient stopped using the mask after 20 h because of discomfort. Only two patients developed abrasions on the nasal bridge, and they healed spontaneously; this was the only obvious complication noted in this group. A nasogastric tube was inserted in all patients before beginning face mask ventilation. This tube resulted in leaking air where the tube exited from the mask, but no patient developed abdominal distention secondary to aerophagia or gaseous insufflation, and no vomiting occurred. We did not observe obvious aspiration clinically or radiographically.

Three patients, lethargic from CO\(_2\) retention (patients 2, 4, and 6), became alert and oriented after beginning face mask ventilation. One patient (7) with refractory hypoxemia was acutely agitated and disoriented before treatment and became comfortable and oriented soon after beginning ventilation.

The mean duration of face mask ventilation was 33 h (range, 3 to 88 h). No patient left the study because of failure to achieve adequate oxygenation or ventilation. Three patients eventually required endotracheal intubation: patient 2 after generalized seizures, patient 4 during cardiopulmonary resuscitation, and patient 9 because of discomfort from the tight-fitting mask. Two patients died. Patient 3 refused to continue after four days of successful ventilation and died 15 h after discontinuing ventilation. Patient 4 had a sudden, unexpected cardiac arrest, and an autopsy showed a myocardial infarction.

The physiologic response to face mask ventilation was considered satisfactory in all patients (Fig 3). In the hypercapnic group, mean PCO\(_2\) was reduced by 18 and 24 mm Hg at the first and sixth hours of ventilation, respectively, while the mean respiratory rate fell by 18 breaths/min. Patients also experienced a marked amelioration of dyspnea and decreased effort to breathe. Overcorrection of respiratory acidosis (from pH 7.25 to 7.50) was, in part, responsible for the development of generalized seizures in patient 2, who had hyponatremia.

The patients with COPD and hypercapnia had the advantage of being able to remove the mask for brief periods (10 to 15 min) during which they could drink fluids, verbalize, receive nebulized \(\beta\)-agonist agents, or expectorate secretions. Weaning from face mask ventilation was achieved by prolonging the periods off the mask, while the patients received additional FIO\(_2\) by nasal cannula or a Venturi mask. Noninvasive face mask mechanical ventilation was successful in achieving adequate alveolar ventilation and correcting the respiratory acidemia (mean rise in pH at 6 h was 0.145) while allowing the respiratory muscles to rest. Compared with mechanical ventilation delivered via an endotracheal tube, face mask ventilation had the advantage of being noninvasive and more comfortable to the patients. It can be used also in an earlier stage of ventilatory decompensation while medical management is initiated. Because of the risk of aspiration, we think that face mask ventilation should not be continued in patients who are lethargic and whose mental status fails to improve after beginning ventilation.

In the group with hypoxemia and low lung compliance, face mask ventilation satisfactorily improved gas exchange and effective alveolar ventilation. Patients 7 and 8 had ARDS with refractory hypoxemia and ventilatory insufficiency (tachypnea and P\(_{CO_2}\) retention) while receiving CPAP delivered by a face mask. In patient 7, tachypnea and the sensation of dyspnea

![Figure 3. View of the face mask and ventilator.](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21592/ on 04/01/2017)
were corrected by instituting face mask ventilation; however, the PaO₂:FiO₂ ratio did not improve for 24 h. Patient S experienced a rapid improvement in oxygen exchange that permitted a reduction in FiO₂ from 0.9 to 0.5 in 13 h, while the PEEP remained constant at 10 cm H₂O. A significant and rapid improvement in PaO₂:FiO₂ ratio was also seen in the two patients with cardiogenic pulmonary edema.

Face mask ventilation was used in a small heterogeneous group of patients with acute respiratory failure due to intrinsic lung disease. This procedure appears to produce physiologic improvements similar to those in intubated patients receiving mechanical ventilation. The mask was well accepted and tolerated for prolonged periods of time without significant complications. While this preliminary report on a new mode of noninvasive mechanical ventilation is encouraging, a much larger study is necessary before firm conclusions and recommendations can be reached.

ACKNOWLEDGMENT: The authors wish to thank the respiratory therapists and the Pulmonary attendings of Norwalk Hospital for their dedication and enthusiasm that have made this study possible. We would like to acknowledge the assistance of Dr. Norman Sooel and Dr. David Armbruster in editing the manuscript, Nancy Smith and Vicky Franke for secretarial support, and Denis Selmont, PhD, for organizing Tables 1-3.

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