A Novel Form of Manually Assisted Ventilation*

Andrew C. Stone, MD; Sheila Nolan; Muhanned Abu-Hijelhia, MD; Dennis McCool, MD; and Nicholas S. Hill, MD, FCCP

We report an individual with limb-girdle muscular dystrophy who has devised a way to assist her respiration by using her hands braced against the tray of her wheelchair. Utilizing this method, she was able to increase her tidal volume (Vt) and lower her respiratory rate compared to unassisted spontaneous breathing, thereby maintaining a stable minute volume. The manually assisted Vt measurements were comparable to those achieved using an intermittent abdominal pressure respirator (pneumatic belt). We believe that others with neuromuscular syndromes could use this technique, possibly decreasing their dependence on mechanical ventilatory assist devices. (CHEST 2003; 123:949–952)

Key words: Duchenne muscular dystrophy; neuromuscular disease; noninvasive ventilation; respiratory failure; ventilatory assistance

Numerous mechanical forms of ventilatory assistance have been devised for patients with neuromuscular diseases, including negative pressure ventilation, intermittent abdominal pressure ventilation (pneumatic belt), the rocking bed, and, currently the most commonly used, noninvasive positive pressure ventilation. However, few noninvasive techniques, other than glossopharyngeal breathing, have been developed that permit independence from mechanical devices. We report here a novel way for patients with respiratory muscle weakness to assist their ventilation without requiring a mechanical device.

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**Case Report**

A 53-year-old woman with severe quadripareisia due to limb-girdle muscular dystrophy was seen in her pulmonologist’s office for routine follow-up. During physical examination, the patient became more dyspneic when the tray of her wheelchair was removed. She stated that she used the tray to help her breathe. She was observed to apply pressure to her mid-epigastrium with the fingers of her left hand during exhalation, pushing on the back of her left hand with her right hand, and using the wheelchair tray as a brace (Fig 1). The fingers of the left hand were fixed in hyperextension, and both hands and wrists were severely weakened.

She first acquired symptomatic respiratory insufficiency in 1984 when her PacO2 was 48 mm Hg and she responded favorably to jacket negative pressure ventilation. In 1989, she switched to nasal positive pressure ventilation using a Knightstar 335 ventilator (Puritan Bennett; Carlsbad, CA) because of convenience and portability advantages. She uses the nasal ventilator for 10 h nightly and 1 h or 2 h during the afternoon at an inspiratory pressure of 23 cm H2O, expiratory pressure of 4 cm H2O, and respiratory rate (RR) of 20 breaths/min. During the daytime, she uses her wheelchair and performs hand-assisted ventilation for 10 to 12 h/d. Her most recent daytime room air arterial blood gas levels are pH 7.31; PacO2, 54 mm Hg; and PaO2, 72 mm Hg (breathing spontaneously without hand-assisted ventilation). Pulmonary functions are FVC of 0.58 L and FEV1 of 0.49 L (both 19% predicted).

A daytime study was performed to assess the efficacy of the manually assisted ventilation technique compared to spontaneous breathing and assisted ventilation using a pneumatic belt at various pressures. A mouthpiece attached to a pneumotachometer was fitted into the patient’s mouth to measure airflow while the nose was pinched and the flow signal was integrated for recording tidal volume (Vt). A lightweight portable magnetometer device (Enertech Consultants; Campbell, CA) was used to measure anterior-posterior displacements of the rib cage and abdomen, as well as the axial displacement of the chest wall, as described previously.

**Results**

As shown in Table 1, the patient’s RR decreased, Vt increased, and minute ventilation (Ve) slightly increased during manually assisted breathing as compared to spontaneous unassisted breathing. When compared to assisted breathing using the pneumatic belt, manually assisted breathing augmented Vt approximately to the same degree as a belt inflation pressure of 60 cm H2O. It was possible to achieve greater Vt readings with the pneumatic belt than with manually assisted breathing, but only at very high inflation pressures (Table 1). The duty cycle (inspiratory time [T]/total breathing cycle time) was unchanged by hand-assisted breathing compared to spontaneous breathing, but was shortened by use of the pneumatic belt, related to setting of the inspiratory/expiratory ratio at 1.5:1 on the PLV 102 ventilator (Respironics;
Murrysville, PA) that was used to power it. Hand-assisted breathing increased inspiratory flow rate ($V_t/T_i$) compared to spontaneous breathing (Table 1).

The patient subsequently tried pneumatic belt ventilation for several months with the supposition that she could use it to relieve hand fatigue. However, she rejected it because it was less comfortable and strapping the belt on was a nuisance.

Figure 2 provides graphic recordings of the excursions of the rib cage and abdomen and $V_t$s achieved during unassisted breathing, hand-assisted breathing, and with different levels of pressure using the pneumatic belt. During unassisted spontaneous breathing, the chest wall and abdominal excursions were synchronous, although the abdominal excursion was barely perceptible. During hand-assisted breathing, $V_t$s were augmented while the chest wall and abdomen moved synchronously. With use of the pneumatic belt, the chest wall moved paradoxically, expanding while the abdomen was compressed.

**DISCUSSION**

We describe a nonmechanical technique of self-assisted breathing that has not been previously reported, developed *de novo* by a patient with neuromuscular disease. The technique utilizes intermittent abdominal thrusts of the hands, braced against a tray attached to a wheelchair. In this way, the technique resembles repeated Heimlich maneuvers, although much less forceful. Although we did not measure esophageal or gastric pressures directly to confirm the mechanism, we presume that by applying force to the mid-epigastrium during expiration, the hands increase abdominal pressure, raise the diaphragm, increase intrathoracic pressure, and actively assist expira-

<table>
<thead>
<tr>
<th>Variables</th>
<th>RR, breaths/min</th>
<th>$V_t$, mL</th>
<th>$V_t$, L/min</th>
<th>$T/T_{tot}$</th>
<th>$V_t/T_i$, mL/s</th>
</tr>
</thead>
<tbody>
<tr>
<td>Spontaneous breathing†</td>
<td>$31 \pm 1</td>
<td></td>
<td>$</td>
<td>$177 \pm 9</td>
<td></td>
</tr>
<tr>
<td>Hand-assisted ventilation†</td>
<td>$22 \pm 1$</td>
<td>$204 \pm 11$</td>
<td>$5.81 \pm 0.29$</td>
<td>$0.53 \pm 0.01$</td>
<td>$225 \pm 12$</td>
</tr>
<tr>
<td>Pneumatic belt pressure, cm H$_2$O‡</td>
<td>40</td>
<td>$23 \pm 1$</td>
<td>$236 \pm 6$</td>
<td>$5.43 \pm 0.24$</td>
<td>$0.40 \pm 0.01</td>
</tr>
<tr>
<td></td>
<td>60</td>
<td>$22 \pm 1$</td>
<td>$291 \pm 9$</td>
<td>$6.40 \pm 0.32$</td>
<td>$0.41 \pm 0.05</td>
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<tr>
<td></td>
<td>80</td>
<td>$20 \pm 1$</td>
<td>$312 \pm 2</td>
<td></td>
<td>$</td>
</tr>
</tbody>
</table>

*Data are presented as mean ± SE. $T_{tot} =$ total breathing cycle time.
†Average of 12 breaths.
‡Average of five breaths.
§$V_t$ derived from the RR and the mean $V_t$.
¶$p < 0.05$ compared to hand-assisted breathing (by analysis of variance).
tion. When the hands relax during inspiration, abdominal pressure falls and gravity pulls the diaphragm downward, assisting inspiration. This mechanism is similar to that of the pneumatic belt, but there is an important difference. As shown by the magnetometry tracings in Figure 2, the chest wall and abdomen move synchronously during abdominal compression by the hands, but paradoxically during use of the pneumatic belt. This indicates that use of the hands truly assists ventilation, whereas the pneumatic belt controls it. When the patient exhales, her hands apply pressure to the mid-epigastrium, forcing the diaphragm into the thorax while the chest wall deflates. The hand force is timed to synchronize with exhalation and does not increase intrathoracic pressure enough to expand the chest wall. By contrast, bladder inflation with the pneumatic belt is not synchronized with the patient’s spontaneous breathing and is quite forceful. The abdominal compression is much greater than that achieved with the hands, increasing intrathoracic pressure more and causing the chest wall to expand and move paradoxically during exhalation. Because there is no paradoxical chest wall motion, manual ventilation must be more efficient than the pneumatic belt. Also, because it uses less force and allows the patient to determine the breathing pattern, the patient senses it as more comfortable.

Although the mechanisms of manual and pneumatic belt ventilation have some differences, there are also similarities, and both are likely to be subject to the same limitations. As with the pneumatic belt, gravity assists inspiration by returning the diaphragm to its resting position, so the technique should be used in an upright position of at least 30°. Also, although our patient demonstrates that only minimal hand strength is required, the technique requires that the hands be braced against an anchored object such as a wheelchair tray. Furthermore, similar to the pneumatic belt, it is likely that the technique works best in the presence of significant diaphragmatic weakness. The markedly diminished abdominal excursions in our patient during spontaneous unassisted breathing suggest that her diaphragm dysfunction is severe.

The potential importance of this technique is that it can afford patients with chronic respiratory failure due to neuromuscular disease more independence from mechanical ventilation, mainly during wheelchair use. Also, unlike glossopharyngeal breathing, another nonmechanical technique that achieves the same end, the manually assisted technique frees the upper airway. Finally, because the technique is simple in concept, it might be taught to other similar patients.

**References**

2. Bach JR, Alba AS, Bodofsky E, et al. Glossopharyngeal breathing and noninvasive aids in the management of post-
Pleural Effusion Caused by Prostaglandin E₁ Preparation*

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We encountered a case of bilateral pleural effusion associated with prostaglandin E₁ (PGE₁) preparation. A 75-year-old man underwent replacement surgery for an amputated hand. PGE₁ was administered at 120 μg/d to maintain circulation after vascularization. From day 7 of administration, respiratory distress developed. On day 12, pleural effusion was observed bilaterally. By discontinuing PGE₁ and improving hypoalbuminemia, pleural effusion resolved rapidly. This is the first case report of PGE₁-induced pleural effusion; like other drug-induced pleural effusions, discontinuing the drug resulted in rapid improvement. Although a rare complication, pleural effusion has to be suspected when a patient receiving PGE₁ experiences difficulty with breathing.

(CHEST 2003; 123:952–953)

Key words: hypoalbuminemia; microsurgery; pleural effusion; prostaglandin E₁

Abbreviations: LDH = lactate dehydrogenase; PGE₁ = prostaglandin E₁

Prostaglandin E₁ (PGE₁) preparation is used clinically to achieve vascular patency in occlusive arterial diseases and to maintain vascular patency until surgery in pediatric cardiac cases. In Japan, this preparation is also used in reattachment surgeries to maintain circulation after revascularization.

A well-known adverse drug reaction of PGE₁ preparations is angiitis. We report the first case of pleural effusion considered to be caused by PGE₁ administration after reattachment surgery for hand amputation.

CASE PRESENTATION

A 75-year-old man had his hand caught in an agricultural machine while cutting straw, resulting in amputation of the palmar area of the right hand except a part of the soft tissues. Reattachment surgery was performed on the same day of the accident. The laboratory findings at presentation were total protein, 7.2 g/dL; albumin, 3.9 g/dL; aspartate aminotransferase, 41 U/L; alanine aminotransferase, 24 U/L; and hemoglobin, 12.6 g/dL. Mildly impaired liver function and mild anemia were observed. Cardiac function tests (ejection fraction, 68.4%; normal range, 58 to 89%) and respiratory function tests (FEV₁ percent predicted, 79.9%; normal range, ≥ 70%; FVC percent predicted, 92.4%; normal range, ≥ 80%) showed no abnormalities.

Administration of urokinase, 240,000 U/d; heparin, 20,000 U/d; and PGE₁, 120 μg/d was started immediately after surgery. Urokinase and heparin were discontinued on day 6 after surgery, and PGE₁ administration was continued until day 12.

From the seventh postoperative day, the patient started to have respiratory distress, which progressed gradually. A chest radiograph and CT conducted on the 12th day showed bilateral pleural effusion and pericardial effusion (Fig 1). Mild edema was also observed in the lower extremities.

Blood tests conducted at the same time showed total protein of 5.2 g/dL; albumin, 2.2 g/dL; and hemoglobin, 7.6 g/dL. Anemia and hypoalbuminemia were observed. Oxygen saturation measured with pulse oximetry was reduced to 93%.

The results of pleural effusion analysis were as follows: positive Rivalta reaction; carcinoembryonic antigen, 1.7 ng/mL; glucose, 113 mg/dL; total protein, 2.9 g/dL; lactate dehydrogenase (LDH), 129 U/L; total cholesterol, 31 mg/dL; and negative culture finding for acid-fast bacteria. Exudative pleural effusion was diagnosed according to Light’s criteria, in which exudative pleural effusion is diagnosed when at least one of the following is met: pleural fluid protein/serum protein > 0.5, pleural fluid LDH/serum LDH ratio > 0.6, and pleural fluid LDH more than two thirds of the normal upper limit for serum.

PGE₁ was discontinued on the 12th postoperative day when pleural effusion was detected. Oxygen was administered immediately together with transfusion of 4 U of RBC concentrate and 1 U of fresh frozen plasma. On the eighth day after PGE₁ was discontinued, pleural effusion disappeared and respiratory distress also improved (Fig 2).

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FIGURE 1. Plain chest CT at day 12 after initiation of PGE₁ administration. Bilateral pleural effusion is observed.