Effectiveness of mask and helmet interfaces to deliver noninvasive ventilation in a human model of resistive breathing

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Submitted 8 December 2004; accepted in final form 11 May 2005


Antonio Patessio, Claudio F. Donner, and V. Marco Ranieri. Effective-ness of mask and helmet interfaces to deliver noninvasive ventilation in a human model of resistive breathing. J Appl Physiol 99: 1262–1271, 2005. First published June 16, 2005; doi:10.1152/japplphysiol.01363.2004.—The helmet, a transparent latex-free polyvinyl chloride cylinder linked by a metallic ring to a soft collar that seals the helmet around the neck, has been recently proposed as an effective alternative to conventional face mask to deliver pressure support ventilation (PSV) during noninvasive ventilation in patients with acute respiratory failure. We tested the hypothesis that mechanical characteristics of the helmet (large internal volume and high compliance) might impair patient-ventilator interactions compared with standard face mask. Breathing pattern, CO₂ clearance, indexes of inspiratory muscle effort and patient-ventilator asynchrony, and dyspnea were measured at different levels of PSV delivered by face mask and helmet in six healthy volunteers before (load-off) and after (load-on) application of a linear resistor. During load-off, no differences in breathing pattern and inspiratory muscle effort were found. During load-on, the use of helmet to deliver pressure support increased inspiratory muscle effort and patient-ventilator asynchrony, worsened CO₂ clearance, and increased dyspnea compared with standard face mask. Autocycled breaths accounted for 12 and 25% of the total minute ventilation and for 10 and 23% of the total inspiratory muscle effort during mask and helmet PSV, respectively. We conclude that PSV delivered by helmet interface is less effective in unloading inspiratory muscles compared with PSV delivered by standard face mask. Other ventilatory assist modes should be tested to exploit to the most the potential benefits offered by the helmet.

Study Population

Six healthy volunteers (all male; 32 ± 11 yr) recruited among medical students or physicians were studied. None was accustomed to breathing with PSV and all were naive to the purpose of the study. The local ethics committee approved the study, and written, informed consent was obtained from all subjects.

Measurements

Flow was measured with heated pneumotachographs (Fleisch no. 2; Fleisch, Lausanne, Switzerland) connected to differential pressure transducers (Diff-Capp, ±1 cmH₂O, Special Instruments, Nordingen, Germany). Volume was calculated from the numerical integration of the flow signal. Pressures were measured with differential transducers (Digima-Clic, ±100 cmH₂O, Special Instruments). Changes in pleural and abdominal pressures were estimated from changes in esophageal (Pes) and gastric (Pga) pressures, respectively, by use of a two-balloon-tipped catheter system (Allegiance, Zutphen, The Nether-
The measuring equipment dead space was 51 ml.

A standard-length and -compliance ventilator circuit was placed between the mainstream capnometry cuvette and the side port Pao (Pao) were placed between the mask inlet and the Y-piece of the ventilator circuit. A pneumotachograph, a mainstream capnometer (Siemens SC 7000, Siemens Elema, Berlin, Germany). Inspiratory triggering was set at a sensitivity of 3 l/min; pressure rise time (RT) was set at 0 s; the threshold for stopping inspiration and initiate expiration was set at an inspiratory flow equal to 25% of the peak inspiratory flow. All experiments were conducted inspiratory and expiratory positive pressure swing synchronous with manual pressure of the shoulders with its armpit braces. A pressure transducer was connected via a side port at the helmet inspiratory access. A pneumotachograph was put in series between the pressure transducer and the inspiratory limb of the ventilator circuit. The expiratory limb of the ventilator circuit was connected to the helmet expiratory port. A baseline mechanical ventilation in the volume-controlled, constant-flow mode with a VT of 1 liter and a breathing frequency of 10 breaths/min was started. PEEP was set at 5 cmH2O to avoid helmet collapse and was maintained unchanged throughout the procedure. While inflation flow was kept at its baseline setting, end-inspiratory occlusions (EIO) were performed in single separate test breaths with different inflation volumes ranging from 0.25 to 1.75 liters (discrete steps of 0.25 liters each). Each EIO was maintained for 5 s. After each test breath, baseline ventilation was resumed. Inflation volumes were varied in random order and measurements were taken in triplicate for each volume. Plateau pressure measured during EIO and volume data were used to plot the pressure-volume relationship of the helmet (14). It is described by the linear equation

\[ \text{helmet pressure} = 0.42 + 19.18 \times \text{volume}; \quad r^2 = 0.999, \quad P < 0.0001 \]

with a slope (i.e., static elastance) of 19.2 cmH2O/l.

The inspiratory and expiratory lines of the ventilator circuit were connected to two dedicated ports placed on the right and left sides of the helmet. Rebreathing valves were not used, because of the fact that the helmet was coupled with an intensive care unit (ICU) ventilator equipped with separate inspiratory-expiratory lines and valves (Evita 4 ventilator). Inside the helmet the subjects wore a nose clip and breathed through a mouthpiece connected to a pneumotachograph, a mainstream capnometer cuvette, and a side port connector for Pao measurement. In the load-on condition, the resistor was connected between the mainstream capnometer cuvette and the side port Pao connector. A standard-length and -compliance ventilator circuit was placed between the mainstream capnometry cuvette and the side port Pao (Pao) were placed between the mask inlet and the Y-piece of the ventilator circuit. A pneumotachograph, a mainstream capnometer (Siemens SC 7000, Siemens Elema, Berlin, Germany). Inspiratory triggering was set at a sensitivity of 3 l/min; pressure rise time (RT) was set at 0 s; the threshold for stopping inspiration and initiate expiration was set at an inspiratory flow equal to 25% of the peak inspiratory flow. All experiments were conducted inspiratory and expiratory positive pressure swing synchronous with manual pressure of the shoulders with its armpit braces. A pressure transducer was connected via a side port at the helmet inspiratory access. A pneumotachograph was put in series between the pressure transducer and the inspiratory limb of the ventilator circuit. The expiratory limb of the ventilator circuit was connected to the helmet expiratory port. A baseline mechanical ventilation in the volume-controlled, constant-flow mode with a VT of 1 liter and a breathing frequency of 10 breaths/min was started. PEEP was set at 5 cmH2O to avoid helmet collapse and was maintained unchanged throughout the procedure. While inflation flow was kept at its baseline setting, end-inspiratory occlusions (EIO) were performed in single separate test breaths with different inflation volumes ranging from 0.25 to 1.75 liters (discrete steps of 0.25 liters each). Each EIO was maintained for 5 s. After each test breath, baseline ventilation was resumed. Inflation volumes were varied in random order and measurements were taken in triplicate for each volume. Plateau pressure measured during EIO and volume data were used to plot the pressure-volume relationship of the helmet (14). It is described by the linear equation

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pressure were also measured proximally to the inspiratory port of the helmet. Flow was not measured distally to the expiratory port of the helmet. This fact precluded the quantitative estimation of leaks through the helmet. They were assessed qualitatively as previously reported (10). Briefly, the physician in charge passed his or her hands around the collar of the helmet to check leaks. Care was taken to minimize them by readjustment of the helmet (1). Other interventions such as the change of the helmet size to a smaller one (1) was never required.

The same equipment used during helmet ventilation (nose clip, mouthpiece, pneumotachograph, mainstream capnometry, and Pao side port connector) was used during 15–20 min of unsupported spontaneous breathing during load-on and load-off conditions.

Intensity of breathlessness was rated with a dyspnea visual analog scale (VAS) at the end of each experimental condition (18). Briefly, subjects were asked to place a vertical mark on a printed 100-mm horizontal scale in response to the question: “How short of breath are you right now?” The line had descriptors below the extreme ends. On the left there was the word “none,” indicating no shortness of breath, and on the right there was the opposite response, “extremely severe.” For each experimental condition, the subjects placed a vertical mark on the line in the spatial position that best represented the intensity of their dyspnea. Intensity was measured as the distance in millimeters from the left side of the horizontal line (corresponding to no dyspnea) to the mark placed by the patient. A fresh scale was presented each time these measurements of breathing comfort were assessed. Before starting the protocol, directions for using the scale were read, and all subjects then practiced marking the scale (18).

Unsupported breathing and each level of PSV lasted 15–20 min in all experimental conditions (mask PSV, helmet PSV, load-off, load on). Measurements were obtained in the last 5 min of each condition, once a stable breathing pattern was observed with air leaks kept to a minimum. The sequence of experimental conditions was random.

Data Analysis

Inspiratory time (TI), expiratory time, total breathing cycle time, and respiratory rate (RRflow) were determined from the flow tracing. Minute ventilation (Vt) was calculated as Vt multiplied by RRflow. Transpulmonary and transdiaphragmatic (Pdi) pressures were obtained by subtracting Pes from Pao and Pga, respectively. Pdi tracing was used to measure the subject’s respiratory rate (central respiratory rate, RRcentral), whereas Pao tracing was used to measure the rate of inspiratory positive pressure boosts delivered by the ventilator (RRPao). The pressure-time product for the diaphragm (PTPdi) was computed for each breath (PTPdi/b) by measuring the area under the Pdi tracing from the beginning of the inspiratory positive deflection to the end of inspiratory flow (38). PTPdi was also computed over 1 min (PTPdi/min) multiplying PTPdi/b by RRcentral. Finally, the diaphragmatic energy expenditure for each liter of Vt produced (PTPdi/l) was determined as mean values of the 5-min continuous recording collected at the end of each experimental condition.

Uncoupling between inspiratory Pdi and ventilator-delivered breath was identified by estimating the following variables: 1) autocycled breaths, defined as ventilator-delivered breaths occurring in the absence of a positive deflection in Pdi and expressed as percent of the total respiratory rate computed on the flow tracing; 2) ineffective inspiratory efforts, defined as a positive deflection in Pdi occurring during expiration that failed to trigger an inspiratory positive pressure boost delivered by the ventilator and expressed as percent of RRcentral (28); 3) multiple inspiratory positive pressure boosts occurring within the same inspiratory effort (Fig. 2) and expressed as percent of both RRflow and RRcentral (6, 7); 4) inspiratory trigger delay (De,insp), defined as the time elapsed from the onset of the positive inspiratory deflection of Pdi and the start of the positive swing of airway opening pressure (interval A-B in Fig. 3) (27); 5) synchronization between neural and ventilator Ti (ΔTi), defined as the time difference between the end of the inspiratory flow at the airway opening and peak inspiratory Pdi. This last was considered an index of neural inspiratory time termination (34); 6) expiratory trigger delay (De,exp), defined as the time elapsed from the beginning of Pao decay and the instant in which inspiratory flow reached the threshold for termination of the inspiratory pressure assistance (interval D–E in Fig. 3) (6, 7); and 7) pressure RT, defined as the time required to reach the set PSV level, starting from the beginning of positive Pao rise (interval B-C in Fig. 3) (11).

Lung elastance (Ei.) and resistance at midinspiratory volume (Ri.) were computed from transpulmonary pressure, flow, and volume recordings as previously described (4). During the load-on condition, respiratory mechanics estimates included the resistor. Dynamic intrinsic PEEP (PEEPi, dyn) was measured as the increase in Pdi preceding the start of inspiratory flow (3, 4, 41).

Inspiratory CO2 was measured by inspection of the capnogram as the CO2 inspiratory plateau value. ETCO2 was assessed as the highest CO2 expiratory value measured on the capnogram.

Statistical Analysis

Results are expressed as means ± SD. Values obtained during the different experimental conditions were compared by two-way ANOVA for repeated measures and Bonferroni/Dunn post hoc test. Linear regression analysis was performed by means of the least squares method. Significance was set at P < 0.05 (StatView, software package; Abacus, Berkeley, CA).
RESULTS

Leaks

During mask ventilation, differences between inspired and expired Vt were on average 17% (17 ± 3%), suggesting the presence of mild air leaks. During helmet ventilation, leaks were not quantitatively assessed (see MATERIALS AND METHODS), but they were always judged at least comparable if not smaller with those obtained with the mask.

Respiratory Mechanics

Figure 4 shows respiratory mechanics during unsupported spontaneous breathing in the load-off (left) and load-on (right) conditions. Et was unchanged by application of the resistor, whereas a tenfold increase in Rt and the presence of PEEPi,dyn were observed during the load-on conditions. The application of the resistor generated some levels of PEEPi,dyn, during both helmet PSV (1.89 ± 0.92 cmH2O) and mask PSV (1.64 ± 0.82 cmH2O) PSV without differences between interfaces and levels of assistance. Et and Rt did not differ between mask and helmet ventilation during all experimental conditions. The application of the resistor increased PTPdi/min from 134.0 (25.9 to 308.6 (38.0 cmH2O)/min (P < 0.05). These values are close to those found in patients with chronic obstructive pulmonary disease (COPD) requiring mechanical ventilation because of acute exacerbation (35).

Breathing Pattern

In the load-off condition, the breathing pattern did not change significantly at any level of PSV applied through the helmet and the mask (Table 1).

In the load-on condition, Vt with 10 and 15 cmH2O of PSV was higher with the helmet than with face mask (P < 0.05) (Table 1). No significant difference was found in RRcentral between helmet and mask ventilation at any level of PSV; however, whereas RRpao, RRflow, and RRcentral were similar during mask PSV, RRpao was systematically greater (P < 0.05) than RRflow and RRcentral during helmet PSV.

Both inspired Pco2 (PiCO2) and ETCO2, measured after application of the resistor, were significantly higher during helmet ventilation compared with mask ventilation at all PSV levels (Table 1).

Uncoupling Between Effort and Ventilator-Delivered Breath

Autocycled breaths were found at any PSV level during both unloaded and resistive breathing, irrespective of the interface used. Even if the percent of autocycled breaths was on average twofold during helmet compared with face mask ventilation, it did not significantly differ between interfaces with or without the resistor. The effects of autocycled breaths on Ve are shown in Table 1 (Ve autocycled breaths). Autocycled breaths accounted for 12 and 25% of the total Ve during mask and helmet PSV, respectively. The fraction of Ve related to autocycled breaths was threefold in the load-off compared with the load-on condition, irrespective of the interface used. No ineffective efforts occurred throughout the procedure.

Values of Del,insp were significantly higher during helmet ventilation than those during mask ventilation at PSV of 10 and 15 cmH2O regardless the application of resistor (Fig. 5A). Del,exp was nearly undetectable during mask ventilation in all

Fig. 3. Experimental record illustrating minor subject-ventilator asynchronies during the inspiratory phase. From top to bottom: flow, Pao, Pdi, and external flow (Flow,ext) tracings during helmet (left) and mask (right) ventilation with PSV of 5 cmH2O superimposed to a PEEP of 5 cmH2O in a representative subject. From left to right: line A, onset of the inspiratory effort; line B, start of positive Pao swing delivered by the ventilator; line C, point at which the pressure reaches its target preset pressure; line D, onset of end-inspiratory Pao decay; line E, point in which the inspiratory flow reaches 25% of peak inspiratory flow.

Fig. 4. Effects of application of the linear resistor on respiratory mechanics during spontaneous breathing. Et,dyn, dynamic lung elastance; Rt, inspiratory lung resistance (resistor included in the load-on condition); PEEPi,dyn, dynamic intrinsic PEEP; load-off, unloaded spontaneous breathing; load-on, resistive loaded spontaneous breathing. Results are expressed as means (SD). §P < 0.01, *P < 0.0001 load-on vs. load-off; ANOVA for repeated measures with Bonferroni/Dunn post hoc test.
Ptpdi/min was always within normal limits, i.e., lower than reported in Fig. 7 and Table 2. In the load-off condition, PTPdi/min during both load-off and load-on conditions are shown in Fig. 6. Average values of Respiratory Muscle Effort

Respiratory Muscle Effort

A representative record illustrating the inspiratory effort in the load-off condition is shown in Fig. 6. Average values of PTPdi/min during both load-off and load-on conditions are reported in Fig. 7 and Table 2. In the load-off condition, PTPdi/min was always within normal limits, i.e., lower than 125 cmH2O·s·min⁻¹ (17). In the load-on condition, increasing levels of PSV delivered by face mask brought the values of PTPdi/min close or below the normal value (Fig. 7). By contrast, PTPdi/min values during helmet ventilation were significantly higher than those found during mask PSV 5 and 10 cmH2O (P < 0.05, Fig. 7), because of both higher tidal Pdi swings and respiratory rate (Fig. 6); PTPdi/min approached the normal value only at PSV of 15 cmH2O (Fig. 7).

The effects of autocycled breaths on PTPdi/min shown in Table 2 (PTPdi/min autocycled breaths) tracked those on V̇E (see in Table 1). Autocycled breaths accounted for 10 and 23% of the total PTPdi/min during mask and helmet PSV, respectively. The fraction of PTPdi/min related to the autocycled breaths was more than threefold in the load-off compared with the load-on condition, irrespective of the interface used.

PTPdi/b and PTPdi/l measured during either helmet or face mask ventilation are shown in Table 2. They were not significantly different in the load-off as well as in the load-on condition, even if in this last condition the diaphragmatic energy expenditure per breath and per liter of V̇E was basically higher during helmet compared with mask ventilation at least at PSV of 5 and 10 cmH2O.

Expiratory muscle activity as detected by expiratory changes of Pga was trivial during unloaded breathing both during helmet and mask PSV (Table 2). By contrast, in the load-on condition expiratory changes of Pga showed little but significant increase during helmet compared with mask ventilation at any level of PSV (Table 2).

Intensity of Dyspnea

In the load-off condition, VAS score was low and stable with increasing level of support during mask PSV. In the load-on condition, it was increased twofold at PSV of 5
cmH₂O, but it decreased to the load-off levels at PSV of 10 and 15 cmH₂O. During helmet ventilation in the load-off condition VAS showed a trend to increase with increasing levels of PSV; in the load-on condition VAS score was twofold its load-off value at PSV of 5 cmH₂O and it increased with increasing levels of PSV (Fig. 8). These opposite trends resulted in a significantly higher VAS during helmet ventilation compared with mask ventilation irrespective of the load-off or the load-on condition (Fig. 8).

DISCUSSION

The present study shows that the helmet interface is less efficient than facial masks to deliver noninvasive PSV, particularly in conditions of increased workload. Our data suggest that the low elastance and high inner volume of the helmet caused a significant overdamp of pressure assistance and a deviation of ventilator-delivered flow from the patient. Moreover, the large inner volume of the helmet may predispose to rebreathing.

Breathing Pattern and CO₂ Washout

During load-off condition, the application of increasing levels of PSV resulted in a similar breathing pattern with either the helmet or the mask interfaces (Table 1). In the load-on condition, the response of the healthy subjects to the increased PICO₂ during helmet PSV was a rise in Vₜ higher during helmet than during mask PSV. However, despite such increase in Vₜ, end-tidal PCO₂ was significantly higher during helmet PSV than during mask PSV at all levels of support (P < 0.05) (Table 1). These data suggest that helmet PSV is less effective than mask PSV in removing CO₂ in a model of increased ventilatory requirement. This could be due to the high volume inside the helmet, which presumably induced CO₂ rebreathing (10).

Patroniti et al. (30) found that PICO₂ was higher during CPAP delivered by helmet compared with CPAP delivered by mask, and that it significantly decreased at increasing gas flow rates. During PSV such control on flow rate can be achieved by manipulating the pressurization rate and the level of the inspiratory pressure. In our study, the former was adjusted to its maximum value, and three increasing levels of PSV were tested. Nevertheless, the PICO₂ measured after application of the resistor was significantly higher during helmet PSV than during to mask PSV at any level of support. Furthermore, no differences in PICO₂ were found by increasing the helmet PSV from 5 to 15 cmH₂O (Table 1).

Patient-Ventilator Interactions

Peculiar mechanical characteristic of the helmet are a large inner volume and a high compliance (1, 2). Both are expected to cause a substantial dissipation of the inspiratory pressure delivered by the ventilator to expand the compliant helmet (in particular the soft collar) (1, 2). Our data confirm that a significant portion of the inspiratory flow is dissipated to expand the helmet (Fig. 3). Because the algorithms regulating the beginning and the end of PSV are pressure and flow based, an influence of the helmet characteristics on the ventilator...
justifies the multiple inspiratory pressure boosts occurring or follow the termination of subjects’ inspiratory effort and finding that termination of ventilator support could anticipate tics and muscle effort (Fig. 3 and 5). Such helmet-controlled changes in flow caused by the subject mechanical characteristics of the helmet rather than by the controlled and cycled off by the changes in flow caused by the subject mechanical characteris-
tically increased as the level of support was increased during the inspiratory effort cycled only by the flow decay inside the helmet (Fig. 3).

Because the pressurization slope was constant and the helmet inflow prevailed on the subjects’ inspiratory flow, RT was longer during helmet PSV than with mask PSV and systematically increased as the level of support was increased during helmet PSV (Fig. 5).

Respiratory Muscle Effort

Inspiratory workload during the load-on condition was similar to those found in patients who required mechanical ventilation because of acute exacerbation of COPD (Fig. 7) (3). Mask PSV was effective in reducing the inspiratory effort to normal values (i.e., PTPdi/min < 125 cmH2O·s·min⁻¹) already at a PSV level of 10 cmH2O (Fig. 7) (3). By contrast, during helmet PSV PTPdi/min remained higher than in normal conditions, up to a PSV level of 15 cmH2O (Fig. 7).

triggering functions should therefore be expected. In fact we found that the helmet PSV was characterized by 1) a significant asynchrony between the beginning and the end of inspiratory support and the beginning and the end of patient inspiratory effort; 2) a relevant number of autocycled breaths that were on average twofold those found during mask ventilation (Table 1) and occurred threefold more frequently during the load-off condition, indicating poor trigger sensitivity during helmet ventilation in conditions of reduced neuromuscular drive; and 3) the fact that the duration of ventilator assistance was controlled and cycled off by the changes in flow caused by the mechanical characteristics of the helmet rather than by the changes in flow caused by the subject mechanical characteristics and muscle effort (Fig. 3 and 5). Such helmet-controlled inspiratory PSV therefore explains the presence of Del,exp, the finding that termination of ventilator support could anticipate or follow the termination of subjects’ inspiratory effort and justifies the multiple inspiratory pressure boosts occurring during the inspiratory effort cycled only by the flow decay inside the helmet (Fig. 3).

The large inner volume and the high compliance of the helmet may cause the overdamping of the inspiratory pressure rise (10). We estimated this phenomenon as the time required to reach the target PSV level set on the ventilator (RT). Because the pressurization slope was constant and the helmet inflow prevailed on the subjects’ inspiratory flow, RT was longer during helmet PSV than with mask PSV and systematically increased as the level of support was increased during helmet PSV (Fig. 5).

Table 2. Effects of different levels of helmet and face mask PSV on diaphragmatic muscle effort and on expiratory gastric pressure changes before and after application of the resistor

<table>
<thead>
<tr>
<th>PSV (cm H2O)</th>
<th>Load-off</th>
<th>Load-on</th>
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<tbody>
<tr>
<td>Helmet</td>
<td>Mask</td>
<td>Helmet</td>
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<tr>
<td>PTPdi/min</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5 cmH2O</td>
<td>62.5 (23.3)</td>
<td>87.9 (37.1)</td>
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<tr>
<td>10 cmH2O</td>
<td>18.0 (26.4)</td>
<td>6.6 (6.5)</td>
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<tr>
<td>15 cmH2O</td>
<td>4.5 (2.0)</td>
<td>6.1 (1.7)</td>
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<tr>
<td>PTPdi/b</td>
<td></td>
<td></td>
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<tr>
<td>5 cmH2O</td>
<td>0.1 (0.1)</td>
<td>0.0 (0.0)</td>
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<td>10 cmH2O</td>
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<td>15 cmH2O</td>
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<tr>
<td>PTPdi/l</td>
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<td>PTPdi/min auto-cycled breaths</td>
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<td>5 cmH2O</td>
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<td>PTPdi/l auto-cycled breaths</td>
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<tr>
<td>PTPdi/min exp</td>
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<tr>
<td>5 cmH2O</td>
<td>4.9 (2.0)</td>
<td>6.5 (2.8)</td>
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<td>10 cmH2O</td>
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Values are means (SD). PTPdi/min, pressure-time product per minute for the diaphragm; PTPdi/min auto-cycled breaths, pressure-time product per minute for the diaphragm computed for auto-cycled breaths only; PTPdi/b, pressure-time product per breath for the diaphragm; PTPdi/l, pressure-time product per liter of minute ventilation for the diaphragm; ΔPgaexp, positive gastric pressure change during expiration. *P < 0.05, mask vs. helmet; †P < 0.01, mask vs. helmet; §P < 0.005, mask vs. helmet, all by ANOVA for repeated measures with Bonferroni/Dunn post hoc test.

Fig. 7. Inspiratory muscle effort during load-off and load-on conditions during mask and helmet PSV. PTPdi/min, pressure-time product for the diaphragm computed over 1 min; continuous horizontal line, PTPdi/min average value during the unsupported load-on condition; dashed horizontal line, threshold of normality for PTPdi/min set at 125 cmH2O·s·min⁻¹. *P < 0.05 mask vs. helmet, ANOVA for repeated measures with Bonferroni/Dunn post hoc test.

Fig. 8. Dyspnea visual analog score (VAS) during load-off and load-on conditions during mask and helmet PSV. VAS load-off, VAS load-on; *P < 0.05 load-off vs. load-on; †P < 0.01, mask vs. helmet; §P < 0.001 helmet vs. mask; ANOVA for repeated measures with Bonferroni/Dunn post hoc test.
At least two mechanisms may explain this reduced effectiveness of helmet PSV in unloading the respiratory muscles during load-on condition. First, during helmet ventilation at all levels of PSV, subjects had to double \( V_E \) to maintain values of end-tidal \( P_{CO_2} \) similar to those observed during mask ventilation. Second, some portion of inspiratory effort was unassisted by the positive pressure boost delivered by the ventilator because of patient-ventilator uncoupling due to the presence of increased \( D_{el,insp} \) and \( D_{el,exp} \) and to a slower pressurization rate compared with mask PSV.

The fact that PTPdi/min and not PTPdi/breath and/or PTDi/l was significantly higher during helmet PSV compared with mask PSV suggests that increased \( V_E \), due to partial rebreathing in the former condition (see above), prevailed over asynchronies in determining the impaired PSV unloading effect on the inspiratory muscles.

Frequent autocycling during helmet PSV accounted for a considerable fraction of the increase in \( V_E \) observed in this condition (see above). This phenomenon could contribute to explain the poor relationship found between PTPdi/breath and PTPdi/l during helmet PSV. As shown in our study, inspiratory effort was associated with substantial levels of Pdi, suggesting that self-triggered ventilator breaths were “triggering” patients’ efforts.

A particular feature of the subject-ventilator asynchrony found in our study can explain totally or at least in part its poor influence on PTPdi/breath and PTPdi/l during helmet PSV. As a matter of fact, the ventilator \( T_I \) was randomly longer (overassistance with some \( V_T \) delivered without any effort) or shorter (underassistance with some \( V_T \) generated by the respiratory muscle effort alone) than neural \( T_I \). This was due to individual natural \( T_I \) variability in face of a quite fixed ventilator \( T_I \) that was markedly affected by the helmet characteristics. These two effects elided each other, ventilator overassistance being prevalent (see \( \Delta T_I \), Table 1) and compensating also for the ventilator underassistance because of both the long inspiratory delay time and the impaired pressurization rate present during helmet PSV (Fig. 5).

The presence of some \( CO_2 \) rebreathing should induce expiratory muscle activity (20). As a matter of fact, an increase of positive Pga swing was found during expiration in our normal subjects during helmet PSV, especially in the load-on condition, thus further increasing the overall respiratory muscle energy expenditure.

**Dyspnea**

Mask PSV significantly decreased the high levels of dyspnea score induced by loaded breathing (Fig. 8). During helmet PSV, VAS was similar to mask PSV only at PSV of 5 cmH\(_2\)O. However, the increase of PSV above this value caused significant increases of dyspnea score both in the load-off and the load-on conditions (Fig. 8).

Patient-ventilator asynchrony has been associated to dyspnea worsening (9). During helmet PSV, numbers of autocycled breaths and length of pressure RT increased from 5 to 15 cmH\(_2\)O of PSV irrespective of the presence or the absence of the resistor. By contrast, both numbers of autocycled breaths and the pressure RT were substantially stable during mask PSV. In particular, in the load-on condition, RT increased from 30% of the inspiratory time during helmet PSV of 5 cmH\(_2\)O to 34% and 57% of the inspiratory time during PSV of 10 and 15 cmH\(_2\)O, respectively, notwithstanding the progressive inspiratory muscle unloading (Fig. 7).

During helmet PSV, the subjects breathed through a mouth-piece connected with the measuring apparatus inside the helmet. This condition could have influenced VAS estimates. However, VAS score was similar at PSV of 5 cmH\(_2\)O both during helmet and mask ventilation, whereas it showed a different behavior in helmet vs. mask ventilation with increasing PSV levels (see above). Thus it is unlikely that differences in VAS depended on the positioning of the measuring apparatus.

**Clinical Implications**

A recent study tested the use of helmet to deliver noninvasive pressure support in patients with acute exacerbation of COPD (2). The study showed that the helmet was as effective as a conventional mask in reducing the need for endotracheal intubation, with similar results on ICU and hospital mortality and length in ICU stay. Helmet NIV was also better tolerated. Both techniques achieved an improvement in arterial \( P_{O_2} \)-to-\( P_{CO_2} \) fraction of inspiratory \( O_2 \) ratio and a significant decrease in arterial \( P_{CO_2} \). However, the reduction in arterial \( P_{CO_2} \) was lower in the group of patients treated with the helmet. The authors speculated that this worse performance of helmet PSV could be due to a smaller reduction of inspiratory effort induced by partial dissipation of the inspiratory pressure due to the mechanical characteristics of the helmet. The authors concluded that, for these reasons, the helmet should be avoided in patients with severe conditions requiring a rapid increase of alveolar ventilation. The present study proposes partial rebreathing and impaired patient-ventilator interactions as the underlying mechanisms responsible for the reduced efficiency of helmet PSV in unloading the respiratory muscles in conditions of increased respiratory muscle workload.

The finding that increasing levels of helmet PSV generate a progressive reduction of the inspiratory effort could suggest that higher levels of PSV level may optimize helmet PSV efficiency. However, this opportunity is partly denied by the observation that progressive increase in helmet PSV level was associated to a parallel worsening of patient-ventilator synchrony and dyspnea sensation (Fig. 8).

It may be argued that the use of higher flows and higher pressures may reduce the compliance of the helmet and be associated with much better results, but on the other hand it may not be well tolerated by normal subjects. However, the flows used to deliver inspiratory support were already maximal, because we used in the protocol the fastest pressurization of the resistor. By contrast, both numbers of autocycled breaths and the pressure RT were substantially stable during mask PSV. In particular, in the load-on condition, RT increased from 30% of the inspiratory time during helmet PSV of 5 cmH\(_2\)O to 34% and 57% of the inspiratory time during PSV of 10 and 15 cmH\(_2\)O, respectively, notwithstanding the progressive inspiratory muscle unloading (Fig. 7).

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It may be argued that the use of higher flows and higher pressures may reduce the compliance of the helmet and be associated with much better results, but on the other hand it may not be well tolerated by normal subjects. However, the flows used to deliver inspiratory support were already maximal, because we used in the protocol the fastest pressurization rate available on the ventilator. Furthermore, the helmet volume-pressure relationship proved to be almost linear above 5 cmH\(_2\)O, precluding significant changes in helmet elastic properties with increasing the pressure. Finally, our data show that...
discomfort increased as the PSV level was raised only when the helmet was used as subject-ventilator interface, but not when the facial mask was used for the same purpose. In other words, ventilator constraints and mechanical properties of the helmet, but not the response of normal subjects to increasing levels of pressure assistance, precluded the hypothesis that such increase would improve the results obtained with the combination helmet + PSV.

Limitations of the Study

Our study simulated a condition of increased inspiratory resistive workload measured in terms of PTPd, such that the normal subjects had to generate inspiratory muscle efforts close to those spent by patients with acute respiratory failure. Furthermore, the trend of PTPd decrease with increasing PSV was similar to that found in the literature in patients with acute or chronic airway obstruction. In this connection, it has to be noted that in our protocol 5 cmH2O corresponded to 10 cmH2O of total pressure delivered during inspiration because of the presence of 5 cmH2O of PEEP, and so on for the other levels of PSV. Thus, at least on the inspiratory workload point of view, our normal subjects were not so dissimilar compared with COPD patients mechanically ventilated because an acute exacerbation.

In awake normal individuals flow-resistive loads cause decreased breathing frequency (13, 42). Similar results were found also in our study. This response appears to be useful in terms of minimization of the mechanical work of breathing (22, 29). By contrast, in awake COPD patients a decreased frequency of breathing is seldom seen, even in the face of markedly increased internal flow resistance (24). This implies that internal respiratory loading is not analogous to external loading (25). Even if the experimental conditions in naive subjects may poorly reflect the clinical conditions of patients with airflow obstruction, this does not discount the fact that PSV delivered through the helmet interface is not the best choice of partial ventilator assistance in these patients. As a matter of fact, at least a couple of reasons suggest that the mechanical limits shown by the combination of helmet with the PSV mode would further disadvantage COPD patients compared with normal subjects. First, increased V̇E showed by normal subjects during resistive breathing with helmet ventilation to maintain stable ETCO₂ would be curtailed, if not impossible, in patients with airflow limitation (42), resulting in even less effective CO₂ washout compared with our normal subjects breathing through an external resistive load. Second, we provide data suggesting that the duration and rate of the positive inspiratory pressure boosts are quite fixed during helmet PSV (RRp200, Table 1), depending on the mechanical properties of the helmet rather than to the subject’s control of breathing. This feature explains most of the asynchronies found in the present study. Such asynchronies would increase until complete dissociation between the subject or patient’s spontaneous breathing pattern and ventilator assistance, with increasing difference between the subject’s or patient’s and the ventilator’s respiratory rate and inspiratory time.

In the present study a nonlinear resistor was used. It can be argued that a very nonlinear pressure-flow relationship could amplify the effect of increasing V̇E observed during helmet ventilation. We believe this was not the case because 1) mean inspiratory flow was not significantly different between mask and helmet conditions and increases of V̇E were matched with parallel increases of the inspiratory time and of the duty cycle (32, 33); and 2) mean inspiratory flows were ~0.5 l/s (range 0.33, 0.59 l/s), a flow interval in which the pressure-flow relationship of the resistor employed is not too steep (average resistive pressure difference between helmet and mask conditions = 3 cmH2O, range 2.3–4.1 cmH2O) (43).

In conclusion, our data show that, given the mechanical characteristics of the helmet and the peculiar algorithms governing inspiratory and inspiratory-to-expiratory PSV triggers, the combination of the helmet with this latter mode of assisted ventilation induces significant patient-ventilator asynchrony, impairs efficacy of NIV to unload the respiratory muscles, and causes discomfort with increasing the inspiratory support. Decrease of the inner volume of the helmet, use of less compliant materials, and/or the use of other modes of pressure-assisted ventilation (e.g., assisted pressure-control ventilation, airway pressure release ventilation) should be evaluated.

ACKNOWLEDGMENTS

We thank Elisabetta Castella, Cristina Cirella, Massimiliano Coha, Carlotta Giacosa, Elena Gianotti, Marco Leopardi, and Alessandro Zito for contributions to the development of our research. We thank Robert Kacmarek for suggesting the study design.

GRANTS

The study has been supported by Università di Torino: Progetti di Ricerca Locali (grant PR60ANRA03)

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