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Effects of Intermittent Negative Pressure Ventilation on Effective Ventilation in Normal Awake Subjects*

Jean-Charles Glé rant, MD, PhD; Vincent Jounieaux, MD, PhD; Veronica F. Parreira, PhD; Myriam Dury; Geneviève Aubert, MD, PhD; and Daniel O. Rodenstein, MD, PhD

Rationale: Previous studies have shown that an increase in inspiratory pressure during nasal intermittent positive pressure ventilation (IPPV) does not result in increased effective minute ventilation (Ve) due to glottic interference.

Study objectives: To test the consequences of increases in negative pressure ventilation (NPV) on Ve.

Material and methods: Eight healthy awake subjects underwent NPV delivered by an iron lung. First, NPV was started at a respirator frequency (f) of 15 cycles per minute with an inspiratory negative pressure (INP) of −15 cm H2O (F15-P15). Then, f was increased to 20 cycles per minute and INP was kept at −15 cm H2O. Next, f was kept at 20 cycles per minute and INP was reduced to −30 cm H2O (F20-P30). Finally, f was decreased to 15 cycles per minute and INP was kept at −30 cm H2O. At each step and for each breath, effective tidal volume (VT), Ve, and end-tidal carbon dioxide pressure were measured. In three subjects, the glottis width was assessed using fiberoptic bronchoscopy.

Results: From spontaneous breathing to the first step of NPV (F15-P15), we observed an inhibition of the phasic inspiratory diaphragmatic electromyogram concomitant to a significant increase in Ve (p < 0.0005). For the group as a whole, the increase in mechanical ventilation (from F15-P15 to F20-P30) resulted in significant increases in VT and Ve leading to hypocapnia (p < 0.0005). Moreover, the glottis width did not decrease with the increase in mechanical ventilation.

Conclusions: We conclude that in normal awake subjects, NPV allowed a significant increase in Ve. These results differ from those previously obtained with nasal IPPV in which the glottis width interferes with the delivered mechanical ventilation.

Key words: control of breathing; glottis; negative pressure ventilation

Abbreviations: ARF = acute respiratory failure; EMGdi = electromyogram of the diaphragmatic muscle; EMGeso = diaphragmatic muscle electromyogram obtained through a bipolar esophageal electrode; EMGrect = filtered, rectified, and integrated diaphragmatic muscle electromyogram signal obtained through a bipolar esophageal electrode; f = respirator frequency; F15-P15 = respirator frequency of 15 cycles per minute with an inspiratory negative pressure of −15 cm H2O; F15-P30 = respirator frequency of 15 cycles per minute with an inspiratory negative pressure of −30 cm H2O; F20-P15 = respirator frequency of 20 cycles per minute with an inspiratory negative pressure of −15 cm H2O; F20-P30 = respirator frequency of 20 cycles per minute with an inspiratory negative pressure of −30 cm H2O; INP = inspiratory negative pressure; IPPV = intermittent positive pressure ventilation; NPV = negative pressure ventilation; PETCO2 = end-tidal carbon dioxide pressure; Ptank = iron lung pressure; Rinsp = inspiratory resistance; Sato2 = arterial oxygen saturation; SB = spontaneous breathing; T1 = inspiratory time; Ttot = total ventilatory cycle duration; V = air flow; Ve = minute ventilation; VT = tidal volume

Noninvasive assisted ventilation can be performed with respirators that deliver positive pressure ventilation into the airways through a nasal or face mask, or that apply intermittent negative pressure around the thorax and abdomen. Nasal intermittent positive1 pressure ventilation (IPPV), using volumetric or barometric ventilators, is used to treat respiratory failure in patients with neuromuscular disease or COPD.1–4 Regarding intermittent negative pres-
sure ventilation (NPV), results reported in several studies\(^5\textendash\textsuperscript{12}\) led to a decrease in its use in COPD patients. Indeed, these trials did not systematically show beneficial results of NPV.\(^5\textendash\textsuperscript{12}\) Moreover, NPV could be detrimental, as this mode of ventilation can induce supraglottic obstruction and glottic closure in both awake or asleep healthy subjects or in patients with neuromuscular respiratory failure.\(^13\textendash\textsuperscript{16}\)

However, studies have shown that the glottis can also interfere with the delivered mechanical ventilation during nasal IPPV.\(^17\) In awake or asleep normal subjects, our group has previously shown that, during nasal IPPV using volumetric ventilators, to achieve the absence of diaphragmatic muscle activity, an increase in mechanical ventilation resulted in a progressive glottic narrowing, leading to a decrease in actual tidal volume (\(V_t\)) and effective minute ventilation (\(V_e\)) \([ie, the V_t and V_e effectively reaching the lungs]\)^.\(^{18,19}\) When using barometric ventilators set in the controlled mode,\(^20\) the response of the glottis to the increases in inspiratory pressure was variable, so that effective \(V_e\) was less predictable than during nasal IPPV using volumetric ventilators. During nasal IPPV using barometric ventilators in the spontaneous mode, the glottis did not interfere with mechanical ventilation, but high levels of inspiratory pressure were required to obtain an effective \(V_e\) higher than during spontaneous unassisted ventilation.\(^21\)

Retrospective and uncontrolled studies\(^22\textendash\textsuperscript{26}\) have shown that NPV could be effective with a high rate of success and reduced requirement for endotracheal intubation in patients with COPD or neuromuscular disorders during acute respiratory failure (ARF). Thus, NPV might well result in significant increases in \(V_e\) during ARF.

The aim of this study was to investigate the effects of NPV on effective \(V_e\) in normal awake subjects. We hypothesize that despite the glottic obstruction previously reported, high levels of \(V_e\) and \(V_t\) can be obtained when increasing respiratory frequency (\(f\)) and inspiratory negative pressure (INP).

## Materials and Methods

### Subjects

Eight healthy medical students (four men and four women) were studied during NPV. They were 21 to 23 years of age (body mass index, 22.5 ± 1.7; range, 21.2 to 25.2) and were without evidence of cardiorespiratory diseases. All subjects were habituated to NPV during one or two previous training sessions when they learned to relax their respiratory muscles and perform the isovolume maneuvers used to calibrate the respiratory inductive plethysmograph.\(^27,28\) No precise explanation on the aim of this study was given to the subjects. The subjects gave written informed consent and received financial remuneration for their participation in the study. The protocol of this study was approved by the ethical committee of the hospital.

### Signal and Recording Equipment

The activity of the respiratory muscles was assessed through the electromyogram of the diaphragmatic muscle (EMGdi) obtained from surface electrodes placed on the chest around the fifth intercostal space between the anterior axillary and midclavicular lines. This signal was filtered between 30 Hz and 3,000 Hz, but was not rectified or integrated. A second but invasive EMGdi recording was obtained through a bipolar esophageal electrode (EMGeso) introduced through the right nostril into the esophagus and positioned to obtain the best possible phasic inspiratory signal when the subject breathed spontaneously. This EMGeso signal was filtered, rectified, and integrated (EMGrect).

The iron lung pressure (\(P_{\text{tank}}\)) was measured with a transducer (model 162 PC 01D; Micro Switch Division, Honeywell; Freeport, IL). Actual \(V_t\) was obtained by respiratory inductive plethysmography (Respiritace; Ambulatory Monitoring; Ardsley, NY) calibrated with the isovolume technique.\(^29,30\) The sum of the thorax and abdominal signals was calibrated against a water-sealed spirometer.

End-tidal carbon dioxide pressure (\(P_{\text{ETCO}_2}\)) was obtained from carbon dioxide recordings made with a catheter passing through a plastic hollow conical piece (Nasal adapter set; Datex; Helsinki, Finland) introduced in the left nostril so that the nostril was kept open and the extremity of the catheter remained in the center of the air stream. The catheter was connected to a carbon dioxide analyzer apparatus (Normocap 200: Datex). Transcutaneous arterial oxygen saturation (\(S_{\text{aO}_2}\)) and pulse rate were recorded by pulse oximetry (N-100; Nellcor; Pleasanton, CA; or Criticare Poni; Criticare Systems; Waukesha, WI) using a finger probe.

All these signals were recorded with a digital acquisition system (OSG Brainlab; Antwerp, Belgium). The following sampling rates were used: 512 Hz for EMGdi, EMGeso, and EMGrect; 64 Hz for the movements of the thorax and abdomen, and for the sum of these parameters (respiratory inductive plethysmography); 32 Hz for \(P_{\text{tank}}\) and \(P_{\text{ETCO}_2}\); and 16 Hz for \(S_{\text{aO}_2}\) and pulse rate.

To specify the role of the glottis, we monitored the glottic width both during spontaneous breathing (SB) and NPV. A pediatric fiberoptic bronchoscope (Olympus BP; type 3c.20 with a 3.5-mm external diameter; Olympus; Tokyo, Japan) was introduced into the right nostril and placed 2 cm above the vocal cords using direct vision. The bronchoscope was attached to a color camera system (Elmo AR-T2; Elmo; Nagoya, Japan) connected to a television screen and a videocassette recorder (model AG-7530-E; Panasonic; Osaka, Japan). The videocassette recorder was synchronized with the digital acquisition system. This allowed for an accurate video image retrieval in synchronization with the recorded signals (accuracy of 1/50 of a second). To assess the glottic width, we measured breath by breath the inspiratory glottic aperture, as previously described,\(^15\) through the angle formed by the vocal cords at the anterior commissure during inspiration. Only three of our eight subjects could tolerate the fiberoptic bronchoscope, which represented a considerable additional burden to the recording equipment and NPV. In the other subjects, the presence of the bronchoscope greatly disturbed the respiratory muscles and led to frequent swallowing and unstable signal patterns.

### Procedure

The nasal and pharyngeal mucosa were sprayed with 10% lidocaine before introducing the bipolar esophageal electrode. The thoracic and abdominal belts of the respiratory inductive
plethysmograph and the finger sensor of pulse oximeter were applied. Subjects were then introduced into the iron lung (J.H. Emerson; Cambridge, MA) in the dorsal decubitus position, with the head outside the respirator. The respiratory inductive plethysmograph was calibrated, and subjects were asked not to move after calibration had been performed. Finally, the carbon dioxide sampling catheter and the bronchoscope were introduced.

The iron lung delivered controlled intermittent negative pressure. After a period of not more than 15 min of SB, mechanical negative ventilation was started according to the following procedure: first (step 1), f at 15 cycles per minute with an INP of −15 cm H2O (F15-P15); next (step 2), f was increased to 20 cycles per minute and INP was kept at −15 cm H2O (F20-P15); then (step 3), f was kept at 20 cycles per minute and INP was reduced to −30 cm H2O (F20-P30); and finally (step 4), f was decreased to 15 cycles per minute and INP was kept at −30 cm H2O (F15-P30). For each step, at least 5 min of good-quality recordings were required before respirator settings were changed from one step to the next step. Subjects were also maintained at each NPV step at the most for 20 min. The recordings were made only when the subjects were awake.

**Measurements**

Except for periods of SB, measurements were only made when signals showed a stable pattern and there was no swallowing activity. During NPV, the duration of these studied stable periods was at least 1 min. Because SB was frequently difficult to analyze (due to swallowing), we have considered stable periods when they lasted <1 min.

For each breath, the corresponding values for actual Vt, inspiratory time (Ti), total ventilatory cycle duration (TTot), PETCO2, Ptank, SatO2, and pulse rate were obtained directly via the digital acquisition system. f was calculated from Ttot (f = 60/Ttot). Effective Ve (Ve effectively reaching the lungs) was calculated from Vt and f (Ve = f × Vt). Inspiratory resistance (Rinsp) was assessed from mean inspiratory air flow (V) and Ptank (Rinsp = Ptank/V), whereas mean inspiratory V was calculated from Vt and Ti (V = Vt/Ti).

**Statistical Analysis**

Data are presented as the mean ± SD. For the group as a whole, comparisons of Vt, Ve, PETCO2, and Rinsp were performed by using a two-way analysis of variance21 based on a two-tailed test. A p value ≤ 0.05 was considered significant.

**RESULTS**

Eight subjects were included in the study. Their anthropometric characteristics are reported in Table 1. In each subject, data were available for SB and the four steps of NPV. For SB, an average of 22 ± 13 breaths were analyzed per subject. For each step of NPV, periods that were analyzed breath by breath had a mean duration of 1.5 ± 0.5 min (26 ± 9 breaths per period).

**Ventilatory Parameters From SB to NPV**

Individual data for Vt, Ve, and PETCO2 during SB and the four steps of NPV are reported respectively in Figures 1–3. Table 2 shows the results for the group considered as a whole.

When applying an INP of −15 cm H2O (F15-P15 and F20-P15), we observed a significant increase in Vt with respect to SB. Moreover, when pressure was increased to −30 cm H2O (F20-P30 and F15-P30), there was a huge and significant increase in Vt, which was more than doubled with respect to SB. Ve significantly increased for all steps. With respect to SB, Ve trebled for steps F20-P30 and F15-P30. Nevertheless, Ve was significantly lower for step F15-P30 than step F20-P30. PETCO2 progressively decreased with each step, all changes being significant and reaching levels <20 mm Hg for step F20-P30. Mean Rinsp was within the usual range of normality during SB, and significantly increased approximately 10-fold during the whole period of NPV.

**Glottic Width From SB to NPV**

In the three subjects who could tolerate the fiberoptic bronchoscope, an obvious decrease in glottic width was observed when subjects were shifted from SB to NPV. During SB, the angle made by the vocal cords at the anterior commissure was 42.6 ± 4.9° on average vs 31.2 ± 7.4° for step F15-P15, 37.4 ± 7.8° for step F20-P15, 36.5 ± 8.2° for step F20-P30, and 38.6 ± 8.3° for step F15-P30. Supraglottic obstructions and/or glottic closures were never observed when INPs were applied (Fig 4).

**DISCUSSION**

The main result of this study is that during NPV generated by an iron lung, increases in f and/or INP result in high levels of Vt and effective Ve, leading to low levels of PETCO2 in spite of a large increase in Rinsp.
Some technical aspects of this study merit consideration. As the analysis of diaphragmatic muscle activity by surface electrodes is known to be fraught with difficulties, we also used a bipolar esophageal electrode that is considered the "gold standard" to assess the muscular activity of the diaphragm (Fig 5). The accuracy of Vt measurements by respiratory inductive plethysmography was as good as possible, since NPV was delivered in the same supine posture as during the calibration of the respiratory inductive plethysmograph, and because subjects were secured in a fixed position in the iron lung that did not allow any change in posture between calibration and recording measurements. Hence, we can estimate that our Vt measurements correspond to the actual Vt.²⁹,³⁰

When comparing with SB, NPV always resulted in a diaphragmatic inhibition with a total disappearance of phasic inspiratory muscle activity on EMGdi. Inhibition of diaphragmatic activity is a well-known phenomenon when PETCO₂ (in fact PACO₂) is driven below the apneic threshold.³² The apneic threshold is known to be influenced by cortical activity that can maintain spontaneous activation despite low levels of PACO₂,¹⁵,²⁷,³³ and by voluntary relaxation that can help in suppressing diaphragmatic activity during NPV.¹⁵,²⁷,³⁴ Our experimental subjects underwent relaxation training that may have facilitated the abolition of SB for PaCO₂ values above the apneic threshold. Inhibition of the diaphragmatic muscle activity suggests that NPV can carry out the entire respiratory work, leading to resting of the respiratory muscles, as previously described in COPD patients.⁷,¹⁵ Sometimes, we observed the appearance of expiratory muscle activity, especially during step F20-P30 (Fig 5). This activity could correspond to an expiratory braking mechanism opposed to rapid emptying of the lungs through the elastic recoil.
forces stored during inspiration,\textsuperscript{35,36} and might be due to hyperventilation induced by NPV.\textsuperscript{27}

We did not include in our protocol periods of SB to allow a return to baseline values between NPV steps. This would have lengthened considerably the study. However, this procedure has the unwanted consequences of a carryover effect of the increased ventilation of one step into the next step, especially concerning $\text{PETCO}_2$. We do not pretend that our values of $\text{PETCO}_2$ represent $\text{PaCO}_2$ during the corresponding step. However, this does not detract from the fact that $\text{VE}$ and $\text{Vt}$ can be greatly increased during NPV.

A significant increase in $\text{VE}$ and a significant decrease of $\text{PETCO}_2$ were systematically observed when comparing SB to each step of NPV. The increase in $\text{VE}$ was due especially to the increase in $f$ for an INP of $-15$ cm H\textsubscript{2}O. Thereafter, the increase in $\text{VE}$ was due to an increase in both $\text{Vt}$ and $f$. It is interesting to note that for steps F20-P30 and F15-P30 delivering the same INP ($-30$ cm H\textsubscript{2}O), the $\text{Vt}$ was significantly higher when $f$ was lower, most probably because of the increase in $\text{Ti}$. It has been previously demonstrated that an increase in inspiratory/expiratory ratio could result in a significant increase in $\text{Vt}$ for an INP of $-20$ cm H\textsubscript{2}O.\textsuperscript{37} However, the reduction in $f$ (from 20 to 15 cycles per minute) resulted in a decrease in $\text{VE}$ despite the increase in the inspiratory/expiratory ratio and $\text{Vt}$ (Table 2). From previous studies,\textsuperscript{11,27,34,38} the ventilatory response to NPV appeared variable. However, in all these studies, diaphragmatic electrical activity was unchanged or decreased but not completely inhibited.

When comparing ventilatory changes during NPV

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{figure2}
\caption{Mean individual $\text{VE}$ measurements during SB and the four levels of NPV. The mean group value for each is also given.}
\end{figure}
and positive pressure ventilation, it has been previously reported that NPV was less effective than positive pressure ventilation. Indeed, Belman et al.\textsuperscript{11} showed that in both COPD and healthy subjects, $V_t$ and $V_E$ were significantly higher during positive pressure ventilation, leading to a significant lower $P_{etco_2}$ with respect to SB or NPV. Moreover, reduction in EMGdi was greater during positive pressure ventilation than other ventilatory modes. Kaneko et al.\textsuperscript{39} demonstrated that in eight patients under general anesthesia, changes in $V_t$ and functional residual capacity were lower during NPV than

**Table 2—Respiratory Findings**

<table>
<thead>
<tr>
<th>Variables</th>
<th>SB</th>
<th>F15–P15</th>
<th>F20–P15</th>
<th>F20–P30</th>
<th>F15–P30</th>
</tr>
</thead>
<tbody>
<tr>
<td>$V_E$, L/min</td>
<td>$6 \pm 2.1$</td>
<td>$9.1 \pm 2.8^*$</td>
<td>$10.4 \pm 3.8^*$</td>
<td>$20.9 \pm 9.3^*$</td>
<td>$18.6 \pm 6.4^*$</td>
</tr>
<tr>
<td>$V_t$, mL</td>
<td>$498 \pm 254$</td>
<td>$615 \pm 186^*$</td>
<td>$531 \pm 200^*$</td>
<td>$1,075 \pm 500^*$</td>
<td>$1,274 \pm 433^*$</td>
</tr>
<tr>
<td>$P_{etco_2}$, mm Hg</td>
<td>$35.7 \pm 4.4$</td>
<td>$27.4 \pm 3.9^*$</td>
<td>$25.9 \pm 3.1^*$</td>
<td>$18.7 \pm 3.7^*$</td>
<td>$17.2 \pm 4^*$</td>
</tr>
<tr>
<td>Rinsp, cm H$_2$O/L/s</td>
<td>$5.4 \pm 4.4$</td>
<td>$48.7 \pm 14.4^*$</td>
<td>$44.8 \pm 13.8^*$</td>
<td>$46.1 \pm 19.7^*$</td>
<td>$49 \pm 19.5$</td>
</tr>
</tbody>
</table>

*Significantly different from the previous step (p < 0.05).
†Significantly different from step F15–P15.
during positive pressure ventilation at the same levels of pressure. Nevertheless, Nava et al.\(^4\) showed that NPV (INP of \(-30\) cm H\(_2\)O) resulted in an increase in V\(_t\) by a factor of three, from approximately 500 mL during SB to 1,500 mL during NPV, with a substantial reduction, though not abolition, of EMGdi. This is similar to our own data,\(^18,20,21\) which showed higher increases in V\(_t\) and V\(\dot{e}\) with NPV than those reported with nasal IPPV (below the apneic threshold, \(\text{i.e.}\) with abolition of EMGdi). In our study of NPV, for the group as a whole, the highest V\(\dot{e}\) was obtained during step F20-P30, whereas the highest V\(_t\) was obtained during step F15-P30 (20.9 ± 9.3 L/min and 1,274 ± 433 mL, respectively). This calls into question the assertion that NPV is less efficient than nasal IPPV at least during wakefulness. A caveat seems worthwhile: we are comparing results obtained in different subjects submitted to only one mode of ventilation and not in subjects submitted to each mode of ventilation. Nevertheless, the differences are so impressive that they most probably reflect a physiologic phenomenon and not simply interindividual differences.

During nasal IPPV, the main obstacle for ventilation is the glottic orifice. The glottis and upper airways have already been identified as serious obstacles for ventilation during NPV, both during wakefulness and sleep.\(^41,42\) That the glottis may narrow during NPV was suggested by the increase in Rinsp we have found in all subjects, as well as by direct glottis observation in our three subjects. However, during NPV, it appears that this glottic hindrance is of a lesser magnitude than that observed during volumetric nasal IPPV, when the glottic width decreased from 29.8 ± 8.4° to 14.8 ± 8.7° with the increase in delivered ventilation.\(^18\) Whereas the disappearance of EMGdi resulted in a glottic narrowing, increases in INP did not result in our study in a further glottic narrowing; 31.2 ± 7.4° for step F15-P15 and 36.5 ± 8.2° for step F20-P30. This can well explain the high level of V\(_t\) seen during NPV in the present study and in the study of Nava et al.\(^4\) Glottic narrowing can be favored by inhibition of the inspiratory activity of the respiratory centers, as during nasal IPPV. However, a reflex abduction of the vocal cords during negative pressure application in the upper airway might counterbalance the tendency of the glottis to narrow\(^43\) and could explain our results. This would not be the case during nasal IPPV.

If the main indication of NPV remains chronic respiratory insufficiency due to neuromusculoskeletal diseases, previous studies\(^22-25\) have suggested some efficacy during ARF in patients with COPD or neuromuscular disorders. In such patients, NPV significantly improved gas exchanges and increased maximal inspiratory pressure. These benefits were less evident in COPD patients who poorly tolerated NPV.\(^23\) More recently, a retrospective study by Corrado et al.\(^26\) demonstrated the efficacy of NPV in patients with ARF and hypoxic hypercapnic coma. NPV generated by an iron lung appeared successful in 105 patients who

**Figure 4.** Example of glottic changes obtained in subject 8 when moving from SB to step F15-P15 and step F20-P30. A: glottic width observed during SB; B: the onset of NPV (F15-P15) led to a decrease in glottic width; C: increasing f from 15 to 20 cycles per minute and INP from \(-15\) to \(-30\) cm H\(_2\)O resulted in an opening of the glottis.

**Figure 5.** Polygraph recording of raw EMGdi, EMGeso, EMGrect, thorax, and abdomen, and sum signals from the respiratory inductive plethysmograph, Ptank, P\(\text{ETCO}_2\), Sa\(_{O2}\), pulse rate (heart rate) recorded in subject 8 during SB and the four steps of mechanical negative ventilation. For recording duration, see scale of 6 s.
regained consciousness after a median of 4 h, reducing
the need of endotracheal intubation and invasive ven-
tilation. The high levels of VT and VE observed in our
study might explain this great efficacy of NPV observed
in patients with COPD or neuromuscular disorders
hospitalized for severe ARF.

In summary, NPV in normal awake subjects can
rest the respiratory muscles with a complete inhibi-
tion of the diaphragmatic muscle activity, and result
in significant increases of VE and VT that lead to a
significant decrease of PtcO₂. During NPV, the
glottis was found to represent a moderate hindrance
that interfered slightly with mechanical ventilation.
These results gathered in normal subjects show that
NPV is an efficient mode of mechanical ventilation;
therefore, the concept of superiority of nasal IPPV is
worth reconsidering. Further studies comparing ven-
tilatory changes during positive pressure ventilation
and NPV in the same subjects are also necessary.

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