

Efficacy of Positive vs Negative Pressure Ventilation in Unloading the Respiratory Muscles*

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We compared the efficacy of positive pressure ventilation (PPV) vs negative pressure ventilation (NPV) in providing ventilatory muscle rest for five normal subjects and six patients with chronic obstructive pulmonary disease (COPD). All participants underwent measurement of transdiaphragmatic pressure (Pdi), pressure time integral of the diaphragm (PTI), integrated diaphragmatic electromyogram (iEMG), minute ventilation (\dot{V}_E), tidal volume (V_T), and end-tidal CO_2 (et CO_2) during 15 minutes of PPV and NPV. For each subject, ventilator adjustments were made to obtain \dot{V}_E similar to levels measured during quiet breathing (QB). We found that the iEMG, Pdi, PTI, and average coefficient of variation of the tidal volume (CV- V_T) were consistently lower during PPV as compared with NPV ($p=0.01$). The iEMG normalized for \dot{V}_E and V_T was also significantly lower during PPV ($p=0.01$). During PPV, subjects were mildly hyperventilated (lower et CO_2 and

higher \dot{V}_E) compared with QB and NPV, but no significant correlation was noted between the change in et CO_2 and the change in iEMG. The change in PTI was significantly correlated with the change in iEMG ($p<0.01$). We conclude that in the short term, PPV is more effective than NPV in reducing diaphragmatic activity. Positive pressure ventilation may be the preferred method of assisted ventilation in future studies of ventilatory muscle rest therapy.

(*Chest* 1990; 98:850-56)

AV = assisted ventilation; VMR = ventilatory muscle rest; NPV = negative pressure ventilation; PPV = positive pressure ventilation; Pdi = transdiaphragmatic pressure; Pes = esophageal pressure; Pga = gastric pressure; et CO_2 = end tidal CO_2 ; fb = breathing frequency; iEMG = integrated electromyogram; QB = quiet breathing; PTI = pressure time integral; dp/dt = decrease of intrathoracic pressure

Assisted ventilation (AV) can improve alveolar ventilation in patients with neuromuscular or thoracic cage abnormalities.¹⁻⁴ Because respiratory muscle fatigue may aggravate the already impaired function of the respiratory muscles in patients with chronic obstructive pulmonary disease (COPD), ventilatory muscle rest (VMR) by means of assisted ventilation has been advocated in these patients.⁵⁻⁷

Most studies have used negative pressure ventilation (NPV) as a means of applying assisted ventilation in patients with COPD. However, not all investigators have shown beneficial results. Ventilatory support has been applied at different intervals ranging from daily to weekly, and from a few days to a few months in duration.⁷⁻¹² In the most extreme case, Gutierrez and coworkers⁸ were able to demonstrate increases in tidal volume, maximal inspiratory mouth pressures, and improved gas exchange after four months of weekly NPV. Zibrak and coworkers¹¹ reported problems with patient acceptance and comfort during NPV and failed to demonstrate any clinical benefit. Celli and colleagues¹² showed that patients randomized to receive rehabilitation alone and rehabilitation plus NPV improved their exercise capacity symptoms to a similar degree. In other words, NPV was of no additional

benefit. Furthermore, it is now well documented that NPV may cause upper airway obstruction with apnea and oxygen desaturation in both normal subjects and patients with neuromuscular respiratory failure.^{13,14} Conversely, positive pressure ventilation (PPV) prevents upper airway obstruction during assisted ventilation and can be used noninvasively via a mouthpiece or nasal mask.¹⁵⁻¹⁷

Most studies of NPV and PPV have not adequately documented that spontaneous inspiratory efforts were actually reduced during periods of AV. Because the rationale for the use of intermittent assisted ventilation is based on the premise that it alleviates muscle fatigue, we believe it is essential to document that a significant reduction in ventilatory muscle activity can be provided.

The purpose of our study was to compare the efficacy of short-term NPV and PPV in reducing spontaneous diaphragmatic activity in awake naive subjects. Our results show that PPV applied noninvasively via a nasal continuous positive airway pressure (CPAP) mask is superior to negative pressure ventilation as a means of resting the diaphragm.

METHODS

Patients

Five normal male volunteers and six ambulatory patients with COPD from the Outpatient Clinic participated in the study. The patients with COPD were medically stable without any exacerbation of their respiratory condition within the previous three months. All

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subjects gave their informed consent prior to the study. The protocol was approved by the Human Subjects Research Committee of Cedars-Sinai Medical Center, Los Angeles.

Measurements

On the day of the study, patients underwent spirometric measurements¹⁸ using a dry seal spirometer (Cardio Pulmonary Instruments, Houston).

Diaphragmatic electromyogram (EMG) was measured using surface electrodes placed in the right sixth and seventh intercostal spaces. An external pacing electrode pad (Padeco, Lake Oswego, Ore) placed over the left scapular area served as the ground. The EMG signal was amplified using a PN22 AC amplifier (TECA, Pleasantville, NY) and filtered using an active band-pass filter (10 to 1,000 Hz) and third-order Paynter filter (time constant = 10 ms).¹⁹

Transdiaphragmatic pressure (Pdi) was obtained by means of the two-balloon technique with balloons positioned in the esophagus and stomach for measurement of esophageal (Pes) and gastric (Pga) pressures using pressure transducers (Validyne MP45-1-871 ± 50 cm H₂O, Northridge, CA). The esophageal and gastric catheters were connected to the negative and positive ports, respectively, of a third differential pressure transducer to obtain Pdi (Pdi = Pga - Pes).

A nasal CPAP mask (Respironics, Inc, Monroeville, PA) was modified to accommodate the balloon catheters. An attachment in the mask allowed continuous sampling of end tidal CO₂ (etCO₂) with a mass spectrometer (Perkins-Elmer 1100 Medical Gas Analyzer, Pomona, CA). Tidal volume (V_T), breathing frequency (fb), and minute ventilation (V̇E) were measured from a turbine flowmeter (ventilation measurement module, Sensormedics, Anaheim, CA) attached to the mask.

Signal Analysis

During the course of an experiment, esophageal, gastric, and transdiaphragmatic pressures, as well as the volume signal from the VMM and the filtered EMG signal were digitized by an A-to-D (analog to digital) converter (Data Translation No. 2801). These five digitized channels were displayed in real-time on a microcomputer (WYSE 386-16) using data acquisition software (Laboratory Technologies Corp, Wilmington, MA), and they were recorded on the hard disk for postexperiment processing. We also recorded Pdi, EMG, and expiratory volume on a strip-chart recorder (Gould 2400, Cerritos, CA).

Software was developed to perform breath-by-breath analysis on the recorded digital data. The algorithm is described herein: Initially, the volume waveform was scanned to mark inspiratory and expiratory cycles. During each inspiration, Pes, Pga, and Pdi waveforms were averaged for measurement of mean Pes (Pes), mean Pga (Pga), and mean Pdi (Pdi). Furthermore, the pressure time integral of the diaphragm (PTI = ∫Pdidt) was calculated during each inspiration.

The software also integrated the digitized EMG signal during each inspiration. Bigland and Lippold²⁰ have shown that integrated EMG (iEMG) signal strength varies directly with the force developed by the muscle it monitors. Usually, in measuring the strength of activity of respiratory muscles, an analog device called a "leaky integrator" with a time constant of 100 to 200 ms is used.²¹⁻²³ The peak amplitude of the leaky-integrated EMG wave is then used as an estimate of the integrated EMG during the inspiratory maneuver. Instead of using a leaky integrator, we digitized the filtered EMG signal and integrated it directly in software during each inspiratory period. This has two advantages: (1) The integration that was done in software was triggered by the inspiratory volume signal. The integration continued until an expiratory volume was detected. This integration had no "leak" associated with it since the period of

integration was certain, and there was no need for integrating over the entire EMG record, as is the case with the analog integrators. From this standpoint, our method of integrating the EMG is an exact software implementation of the analog integrator used by Bigland and Lippold.²⁰ (2) The software marked and removed some of the cardiac electrical activity present in the EMG signal (such as the QRS complex). To accomplish this, we wrote a simple algorithm that searched in a moving window of length 0.4 s for epochs of very high impulse-like electrical activity. This portion of the signal was marked (mean duration = 120 ms) and replaced by the average of an equal length EMG period that immediately followed.

In summary, after the digitized record of an experiment was processed by the software, the following variables were printed out on a breath-by-breath basis: V_T, fb, V̇E, Pes, Pga, Pdi, peakPdi, PTI, and iEMG.

Study Protocol

After adjustments were made to ensure optimal pressure and EMG signals, all subjects were studied with the following protocol: Initial measurements were made during 10 minutes of quiet breathing (QB). Breath-by-breath samples obtained during the last 3 minutes of QB were considered baseline values.

Subsequently, each subject received 15 minutes of both NPV and PPV. Negative pressure ventilation was applied via a Thomson Maxivent ventilator (Puritan-Bennett, Boulder, CO) attached to a poncho-wrap. A plastic frame, which allowed sufficient room for chest expansion, was positioned over the chest, and the wrap was placed over this frame. Positive pressure ventilation in the control mode was delivered through a nasal CPAP mask via a volume cycled ventilator (Lifecare, Portable Lifecare PLV 100, Lafayette, CO). Between each ventilator session, the subjects resumed QB for 10 to 15 minutes to allow the breathing pattern to return to baseline conditions. The order of assisted ventilation was randomly assigned. Because of the effect of PaCO₂ on respiratory drive, initial ventilator adjustments of V_T, flow rate, and frequency during NPV and PPV were made to achieve V̇E and etCO₂ similar to that recorded during the QB. If iEMG activity persisted at equal etCO₂ levels, ventilator pressures were increased to reduce iEMG. This was usually a problem with NPV during which negative pressures to -40 cm H₂O were generally unsatisfactory in reducing iEMG. The subjects were repeatedly encouraged to relax in order to allow the ventilator to control breathing. If patients indicated that the ventilatory volumes were insufficient, these were adjusted until the patient felt comfortable. At the end of each session of ventilatory support, the patient was disconnected and measurements during spontaneous breathing were repeated. It should be emphasized that the subjects remained awake during the study and kept their mouths closed to prevent escape of air. We believe, therefore, that the volume recordings accurately reflect the true volume changes. The entire study lasted from 1½ to two hours.

Analysis of Data

Comparisons were made using the averages of the last 3 minutes of the two ventilator sessions with QB. The values during quiet breathing, PPV, and NPV were compared by an analysis of variance (ANOVA) for repeated measures. Post hoc comparisons of significant differences were performed using the Tukey-A test (CRUNCH Software, CRUNCH-3, Oakland, CA).

RESULTS

We investigated two groups of subjects. The first group consisted of five men with normal pulmonary function. The second group was composed of six male patients with COPD, ex-cigarette smokers (48 ± 28

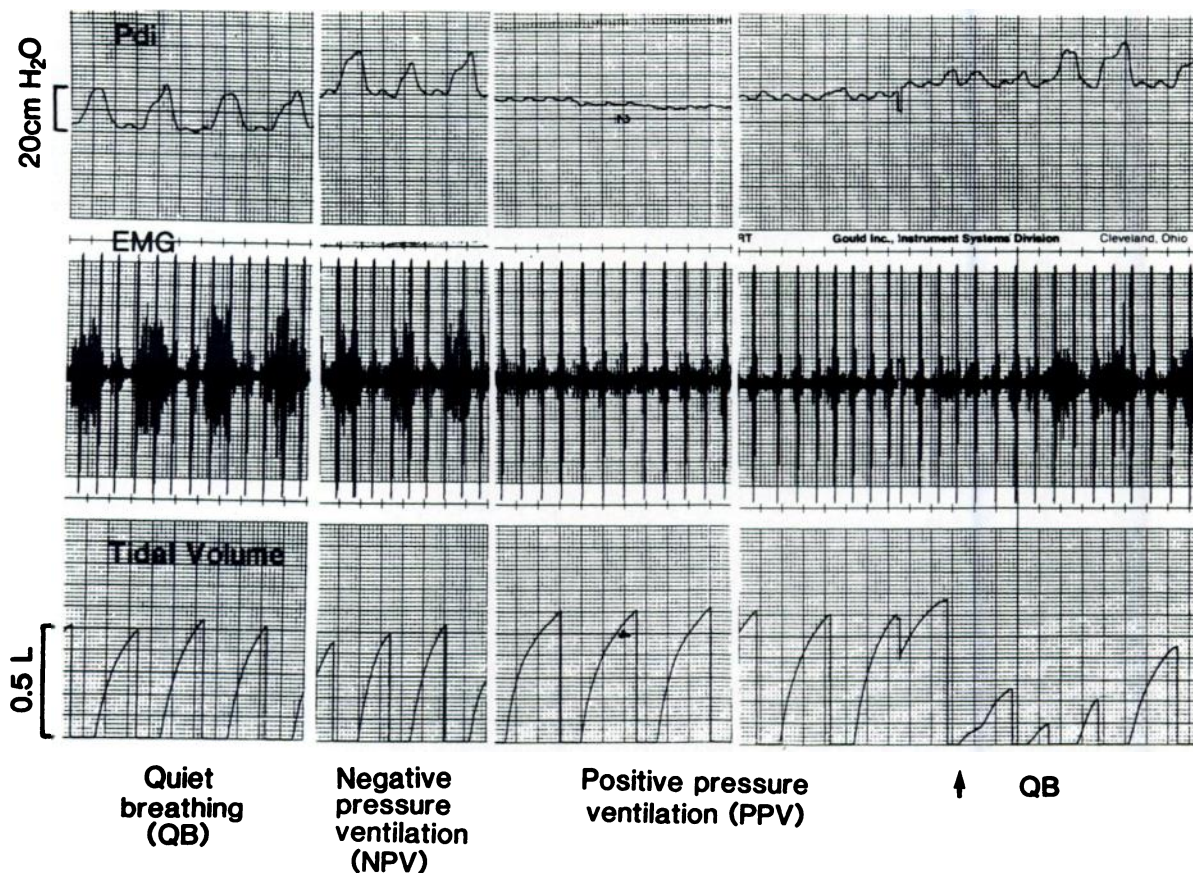


FIGURE 1. Representative tracings during QB, NPV, and PPV sessions in a patient with COPD. Note the larger reduction in Pdi and EMG during PPV. After discontinuation of PPV (vertical arrow), return of diaphragmatic activity is demonstrated.

pack years, mean \pm SD), and average age of 71 ± 5 years (range, 63 to 77 years). The study day FEV₁ was 1.13 ± 0.2 L with a mean FEV₁/FVC of 44 percent \pm 11 percent. Previous arterial blood gases demonstrated an average PaO₂ of 78 ± 11 mm Hg and a mean PaCO₂ of 38 ± 2 mm Hg. No subject was hypercarbic (PaCO₂ >45 mm Hg).

Using ANOVA for repeated measures, we found that patients with COPD had higher \dot{V}_E ($p < 0.01$), lower etCO₂ ($p < 0.05$), and higher peak Pdi ($p < 0.05$) than normals during QB, NPV, and PPV (Table 1). However, the response of the measured indices to assisted ventilation was the same in both groups. Therefore, we pooled the data from both groups to compare the effects of AV on \dot{V}_E , peak Pdi, and etCO₂.

Figure 1 is representative of tracings obtained from one of our patients with COPD during QB, NPV, and PPV. The tracings of Pdi, EMG, and expired volume during QB represent baseline data. Comparison of the tracings during AV illustrate minimal reduction in Pdi and EMG during NPV, with much greater reduction during PPV. Another measure of diaphragmatic capture is illustrated by the small variation of the V_T during PPV as compared with the variability during NPV.

Figures 2 and 3 summarize the effect of PPV and NPV in both normals and patients while receiving AV. To maintain patient comfort, the V_T and \dot{V}_E were higher during PPV as compared with QB and NPV ($p = 0.01$ or $p = 0.05$). Consequently, the etCO₂ was significantly lower during PPV ($p = 0.01$). A comparable \dot{V}_E could not be achieved during NPV despite high negative pressures (mean = -48 cm H₂O). The iEMG in absolute values showed a significant reduction for PPV as compared with QB ($p = 0.01$) and for PPV compared with NPV ($p = 0.05$). The difference between NPV and QB was not significant. Because V_T and \dot{V}_E were noted to be higher during PPV, the iEMG was normalized for both V_T and \dot{V}_E . This did not alter the results apart from a significant decrease in iEMG/ \dot{V}_E when NPV was compared with QB.

Figure 2 represents the iEMG during assisted ventilation. The iEMG during PPV was reduced to 47 percent of the QB value while iEMG/ \dot{V}_E and iEMG/V_T were reduced to 29 percent and 24 percent, respectively. The reduction of iEMG during NPV was not significant. The remaining graphs in Figure 2 illustrate the greater reduction in iEMG/ \dot{V}_E and iEMG/V_T from QB and NPV achieved during PPV ($p = 0.01$). Although there was significant reduction in

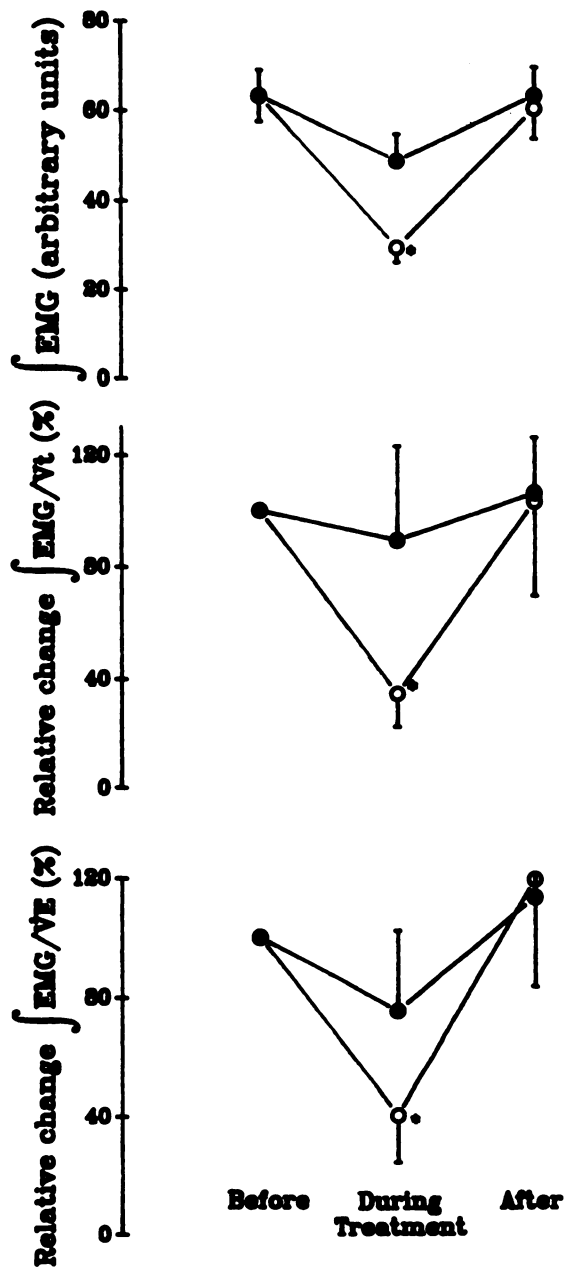


FIGURE 2. This illustrates the reduction in EMG, EMG/V_T, and EMG/V_E for all subjects during PPV (open circles) and NPV (filled circles). (* PPV < QB, Tukey A p = 0.01; + NPV < QB p = 0.05; and PPV < NPV during ventilation, p = 0.05 for EMG and EMG/V_E and p = 0.01 for EMG/V_T.)

iEMG/V_E during NPV as compared with QB (p = 0.05), this was not seen with iEMG/V_T.

The other measures of diaphragmatic activity also demonstrated greater reduction during PPV. Figure 3 and Table 1 illustrate the reduction in the PTI, peak Pdi, and CV-V_T. The reduction of PTI during NPV was different from QB (58 percent, p = 0.05) and the reduction during PPV was different from both QB (29 percent, p = 0.01) and NPV (p = 0.05). The peak Pdi was similarly reduced during PPV to 47 percent of baseline (p = 0.01). The CV-V_T during PPV was signif-

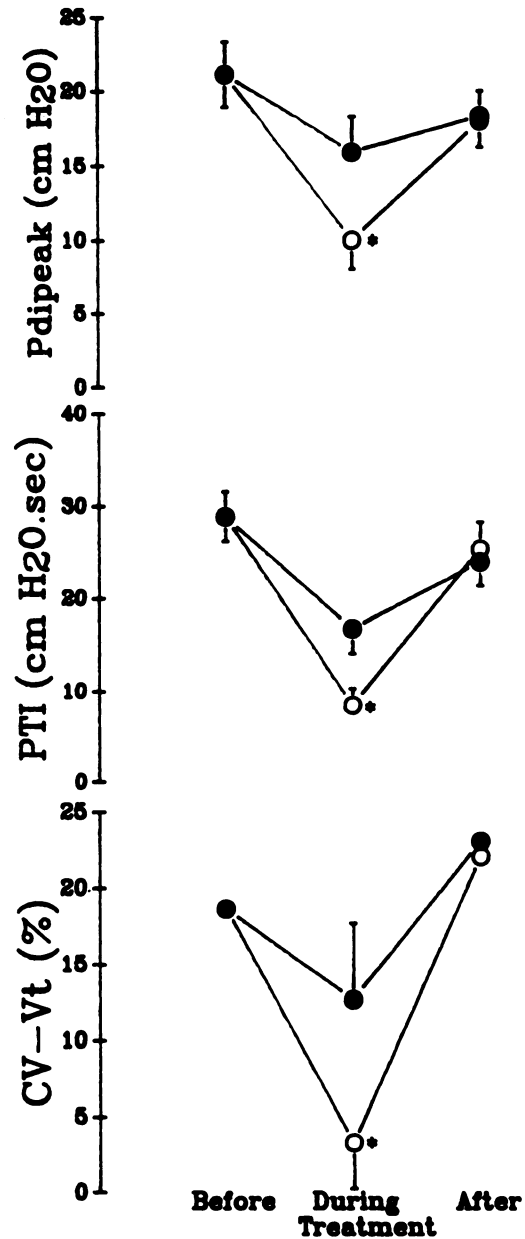


FIGURE 3. Reduction in peak Pdi, PTI, and CV-V_T for all subjects during PPV (open circles) and NPV (filled circles). (* PPV < QB, Tukey A p = 0.01; + NPV < QB p = 0.01; and PPV < NPV, p = 0.05 for PTI and p = 0.01 for CV-V_T.)

icantly lower (3 percent) when compared to NPV (CV-V_T = 13 percent) and QB (CV-V_T = 18 percent) (p = 0.01).

Figure 4 shows the relationship between the change in etCO₂ and iEMG during both PPV and NPV. Both combined (PPV + NPV) and separate analysis failed to show a significant relationship between the two variables (r = 0.210, p = 0.349).

The relationship between iEMG and peak Pdi and PTI was examined. During QB, there was a significant relationship between iEMG and both indices

Table 1—Means of Breath-by-Breath Sampling during Last Three Minutes of Each Study Session Expressed as Mean \pm SD

	\dot{V}_E ,* L/min	Peak Pdi,† cm H ₂ O	ETCO ₂ ,† %
Normals (n = 5)			
QB	7.3 \pm 1.3	16.8 \pm 5.2	5.8 \pm 0.5
PPV	8.6 \pm 1.2	8.2 \pm 4.4	5.0 \pm 0.4
NPV	7.8 \pm 1.3	11.3 \pm 6.7	5.4 \pm 0.4
COPD (n = 6)			
QB	9.9 \pm 1.7	24.6 \pm 7.3	4.7 \pm 0.6
PPV	12.7 \pm 2.8	11.4 \pm 8.0	4.0 \pm 0.7
NPV	10.6 \pm 1.3	19.8 \pm 6.7	4.6 \pm 0.6

*ANOVA $p < 0.01$ COPD = normals. See text for explanation of abbreviations.

† $p < 0.05$ COPD = normals.

($r = 0.818$ and $r = 0.801$, respectively, $p < 0.01$). There was also a significant relationship between the change in iEMG and change in PTI during NPV and PPV ($y = 0.90x - 23.04$; $r = 0.88$, $p < 0.01$) (Fig 5).

DISCUSSION

We have shown that PPV is more effective than NPV in achieving an acute reduction of diaphragmatic activity in the naive awake subject. We interpret the reduction in integrated EMG during PPV to represent a decrease in neural drive. The decrease in peak Pdi and PTI of the diaphragm is consistent with reduced diaphragmatic energy expenditure.²⁴ Furthermore, the small coefficient of variation of V_T during PPV as compared with NPV is consistent with ventilator capture of the breathing pattern.

We recorded diaphragmatic EMG via surface electrodes placed in the sixth and seventh intercostal spaces. Previous investigators have described that there is a good correlation between surface and esophageal EMG recordings of diaphragmatic activity,^{21,25} a finding corroborated by our experience (unpublished observations). In addition to the EMG, we also used other measures of diaphragmatic activity such as peak Pdi and PTI. The changes in these two indices were significantly correlated with the change in the iEMG as noted by Rochester et al²⁶ ($r = 0.818$ and $r = 0.801$, respectively) and support the use of surface iEMG as indicative of diaphragmatic activity.

Although the AV was applied for short periods, there were large reductions in diaphragmatic activity during PPV. During NPV the changes were considerably smaller and not different from the activity during QB. Comparable reductions in iEMG during PPV were described previously,²¹ but in this study, measurements were made during non-REM sleep. Our study shows that significant reductions in iEMG, Pdi, and PTI are apparent within a short period of time after initiating nasal PPV in awake naive normal subjects and patients with COPD.

We attempted to match \dot{V}_E during assisted ventila-

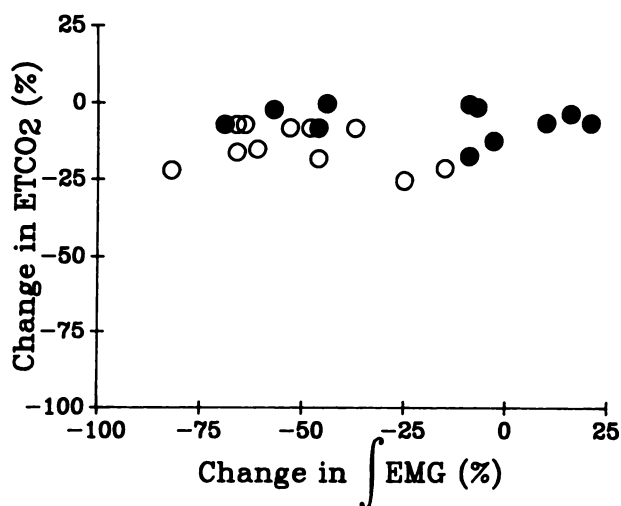


FIGURE 4. Scattergram compares the change in iEMG and the change in etCO₂ for all subjects. No significant relationship was found. Symbols as defined in Figures 2 and 3.

tion to the level during QB. However, an additional goal was to maximize subject comfort during the AV. The small differences in \dot{V}_E and etCO₂ during PPV as compared with NPV were not significant. Although reduction in diaphragm EMG can be achieved with hyperventilation by mechanical means, we do not believe that this is the main reason for reduced iEMG in our patients.^{21,27} In the study of Henke and coworkers,²¹ reduction of iEMG was related mainly to reduction of the etCO₂ below the apneic threshold of normal subjects during non-REM sleep. In awake subjects, considerably greater reductions in etCO₂ are required to silence spontaneous respiratory activity.²⁷ Our subjects showed no correlation between the reduction in PaCO₂ and iEMG. These data agree with those of Rochester et al²⁶ and Rodenstein et al^{22,23} who also failed to find relationships between changes in CO₂ tensions and decreases in iEMG. Conversely, our data

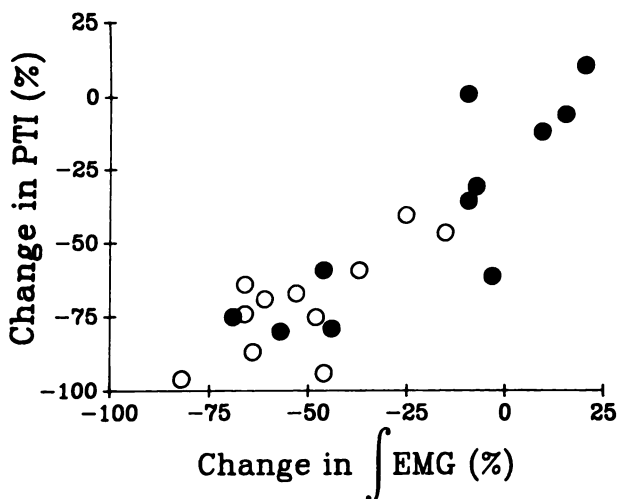


FIGURE 5. Significant relationship between the change in iEMG and the change in PTI ($p < 0.01$) for all subjects. Symbols as defined in Figures 2 and 3.

strongly suggest that the reduction in diaphragmatic activity resulted from mechanical unloading of the diaphragm. This is shown by the excellent correlation between the changes in the PTI of the diaphragm and iEMG. Even in the study of sleeping subjects,²¹ a partial role for mechanical factors was demonstrated by the fact that a reduction of iEMG persisted even after CO₂ tensions were restored to isocapnic levels. Previous studies have shown that nonchemoreceptor influences, including a reduction in mechanical loading, can produce an inhibition of the respiratory system.²¹

The reduction in iEMG during NPV in our study is comparable to that achieved by other investigators.^{22,23,27} Some investigators^{5,26} have found larger increases, but we agree with the conclusions of Rodenstein and coworkers^{22,23} who stated that the reduced EMG during NPV is most likely the result of practice with the negative pressure ventilators. In the preliminary data of Levy and coworkers,²⁵ it is emphasized that considerable practice with the negative pressure ventilator was necessary before reductions in the EMG activity could be found. Rodenstein and coworkers^{22,23} were unsuccessful in achieving adequate reductions in iEMG during NPV in naive subjects. Criner and Celli²⁸ compared the rate of decrease of the intrathoracic pressure (dp/dr) during spontaneous and assisted NPV in patients with COPD and neuromuscular disease. They showed that in contrast to patients with neuromuscular disease, spontaneous dp/dr was in excess of the ventilator dp/dr in patients with COPD and speculated that NPV would generally be unsuccessful in capturing the diaphragm. The patients of Rochester and coworkers,²⁶ in whom significant reductions in iEMG were seen, had had considerable previous experience with NPV. Furthermore, in this study, the iEMG response was greater during NPV with an iron lung as compared with a cuirass. Naive subjects do not show this response, although when actively encouraged to relax, there is a decrement in the EMG.^{22,23} The importance of practice and relaxation is underscored by the fact that in some patients, persistence of the EMG is seen even in the presence of hypocapnia.²¹⁻²³

In our study, we used only very short exposures (15 minutes) to ventilatory support. It could be argued that with further training and practice, our subjects would have achieved further reductions in diaphragmatic activity with NPV. However, the time and effort required to produce significant diaphragm capture and rest with NPV should be compared with the rapid and larger reduction in iEMG with PPV. It is also important to remember that upper airway obstruction, a complication frequently described with NPV,¹³⁻¹⁵ is avoided with the use of PPV. In a study of patients with restrictive lung disease,¹³ nasal CPAP or a tricyclic

antidepressant (protriptyline) in combination with the NPV was necessary to circumvent the problem of upper airway closure.

Much of the current enthusiasm for ventilatory support is based on the assumption that assisted ventilation rests the ventilatory muscles.⁵⁻⁷ By this means, inspiratory impedance is reduced allowing recovery of chronic fatigue of the ventilatory muscles. The early enthusiasm for NPV has diminished because of conflicting results reported in several recent studies.⁷⁻¹² However, the lack of documentation of actual unloading of the respiratory muscles in the negative studies detracts from the validity of their conclusions. The fact that PPV is able to substantially reduce diaphragmatic work in both awake and sleeping subjects suggests that this may be the preferred method of AV to be used in future studies. Other questions that must be examined include the effect of PPV during both non-REM and REM sleep, and patient acceptance of and comfort with the nasal mask. Previous experience in patients with neuromuscular disease suggests that long-term nasal nocturnal PPV is well tolerated.^{4,15,16} In these studies, PPV was delivered nightly for three to five months. In one study, PPV¹⁷ was delivered to patients with both COPD and restrictive disease for three to nine months. Improvements in control of oxygenation and quality of sleep were superior in the restricted vs the obstructed patients. However, both groups demonstrated an improvement in arterial gas tensions and good acceptance of the nasal masks.

ADDENDUM

Since submission of this article, a study by Carrey Z, Gotfried SB, and Levy RD, "Ventilatory Muscle Support in Respiratory Failure with Nasal Positive Pressure Ventilation," (*Chest* 1990; 97:150-58) has appeared. The results showed that positive pressure ventilation applied through a nasal mask reduced inspiratory muscle activity in normals and patients with restrictive and obstructive pulmonary disease.

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