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Submitted 16 August 2006; accepted in final form 28 March 2007

Sternomastoid, rib cage, and expiratory muscle activity during weaning failure

Parthasarathy S, Jubran A, Laghi F, Tobin MJ. Sternomastoid, rib cage, and expiratory muscle activity during weaning failure. J Appl Physiol 103: 140–147, 2007. First published March 29, 2007; doi:10.1152/japplphysiol.00904.2006.—We hypothesized that patients who fail weaning from mechanical ventilation recruit their inspiratory rib cage muscles sooner than they recruit their expiratory muscles, and that rib cage muscle recruitment is accompanied by recruitment of sternomastoid muscles. Accordingly, we measured sternomastoid electrical activity and changes in esophageal (ΔPeso) and gastric pressure (ΔPga) in 11 weaning-failure and 8 weaning-success patients. At the start of trial, failure patients exhibited a higher ΔPeso-to-ΔPga ratio than did success patients (P = 0.05), whereas expiratory rise in Pga was equivalent in the two groups. Between the start and end of the trial, failure patients developed additional increases in ΔPeso-to-ΔPga ratio (P < 0.0014) and the expiratory rise in Pga also increased (P < 0.0004). At the start of trial, sternomastoid activity was present in 8 of 11 failure patients contrasted with 1 of 8 success patients. Over the course of the trial, sternomastoid activity increased by 53.0 ± 9.3% in the failure patients (P = 0.0005), whereas it did not change in the success patients. Failure patients recruited their respiratory muscles in a sequential manner. The sequence began with activity of diaphragm and greater-than-normal activity of inspiratory rib cage muscles; recruitment of sternomastoids and rib cage muscles approached near maximum within 4 min of trial commencement; expiratory muscles were recruited slowest of all. In conclusion, not only is activity of the inspiratory rib cage muscles increased during a failed weaning trial, but respiratory centers also recruit sternomastoid and expiratory muscles. Extradaphragmatic muscle recruitment may be a mechanism for offsetting the effects of increased load on a weak diaphragm.

sternomastoid muscles; respiratory muscles; mechanical ventilation

PATIENTS WHO FAIL a trial of weaning from mechanical ventilation develop marked and progressive increases in mechanical load (16, 40, 49). In an attempt to maintain alveolar ventilation over the course of a failed weaning trial, patients increase respiratory effort to more than four times the normal level (15, 16, 19). In addition to experiencing an increased load, patients undergoing ventilator weaning display severe diaphragmatic weakness (19). Accordingly, patients failing a weaning trial may become more dependent on assistance from other muscles of respiration in achieving the heightened respiratory effort. During resting tidal breathing, patients with chronic obstructive pulmonary disease (COPD) recruit both their inspiratory rib cage muscles and expiratory muscles (31, 32, 51) to compensate for an overloaded and disadvantaged diaphragm. As patients exercise to exhaustion, they further recruit their rib cage muscles, and the magnitude of this recruitment appears to depend on rib cage muscle reserve during resting breathing (51). With further increases in respiratory load, patients also recruit their expiratory muscles (23, 51). This pattern of respiratory muscle activity suggests the existence of a possible hierarchy of muscle recruitment (specific muscle groups recruited in a particular sequence) when patients with a weakened diaphragm are subjected to increased respiratory loads.

We previously showed that weaning-failure patients displayed greater recruitment of rib cage and expiratory muscles than did weaning-success patients (19). We did not, however, separate the relative contribution of each muscle group or the timing of recruitment. Defining the relative activity of respiratory muscle groups during a failed weaning trial may shed light on how the respiratory controller apportions work between these muscle groups in patients with acute respiratory failure. It is commonly believed that patients recruit not only the scalene muscles but also the sternomastoids when they develop respiratory distress (8, 25, 29). This reasoning is based on findings from surface electromyographic (EMG) recordings of the sternomastoids or direct palpation of the neck muscles (3, 11). Surface EMG recordings of the sternomastoids, however, may be unreliable in determining sternomastoid activity because of contamination from scalene muscle activity (9). On the basis of surface EMG recordings, it had generally been accepted that patients with severe COPD commonly recruit their sternomastoids (13, 42, 43). When EMG recordings were obtained using needle electrodes, however, only 4% of patients displayed phasic activity of the sternomastoids; in contrast, scalene contractions were present in all patients (9). That few patients with COPD recruit their sternomastoids suggests that these muscles have a high threshold for activation. Sternomastoid recruitment has also been reported in patients with extensive respiratory muscle weakness, such as patients with transection of the upper cervical cord (7). Because weaning-failure patients display respiratory muscle weakness and experience a rapid and progressive increase in respiratory load, it is conceivable that the sternomastoids might be recruited in the early phase of a weaning trial. The pattern of sternomastoid activation during a weaning trial using needle electrodes has not been previously reported.

Accordingly, the aim of the study was to examine for the first time the pattern of recruitment of inspiratory rib cage, expiratory, and sternomastoid muscles during a trial of spontaneous breathing. We hypothesized that weaning-failure patients recruit their rib cage muscles and sternomastoids at an earlier point in time than they recruit their expiratory muscles during the course of a weaning trial.

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RESPIRATORY MUSCLE ACTIVITY DURING WEANING FAILURE

METHODS

Patients

Nineteen critically ill male patients who were receiving mechanical ventilation and whose primary physician considered them ready to undergo a trial of weaning were recruited on a nonconsecutive basis (Table 1). The patients had received 18.8 ± 4.2 (SE) days of ventilator support. The decision to extubate patient or reinitiate mechanical ventilation was made solely by the primary physician. The physician was blinded to the study design and the measurements obtained, although arterial blood gas values were available. The study was approved by the local Human Studies Subcommittee and informed consent was obtained from each patient. Some aspects of data on esophageal pressure measurements have been included in one other report that addresses a different research question (15).

Experimental Setup

Flow and pressure measurements. Flow was measured with a heated Fleisch pneumotachograph (Hans Rudolph, Kansas City, MO) placed between the endotracheal tube and the Y-piece of the ventilator circuit. Airway pressure (Paw) was measured proximal to the endotracheal tube. Esophageal pressure (Pes) and gastric pressure (Pga) were separately measured with two thin-walled, balloon-tipped catheters (Erich Jaeger, Wurzberg, Germany) coupled to pressure transducers (MP-45, Validyne, Northridge, CA; Refs. 19, 35). Proper positioning of the esophageal balloon catheter was ensured with the occlusion technique (2). Transdiaphragmatic pressure (Pdi) was obtained by subtracting Peso from Pga.

EMG measurements of the sternomastoid muscle. The EMG of the sternomastoid muscle was obtained using bipolar fine-wire electrodes introduced in the muscle’s belly midway between the mastoid process and the medial end of the clavicle (7, 9, 20). EMG signals were filtered below 10 Hz and above 1,000 Hz, EMG, flow, and pressures (Paw, Pes, Pga) were acquired at a sampling rate of 2,000 Hz and recorded on a personal computer using digital acquisition systems (DATAQ).

Table 1. Characteristics of patients

<table>
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<th>No.</th>
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Success Group

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ET tube, endotracheal tube; ID mm, internal diameter in millimeters; COPD, chronic obstructive pulmonary disease; CHF, congestive heart failure.

Protocol

After placement of all transducers, an arterial blood gas measurement was obtained while the patient was still receiving mechanical ventilation. The patient was then disconnected from the ventilator, and maximum inspiratory airway pressure (PImax) was measured during a 20-s occlusion (17, 30). The measurement was made using a one-way valve that allowed exhalation but prevented inhalation, thus ensuring that PImax was measured at low lung volume (17, 30). The patient was then placed back on the ventilator for 2–3 min while the T-tube system for the weaning trial was set up. Next, the patient was disconnected from the ventilator and began to breathe spontaneously through the T-piece circuit with oxygen delivered at the same concentration as during mechanical ventilation. Arterial blood samples were collected at 2 min after starting the trial and at its end. The criteria for weaning failure used by the primary physician were tachypnea, hypoxemia (O2 saturation <90% with a fraction of inspired oxygen ≥0.4), tachycardia, arrhythmias, hypotension, diaphoresis, or evidence of increasing effort (16, 19). Patients who met these criteria were returned to the ventilator and designated as weaning-failure patients. Patients who met none of these criteria at the end of the trial were extubated. Patients who were extubated and sustained spontaneous breathing for >48 h were designated as weaning-success patients. Throughout data acquisition, patients were studied while lying at 30° with their neck in the neutral position.

Physiological Measurements

Intrinsic positive end-expiratory pressure. During spontaneous breathing trial, total intrinsic positive-end-expiratory pressure (PEEP) was measured as the negative deflection in Peso between the point of its rapid decline and the onset of inspiratory flow (33, 34; Fig. 1). Expiratory muscle contribution to total PEEP was measured as the rise in Pga between the onset of expiratory flow and the point of rapid decline in Peso (26, 35, 52; Fig. 1). The rise in Pga during expiration may result from activation of the abdominal muscles, expiratory rib cage muscles, or a combination of the two; the relative contribution of each muscle group to the expiratory rise of Pga cannot be determined.

Respiratory pressures. Maximal inspiratory pressures were calculated as previously described (16, 19). Changes in Pes (∆Pes) during spontaneous breathing were used as an estimate of overall respiratory muscle pressure output (16, 21). ΔPes was measured from the beginning of effort to its nadir. The inspiratory change in Pga (∆Pga) was measured from the beginning of effort to its maximum excursion (19). When present, expiratory muscle contraction can contribute to ΔPes and ΔPga (34). To correct for expiratory muscle contraction, the rise in Pga during the preceding expiration was subtracted from ∆Pes to yield corrected ∆Pes (c∆Pes) and from ∆Pga to yield corrected ∆Pga (c∆Pga). We reasoned that c∆Pes represents an estimate of inspiratory muscle effort and that c∆Pga represents an estimate of diaphragmatic activity during inspiration, free from the contribution of expiratory muscle contraction. The relative contributions of the diaphragm and inspiratory rib cage muscles to inspiratory effort were then estimated as the ratio of corrected ∆Pga (c∆Pga) to corrected ∆Pes (c∆Pes). In healthy volunteers, the ∆Pga/∆Pes ratio during resting breathing is normally more negative than −1 [n = 18; normal = −1.95 (28)]. A ∆Pga/∆Pes ratio of +1 or greater indicates a totally ineffective diaphragm (diaphragmatic paralysis). A ∆Pga-to-∆Pes ratio between −1 and +1 is highly suggestive of impaired diaphragmatic activity (diaphragmatic weakness; Ref. 47); it could also result from greater activity of the rib cage muscles (relative to the diaphragm), relaxation of the abdominal muscles (5), or any combination of the above (54). Change in Pes over time (dP/dt; Ref. 27) was taken as an estimate of respiratory drive.

Electromyography analysis. For each patient, the number of breaths with any sternomastoid EMG activity during inspiration was expressed as a percentage of the total number of breaths during the entire spontaneous breathing trial (45, 46). To assess extent of ster-
Fig. 1. Representative tracings from the first and last minute of a spontaneous breathing trial in a weaning-failure patient. Flow, esophageal pressure (Pes), and gastric (Pga) pressure are shown. Total intrinsic positive end-expiratory pressure (PEEP) was estimated as the drop in Pes from the onset of inspiratory effort (2nd vertical line) to onset of inspiratory flow (3rd vertical line). Estimation of expiratory muscle contribution to changes in Pes, and PEEP, were obtained by measuring rise in Pga from onset of expiratory flow (1st vertical line) to onset of inspiratory effort (2nd vertical line). Note increases in changes in Pes swing, PEEPi, and expiratory rise in Pga between start and end of trial.

Respiratory Muscle Activity

Eleven patients met the criteria for weaning failure after 21 ± 6 min of spontaneous breathing, and mechanical ventilation was re instituted. Eight patients tolerated the trial without distress and were extubated after 31 ± 3 min. Pmax (before the trial) was lower in the failure patients than in the success patients: 32.7 ± 3.5 (36% of predicted) vs. 51.6 ± 9.2 cmH2O (47% of predicted), P = 0.05.

RESULTS

For the spontaneous breathing trial, EMG and pressure data were analyzed at six points in time (sixties): the first and last minute of the trial and four 1-min periods taken at equal time intervals between the first and last minute. Mean EMG and pressure data were calculated based on eight representative breaths within each sextile. The mean activity of sternomastoid muscle for each sextile was then referenced to the sextile in which the patient had achieved the maximum sternomastoid phasic activity during the entire weaning trial. To ensure that our data were normally distributed, we used the Kolmogorov-Smirnov test of normality. Within a group, data at the six time points were compared by one-way ANOVA with repeated measures and by Newman-Keuls test of multiple comparisons between individual means when appropriate. To define the determinants of sternomastoid activity, the relationship between the EMG of the sternomastoids with various physiological indexes was examined using single and multiple linear regression analysis. The breath-to-breath variability in the activity of the various muscle groups was quantified using coefficient of variation, calculated as standard deviation divided by mean. Data between the groups were compared by two-way ANOVA with repeated measures across time. Results are expressed as means ± SE.

Data Analysis

For the spontaneous breathing trial, EMG and pressure data were analyzed at six points in time (sixties): the first and last minute of the trial and four 1-min periods taken at equal time intervals between the first and last minute. Mean EMG and pressure data were calculated based on eight representative breaths within each sextile. The mean activity of sternomastoid muscle for each sextile was then referenced to the sextile in which the patient had achieved the maximum sternomastoid phasic activity during the entire weaning trial. To ensure that our data were normally distributed, we used the Kolmogorov-Smirnov test of normality. Within a group, data at the six time points were compared by one-way ANOVA with repeated measures and by Newman-Keuls test of multiple comparisons between individual means when appropriate. To define the determinants of sternomastoid activity, the relationship between the EMG of the sternomastoids with various physiological indexes was examined using single and multiple linear regression analysis. The breath-to-breath variability in the activity of the various muscle groups was quantified using coefficient of variation, calculated as standard deviation divided by mean. Data between the groups were compared by two-way ANOVA with repeated measures across time. Results are expressed as means ± SE.

Data Analysis

Respiratory Muscle Effort

Estimates of electrical activation of sternomastoids were available for all 19 patients. Because the gastric balloon malfunctioned in one patient, estimates of diaphragmatic pressure output were available in 18 patients (10 of whom failed).

Mechanical estimates. At the start of the trial, the generation of respiratory muscle pressure, inferred from ΔPes, was equivalent in the failure and success groups, 10.7 ± 1.5 and 11.4 ± 1.7 cmH2O, respectively (P = 0.4). Likewise, when ΔPes was corrected for expiratory muscle contraction (cΔPes), values were equivalent in the failure and success groups, 8.9 ± 1.3 and 11.3 ± 1.7 cmH2O, respectively (P = 0.27). At the end of the trial, ΔPes (not corrected for expiratory muscle contraction) increased to 23.0 ± 1.5 cmH2O in the failure group (P < 0.0001) and to 14.6 ± 1.7 cmH2O in the success group (P = 0.005). At the end of the trial, cΔPes (corrected for expiratory muscle contraction) was 18.7 ± 1.5 cmH2O in the failure group. The values of cΔPes and ΔPes in the success group at the end of the trial were nearly identical. Over the course of the trial, ΔPes (not corrected for expiratory muscle contraction) was higher in the failure group than in the success group (P = 0.0004); a similar pattern was observed when ΔPes was corrected for expiratory muscle contraction (P = 0.0015).

At trial onset, the cΔPga/cΔPes ratio was greater in the failure group than in the success group: 0.11 ± 0.08 vs. −0.15 ± 0.09 (P = 0.05) (Fig. 2, top). Over the course of the trial, cΔPga/cΔPes remained greater in the failure patients (P = 0.0014). At the end of the trial, the ratio had increased to 0.39 ± 0.12 in the failure group (P = 0.04) and was unchanged in the success group (−0.14 ± 0.09).

Expiratory muscle pressure output (i.e., increase in Pga during exhalation), expressed as a percentage of the subsequent ΔPes (i.e., global respiratory muscle pressure output), increased from 9.1 ± 3.7% at the onset to 22.6 ± 7.3% at the end of the trial.
trial in the failure group; in some patients, respiratory muscle effort constituted as much as 40% of the subsequent global respiratory muscle pressure output. In the success group, expiratory rise in \( P_{ga} \) remained unchanged at 0.9 ± 0.9% of \( \Delta P_{es} \) over the course of the trial.

Electrical estimates. Sternomastoid activity was evident in 82.5 ± 9.1% of all the breaths in the failure group and in 18.6 ± 10.1% of all breaths in the success group (\( P = 0.002 \)). Plot of sternomastoid EMG activity during a weaning trial in a representative failure patient are shown in Fig. 3. Sternomastoid activity became evident within the first minute of the trial in 8 of the 11 failure patients and 1 of the 8 success patients. By the end of the trial, sternomastoid activity was noted in all failure patients. In contrast to the failure patients, only 3 of the 8 success patients exhibited sternomastoid activity during the trial, and even this activity was modest compared with that recorded in the failure patients (Fig. 2, middle).

Sternomastoid activity (expressed as the percentage of highest activity that an individual patient manifested during the course of the trial) increased by 53.0 ± 9.3% in the failure group over the course of the trial (\( P = 0.0005 \)), whereas it did not change in the success group (\( P = 0.91 \); Fig. 2, middle).

Sternomastoid activity correlated with \( c\Delta P_{ga}/c\Delta P_{es} \) ratio (\( r = 0.54 \) (0.1–0.8, 95% confidence interval), \( P = 0.02 \)) and PEEP, \( [r = 0.66 \ (0.28–0.86), \ P = 0.002] \), and it tended to correlate with \( P_{\text{max}} \) \( [r = -0.43 \ (-0.74–0.03), \ P = 0.07] \). On multiple linear regression analysis, in which sternomastoid EMG activity recorded throughout the trial was the dependent variable and \( P_{\text{max}}, c\Delta P_{ga}/c\Delta P_{es} \) ratio, and PEEP, recorded throughout the trial were the independent variables, 70% of the variance in sternomastoid activity resulted from these three variables (adjusted \( R^2 = 0.70 \)).

**PEEP**

At the onset of the trial, total \( P_{\text{EEP}} \) (not corrected for expiratory muscle contraction) was similar in the failure group, 2.5 ± 0.7 cmH\(_2\)O, and success group, 2.3 ± 0.6 cmH\(_2\)O (\( P = 0.8 \)). At the end of the trial, total \( P_{\text{EEP}} \) increased to 6.9 ± 1.2 cmH\(_2\)O in the failure group (\( P = 0.0001 \)), but it did not change in the success group, 2.5 ± 0.7 cmH\(_2\)O (\( P = 0.6 \)). Over the course of the trial, total \( P_{\text{EEP}} \) was higher in the failure group than in the success group (\( P = 0.04 \)). At the onset of the trial, \( P_{\text{EEP}} \), corrected for expiratory muscle contraction was not different between the failure and success groups: 1.6 ± 0.5 vs. 2.2 ± 0.6 cmH\(_2\)O (\( P = 0.36 \)). At the end of the trial, corrected \( P_{\text{EEP}} \) was 2.6 ± 0.8 cmH\(_2\)O in the failure patients (\( P = 0.3 \)) and 2.5 ± 0.7 cmH\(_2\)O in the success patients (\( P = 0.75 \)). Over the course of the trial, corrected \( P_{\text{EEP}} \) was not different between the failure patients and the success patients (Fig. 4).

**Expiratory Muscle Activity**

Expiratory muscle activity, as indicated by an expiratory rise in \( P_{ga} \), was present in all but one of the failure patients, the exception being a patient with paraplegia (excluding this patient from analysis does not change the findings of the study). Expiratory muscle activity was absent in all but three of the success patients. At the onset of the trial, the expiratory rise in \( P_{ga} \) was equivalent in the failure and success groups, 0.9 ± 0.5 and 0.1 ± 0.1 cmH\(_2\)O, respectively (\( P = 0.3 \); Fig. 2, bottom). At the end of the trial, the expiratory rise in \( P_{ga} \) increased to 4.4 ± 1.1 cmH\(_2\)O in the failure group (\( P = 0.0005 \)), whereas it did not change, 0.1 ± 0.1 cmH\(_2\)O, in the success group (\( P = 0.4 \); Fig. 2, bottom). Compared with the success group, the failure group exhibited larger increases in expiratory rise in \( P_{ga} \) (\( P = 0.004 \)). In the failure group, expiratory muscle activity accounted for 53 ± 4% of total \( P_{\text{EEP}} \) throughout the weaning trial. Throughout the trial, expiratory rise in \( P_{ga} \) correlated with drive, estimated as change in \( P_{es} \) over time (dp/dt) \( [r = 0.57 \ (0.12–0.82), \ P = 0.02] \).

**Variability in the Pattern of Muscle Activation in Weaning Failure**

During the first sextile, the coefficient of variation for sternomastoid activity was higher than that for \( c\Delta P_{ga}/c\Delta P_{es} \) ratio (41 ± 10 vs. 6 ± 3%, \( P < 0.006 \)); the coefficient of variation for sternomastoid activity was similar to that of the expiratory rise in \( P_{ga} \) (57 ± 23%, \( P = 0.54 \)). Likewise, at the
last sextile, the coefficient of variation for sternomastoid activity remained higher than that for \( \frac{c\Delta P_{es}}{c\Delta P_{ex}} \) ratio (65 ± 20 vs. 10 ± 5%, \( P < 0.01 \)) but was similar to that of the expiratory rise in \( P_{ga} \) (36 ± 12%, \( P = 0.23 \)).

**Arterial Blood Gas Measurements**

During mechanical ventilation, \( P_{aco2} \), \( P_{aco2} \), and \( pH \) were not different between the groups (Table 2). By the end of the trial, the failure group developed an increase in \( P_{aco2} \) (\( P = 0.001 \)) and a decrease in \( pH \) (\( P = 0.001 \)). None of the success patients developed hypoxemia (\( P_{aO2} < 60 \text{ mmHg} \) with a \( F_{IO2} \) of 0.40) or respiratory acidosis (\( pH < 7.35 \)).

**DISCUSSION**

This is the first study of systematic measurements of respiratory muscle recruitment in patients being weaned from mechanical ventilation. In patients failing a weaning trial, the sequence of respiratory muscle recruitment began with greater activity of inspiratory rib cage muscles than was the case in the success patients; recruitment of sternomastoids and rib cage muscles was near maximum early in the weaning trial in the failure patients and was followed by progressive activity of the expiratory muscles.

**Sternomastoid Muscle and Rib Cage Inspiratory Muscle Recruitment**

Within the first minute of the spontaneous breathing trial, three-quarters of our failure patients recruited their sternomastoids; in contrast, only one of eight success patients recruited their sternomastoids within the same time frame. Similarly, within the first minute of the spontaneous breathing trial, the \( \frac{c\Delta P_{es}}{c\Delta P_{ex}} \) ratio, a surrogate of rib cage inspiratory muscle recruitment, was greater in the failure patients than in the success patients.

The preferential (more prevalent) recruitment of the sternomastoids and greater inspiratory rib cage muscle contribution to tidal breathing in the failure patients at the start of the trial is most likely secondary to decreased capacity of the inspiratory muscles to generate pressure. This notion is supported by two observations. First, overall inspiratory muscle strength (\( P_{max} \)) before the trial was less in the failure group than in the success group. Second, from the start of the trial, \( c\Delta P_{es}/c\Delta P_{ex} \) ratio was less negative (positive) in the failure group than in the success group: 0.11 and −0.15 (\( P = 0.05 \); Fig. 2). While a \( c\Delta P_{es}/c\Delta P_{ex} \) ratio of less than one indicates that the diaphragm was active and capable of generating pressure (in both patient groups; Ref. 14, 47), the higher \( c\Delta P_{es}/c\Delta P_{ex} \) ratio in the failure group suggests greater diaphragmatic impairment than in the success group (resulting in recruitment of extradiaphragmatic inspiratory muscles). Although the increase in \( c\Delta P_{es}/c\Delta P_{ex} \) ratio could be secondary to relaxation of the abdominal muscles (5), this is unlikely. When computing the \( c\Delta P_{es}/c\Delta P_{ex} \) ratio, the expiratory rise in \( P_{es} \) (an estimation of the magnitude of expiratory muscle recruitment) was subtracted from tidal excursions in \( P_{ga} \) and \( P_{es} \).

Decreased capacity of the inspiratory muscles to generate pressure is also one of the likely mechanisms for greater recruitment of the sternomastoids and rib cage inspiratory muscles during the course of the trial in the failure patients. First, the degree of sternomastoid activity throughout the trial tended to correlate negatively with \( P_{max} \) recorded before the trial (\( r = −0.43, P = 0.07 \)). Second, development of dynamic hyperinflation during a trial will additionally aggravate respiratory muscle weakness (10, 24, 36). Of the 10 failure patients, 7 developed an increase in corrected PEEPi between the start and end of the trial: 1.6 ± 0.5 to 2.6 ± 0.8 cmH2O (\( P = 0.01 \)).

Recruitment of the sternomastoids as a compensatory mechanism for a decrease in the capacity of the diaphragm and rib cage muscles to generate pressure has also been reported in patients with high tetraplegia (7). Sternomastoid and inspiratory rib cage muscle recruitment can also occur in response to an increase in mechanical load.
An increase in load (assessed by $cP_{es}$) at the beginning of the trial, however, is an unlikely cause of sternomastoid and inspiratory rib cage muscle recruitment in the failure patients, because $cP_{es}$ at the beginning of the trial was similar in the two groups of patients. In contrast, increased load (combined with decreased capacity of the inspiratory muscles and diaphragm to generate pressure) was a likely mechanism for sternomastoid and rib cage inspiratory muscle recruitment observed over the course of the trial. This notion is supported by the progressive increase in $cP_{es}$, PEEP, and in $cP_{es}/cP_{ga}$ ratio observed between the start and end of the trial in the failure patients. Moreover, multiple regression analysis revealed that 70% of the variance in EMG activity resulted from $P_{max}/cP_{ga}/cP_{es}$ ratio, and PEEP.

That heightened sternomastoid and rib cage muscle activity in the failure patients represents a compensatory response to the high mechanical load and weak diaphragm is supported by observations in healthy volunteers (50) and in ambulatory patients with COPD (51). When healthy volunteers sustain fatiguing inspiratory loads (tidal excursions in $P_{di}$ 50% of maximum), they demonstrate sternomastoid recruitment and proportionately greater use of rib cage muscles than of the diaphragm (50). Similarly, in patients with COPD during exercise to exhaustion, increased respiratory loads are met with a proportionately greater use of rib cage muscles than of the diaphragm (51). Rib cage pressure contribution predominates during the period of inspiratory flow, not only for overcoming the elastic load of the respiratory system but also in compensating for the gradual loss of diaphragmatic contribution to inspiratory flow (51).

Expiratory Muscle Recruitment

Over the course of the trial, most failure patients activated their expiratory muscles, indicated by a rise in $P_{ga}$ during exhalation (26, 35). In contrast, expiratory muscle recruitment was negligible to absent in success patients.

Increased activation of the expiratory muscles represents an automatic component of the response of the respiratory system to very high levels of ventilatory stimulation (12, 33, 55, 56). Consistent with this viewpoint is the observed correlation between expiratory muscle activation and respiratory drive ($r = 0.57, P = 0.02$).

It has been reasoned that the goal of expiratory muscle recruitment is to assist the inspiratory muscles by decreasing end-expiratory lung volume (22). Most of our patients had COPD and airflow limitation. Therefore, it is unlikely that expiratory muscle recruitment in the failure patients lowered end-expiratory lung volume. If anything, end-expiratory lung volume appeared to have increased despite the presence of expiratory muscle contraction: PEEP, (after correcting for expiratory muscle recruitment) increased between the start and end of the trial in 7 of the 10 patients. Finally, expiratory

Table 2. Arterial blood gas measurements*

<table>
<thead>
<tr>
<th></th>
<th>Mechanical Ventilation</th>
<th>Spontaneous Breathing</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>2 min</td>
<td>End</td>
<td></td>
</tr>
<tr>
<td><strong>Failure Group</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$PH$</td>
<td>7.44±0.01</td>
<td>7.4±0.02</td>
<td>7.37±0.02</td>
</tr>
<tr>
<td>$P_{aCO2}$ mmHg</td>
<td>36.1±3.0</td>
<td>39.3±6.3</td>
<td>45.01±3.6</td>
</tr>
<tr>
<td>$P_{aO2}$ mmHg</td>
<td>110.7±11.1</td>
<td>134±15.8</td>
<td>122.1±19.3</td>
</tr>
<tr>
<td>$F_{O2}$</td>
<td>38.9±0.7</td>
<td>40</td>
<td>40</td>
</tr>
<tr>
<td><strong>Success Group</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$PH$</td>
<td>7.44±0.01</td>
<td>7.40±0.02</td>
<td>7.41±0.02</td>
</tr>
<tr>
<td>$P_{aCO2}$ mmHg</td>
<td>36.6±2.9</td>
<td>40.1±2.5</td>
<td>39.5±2.5</td>
</tr>
<tr>
<td>$P_{aO2}$ mmHg</td>
<td>105.6±14.7</td>
<td>78.8±9.8</td>
<td>101.3±16.7</td>
</tr>
<tr>
<td>$F_{O2}$</td>
<td>39.2±0.8</td>
<td>40</td>
<td>40</td>
</tr>
</tbody>
</table>

Values are mean ± SE. $F_{O2}$, fractional inspired oxygen concentration; NS, not significant.
muscle recruitment induces additional energy expenditure during respiration (54). This consideration raises the possibility that expiratory muscle recruitment itself could have contributed to weaning failure.

Hierarchy of Muscle Recruitment During Weaning Failure

The extent of sternomastoid recruitment and inspiratory rib cage muscle activity in failure patients increased over the course of the trial: sternomastoid activity was 25, 76, 71, 88, 47, 59, and 100% of the normalized value (expressed as percentage of highest activity that an individual patient manifested during the course of the trial; Fig. 2, middle), and \( c\Delta P_{pa}/c\Delta P_{es} \) ratio was 27, 79, 47, 80, 85, and 100% of the value obtained at the final sextile (Fig. 2, top). As such, more than three-quarters of the increase in sternomastoid activity and inspiratory rib cage muscle activity were reached by the second sextile (~4 min into the trial). The immediate increase in sternomastoid activity with little change thereafter casts doubt on the notion that sternomastoid activity is a marker of impending diaphragmatic fatigue (3, 37). Instead, activation probably results from a combination of decreased capacity of the respiratory muscles to generate pressure and [as we previously showed (16)] an increase in respiratory load that occurs early on in the weaning trial.

While the sternomastoid and rib cage muscles had similar timings of activation, indirect evidence suggests that their patterns of activation differed. At a given level of \( \Delta P_{es} \), the coefficient of variation of sternomastoid EMG activity was higher than that for the \( c\Delta P_{pa}/c\Delta P_{es} \) ratio. The greater variability in activation of sternomastoids than in that of the rib cage muscles raises the possibility that behavioral factors may have a greater influence on activation of the sternomastoids than of the rib cage muscles in weaning-failure patients (4).

Unlike the rapid increases in sternomastoid and inspiratory rib cage muscle activities, recruitment of the expiratory muscles was slower throughout the trial (Fig. 2, bottom). Moreover, half the increase in expiratory muscle activity in the failure patients did not occur until the fourth sextile (~13 min into the trial): the expiratory rise in \( P_{pa} \) was 22, 44, 47, 59, 69, and 100% of the final value for each successive sextile between the start and end of the trial. Of note, the largest increase in expiratory rise in \( P_{pa} \) occurred between the fifth and sixth sextile (17–20 min into the trial). The relatively late activation of the expiratory muscles suggests a hierarchy of muscle recruitment (specific muscle groups may be recruited in a particular sequence). The existence of such a hierarchy is supported by the known delayed activation of the expiratory muscles in healthy volunteers (23, 53) and in ambulatory patients with COPD (6).

In summary, the respiratory muscles of patients who fail a weaning trial present a sequential pattern of recruitment. The sequence begins with activity of the diaphragm and greater-than-normal activity of the inspiratory rib cage muscles; recruitment of sternomastoids and rib cage muscles is near maximum within 4 min of trial commencement; and the expiratory muscles are recruited at the slowest pace of all. In conclusion, not only is activity of the inspiratory rib cage muscles increased during a failed weaning trial, but the respiratory centers also recruit the sternomastoid and expiratory muscles as a mechanism for offsetting the effects of an increased load on a weak diaphragm.

GRANTS

This work was supported by grants of the Veterans Administration Research Service.

REFERENCES
