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Lung Volumes, Mechanics, and Oxygenation during Spontaneous Positive-Pressure Ventilation: the Advantage of CPAP Over EPAP

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To determine if continuous positive airway pressure (CPAP) or expiratory positive airway pressure (EPAP) is superior for achieving or maintaining effective lung volume in spontaneously breathing critically ill patients in acute respiratory failure, the authors measured functional residual capacity (FRC), airway and esophageal pressures, and arterial oxygen tensions when CPAP and EPAP were 5 and 10 cm H_2O. Arterial oxygenation, FRC, and transpulmonary pressure at end-expiration were greatest when CPAP was 10 cm H_2O. Lung compliance did not change. The authors conclude that CPAP at 10 cm H_2O is the more effective technique, either because it allows relaxation of chest wall musculature on expiration, or because EPAP at 10 cm H_2O increases chestwall muscle tone. (Key words: Lung: compliance; functional residual capacity. Ventilation: airway pressure; failure; oxygen tension (gradients); positive end-expiratory pressure.)

POSITIVE AIRWAY PRESSURE has been widely used in spontaneously breathing intubated patients during weaning from mechanical ventilation[1] and in the treatment of respiratory distress syndrome[2–6] and atelectasis.[4] At present, it can be applied in two ways: as continuous (CPAP) or expiratory positive airway pressure (EPAP).[5,7–9,¶] During CPAP, airway pressure remains poitive during the entire respiratory cycle; during EPAP, the inspiratory phase returns to ambient airway pressure. The relative efficacy of these two modes in critically ill patients is unclear.

Without actually comparing the effects of these two modes, Gillick[5] and Greenbaum *et al.*[8] suggested that EPAP is an effective yet simplified alternative to CPAP in the treatment of selected patients with adult respiratory distress syndrome (ARDS). Sturgeon *et al.*[7] demonstrated that the effects of EPAP on cardiovascular function were superior to those of CPAP when applied to postoperative aortocoronary bypass patients who presumably had normal lungs. It has been suggested that the work of breathing with EPAP is greater than with CPAP.[5,8,10] This was confirmed in a study of athletes who had excellent pulmonary function.[11]

We describe the comparative effects of CPAP and EPAP on functional residual capacity (FRC), transpulmonary pressures, and arterial oxygenation in two groups of spontaneously breathing critically ill patients in acute respiratory failure.

ABBREVIATIONS

BTPS = body temperature and pressure saturated with water vapor
C_L = dynamic pulmonary compliance
$C_{L_{sp}}$ = the ratio of specific dynamic pulmonary compliance to C_L/FRC
$\Delta C_{L_{sp}}$ = difference (between EPAP and CPAP) in specific dynamic pulmonary compliance
CPAP = continuous positive airway pressure
EPAP = expiratory positive airway pressure
$F_{I_{O_2}}$ = fractional concentration of inspired oxygen
FRC = functional residual capacity
ΔFRC = difference in FRC (between EPAP and CPAP)
$P(A\text{-}a)_{O_2}$ = alveolar-arterial oxygen partial pressure difference
$P(A\text{-}a)_{O_2}$ 1.0 = $P(A\text{-}a)_{O_2}$ at an $F_{I_{O_2}}$ of 1.0
Pa_{CO_2} = arterial carbon dioxide partial pressure
Pa_{O_2} = arterial oxygen partial pressure
$P_{A_{O_2}}$ = alveolar oxygen partial pressure
P_{aw} = airway pressure
ΔP_{aw} = difference in airway pressure at end-inspiration and end-expiration
P_{es} = esophageal pressure
ΔP_{es} = absolute change (inspiration minus expiration) in esophageal pressure
$P_{I_{O_2}}$ = inspired oxygen partial pressure
P_{tp} = transpulmonary pressure
ΔP_{tp} = difference (between EPAP and CPAP) in transpulmonary pressure
STPD = O°C, 760 torr, dry
TLC = total lung capacity
$\dot{V}_{O_2}$ = oxygen consumption

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¶ As Greenbaum *et al.*[8] and Grenvik,[7] we prefer the more descriptive term "EPAP" to "PEEP" and "sPEEP", because PEEP is usually associated with mechanical ventilation.

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Materials and Methods

We studied 18 intubated, spontaneously breathing, supine patients in the ICU at San Francisco General Hospital. Twelve were comatose due to severe cranial trauma or metabolic encephalopathy, and six had recently been weaned from mechanical ventilation after major abdominal trauma (five patients) and smoke inhalation (one patient). Three of 18 patients had a history of chronic obstructive pulmonary disease. The study consent was obtained according to protocols approved by the Committee on Human Research of the University of California, San Francisco.

CPAP was produced by a high-flow air-oxygen blender through a humidifier, a reservoir bag, and a T-piece leading to the patient.[9] Exhalation occurred through a water-column threshold resistor (fig. 1*A*). The system was thus pressurized from the reservior during inspiration and from the water column during exhalation. Although the expiratory limb for EPAP was identical to that of CPAP, the inspiratory limb received its air-oxygen mixture from a venturi nebulizer and had a small reservoir that was open to ambient pressure (fig. 1*B*). A one-way valve ensured unidirectional flow. The patient thus inspired at ambient pressure and exhaled at positive expiratory pressure.

Experimental Protocol

In the first ten patients (Group I), we compared the effects of CPAP with EPAP (on FRC and arterial oxygenation) at end-expiratory pressures of 5 and 10 cm H_2O in the supine position. Each patient received CPAP = 5, CPAP = 10, EPAP = 5, and EPAP = 10. We also evaluated the possible effects of the application sequence of CPAP and EPAP. Figure 2 shows the sequence used for half the patients in this study group. The opposite mode sequence, EPAP-CPAP-EPAP, was used in the other five patients. Thus, the initial mode was always repeated after the opposite mode. Similarly, the initial end-expiratory pressure of 5 cm H_2O was always repeated after the 10 cm H_2O level. Measurements were made over a 5-min interval after a 15-min period of equilibration for each mode and level of end-expiratory pressure. The three patients who had chronic obstructive pulmonary disease were in this group.

After finding comparative increases in FRC and arterial oxygenation with CPAP = 10, we compared CPAP = 10 with EPAP = 10 in eight additional patients (Group II) who were in Fowler's position (truncal elevation of 45–60°). The second protocol was simplified to two steps (CPAP before EPAP in six patients, CPAP after EPAP in two patients), and now included esophageal pressure (P_{es}) to separate chest wall and lung factors.

Measurements

Functional residual capacity was measured by the closed-circuit helium-dilution technique. We used an apparatus described by Suter and Schlobohm[12] that was modified for use in spontaneously breathing patients, and that allowed for application of EPAP or CPAP during measurements of FRC. Tidal volumes and respiratory

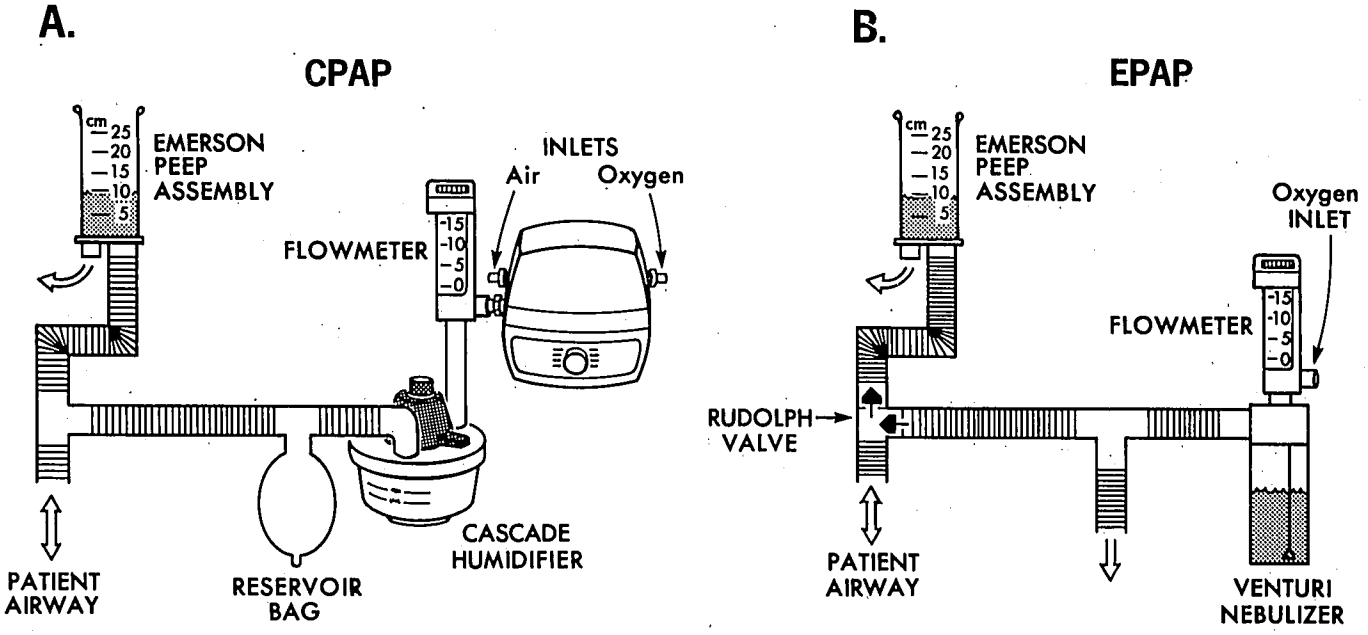

FIG. 1. *A*. Schematics of the continuous positive airway (CPAP) and *B*. The expiratory positive airway pressure (EPAP) circuits used in the study.

rates were determined using the spirometric recordings of this apparatus. Oxygen consumption ($\dot{V}_{O_2}$) was determined by measuring the slope of the spirometric tracing. Arterial oxygen and carbon dioxide tensions and *p*H were measured with a Corning® 175 blood-gas analyzer, and the results were corrected to the patient's temperature.

In Group I patients, alveolar-to-arterial oxygen partial pressure difference $P(A\text{-}a)_{O_2}$ was calculated prior to system closure (for determination of FRC), but after 7 min at a fractional concentration of inspired oxygen ($F_{I_{O_2}}$) of 1.0, assuming

$$P_{A_{O_2}} = P_{I_{O_2}} - P_{a_{CO_2}}\left[F_{I_{O_2}} + \frac{1 - F_{I_{O_2}}}{0.85}\right]$$

where $P_{A_{O_2}}$ is the calculated ideal alveolar oxygen partial pressure.

In the Group II patients, $P_{a_{O_2}}$ was measured at the $F_{I_{O_2}}$ that resulted in clinically acceptable arterial oxygenation. This $F_{I_{O_2}}$ was not changed throughout ($F_{I_{O_2}}$ range = 0.21–0.40; $P_{a_{O_2}}$ range = 66–163 torr).

Airway and esophageal pressures (P_{aw} and P_{es}) were measured simultaneously with Statham® pressure transducers (Models P23DB and PM 131 TC), which were precalibrated with a water manometer. Esophageal pressure was measured with a nasogastric esophageal balloon** 10 cm long and 3.8 cm in circumference.[13] The tip of the balloon was placed in the mid to lower third of the esophagus[14] by passing the empty balloon into the stomach and inflating it with 0.5 ml of air. Using continuous recordings of P_{es}, we verified the balloon to be in the stomach by observing a positive deflection of P_{es} during inspiration. The balloon was then withdrawn to a position in the esophagus where the cardiac artifact was minimal and the end-expiratory P_{es} most negative.[15] This position was 36–44 cm from the tip of the balloon to the nares.

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Calculations and Statistical Analysis

Using physical characteristics of sex, age, height, and weight, we calculated a predicted normal value for FRC (Appendix A). The transpulmonary pressure (P_{tp}) was calculated as the difference between P_{aw} and the corresponding P_{es}, assuming P_{aw} and P_{es} to be representative of alveolar and intrapleural pressures, respectively.[16] An approximate dynamic lung compliance (C_L) for each condition was calculated from the tidal volume and change in transpulmonary pressure (ΔP_{tp}) between inspiration and expiration. To correct for differences in lung volume between patients, specific dynamic compliance ($C_{L_{sp}}$), a ratio of C_L/FRC, was derived. It has been observed that in the absence of alterations associated with increased airway resistance, dynamic compliance relationships are a reflection of static compliance.[17] Calculations were based on the average of data obtained from at least ten consecutive breaths during measurements in each condition.

To assess the relationship of ventilatory mode, sequence of previous ventilatory mode, and level of end-expiratory pressure in Group I, data were analyzed using a three-factor repeated-measures analysis of variance.[18] When significant differences were found, the method of least-significant difference was used to compare specific group means.[18] In Group II, the data generally lacked a normal distribution and were analyzed using the nonparametric Wilcoxon's signed rank test and Spearman's rank correlation.[19] Data are presented as means ± SEM.

Results

Sequence Effects in the First Protocol

We assumed that the effects of the four conditions (CPAP = 5, CPAP = 10, EPAP = 5, and EPAP = 10) would not carry forward to the next condition. The sequence of their application (fig. 2) in the first protocol was designed to test this hypothesis. In general, measurements that repeated those of earlier applications were

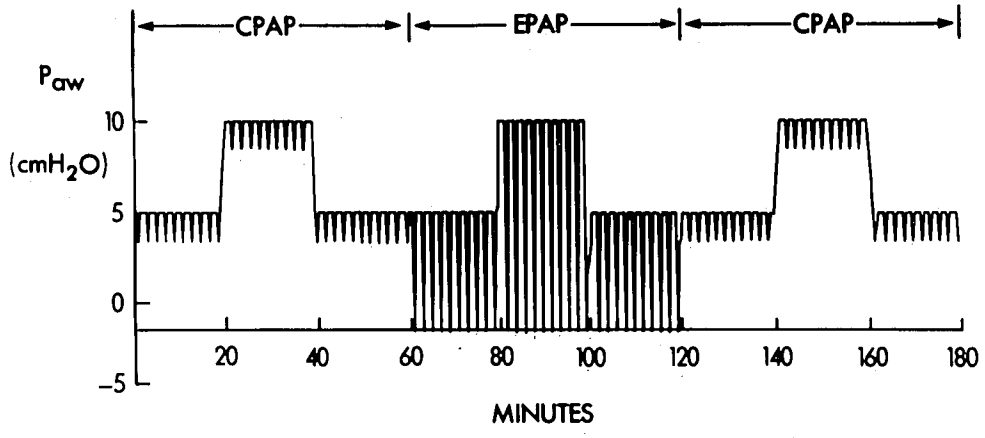

FIG. 2. A representation of the sequence used for applying CPAP and EPAP in one-half the Group I patients. The airway pressure wave forms represent typical levels occurring during each mode and at each level of end-expiratory pressure; however, the typical respiratory rate was 18 breaths/min.

TABLE 1. Comparison of EPAP and CPAP at 5 cm H_2O End-expiratory Pressure (Mean ± SE) in the Ten Group I Supine Patients

	EPAP	CPAP	*P*
FRC, ml (BTPS)	2,240 ± 460	2,429 ± 456	< 0.05
Per cent predicted	98 ± 13	114 ± 16	< 0.025
Tidal volume, ml (BTPS)	494 ± 35	495 ± 34	NS
$P(A\text{-}a)_{O_2}$ 1.0, torr	287 ± 12	266 ± 11	NS
ΔP_{aw}, cm H_2O	5.6 ± 0.2	2.5 ± 0.2	< 0.001
O_2 consumption, ml/min (STPD)	247 ± 25	239 ± 15	NS

See list for abbreviations.
P levels were derived using a three-factor repeated-measures analysis of variance. When significant differences were found, the method of least-significant difference was used to compare specific group means.[18]

not statistically different from measurements during any preceding identical application. Three specific exceptions are noted in Appendix B.

EFFECTS OF END-EXPIRATORY PRESSURE ON FRC

In Group I, FRC at EPAP = 5 was close to the value predicted for normal supine subjects (table 1). Elevation to EPAP = 10, along with both levels of CPAP, increased FRC to as much as 149 per cent of predicted normal (tables 1 and 2). FRC was higher with CPAP than with EPAP (tables 1–3) in both groups. Although elevation of end-expiratory pressure from 5 to 10 cm H_2O increased FRC, CPAP produced an even greater increase (tables 1 and 2). Tidal volumes were not affected by mode or level of end-expiratory pressure (tables 1–3).

EFFECTS OF END-EXPIRATORY PRESSURE ON OXYGENATION

At CPAP = 10, $P(A\text{-}a)_{O_2}$ 1.0 was smaller (table 2) and Pa_{O_2} was larger (table 3) than at EPAP = 10. However, no difference in $P(A\text{-}a)_{O_2}$ 1.0 occurred between CPAP = 5 and EPAP = 5 (table 1). Neither mode nor level of end-expiratory pressure altered oxygen consumption, *p*H, or P_{CO_2}.

EFFECTS OF END-EXPIRATORY PRESSURE ON LUNG MECHANICS

The difference in P_{aw} at end-inspiration and end-expiration (ΔP_{aw}) was greater with EPAP than with the corresponding level of CPAP (tables 1–3). Elevation of EPAP from 5 to 10 cm H_2O increased ΔP_{aw} (tables 1 and 2). The large ΔP_{aw} during EPAP = 10 is to be expected, because the circuit design requires inspiration to occur at ambient pressure (fig. 1*B*).

The pressure-volume relationships of Group II patients at CPAP = 10 and EPAP = 10 are illustrated in figure 3. Transpulmonary pressures and lung volumes were greater in the CPAP = 10 mode at end-expiration (table 3). Lung compliance and the end-inspiratory pressure-volume values were not different between modes. This suggests that the pressure-volume relationship for CPAP = 10 is identical to that for EPAP = 10. The difference in FRC between the two modes (ΔFRC) correlated positively with the corresponding difference in P_{tp} (ΔP_{tp}) ($r_s = 0.80$, $P < 0.05$). However, specific dynamic compliance ($C_{L_{sp}}$) did not differ between EPAP = 10 and CPAP = 10 (table 3), and there was no correlation when ΔFRC was compared with the corresponding difference in $C_{L_{sp}}$ ($\Delta C_{L_{sp}}$).

Since there was no difference in end-expiratory P_{aw} during breathing with EPAP and CPAP, the greater end-expiratory P_{tp} during CPAP was entirely related to a lower P_{es} (table 3). Figure 4 illustrates this difference in a P_{es} tracing from a representative subject. No difference was found in P_{tp} between the two modes at end inspiration. However, absolute change (inspiration minus expiration) in P_{es} (ΔP_{es}) and P_{aw} was greater with EPAP (table 3).

Discussion

Positive airway pressure increases FRC in spontaneously breathing healthy subjects.[11,20-22] Whether ad-

TABLE 2. Mean (± SE) Values for EPAP and CPAP at 10 cm H_2O End-expiratory Pressure in Ten Group I Supine Patients

	EPAP	CPAP	*P*
FRC, ml (BTPS)	2,538 ± 556	3,446 ± 596	< 0.001
Per cent predicted	112 ± 17	149 ± 15	< 0.005
Tidal volume, ml (BTPS)	456 ± 34	480 ± 41	NS
$P(A\text{-}a)_{O_2}$ 1.0, torr	307 ± 22	249 ± 23	< 0.01
ΔP_{aw}, cm H_2O	11.0 ± 0.4	2.8 ± 0.5	< 0.001
O_2 consumption, ml/min (STPD)	246 ± 25	249 ± 20	NS

See list for abbreviations
P levels were derived as in Table 1.

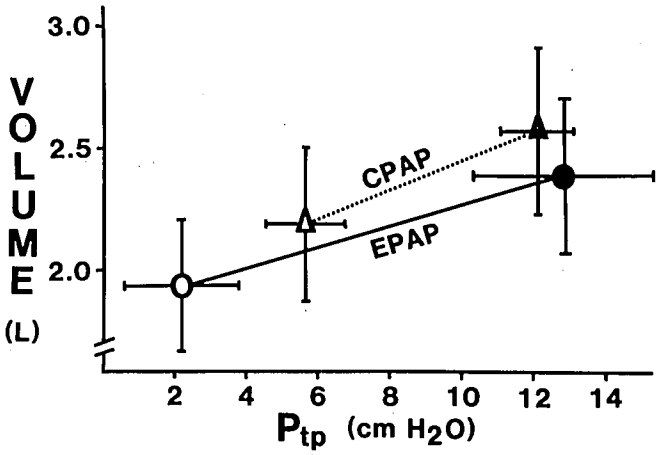

FIG. 3. Pressure-volume relationships for CPAP = 10 and EPAP = 10 in eight patients in Fowler's position. Solid symbols represent end-inspiration and open symbols represent FRC. Triangles represent the CPAP mode and circles indicate EPAP. At end-expiration, FRC and transpulmonary pressures during CPAP were greater than during EPAP. Bars represent ± 1 SEM.

ministered as CPAP[1-4,6,8] or as EPAP,[5,8] it improves arterial oxygenation in patients with respiratory failure. Our data indicate that CPAP is the more effective mode (tables 2 and 3). FRC at both CPAP = 5 and 10 was greater than the corresponding level of EPAP. These findings are consistent with those of Gherini *et al.*,[11] who also found larger FRCs with CPAP in healthy subjects. In our patients, oxygenation also improved, even though at CPAP = 10 (Group I), FRC was 50 per cent above predicted levels.

We observed that CPAP = 10 resulted in a higher P_{tp} at end-expiration than EPAP = 10, and that the higher P_{tp} correlated with an increase in FRC. These findings necessarily result from differences in the mechanical properties of either the lung or the chest wall, or both. A change in the slope (compliance) of the pressure-volume curve of the lung in the spontaneous-breathing tidal-volume range usually reflects a change in the elastic properties of the lung. Since there was no difference in the specific dynamic lung compliance between CPAP = 10 and EPAP = 10, it is unlikely that the observed increase in FRC with CPAP is a result of differences in the elastic properties of the lung.

Alternatively, chest wall factors alone might explain the observed differences in FRC and P_{tp}. A change in chest wall elastic recoil may alter FRC and P_{tp} but not lung elastic properties.

Two mechanisms may be responsible for a difference in chest wall elastic recoil between EPAP and CPAP. First, EPAP could increase expiratory muscle tone. Our common clinical observation that many patients receiving EPAP breathe with an end-expiratory squeeze (which will decrease FRC and P_{tp}) is evidence supporting this mechanism. Second, CPAP could have produced a progressive relaxation of the chest wall musculature, resulting in an increase in chest wall elastic recoil and a consequent increase in lung volume and P_{tp}. This could have occurred due to the phenomenon of CREEP.[23] CREEP is a viscoelastic property of both the lungs and chest wall, the definition of which is a time-dependent increase in lung volume at a constant pressure. This has been observed in normal dogs,[23] humans,[24] and in patients with acute respiratory failure receiving continuous positive-pressure ventilation.[25] Therefore, the observed increases in FRC may be related to an above-ambient

TABLE 3. Lung Volumes, Gas Exchange, and Respiratory Pressure during EPAP and CPAP at 10 cm H_2O in Eight Group II Patients (Fowler's Position)

	EPAP	CPAP	*P*
Function residual capacity, ml (BTPS)	1,935 ± 268	2,188 ± 315	<0.05
Per cent predicted at 30 degrees*	85 ± 10	95 ± 10	<0.05
Tidal volume, ml (BTPS)	500 ± 67	426 ± 39	NS
Respiratory rate, breaths/min	21 ± 2	19 ± 2	<0.05
Pa_{O_2}, torr	111 ± 13	126 ± 13	<0.05
Dynamic lung compliance, ml × cm H_2O^{-1}	68 ± 14	74 ± 11	NS
Specific dynamic compliance, cm H_2O^{-1}	0.046 ± 0.015	0.041 ± 0.010	NS
Airway pressure, cm H_2O			
End-inspiratory	−1.8 ± 0.7	8.3 ± 0.2	<0.05
End-expiratory	10.3 ± 0.2	10.2 ± 0.2	NS
Absolute change	12.4 ± 0.7	1.9 ± 0.2	<0.05
Esophageal pressure, cm H_2O			
End-inspiratory	−14.4 ± 2.9	−4.0 ± 1.0	<0.05
End-expiratory	8.0 ± 1.5	4.6 ± 1.1	<0.05
Absolute change	22.4 ± 4.1	8.6 ± 1.0	<0.05
Transpulmonary pressure, cm H_2O			
End-inspiratory	12.9 ± 2.3	12.2 ± 1.0	NS
End-expiratory	2.2 ± 1.5	5.7 ± 1.0	<0.05
Absolute change	11.0 ± 3.4	6.5 ± 0.9	<0.10

See list for abbreviations.
* See Appendix A.
P levels were derived using the Wilcoxon's Signed Rank Test.[19]

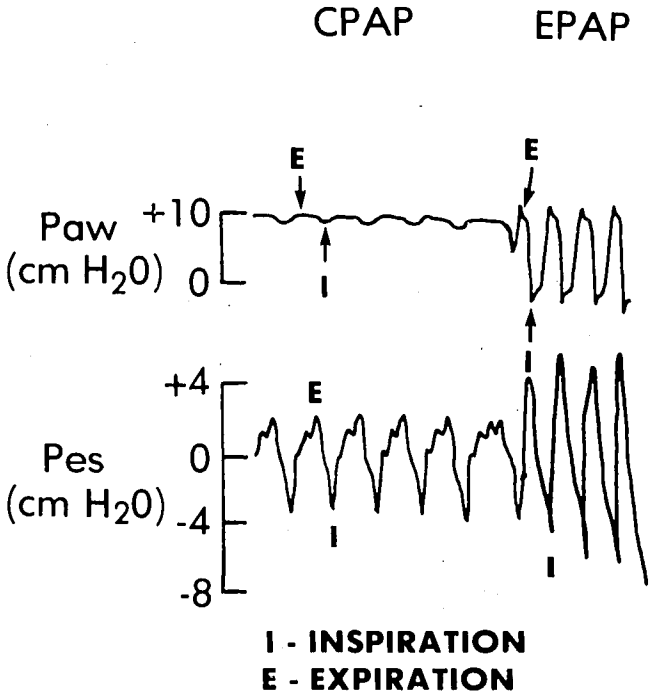

FIG. 4. Typical recordings of airway (P_{aw}) and esophageal (P_{es}) pressures during CPAP = 10 and EPAP = 10. End-expiratory points are at (E) while end-inspiratory points are at (I). Although end-expiratory P_{aw} during CPAP does not differ from that occurring during EPAP, end-expiratory P_{es} is lower during CPAP.

airway pressure that is constantly present during all phases of the respiratory cycle with CPAP, but only intermittently present during expiration with EPAP.

The mechanisms responsible for the greater efficiency for CPAP must be effective only during expiration, since with both CPAP = 10 and EPAP = 10, the comparative end-inspiratory transpulmonary pressures were not significantly different.

The change in FRC between modes (CPAP = 10 *vs.* EPAP = 10) was greater in the supine Group I patients (3,446 ml *vs.* 2,538 ml, or 112 *vs.* 149 per cent, table 2), than in Group II patients (2,188 ml *vs.* 1,935 ml, or 85 *vs.* 95 per cent, table 3), who were in Fowler's position. Therefore, mechanisms that account for the greater effects of CPAP are probably more pronounced in the supine position. Comparison of data between groups must be approached with caution as they may only identify different populations of patients. Thus, population and methodology may account for the relatively larger FRC values seen in CPAP = 10 and EPAP = 10 of Group I compared with Group II (tables 2 and 3). Similarly, for Group I, data for FRC when CPAP = 5 appears larger than that reported by Annest *et al.*,[26] or the EPAP = 5 of Quan *et al.*[27]

Although FRC and Pa_{O_2} were greater during CPAP, the subjects were in each mode for minimum of 15 min before measurements were obtained. The effect of a longer time course of breathing in each mode is not known.

Oxygen consumption was not different between modes (table 2), but the wide difference in absolute esophageal pressure (ΔP_{es}) between modes (table 3) is consistent with a recent observation that the work of breathing is greater with EPAP in normal individuals.[11]

In conclusion, CPAP is more effective than EPAP in increasing FRC and improving arterial oxygenation. These findings are probably related to chest wall factors occurring during end-expiration.

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APPENDIX A

There are many sets of normal FRC data for sitting men and women of different heights, ages, and weights, but few that correlate these attributes with normal subjects in the semi-recumbent and supine positions. FRC progressively decreases 500–1000 ml from the upright to supine positions.[28]

1) *Normal FRC in the supine position (FRC_{sup}).* Our algorithm required these three steps:

a) We computed total lung capacity for the sitting position (TLC_{sit}) using equations from Grimby and Söderholm,[29] who studied 152 normal men (20–65 years of age) and 58 normal women (18–72 years of age):

$$TLC_{sit} = 6.92 \times m - 0.017 \times kg - 4.30 \pm 1.34 \text{ in men}$$

$$TLC_{sit} = 6.71 \times m - 0.015 \times yr - 5.77 \pm 0.96 \text{ in women}$$

where TLC_{sit} is given in liters, m is height in meters, kg is weight in kilograms, and yr is age in years. The limit of statistical reliability is ± two standard deviations from the mean.

b) TLC_{sit} was converted to FRC_{sup} using the data of Whitfield and co-workers,[30] who studied and collated data from 148 normal men and 66 normal women:

$$FRC_{sup} = (0.393 \pm 0.011) \times TLC_{sit} \text{ in men}$$

$$FRC_{sup} = (0.430 \pm 0.008) \times TLC_{sit} \text{ in women}$$

where FRC supine is given in liters and the limit of statistical reliability is one standard error of the mean.

c) By combining the above equations, we derived the following equations:

$$FRC_{sup} = [(6.92 \times m) - (0.017 \times kg) - (4.30)] \times 0.393 \text{ in men}$$

$$FRC_{sup} = [(6.71 \times m) - (0.015 \times yr) - (5.77)] \times 0.430 \text{ in women}$$

2) *Normal FRC at 30 degrees above supine ($FRC_{30°}$).* Data sufficient for correlation with the above attributes at the 45- to 60-degree elevation of study Group II do not exist. As a substitute, we derived algorithms for the 30-degree position FRC as follows:

a) We utilized the following equation from Boren *et al.*,[31] who studied 422 normal men (20–66 years of age) related to

15 different Veterans Administration and Army Hospitals (69 per cent of the patients had a history of smoking, which produced a slightly larger FRC):

$$FRC_{30°} = 3.2 \times m - 2.94 \pm 0.63 \text{ in men}$$

where $FRC_{30°}$ is given in liters, and the limit of statistical reliability is one standard error of the mean.

b) Finding no comparable data for women, we relied on the data of Grimby and Soderholm[29] to assume that, at equal heights, the female FRC is about 90 per cent of the male. Thus, we computed the "normal $FRC_{30°}$" of the four men and four women of Group II.

Appendix B

Repetition of CPAP and EPAP at 5 and 10 cm H_2O throughout the first study protocol did not yield statistically different results, except in the following three comparisons. In CPAP = 5, initial FRC was smaller than its final value measured eight steps later (fig. 2) ($2285 \pm 442 < 2575 \pm 415$ ml, SE, $P < 0.025$, n = 5). In CPAP = 10, initial FRC was greater than its final value determined six steps later (fig. 2) ($3624 \pm 534 > 3127 \pm 446$ ml, $P < 0.05$, n = 5). In EPAP = 5, initial $P(A\text{-}a)_{O_2}$ 1.0 was greater than its final value determined eight steps later ($364 \pm 47 > 226 \pm 20$ torr, $P < 0.05$, n = 5).

This failure to return to original values suggests the existence of a long-term CPAP, EPAP, or end-expiratory pressure effect and/or an additional effect of the protocol sequence itself. These effects may produce the step expansion of FRC seen in exceptions one and three, but do not similarly apply to the second exception. Also, the expected complementary findings did not accompany any of these exceptions, such as a decreased $P(A\text{-}a)_{O_2}$ 1.0 accompanying an increased FRC, and vice versa. Thus, these data are presented without explanation. We are unable to offer a uniform thesis to explain these three exceptions and believe that the findings of the first and third exceptions are diametrically opposed to those of the second exception.

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