Lung Volumes and Closing Capacity with Continuous Positive Airway Pressure

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Total lung capacity, vital capacity, residual volume, and functional residual capacity were determined by body plethysmography and the single-breath oxygen (SBO2) test was performed at 0, 5, and 11 cm H2O continuous positive airway pressure in healthy, awake, seated, spontaneously breathing subjects. Mean values for the absolute lung volume at which phase IV of the SBO2 test begins (closing capacity) did not change significantly with continuous positive airway pressure at 5 or 11 cm H2O. Mean total lung capacity, functional residual capacity, and residual volume increased significantly, and the mean closing volume, the lung volume above residual volume at which phase IV begins, decreased significantly with 11 cm H₂O continuous positive airway pressure; differences at 5 cm H2O were not significant. The slope of the alveolar nitrogen plateau (phase III) obtained during the SBO2 test did not change with continuous positive airway pressure. (Key words: Lung: closing capacity; Ventilation, mechanical: closing capacity.)

ARTERIAL HYPOXEMIA is said to occur during tidal breathing if closing capacity (CC)§

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Closing capacity is the absolute lung volume at which phase IV of the single-breath oxygen test begins; closing volume is the volume above residual volume at which phase IV begins.

exceeds functional residual capacity (FRC).1 The marked decrease in FRC frequently observed in patients with acute respiratory failure2 might therefore lead to arterial hypoxemia during tidal breathing. Indeed, the improvement in arterial oxygen levels associated with continuous positive airway pressure (CPAP) during spontaneous breathing or with positive end-expiratory pressure (PEEP) during mechanical ventilation has been attributed, at least in part, to an increase in FRC.3 which apparently re-establishes the normal relationship between FRC and CC. An optimal effect of CPAP or PEEP on pulmonary gas exchange can be expected if FRC increases and CC remains unaffected or becomes smaller. A communication recently published4 and discussed5 in this Journal stated that CC** increased with CPAP: when airway pressure was increased from 0 to 10 cm H₂O CPAP, marked increases in FRC (1.78 liters) and in CC (0.47 liter) were observed; thus, mean FRC-CC increased by 1.31 liters with CPAP. However, this increase in CC was unexpected and was attributed to the

ABBREVIATIONS

CC = closing capacity

CPAP = continuous positive airway pressure

CV = closing volume FRC = functional residual capacity

FRC = functional residual capacity

MEFV = maximal expiratory flow volume P_{ao} = pressure at the airway opening

PEEP = positive end-expiratory pressure

RV = residual volume

SBO₂ test = single-breath oxygen test

TLC = total lung capacity

VC = vital capacity

Vt = thoracic gas volume

^{**} In that communication, "CV (1)" is the same as "CC" in this paper.

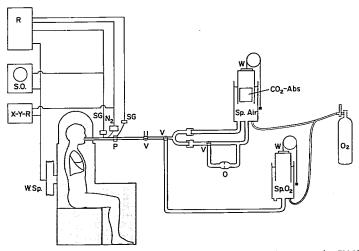


FIG. 1. Breathing circuit. W. Sp. = wedge spirometer; SG = strain gauge (SG closest to mouth is PM-I31 for measurement of P_{ao}); N_2 = nitrogen meter; P = pneumotachograph; V = three-way valves; O = 3-mm-diameter orifice; Sp. Air = air-filled spirometer; Sp. O_2 = O_2 -filled spirometer; W = weights; CO_2 -Abs = CO_2 absorber; R = recorder; S.O. = storage oscilloscope; X-Y-R = X-Y recorder; O_2 = tank containing compressed analyzed oxygen.

large increase, 0.81 liter, in residual volume (RV). In that study, FRC was determined at 0 cm H₂O airway pressure and the changes in FRC with CPAP were measured from the displacement of the weighted spirometer bell.

Because accurate determination of the absolute lung volume is critical for the estimation of CC, RV, and total lung capacity (TLC), it seemed important to measure the effects of CPAP on CC and lung volumes in normal man in a body plethysmograph to achieve conditions such that absolute lung volumes could be determined repeatedly. When we did these studies, we found no increase in CC with CPAP and observed an increase of only 0.2 liter in the mean values of RV and TLC, a finding that agrees with the results of Rahn and co-workers.⁶

Methods

Total lung capacity (TLC), its subdivisions (RV, VC, FRC), and closing volume (CV) were determined at 0, 5 (range, 4.5 to 6.0), and 11

(range, 10.0 to 12.0) cm H₂O CPAP in four subjects (ages, 26 to 43 years; heights, 1.63 to 1.83 m; weights, 64 to 93 kg) and at 0 and 11 cm H₂O CPAP in five additional subjects (ages, 30 to 45 years; heights, 1.58 to 1.88 m; weights, 59 to 83 kg). Maximal expiratory flow-volume (MEFV) curves were obtained for five of the nine subjects at 0, 5, and 11 cm H₂O CPAP.

The breathing circuit (fig. 1) for the study consisted of an air-filled spirometer with CO₂ absorber. To maintain expiratory flow rates of approximately 0.5 liter/s during the single-breath oxygen (SBO₂) test, expiration was directed through a 3-mm-diameter orifice incorporated into a bypass of the expiratory line. An O₂-filled spirometer served as an O₂ source for the SBO₂ test and could be connected to the circuit by means of a large-bore three-way valve. The desired level of CPAP was achieved by placing weights on the bells of the spirometers. Pressure at the airway opening (P_{ao}) (Statham strain gauge PM-131), inspiratory and expiratory flow rates (Fleisch

	Mean (#SE) Airway Pressure			
	0	5 ± 0.4	0	11 ± 0.1
TLC (liters)	6.6 ± 0.2	6.8 ± 0.2	6.9 ± 0.3	7.1 ± 0.4†
VC (liters)	5.2 ± 0.2	5.2 ± 0.3	5.5 ± 0.3	5.5 ± 0.3
RV (liters)	1.4 ± 0.1	1.6 ± 0.1	1.4 ± 0.1	1.6 ± 0.1†
FRC (liters)	3.7 ± 0.2	4.1 ± 0.3	3.8 ± 0.2	4.6 ± 0.2†
CC (liters)	2.5 ± 0.2	2.7 ± 0.2	2.5 ± 0.1	2.5 ± 0.2
CV (per cent VC)	21 ± 3	20 ± 3	. 19 ± 2	17 ± 2†
FRC-CC (liters)	1.2 ± 0.1	1.4 · ± 0.3	1.3 ± 0.2	2.1 ± 0.2†
CC/FRC	0.68 ± 0.03	0.65 ± 0.05	0.67 ± 0.03	0.56 ± 0.041
Slope of phase III (per cent N ₂ /liter)	0.7 ± 0.1	0.7 ± 0.1	0.9 ± 0.1	0.9 ± 0.2
Subjects (number)	4	-4	9	9

^{*} Abbreviations: TLC = total lung capacity; VC = vital capacity; RV = residual volume; FRC = functional residual capacity; CC = closing capacity; CV = closing volume.

† Significant difference between 0 and 11 cm H₂O mean airway pressure, P < 0.05.

pneumotachograph), and N₂ concentrations (N₂ meter, 90 per cent response time, 30 ms) of respired gases were detected distal to the mouthpiece and recorded simultaneously. Apparatus deadspace was 212 ml. Thoracic gas volumes (V_{tt}) were determined by a volume-displacement body plethysmograph, and lung volumes (BTPS) were calculated by an on-line computer. Expired N₂ concentrations and gas volumes (wedge spirometer attached to body box) during the SBO₂ test were recorded simultaneously on an analog frequency-modulated tape recorder and later transcribed to an X-Y recorder.

The subject, seated in the body plethysmograph with nose occluded, was connected to the breathing circuit and breathed room air for approximately 4 minutes at ambient mean airway pressure. Each SBO₂ test was initiated by the determination of V₁₆, so that all events could be expressed at absolute lung volume. The subject exhaled to RV, inspired 100 per cent O₂ slowly to TLC, and expired immediately at a flow rate of approximately 0.5 liter/s through the 3-mm-diameter orifice. Three satisfactory maneuvers were performed. The process was repeated after 4 minutes at each of the two levels of CPAP.

No correction for the 30-ms time delay of the N2 meter was used. A visual best-fit straight line was drawn through the last two thirds of phase III, and the beginning of the permanent deviation from this line was taken as the beginning of phase IV. We found it difficult, in some instances, to identify exactly the take-off point of phase IV. To decrease observer bias, the phase IV measurements were carried out independently by four observers who had no knowledge of the experimental condition. In a previous study,9 one of us (R.E.H.) had measured phase IV in duplicate on the same record and found a mean difference of approximately I per cent of the average VC. The slope of phase III is expressed in per cent N2/liter expired gas volume.

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Statistical significance was determined by Student's two-tailed t test for paired data.

Results

The results are summarized in table 1. Mean values for VC and CC did not change significantly with 5 or 11 cm H₂O CPAP. Mean values for TLC, FRC, and RV and the difference between FRC and CC (FRC-CC)

increased with CPAP, while mean values for CV and CC/FRC decreased with CPAP. Differences in mean values achieved statistical significance only at 11 cm H₂O CPAP (fig. 2). The mean (±SE) slopes of phase III obtained during the SBO₂ test were 0.7 ± 0.1 and 0.9 ± 0.1 per cent N₂/liter at ambient mean airway pressure in the four and nine subjects, respectively; the slope was unchanged with CPAP. We could detect no consistent effect of CPAP on the MEFV curve.

Discussion

Although Craig and McCarthy have reported a mean increase in CC of 0.47 liter with 10 cm H₂O CPAP, we found no change in mean CC with 11 cm H₂O CPAP.

Consideration of the determinants of CC suggests no reason that CC should increase with CPAP. As measured by the SBO₂ test, CC depends on 1) the vertical differences in regional RV/TLC ratios, 2) the distribution of the inspired deadspace gas, and 3) the sequence of regional lung emptying. Since CC is the same whether inspiration is initiated from RV or FRC,* the increase in RV with CPAP should not affect CC. Likewise, the small increase in TLC should have little if any effect on the vertical gradient of alveolar N₂ concentration.

A change in the regional pressure-volume behavior of the lung could change regional RV/TLC ratios and the sequence of emptying. However, we found no change in the overall lung pressure-volume curve as estimated with the esophageal balloon technique (Rehder et al., unpublished data). It seems unlikely that there could be appreciable changes in regional lung pressure-volume behavior that fortuitously would result in the same overall pressure-volume curve. Similarly, we found no change in the vertical gradient of the transpulmonary pressure with CPAP in awake man (Rehder et al., unpublished data), unlike the observations by Agostoni and associates10,11 in anesthetized animals. MEFV curves are another means of assessing the overall mechanical behavior of the lung and are dependent on the lung pressure-volume curve.12,13 We found no change in the MEFV curve with CPAP.

Lemelin and co-workers14 showed that the

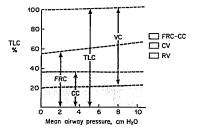


FIG. 2. Total lung capacity (TLC) and subdivisions (expressed as percentage of control TLC) at ambient (zero) and positive mean airway pressure. Note the decrease in closing volume (CV) and the related increase in residual volume (RV), resulting in an unchanged closing capacity (CC). Note also the small increase in TLC, which was similar to that of RV, resulting in an unchanged vital capacity (VC).

distribution of inspired gas was not significantly affected by CPAP. In our study, the slope of phase III was not changed by CPAP, suggesting that the intrapulmonary distribution of inspired gas and the sequence of emptying during expiration were unchanged with CPAP. In summary, it appears that CC should not increase with CPAP in normal man.

What then is the cause for the difference between our finding of no increase in CC with CPAP and the opposite finding by Craig and McCarthy?4 Both studies showed that VC was unchanged and that CV decreased with CPAP. Clearly, then, the difference lies in the measurement of the absolute lung volume. Craig and McCarthy determined the absolute volume of FRC at ambient airway pressure. FRC during CPAP was measured from the displacement of the weighted spirometer bell, corrected for the compression volume of the system. They found an increase of 1.78 liters with 10 cm H2O CPAP. This increase is considerably greater than what has been reported15 in a previous study utilizing the weighted spirometer and also is greater than what would be anticipated from the data of Rahn and coworkers.6 Both these studies6.15 pointed out that, if special efforts are not made to achieve complete muscular relaxation, respiratory reflexes tend to diminish the increase in FRC with CPAP. Our finding of a mean increase

in FRC of 0.8 liter, which is lower than the increase (1.1 liters) that would be predicted from the normal compliance of the respiratory system (0.1 liter/cm H₂O),¹⁶ is consistent with incomplete muscular relaxation.

If the increase of the FRC were overestimated in Craig and McCarthy's study,* TLC, RV, and CC would be overestimated by the same amount. The increases in TLC and RV in Craig and McCarthy's study exceed the increases of TLC and RV in our study by an amount approximately the same as the increase in CC they reported. The increases of RV and TLC that we observed are consistent with the data of Rahn and co-workers.⁶

In conclusion, we have reservations about the changes in lung volumes and closing capacity reported by Craig and McCarthy.⁴ Our data show that, in normal man, closing capacity is not increased by continuous positive airway pressure.

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