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Extubation from Ambient or Expiratory Positive Airway Pressure in Adults

Stuart F. Quan, M.D., Robert T. Falltrick, M.D.,† Richard M. Schlobohm, M.D.‡*

End-expiratory pressure is often used to improve arterial oxygenation and prevent atelectasis in intubated spontaneously breathing patients. To compare the effect of extubation from low levels of expiratory positive airway pressure (EPAP) to extubation from ambient airway pressure, functional residual capacity (FRC) and arterial blood oxygen tension (Pa_{O_2}) were measured in 12 spontaneously breathing patients during three conditions in the peri-extubation period: 1) intubated at 5 cm H_2O EPAP (EPAP 5); 2) intubated at ambient airway pressure (EPAP 0); and 3) within one hour after extubation. During EPAP 5, mean ± SE values for FRC (1864 ± 230 ml) and Pa_{O_2} (114 ± 8 torr) were the same as those obtained after extubation (FRC = 1794 ± 159 ml, Pa_{O_2} = 117 ± 5 torr). However, both FRC (1600 ± 186 ml) and Pa_{O_2} (106 ± 8 torr) were lower during EPAP 0 than after extubation or EPAP 5 ($P < 0.01–0.05$). The magnitude of increase in FRC and Pa_{O_2} on extubation from EPAP 0 varied inversely with the patient's lung thorax compliance ($r = -0.84$, $P < 0.005$). It was concluded that a period of EPAP 0 is not necessary in the weaning period, and that it may be deleterious in patients with compromised lung thorax mechanics. (Key words: Airway: extubation. Lung: functional residual capacity. Ventilation: positive end-expiratory pressure.)

END-EXPIRATORY POSITIVE PRESSURE may be useful in preventing hypoxemia in spontaneously breathing intubated patients who do not require mechanical ventilation.[1] In addition, it may minimize hypoxemia during weaning from mechanical ventilation[2] and prevent atelectasis in patients who have been intubated for protection of their airway.[1] In infants with pulmonary disease, it has been observed that intubation without use of end-expiratory pressure has an adverse effect on arterial blood oxygenation,[3,4] and that weaning and extubation are optimally performed at low levels of end-expiratory pressure.[4,5] In these spontaneously breathing infants with pulmonary dysfunction, it has been demonstrated that this is related to a dependence on "grunting" produced by glottic closure at end expiration.[3] This mechanism may not necessarily occur in adults, and it is not established whether adults should be extubated from low levels of end-expiratory pressure.[6–8] Therefore, this study compares changes in lung volume and arterial blood oxygenation after extubation from ambient airway pressure to those from low levels of end-expiratory pressure in an adult population.

Methods

Twelve adult patients in the Medical-Surgical Intensive Care Unit of San Francisco General Hospital who were intubated and breathing spontaneously were studied (table 1) according to a protocol approved by the University of California, San Francisco, Committee on Human Research. Ten of these patients were intubated to permit mechanical ventilation, and were studied 1–2 hours after successful weaning. The other two patients were intubated for airway protection, and were studied just prior to extubation.

Measurements of maximum inspiratory force (MIF) and vital capacity (VC) were made in all patients upon initiation of the study. In the mechanically ventilated patients, total static lung thorax compliance (C_T) was calculated shortly before cessation of mechanical ventilation from the ratio of changes in expired tidal volume and airway pressures [end-inspiratory (after zero flow for one second) minus end-expiratory].[9]

With the patient in the supine position, functional residual capacity (FRC), arterial blood oxygen tension (Pa_{O_2}), tidal volume (V_T) and respiratory rate were measured in each of these three conditions: 1) intubated, with 5 cm H_2O expiratory positive airway pressure (EPAP 5); 2) intubated, at ambient airway pressure (EPAP 0); and 3) within 1 hour after extubation (EXT). The first condition, EPAP 0 or EPAP 5 was assigned randomly. At least 15 min were allowed for equilibration before measurements were made while on EPAP 5 or EPAP 0. Although all patients were breathing an inspired oxygen concentration which resulted in a clinically acceptable Pa_{O_2} (mean FI_{O_2} = 0.34, range = 0.21–0.40; mean Pa_{O_2} = 109, range = 68–144 torr) each patient had only one FI_{O_2} throughout the study.

FRC was determined by the closed circuit helium dilution method using an apparatus described by

* Fellow in Emergency Medicine; currently Instructor in Medicine, University of Arizona.

† Assistant Clinical Professor of Anesthesia.

‡ Associate Clinical Professor of Anesthesia.

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Address reprint requests to Dr. Stuart F. Quan: Division of Respiratory Sciences, College of Medicine, University of Arizona, Tucson, Arizona 85724.

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TABLE 1. Patient Clinical Data

Patient Number	Age	Sex	Diagnosis	Number of Days Intubated at Study Date
1	27	M	Epiglottitis	2
2	30	M	Resection of spinal cord ependymoma	1
3	49	M	Multiple traumatic abdominal injuries, ARDS	30
4	18	M	Rib fractures, lung contusion	5
5	28	M	Rib fractures, chest trauma	1
6	56	M	Post portal caval shunt; pulmonary edema	5
7	39	M	Multiple traumatic abdominal and chest injuries	2
8	62	M	Postgastric resection, chronic bronchitis	2
9	38	M	Perforated gastric ulcer	2
10	63	F	Multiple traumatic abdominal injuries, ARDS	24
11	26	M	Flail chest, lung contusion	12
12	38	M	Multiple traumatic abdominal and chest injuries	2

Suter and Schlobohm[10] modified for spontaneous ventilation. On repetitive testing, the apparatus measured model gas volumes of 1000 and 1500 ml as 1010 ± 15 and 1506 ± 11 ml, respectively (mean ± SE). FRC results were compared to predicted normal values corrected for the supine position, and expressed both as a volume of gas (BTPS) and as a per cent of each patient's predicted value.[11,12] V_T and respiratory rate were measured from the spirometric tracing on the helium dilution apparatus. Pa_{O_2}, Pa_{CO_2}, and *p*H were determined with a standard blood gas analyzer.

The data were analyzed using Student's *t* test for paired data with Bonferroni's correction, and the method of least squares correlation and regression.[13] Results are given as means ± SE.

Results

The results are summarized in table 2. Mean values for FRC, per cent predicted FRC (per cent PFRC), and Pa_{O_2} at EPAP 0 were lower than at both EPAP 5 and after extubation. The values at EPAP 5, however, were not different than those after extubation. V_T after extubation was larger than at either EPAP 5 or EPAP 0, but the latter values were not different from each other. Respiratory rate was not different among the three conditions. The order of application of the first condition, EPAP 5 or EPAP 0, did not affect the changes observed in any of the variables after extubation.

Figure 1 shows the relationship of C_T, measured shortly before termination of mechanical ventilation, to change in per cent PFRC between EPAP 0 and after extubation. A negative correlation is present ($r = -0.84$, $P < 0.005$), indicating that lower values for C_T were associated with larger increases in FRC after extubation. MIF and VC were not found to be predictors of change in FRC in response to EPAP or after extubation.

Discussion

The application of end-expiratory pressure (EEP) in spontaneously breathing intubated patients often results in an improvement in arterial blood oxygenation[1,2] presumably by increasing FRC[14] and thereby improving ventilation perfusion ($\dot{V}/\dot{Q}$) relationships in the lung. EEP can be administered as continuous (CPAP) or expiratory (EPAP) positive airway pressure in spontaneous breathing patients. We utilized the simpler EPAP method in this intensive care unit until recently, when central compressed air and the additional necessary airway equipment became available. In this study, the effect on pulmonary function produced by extubation from a low level of EEP (EPAP 5) was compared to extubation from ambient airway pressure (EPAP 0). Our results demonstrate that there is no change in FRC and Pa_{O_2} after extubation from EPAP 5, whereas intubation without low levels of EEP (EPAP 0) results in a deterioration in FRC and Pa_{O_2}. While the observed mean differences were not large, the largest individual differences in FRC and Pa_{O_2} between EPAP 0 and after extubation were 625 ml and 44 torr, respectively. This indicates that a clinically significant decline in Pa_{O_2} and FRC may occur with EPAP 0 in some patients. This finding is similar to that found in infants[4] and adults,[8] when low levels of EEP are produced by CPAP. We thus agree that patients should be extubated from low levels of EEP, avoiding the possibility of hypoxemia during breathing at ambient airway pressure. Although there is data sug-

TABLE 2. Pulmonary Function and Arterial Blood Gas Data

	EPAP 5*	EPAP 0*	Extubation
FRC (ml)	1864 ± 230†	1600 ± 186	1794 ± 159†
Per cent predicted FRC	75.7 ± 8.3†	65.8 ± 6.9	73.2 ± 5.8†
Pa_{O_2} (torr)	114 ± 8‡	106 ± 8	117 ± 5‡
Pa_{CO_2} (torr)	38 ± 1	40 ± 1	38 ± 1
*p*H	7.45 ± 0.01	7.44 ± 0.01	7.45 ± 0.01
Respiratory rate	22 ± 2	22 ± 2	23 ± 2
V_T (ml)	456 ± 20	471 ± 15	544 ± 33‡

* See text definition.
† $P < 0.01$ compared with EPAP 0.
‡ $P < 0.05$ compared with EPAP 0.

gesting that CPAP may be a more efficient technique of applying EEP in spontaneously breathing patients,[15] the comparative virtues of EPAP *vs.* CPAP in the peri-extubation period have not been addressed.

In adults, the mechanism by which FRC and oxygen gas exchange might be greater after extubation in comparison to EPAP 0 is not clear, but may be related to the presence of a functioning glottis. First, the glottis may act to increase expiratory upper airway resistance. This is the presumed function of expiratory grunting due to glottic narrowing in infants with hyaline membrane disease.[3–5] Circumventing glottic function with an endotracheal tube in these infants leads to a fall in Pa_{O_2}.[3,4] It has not been widely recognized that glottic narrowing can also occur in adults. Increased expiratory upper airway resistance due to glottic narrowing has been demonstrated in some adults with chronic obstructive pulmonary disease presumably as a compensatory mechanism to prevent airway closure during expiration.[16] Furthermore, glottic narrowing has been implicated in producing the increase in subglottic expiratory airway pressure and presumably expiratory upper airway resistance observed after extubation in a group of adult surgical patients.[8] An increase in expiratory airway resistance has been shown to increase FRC.[17] Therefore, in adults with acute respiratory insufficiency, the glottis may act to increase expiratory airway resistance, thus leading to an increase in FRC. Second, elimination of glottic function with an endotracheal tube may impair cough production resulting in retention of pulmonary secretions, atelectasis, and a decrease in FRC. Coughing during endotracheal intubation in healthy individuals is associated with decreased maximum expiratory air flows and in some instances with decreased maximum transpulmonary pressures.[18] Although glottic function may not be essential for cough production,[19] cough effectiveness has been proposed to be improved when it is intact.[20] Therefore, the increase in FRC after extubation from EPAP 0 might be a result of a reduction in atelectasis produced by a more efficient cough.

In this study, tidal volume was larger after extubation than during the EPAP 0 or EPAP 5 conditions. Although the explanation for this finding is not clear, it may provide an alternative mechanism for the increase in FRC after extubation from EPAP 0. A larger tidal volume may reduce atelectasis, and thus increase FRC and Pa_{O_2}.

In patients studied in the immediate postoperative period, FRC and intrapulmonary shunt,[6] and Pa_{O_2}[7] have been observed to remain unchanged after extubation from ambient airway pressure. However, factors such as incisional pain and abdominal distension

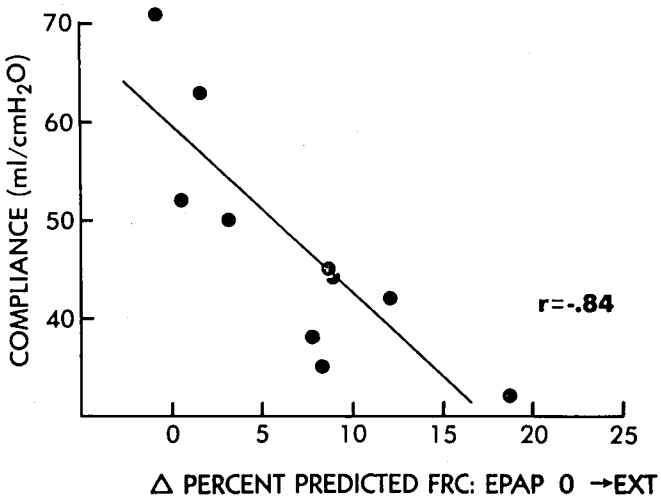

FIG. 1. Relationship between compliance (C_T) and change in functional residual capacity (FRC) measured while intubated at ambient airway pressure (EPAP 0) and after extubation (EXT). Change (Δ) in FRC is shown as the difference between each patient's FRC, as a per cent of predicted (per cent PFRC), in the two conditions. As demonstrated by the negative correlation ($r = -0.84$), low compliance values were associated with the greatest changes in FRC after extubation. This relationship is described by the regression equation: $C_T = 59 - 1.7$ (Δ per cent PFRC).

act to decrease lung-thorax compliance, and reduce FRC in the immediate postoperative period.[21] Therefore, these factors may mask the influence of those mechanisms which would increase FRC after extubation from ambient airway pressure (EPAP 0).

The degree of existing respiratory impairment may also be an important factor in determining whether FRC will increase after extubation from ambient airway pressure. In patients with acute respiratory insufficiency the amount of reduction in C_T may be a reflection of the degree of respiratory impairment. The greater increase in FRC after extubation from EPAP 0 in patients with a low C_T suggests that mechanisms which may increase FRC after extubation are not as important when the C_T is normal.

In conclusion, removal of end-expiratory pressure prior to extubation is not necessary, and may be associated with a clinically significant deterioration in FRC and arterial oxygenation, especially in patients with compromised lung thorax mechanics. We suggest that spontaneously breathing intubated patients recovering from acute respiratory insufficiency should be maintained on low levels of end-expiratory pressure. Furthermore, weaning and extubation should be performed with this regimen without a trial of breathing at ambient airway pressure.

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References

1. Gillick JS: Spontaneous positive end-expiratory pressure (sPEEP). Anesth Analg (Cleve) 56:627–632, 1977
2. Feeley TW, Saumarez R, Klick JM, et al: Positive end-expiratory pressure in weaning patients from controlled ventilation. Lancet 2:725–728, 1975
3. Harrison VC, de V Heese H, Klein M: The significance of grunting in hyaline membrane disease. Pediatrics 41: 549–559, 1968
4. Berman LS, Fox WW, Raphaely RC, et al: Optimum levels of CPAP for tracheal extubation of newborn infants. J Pediatr 89:109–112, 1976
5. Fox WW, Berman LS, Dinwiddie R, et al: Tracheal extubation of the neonate at 2–3 cm H_2O continuous positive airway pressure. Pediatrics 59:257–261, 1977
6. Millbern SM, Weinstein ME, Smith DE, et al: Physiological CPAP: fact or fiction. Crit Care Med 6:97, 1978 (abstract)
7. Colgan FJ, Liang JQ, Barrow RE: Noninvasive assessment by capacitance respirometry of respiration before and after extubation. Anesth Analg (Cleve) 54:807–813, 1975
8. Annest SJ, Gottlieb M, Paloski WH, et al: Detrimental effects of removing end-expiratory pressure prior to endotracheal extubation. Ann Surg 191:539–545, 1980
9. Suter PM, Fairley HB, Isenberg MD: Effect of tidal volume and positive end-expiratory pressure on compliance during mechanical ventilation. Chest 73:158–162, 1978
10. Suter PM, Schlobohm RM: Determination of functional residual capacity during mechanical ventilation. ANESTHESIOLOGY 41:605–607, 1974
11. Grimby G, Soderholm B: Spirometric studies in normal subjects, III. Static lung volumes and maximum voluntary ventilation in adults with a note on physical fitness. Acta Med Scand 173:199–206, 1963
12. Whitfield AGW, Waterhouse JAH, Arrott WM: The total lung volume and its subdivisions. A study in physiological norms. Br J Soc Med 4:1–25, 1950
13. Zar J: Biostatistical Analysis. Englewood Cliffs, Prentice Hall, 1974, pp 121–130, 236–241
14. Craig DB, McCarthy DS: Airway closure and lung volumes during breathing with maintained airway positive pressures. ANESTHESIOLOGY 36:540–543, 1972
15. Gherini S, Peters RM, Virgilio RW: Mechanical work on the lungs and work of breathing with positive end-expiratory pressure and continuous positive airway pressure. Chest 76: 251–256, 1979
16. Campbell AH, Imberger H, MacJones B: Increased upper airway resistance in patients with airway narrowing. Br J Dis Chest 70:58–65, 1976
17. Zechman F, Hall FG, Hull WE: Effect of resistance to tracheal airflow in man. J Appl Physiol 10:356–362, 1957
18. Gal TJ: Effects of endotracheal intubation on normal cough performance. ANESTHESIOLOGY 52:324–329, 1980
19. Irwin RS, Rosen MJ, Braman SS: Cough—A comprehensive review. Arch Intern Med 137:1186–1191, 1977
20. Harris S, Jonson B: Lung function before and after laryngectomy. Acta Otolaryngol (Stockh) 78:287–294, 1974
21. Alexander JI, Spence AA, Parikh RK: The role of airway closure in post-operative hypoxemia. Br J Anesth 45:34–40, 1973