

Reduction in Resting End-Expiratory Position of the Respiratory System with Induction of Anesthesia and Neuromuscular Paralysis

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Resting end-expiratory position (REEP) of the respiratory system was monitored continuously using spirometric recording in eleven patients during transition from consciousness to thiopental hypnosis and following subsequent administration of succinylcholine. REEP decreased following thiopental and was little affected by subsequent relaxant in most patients. A fall in REEP was observed within 30 s after thiopental, and a lower, stable level of REEP was attained within approximately 15-45 s. Mean volume of gas expelled from the lungs was 189 (SE 32) ml BTPS. It is concluded that the previously extensively documented decrease in functional residual capacity associated with anesthesia occurs immediately on induction and requires a short, but finite, time for apparent stabilization. (Key words: Anesthetics, intravenous: thiopental. Lung: function; functional residual capacity; mechanics; volume. Neuromuscular relaxants: succinylcholine.)

THE TEMPORAL RELATIONSHIP between fall in functional residual capacity (FRC) and induction of anesthesia, as well as the rate at which FRC diminishes, is not known. Several investigators have compared FRC in the conscious state and in the anesthetized state both during spontaneous and artificial ventilation and have reported 15-30 per cent reductions. These studies using gas washout, gas dilution, or plethysmographic methods appear to include no measurement made earlier than five minutes (but possibly longer) following induction of anesthesia by which time FRC is already small and stable. The present study therefore was undertaken to determine whether a decrease in FRC occurs immediately with induction of anesthesia with thiopental and with subsequent muscle paralysis produced by succinylcholine. Change in FRC was detected by spirometric recording of the resting end-expiratory position of the respiratory system beginning in the conscious state and continuing throughout all events associated with anesthetic induction and neuromuscular paralysis.

Methods

Subjects of the study were eleven consenting patients scheduled for elective surgery under general anesthesia.

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They were premedicated with a variety of agents according to the choice of the anesthesiologist responsible for clinical management of the patient. The apparatus consisted of a 9-liter spirometer, a self-inflating AMBU bag, and two giant three-way respiratory stopcocks arranged as in figure 1. This apparatus was substituted for the breathing bag of an anesthesia circle system. Three configurations of the equipment were attainable at constant total system volume by appropriate positioning of the stopcocks. The patient could be isolated from the AMBU bag and respire spontaneously from the spirometer. Alternatively, the patient could be isolated from the spirometer and receive manual artificial ventilation from the AMBU bag. Finally, patient, spirometer, and bag could be placed in continuity permitting pressure equilibration throughout the system. The ability of the system to operate without loss of gas in these configurations was verified with an artificial thorax (Training Thorax, North American Drager Co.). Oxygen concentration in the circuit was monitored continuously with an oxygen analyzer whose sensor was in the inspiratory limb of the circle system.

To begin the study, a tight mask fit was obtained and FI_{O_2} was adjusted to 0.70-0.75 (balance nitrogen). Then, for the remainder of the observations, total rebreathing occurred from the closed-circle system with CO_2 absorber and no further fresh gas added. The patient breathed until the spirometric recording indicated that a convincing resting end-expiratory position (REEP) could be established for the conscious state. Then, thiopental, 3-5 mg/kg (with 3 mg *d*-tubocurarine) was given while the mask fit was maintained. If a prolonged period of apnea ensued, manual artificial ventilation was provided as indicated. After a convincing thiopental REEP had been established 1.5 mg/kg succinylcholine was given. Periods of artificial ventilation alternating with periods of measurement continued until a convincing succinylcholine REEP was established by inspection of the spirometer recording. To terminate observations, the experimental apparatus was replaced by the anesthesia breathing bag and an endotracheal tube was inserted in preparation for surgery after a brief period of hyperventilation with oxygen.

Validity of experimental results was critically dependent on integrity of the circuit. Because of the ever-pres-

ent possibility of an imperfect mask fit and leakage of gas, criteria for rejection of experimental records were established in advance of the study. In addition to a grossly identifiable episode of loss of gas, a sustained apparent increase in REEP or an apparent basal oxygen uptake greater than 350 ml/min were assumed to represent gas leakage and the involved study was discarded.

Change in REEP was measured directly from the spirometer recording and was corrected to BTPS. Time required to attain an altered REEP was taken as the interval from the first departure of the end-expiratory position from a previously constant level until the convincing establishment of a new constant level. This interval was estimated only in subjects who did not have sustained apnea following thiopental and in whom continuous patency of the airway was evident. FRC was predicted for each patient using published tables,¹ and percentage reduction in FRC was estimated from the observed change in REEP. Statistical significance of data was tested, when necessary, using a single-tailed *t* test.²

Results

Details concerning subjects and results of observations are presented in table 1. A representative spirometric recording is shown in figure 2. In ten of eleven subjects REEP decreased after administration of thiopental. In six of the ten who maintained spontaneous ventilation or had transient apnea, the alteration in REEP was usually apparent within about 30 s following thiopental administration. In these patients, in whom REEP could be continuously observed, 13–41 s were required to attain a new REEP. The four patients who had more prolonged apnea requiring one or more periods of artificial ventilatory support commenced spontaneous ventilation at a lower REEP. An initial, transitory effect of thiopental administration in some individuals was 3–4 large breaths with increased REEP before the rapid, shallow breathing at declining REEP characteristic of thiopental induction was observed. Mean reduction in REEP was 188 ml (range 0–326 ml; SE 31 ml). Three patients had apparent further decreases in REEP following succinylcholine. Mean reduction was 47 ml (SE 26 ml) and not significantly different from zero. The one individual who had a striking reduction in REEP following succinylcholine (patient 8) was the patient who had no change after thiopental. No further change in REEP was apparent in any patient after stabilization in the anesthetized state.

Discussion

Howell and Peckett³ reported a decrease in REEP of about 200 ml following administration of thiopental and no further change with establishment of neuromuscular

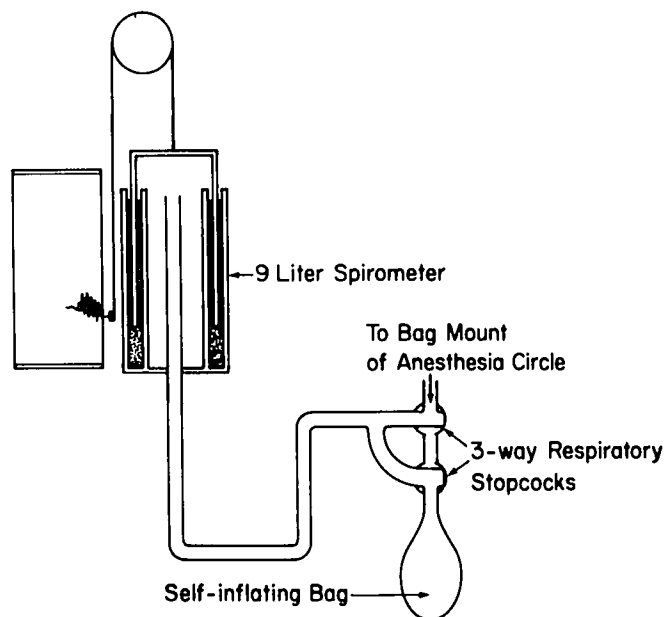


FIG. 1. Diagram of experimental apparatus. The apparatus could be substituted for the breathing bag on any standard circle system.

paralysis with succinylcholine. The format of presentation of results in their report might cause misinterpretation as to the number of experimental subjects involved, but the Methods section of the paper indicates that the thiopental study was done in only one subject. Don *et al.*⁴ failed to observe a change in position assumed by the respiratory system during apnea following administration of 5 mg/kg thiopental in four subjects. It is surprising that these two studies apparently represent the only previous application of the simple technique of spirometry to monitor changes in lung volume during transition from the conscious to the anesthetized state. Spirometric measurements of effects of relaxant administration on resting lung volume in subjects already anesthetized for considerable periods are more frequent. Most such studies suggest that relaxants produce no further decrease in REEP, although Kallos *et al.* reported that succinylcholine increased FRC in subjects who had received Innovar,⁵ and Nims *et al.* reported an increase in REEP following succinylcholine in anesthetized patients.⁶ Virtually all individuals who have measured FRC using various other techniques after establishment of anesthesia conclude that a significant reduction in FRC characterizes the anesthetized state.⁷ Previous investigators also agree that FRC stabilizes during anesthesia and the reduction is not progressive with time.

The present study confirms that FRC becomes smaller during anesthesia, as manifested by a decrease in the resting end-expiratory position of the respiratory system. The new information presented is that this reduction in FRC begins immediately as anesthesia is induced and

TABLE 1. Summary of Observations in the Patients Studied

Patient	Sex	Age	Ht (cm)	Wt (kg)	Δ REEP (ml)		Total Per Cent Δ REEP	Δ T(s)
					Conscious to Thiopental	Thiopental to Succinylcholine		
1	F	18	168	81	-320	I	-18	26
2	F	19	165	61	-146	-90	-13	17
3	M	21	180	68	-109	0	-8	—
4	F	21	163	83	-136	I	-8	—
5	M	22	175	77	-81	-81	-7	13
6	M	26	183	82	-298	0	-14	—
7	F	29	175	125	-190	0	-10	41
8	F	37	168	55	0	-255	-13	—
9	F	39	168	105	-326	0	-17	19
10	M	55	178	59	-260	0	-7	—
11	F	61	152	45	-212	0	-10	15
MEAN					188	47		
SE					31	26		

REEP = resting end-expiratory position. Total Δ REEP = change in REEP expressed as a percentage of predicted FRC. Δ T = time

interval from beginning of change in REEP to apparent stabilization at a new level. I = indeterminable because of technical problem.

stabilization of REEP at a new lower level has occurred within approximately 15–45 s after loss of consciousness. Once this has happened, subsequent administration of succinylcholine has no further effect. Examination of the spirometric recording of the one patient (number 8) who had no change in FRC with thiopental but a large decrease following succinylcholine suggests that she sustained irremediable upper airway obstruction with thiopental which could be corrected following succinylcholine. If this interpretation of events in this patient is correct, then diminution of FRC with thiopental occurred in every patient tested who provided a recording suitable for analysis. Records from four more patients not included in the data analysis were discarded because of

easily recognizable episodes of loss of gas from the circuit. Records from one additional patient were excluded due to apparent excessive oxygen consumption. (It is of interest that at surgery this patient proved to have intra-abdominal malignancy with extensive metastatic involvement.)

This simple study provides little information on the mechanism of reduction of FRC during anesthesia. The generally slow fall of REEP with the gradual approach to the new, lower level indicates that an abrupt loss of tonic muscle activity, as must occur with development of upper airway obstruction during the course of a single breath associated with loss of consciousness, is not involved in FRC reduction with anesthesia. Rather, progressive loss of inspiratory muscle tone mediated through some mechanism subject to anesthetic depression might be postulated. Lack of effect of succinylcholine in the unconscious patient confirms that enhanced expiratory muscle activity is probably of little significance in reducing FRC. Absolute lung volumes were not determined in the present study. Therefore, fractional reduction in FRC cannot be stated with confidence. Observed estimated percentage reductions seem somewhat less than changes measured by other investigators using more conventional techniques for FRC determinations. One therefore might speculate that in addition to gas expelled from the lungs on induction of anesthesia manifested by a reduction in REEP, there may have been further gas trapped behind closed airways.⁸ This uncertainty is emphasized in this communication by designation of the end-expiratory point of equilibrium of the respiratory system as "REEP" rather than as "Resting End-Expiratory Volume." It is also possible that REEP, having quickly plateaued at a new level immediately following induction, continued to diminish slowly during the early

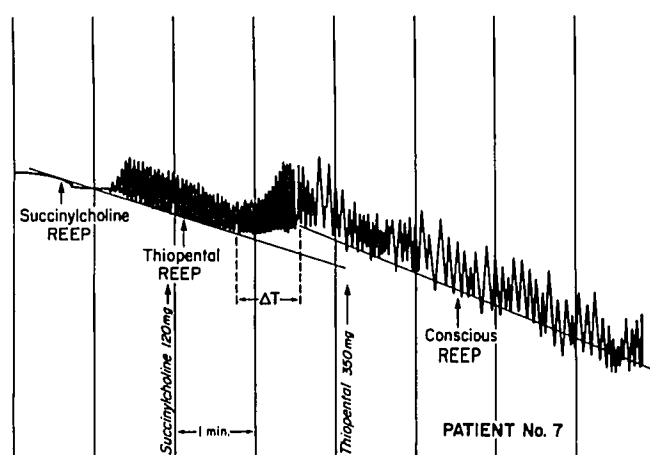


FIG. 2. The spirometric recording obtained in patient 7. Breathing pattern is somewhat irregular in the conscious state but an unambiguous resting end-expiratory position can be established. REEP begins to decrease within 30 s of thiopental administration, falls 190 ml (BTPS) over the next 41 s, and is not influenced further by neuromuscular blockade.

period of anesthetic maintenance. Such behavior cannot be distinguished, on the basis of spirometry, from the small decrease in oxygen consumption usually observed during anesthesia. The small "defasciculating" dose of *d*-tubocurarine given with the thiopental seems unlikely to have contributed to the change in REEP. Howell and Peckett observed the identical phenomenon in their patient who received thiopental without relaxant.³

Patients in the present study had been recumbent for more than one hour before anesthetic induction. Therefore, the small, slowly progressive decrease in functional residual capacity occurring during the first 25–30 min of recumbency⁹ was presumably complete and did not contribute to the observed results. Redistribution of blood from peripheral to central reservoirs is another possibility. However, in such peripheral to central fluid shifts in experimental animals, an increase in abdominal and thoracic girth need not be associated with displacement of gas from the lungs.⁹

The relationship between changes in many respiratory variables which have been reported in association with anesthesia and the extensively documented decrease in FRC characteristic of this state remains uncertain. It seems probable that most changes in mechanical properties of the respiratory system associated with anesthesia can be explained by its smaller size. For example, if one examines the data of Westbrook *et al.*,¹⁰ it is apparent that the proportional decreases in FRC and total respiratory compliance with anesthesia are identical so that specific compliance during consciousness and in the anesthetized state remains unchanged. The apparent increased stiffness of the respiratory system during anesthesia, attributed by these investigators to increased elastic recoil of the lung, might alternatively be explained by a smaller initial quantity of lung participating in volume changes. Lehane and associates emphasize the importance of considering lung volume at which measurements of respiratory resistance are made during anesthesia. They point out that changes in lung volume known to occur with anesthesia can produce very large

changes in respiratory resistance without change in bronchomotor tone.¹¹ The role of FRC diminution in gas exchange abnormalities associated with anesthesia is more nebulous. In the anesthetized patient, as in the conscious subject, closing capacity remains below FRC in patients younger than about 45 years. Nevertheless, the majority of anesthetized patients seem to have some impairment of oxygenation in spite of the fact that in many, decrease in FRC is not sufficient to drop REEP below the volume where lung units begin to close. In addition, artificial elevation of FRC above closing capacity with positive end-expiratory pressure is only partially effective in overcoming anesthesia-induced pulmonary dysfunction.¹² Further investigation of the role of FRC in anesthesia is needed.

References

1. Dittmer DS, Grebe RM: Handbook of Respiration. Philadelphia, WB Saunders, 1958, pp 38–39
2. Huntsberger DV: Elements of Statistical Inference. Boston, Allyn and Bacon, 1967, pp 179–202
3. Howell JBL, Peckett BN: Studies of the elastic properties of the thorax of supine anaesthetized paralyzed human subjects. *J Physiol (Lond)* 136:1–19, 1957
4. Don HF, Wahba M, Cuadrado L, Kelkar K: The effects of anesthesia and 100 per cent oxygen on the functional residual capacity of the lungs. *ANESTHESIOLOGY* 32:521–529, 1970
5. Kallos T, Wyche MQ, Garman JK: The effects of Innovar on functional residual capacity and total chest compliance in man. *ANESTHESIOLOGY* 39:558–561, 1973
6. Nims RG, Conner EH, Comroe JH: The compliance of the human thorax in anesthetized patients. *J Clin Invest* 34:744–750, 1955
7. Nunn JF: Applied Respiratory Physiology. Second edition. London, Butterworths, 1977, pp 66–69
8. Don HF, Wahba WM, Craig DB: Airway closure, gas trapping, and the functional residual capacity during anesthesia. *ANESTHESIOLOGY* 36: 533–539, 1972
9. Tenney SM: Fluid volume redistribution and thoracic volume changes during recumbency. *J Appl Physiol* 14:129–132, 1959
10. Westbrook PR, Stubbs SE, Sessler AD, et al: Effects of anesthesia and muscle paralysis on respiratory mechanics in normal man. *J Appl Physiol* 34:81–86, 1973
11. Lehane JR, Jordan C, Jones JG: Influence of halothane and enflurane on respiratory airflow resistance and specific conductance in anaesthetised man. *Br J Anaesth* 52:773–789, 1980
12. Tien YK, Bergman NA: Airway closure, PEEP, and AaDO₂ during anesthesia. *ANESTHESIOLOGY* 53:S413, 1980