## Resistance Breathing (Blow Bottles) and Sustained Hyperinflations in the Treatment of Atelectasis

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Resistance breathing (blow bottles) and sustained hyperinslations employing the Elder valve were evaluated in the treatment of postoperative atelectasis. Neither maneuver evoked major improvement, yet FRC increased significantly and shunting was reduced following resistance breathing. Laboratory analyses of the pressure relationships across the lung during both maneuvers and during the Valsalva maneuver were done. A sustained deep breath, involved in all maneuvers was necessary to achieve a maximal transpulmonary pressure gradient. Evoked responses of lung to sustained inflation are discussed in relation to alveolar opening. (Key words: Resistance breathing: Blow bottles; Hyperinslations; Atelectasis.)

ATELECTASIS in the postoperative patient usually is associated with an increase in physiologic shunting and a reduction in functional residual capacity (FRC). Many procedures have been employed in attempts to expand atelectatic lung and increase the resting lung volume (FRC). The present study evaluates the rationales for and clinical effectiveness of two such forms of therapy; resistance breathing (blow bottles) and intermittent sustained hyperinflation of the lung with the Elder valve.

#### Methods

### RESISTIVE BREATHING

Eight postoperative patients with x-ray and laboratory evidence of atelectasis were studied. Monitoring and sampling catheters had been placed in the radial artery and in the right atrium or ventricle prior to the surgical operation. Control measurements of FRC

were made by the closed-circuit helium-dilution technique when postoperative atelectasis was suspected. With the patient breathing air, arterial and mixed venous blood samples were drawn and expired air was collected in a meteorological balloon for three minutes. Following ten minutes of oxygen breathing, samples of end-expired gas and arterial and mixed venous blood again were drawn for determination of intrapulmonary shunting. Following a deep inspiration, each patient blew into a prototype of a commercially available blow bottle. The patients were instructed to blow a liter of water from one bottle to another. Some were able to do this in two or three breaths. Three patients who were receiving supplementary oxygen required several breaths to empty the bottle, although every patient eventually was able to do so. Following a ten- to 15-minute rest period, the initial measurements were repeated.

The blow-bottle system consisted of two one-liter plastic bottles connected by a short loop of plastic tubing at the caps (fig. 1). A standard U-tube water manometer was used to monitor airway pressure developed while the patient was blowing into the system. A pressure of approximately 18 cm H<sub>2</sub>O was necessary to initiate the flow of water from one bottle to the other. However, as the water level in the first bottle fell with each breath, a greater pressure was required to raise the column of water in the connecting loop and to start the flow. When the bottle was nearly empty, a pressure of about 36 cm H2O was necessary to initiate flow. Once the loop of the connecting tube was filled with water, a siphon effect was created and the pressure required to maintain flow was approximately 12 cm H<sub>2</sub>O less than the starting pressure. Because of the resistance to flow of water through the tubing, it was possible for a spirited patient to generate airway pressures

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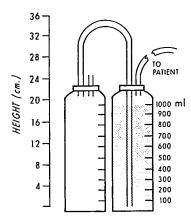


Fig. 1. Schematic of plastic blow-bottle system used to create resistance breathing.

as high as 70 cm H<sub>2</sub>O, resulting in more rapid transfer of water from one bottle to the other.

# SUSTAINED INFLATION WITH THE ELDER VALVE

Eight additional postoperative patients with atelectasis received four or five sustained hyperinflations lasting five to 15 seconds with the Elder Demand Valve Resuscitator.<sup>1</sup>

The patients were instructed to inhale slowly to capacity, and airway pressure was increased gradually by simultaneous depression of the inflation button. With instruction and reassurance, patients were able to sustain airway pressures at or near 50 cm H<sub>2</sub>O for five to 15 seconds. All patients experienced some discomfort at high inflation pressures but cooperated readily. Two patients with tracheostomics each received four sustained hyperinflations at peak pressure for 15 seconds each.

Measurements of FRC and physiologic shunting during inhalation of air and oxygen were made before and after use of the Elder valve.

Shunt was calculated using standard equations and oxygen content determined directly using a mixture of deoxygenated potassium ferricyanide and blood.<sup>2, 3</sup> P values relating to significance were obtained by the method of paired comparisons.

# EFFECTS OF THE VALSALVA MANEUVER, RESISTANCE BREATHING AND SUSTAINED HYPERINFLATIONS ON THE PRESSURE GRADIENT ACROSS THE NORMAL LUNG

Both resistance breathing and sustained hyperinflation may produce markedly increased airway pressures, which also occur during the Valsalva maneuver. Because of this similarity, the pressure gradients across the lung induced by the three maneuvers were studied in two normal trained subjects in the sitting position. In order to compare transpulmonary pressure changes induced by the three maneuvers, sustained airway pressures were kept between 30 and 35 cm H2O. Simultaneous recordings of airflow, airway, esophageal and transpulmonary pressures were made during the maneuvers and calculations of tidal volume and dvnamic lung compliance were made from the tracings. Esophageal pressure changes used to assess intrapleural pressure changes were recorded from a 51/2-inch rubber esophageal balloon passed through the nose into the lower third of the esophagus. Esophageal pressure at zero flow at the end of expiration was recorded as zero. Sanborn 268 pressure transducers were used for airway, esophageal and transpulmonary pressures, and a 270 lowpressure transducer was used for the pressure signal from a Fleisch #2 pneumotachograph.

Valsalva Maneuver. Each subject, while sitting, inhaled air through a mouthpiece and a nonrebreathing swivel Y-piece. A pneumotachograph was attached to the inspiratory side and airway pressure was monitored between the nonrebreathing valves. The subject accomplished the Valsalva maneuver by taking a deep breath of approximately three times tidal volume, occluding the exhalation port of the swivel Y-piece and, with an open glotttis, achieving pressure of 30 to 50 cm H2O in the airway by contracting the thoracic and abdominal muscles as during a forced expiration. Continuous recordings of airflow, transpulmonary, airway and esophageal pressures were made before, during and after Valsalva maneuvers of 15 seconds' duration. ments of dynamic lung compliance were made from the transpulmonary pressure and the integrated flow signal (volume) at peak inspiration, one minute before and one minute after the maneuver.

Resistance Breathing (Blow Bottles). Resistance breathing differs from the Valsalva maneuver with an open glottis only in that a gradual reduction in lung volume, commensurate with the amount of water displaced from the bottle, occurs while the patient is blowing. During resistance breathing, the exhalation port of the swivel Y-piece was connected to the blow-bottle tubing at the peak of each deep inspiration. Measurements similar to those taken during the Valsalva maneuver were made.

Sustained Inflations with the Elder Valve. An Elder demand valve was placed on the inspiratory side of the swivel Y-piece in series with the pneumotachograph. During the inspiration of a deep breath, inspiratory flow was augmented by pressing the flow button so as to achieve a positive airway pressure. Simultaneously, the exhalation side of the swivel Y-piece was occluded. Hyperinflation of the lungs was achieved with an open glottis and with sustained airway pressure of 30 to 50 cm H<sub>2</sub>O. As with the other two maneuvers, dynamic lung compliance, tidal volume, airway, esophageal and transpulmonary pressures were measured.

Dynamic lung compliance was measured in each normal subject before and within two

minutes of completion of three consecutive Valsalva maneuvers, then three episodes of resistance breathing, followed by three sustained hyperinflations using the Elder valve. A minimum of 15 minutes separated each series of maneuvers.

#### Results

RESISTANCE BREATHING (BLOW BOTTLES)

The effects of resistance breathing on FRC, blood gases and shunting are compiled in table 1. FRC increased significantly (P = 0.04) and true shunt decreased slightly (P = 0.07) after use of the blow bottles. Arterial oxygen tensions during inhalation of air or oxygen were unaffected.

#### SUSTAINED HYPERINFLATIONS

No significant change in FRC, blood gases or shunting occurred following four to five sustained hyperinflations of five to 15 seconds' duration employing the Elder valve (table 1).

PRESSURE CHANGES ACROSS THE LUNG DURING
RESISTANCE BREATHING, SUSTAINED
HYPERINFLATIONS AND THE VALSALVA MANEUVER IN NORMAL
TRAINED SUBJECTS

Figure 2 is a record of typical changes in airway, esophageal and transpulmonary pressures during resistance breathing. When the

Table 1. Effects of Resistance Breathing and Sustained Hyperinflations on FRC and Intrapulmonary Shunting in Postoperative Patients with Atelectasis

	Resistance Breathing (Blow Bottles) (n = S)		Sustained Hyperinflations (Elder Valve) (n = S	
	Before	After	Before	After
			o:	
FRC (ml)	$1,861 \pm 137$	$2,170 \pm 179$	$1,746 \pm 204$	$1,874 \pm 339$
A-aDo <sub>2</sub> (torr)	$325 \pm 35$	$292 \pm 27$	$348 \pm 27$	$354 \pm 24$
O./Ot (per cent)	$19 \pm 2$	16 ± 2	18 ± 2	$18 \pm 1$
PAO <sub>2</sub> (torr)	$603 \pm 36$	$584 \pm 54$	$626 \pm 21$	$642 \pm 16$
Paga (torr)	$275 \pm 32$	$296 \pm 28$	278 ± 41	$305 \pm 33$
Paco <sub>2</sub> (torr)	$38 \pm 3$	$36 \pm 3$	36 ± 2	$35 \pm 2$
pH	$7.47 \pm 0.02$	$7.49 \pm 0.02$	$7.51 \pm 0.05$	$7.51 \pm 0.05$
			Air	
$\dot{Q}_s/\dot{Q}_t$ (per cent)	$27 \pm 5$	$26 \pm 2$	25 ± 4	$20 \pm 2$
Pao. (torr)	$55 \pm 4$	$54 \pm 3$	59 ± 5	$58 \pm 4$
Paco <sub>2</sub> (torr)	$36 \pm 2$	$35 \pm 2$	34 ± 2	$33 \pm 2$
pH	$7.48 \pm 0.02$	$7.49 \pm 0.02$	$7.50 \pm 0.02$	$7.50 \pm 0.02$

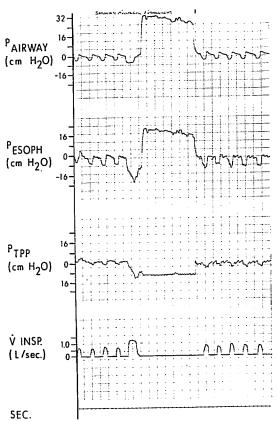


Fig. 2. Airway, esophageal, and transpulmonary pressures and inspiratory flow rate during a deep breath and resistance breathing (blow bottles).

subject took a deep breath of 2,250 ml, esophageal pressure fell to -20 cm H<sub>2</sub>O. Since airway pressure at that time was -5 cm H<sub>2</sub>O, the net force acting on the lung during inspiration was -15 cm H<sub>2</sub>O. When blowing began, airway pressure rapidly rose to 32 cm H<sub>2</sub>O, and esophageal pressure also rose, to about 20 cm H<sub>2</sub>O. Thus, the net transpleural pressure gradient was only 12 cm H<sub>2</sub>O. Assuming a near-linear pressure-volume relationship of the lung, the 12 cm difference between airway and esophageal pressures can be ac-

counted for by the increased elastic retractive force exerted by the distracted lung. Lung compliance measured one minute before and one minute after the episode of resistance breathing was unchanged at 0.176 l/cm H<sub>2</sub>O.

In figure 3, changes in pressure during resistance breathing are compared with those occurring during a Valsalva maneuver and during sustained hyperinflations in the same subject. The esophageal pressure baseline in each case was transposed to zero in order to emphasize the cyclic changes in transpulmo-

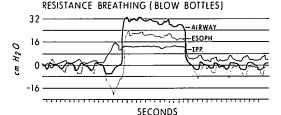
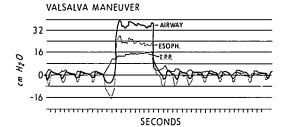
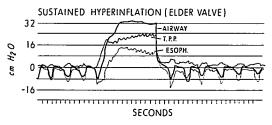


Fig. 3. The effects of resistance breathing, sustained hyperinflations and the Valsalva maneuver on airway, esophageal and transpulmonary pressures.





nary pressure occurring with respiration and with the deep breath. During the blow-bottle and Valsalva maneuvers, thoracic and abdominal muscular contractions persisted after the subject took a breath of approximately two liters and were necessary to maintain elevated airway pressure. The esophageal and airway pressures differed throughout both maneuvers by approximately 12 to 14 cm H<sub>2</sub>O. This difference can be accounted for by the increased elastic retractive force of the lung exnanded by the deep breath.

During sustained hyperinflation, the subject made a conscious effort to relax the thoracic and abdominal wall to accommodate a larger volume of air (2,900 ml). Again, the airwayesophageal pressure gradient of approximately 20 cm H<sub>2</sub>O can be accounted for by the compliance of the greatly-distracted lung.

Dynamic lung compliance, measured in two seated subjects, ranged from 0.166 l/cm H<sub>2</sub>O and 0.201 l/cm H<sub>2</sub>O before each series of three maneuvers; individual changes in compliance measured two minutes following a se-

ries of maneuvers amounted to less than ±9 per cent.

#### Discussion

THE TRANSPULMONARY PRESSURE GRADIENT AND ALVEOLAR OPENING

Therapeutic measures designed to re-expand atelectatic (gas-free) alveoli must provide a sufficient transpulmonary pressure gradient across atelectatic lung to overcome those forces opposing alveolar opening. The critical opening pressure in cat and dog lungs, and in newborn human lungs, is near 8 to 10 cm H<sub>0</sub>O; it is somewhat less for adult humans. 4-6 Whether resistance breathing or sustained hyperinflation maneuvers produce a gradient sufficient to inflate atelectatic lung depends on the state of inflation of the nonatelectatic lung and lung compliance rather than on the magnitude of the induced airway pressure. At normal resting lung volume (40 per cent VC) the transpulmonary pressure in man remains constant at about 5 cm HaO regardless of how much positive or negative pressure is applied to the airway (±40 cm H2O).7 At such a lung volume, atelectatic lung, if present, would exert no retractive force and would be subject to a net distracting force of 5 cm H2O. This pressure probably would be insufficient to open alveoli regardless of airway pressure. The present study, however, has demonstrated that when lung volume is increased to nearcapacity, the gradient across normal lung increases during the Valsalva maneuver, resistance breathing and sustained hyperinflations, but only in proportion to the increased retractive forces exerted by the nonatelectatic, dis-The gradient across the untracted lung. expanded lung will increase directly with the increase in transpulmonary pressure. Finley has demonstrated experimentally that the airways distal to a site of occlusion in atelectatic lung tissue reflect directly and quantitatively the intrapleural pressures during both spontaneous and positive-pressure ventilation.8 If airways leading to atelectatic lung are patent, then the transpulmonary pressure gradient across atelectatic lung must increase as inflatable lung expands with a deep breath or The full magnitude of this hyperinflation. gradient should act directly on atelectatic alveoli, when large areas of atelectasis are exposed to the pleural surface. Whether the

full effect of the pressure gradient acts on areas of miliary atelectasis is not known. Gradients of 16 to 24 cm H2O were obtained in the laboratory during both resistance breathing and hyperinflations with the Elder valve, but were dependent on an associated deep breath. The clinical significance of the above observations is readily apparent: efforts to expand atelectatic lung when applied at or near the tidal-volume range will not generate a transpulmonary pressure gradient sufficient to open closed alveoli regardless of high airway pressures. Similarly, sustained inflation of the lung is helpful in mobilizing atelectatic lung only insofar as the lung volume, not the actual airway pressure achieved is increased. It is conceivable that in an attempt to inflate the lungs against increased chest-wall resistance, airway pressure may reach levels sufficient to occlude airways to atelectatic segments of the lung by overexpanding adjacent lung tissue, and thereby hinder alveolar opening. In such a situation, thoracic-cage resistance could be reduced by narcotics or muscle relaxants during controlled respiration. Any increase in tidal ventilation and transpulmonary pressure could then be accomplished with a smaller increment of airway pressure.

# OTHER FACTORS AFFECTING ALVEOLAR OPENING

Position. With the subject in the sitting position, the gradient across the lung is approximately 5 cm H<sub>2</sub>O. The gradient is reduced to 1 cm H<sub>2</sub>O by reclining, leading to lung retraction and a smaller FRC. When FRC and alveolar surface area are decreased, the stability of the smaller alveoli may be reduced.

Inflation Time. Sustained hyperinflations and resistance breathing both evoke three responses of human lung tissue, not usually evoked by IPPB, which favor re-expansion of atelectatic lung: 1) sustained hyperinflation tends to replenish the surface-active material in the alveolus, momentarily increases lung compliance and retards the development of atelectasis. 2) Lung tissue, particularly at the limits of inflation, exhibits "stress relaxation" characterized by a gradual decrease in intraesophageal pressure for ten to 20 seconds while the subject is holding a deep breath. 19

The phenomenon evident in the esophageal tracings of figure 3 may be due to redistribution, opening of atelectatic alveoli, or alterations in the pulmonary blood volume during hyperinflation. The net effect with time is a gradual increase in the transpulmonary pressure gradient (fig. 3) during sustained inflation, favoring re-expansion of collapsed alveoli. 3) Alveolar opening at a given transpulmonary pressure is highly erratic: some alveoli open immediately, while others require much longer sustained pressures before opening. In laboratory studies 20 to 30 minutes sometimes are required for final volume equilibration at a given transpulmonary pressure, and recruitment of terminal units may continue on progressive inflation to as much as 70 per cent of the total lung capacity.4,6

Inflation Pressure. A high airway pressure without a change in lung volume has no direct effect on increasing the transpulmonary pressure gradient. Evidence is available, however, that sustained end-expiratory airway pressures of 2 to 5 cm H<sub>2</sub>O, by increasing the FRC and thus the transpulmonary pressure gradient, probably account for some alveolar recruitment of atelectatic lung as well as forestalling alveolar collapse.<sup>9, 11</sup> The effectiveness of this maneuver emphasizes the importance of a small increase in transpulmonary pressure on lung inflation and atelectasis.

## Untoward Effects of Increased Transpulmonary Pressure Gradient

When the transpulmonary pressure gradient is increased during spontaneous respiration, pulmonary blood flow through atelectatic lung increases.8 This probably occurs during a sustained deep breath, following a change from the supine to the sitting position, during the Mueller maneuver, and during sustained expiratory-resistance maneuvers designed to increase the transpulmonary pressure gradient. Cheney, Hornbein and Crawford concluded that using expiratory resistance during anesthesia probably reversed airspace collapse, vet the maneuver evoked either a redistribution of pulmonary blood flow or a change in cardiac output.12 In patients with very large intrapulmonary shunts, however, expiratory resistance may provide alveolar recruitment when other methods fail.11

Both resistance breathing (blow bottles) and sustained hyperinflations produce pressure effects similar to those of the Valsalva maneu-These maneuvers, therefore, should be used with caution in patients with emphysema and reduced cardiac output, since they may cause further marked reductions in cardiac output.13 Both syncope and convulsions are known to result from the decreased cardiac output associated with a sustained Valsalva maneuver. Chronic cardiac disease should be no deterrent to resistance breathing or sustained hyperinflations, however. Patients with congestive failure 14 or advanced myocardial and valvular disease 15 have markedly attenuated circulatory responses to sustained airway pressures. The Valsalva maneuver actually has been used therapeutically in the treatment of angina pectoris.16 Any maneuver which evokes an increase in vagal tone, however, should be avoided in the presence of acute coronary disease.

If the transpulmonary pressure is increased by airway inflation, simultaneous inspiratory efforts by the patient may augment the pressure gradient across the lung. The maximum safe intrapulmonary pressure in man is about 70 cm H<sub>2</sub>O when the lungs are supported by the thoracic cage and abdominal musculature.<sup>17</sup> If a vigorous inspiratory effort is associated with positive airway inflation the distending gradient could be momentarily excessive, and emphysematous lungs with bullous areas of relatively high compliance would be subject to damage.

#### INTERPRETATION OF RESULTS

Sustained hyperinflations of the lung by positive airway pressure did not appear to mobilize atelectatic lung since shunt did not decrease while the subjects were breathing oxygen. Some improvement in shunt during breathing of air (P=0.07) suggests a better distribution of ventilation relative to perfusion and is consistent with the minimal increase in FRC. Although in trained subjects it is possible to inflate to lung capacities in excess of those achieved with unassisted maximal inspiration, we suspect that the lungs of our patients were not inflated to capacity because of resistance to inflation. Unfortunately, it was not convenient to measure inspired or expired

volume with either hyperinflation or resistancebreathing maneuvers to confirm this suspicion. Regardless of the airway pressure achieved, with a submaximal lung volume the transpulmonary pressure would be small.

Resistance breathing was followed by some increase in the mean FRC and decrease in the shunt due to atelectasis. Although small, the changes in lung volume and shunting suggest that sustained inflations may be of value in the treatment of atelectasis. Multiple unsustained hyperinflations or deep breaths have proved of little value in altering lung volume and function, while there is other evidence that sustained deep breaths are beneficial.<sup>2, 18-20</sup>

Resistance breathing, which incorporates the sustained deep breath as a basic maneuver, can be easily taught to patients and offers the advantage of patient participation in therapy. The efficacy of resistance breathing as a form of therapy depends on an initial large and sustained deep breath, with prolonged gradual transfer of water from one bottle to another. Rapid transfer generates excessive and useless airway pressure and a more rapid reduction in lung volume. While a single sustained deep breath offers the same favorable transpulmonary gradient that occurs with resistance breathing, we feel that some patients benefit from the challenge offered by resistance breathing. For other, less motivated, patients the transfer of water from one bottle to another gives tangible evidence of progressing therapy.

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## References

- Pearson, J. W., and Redding, J. S.: Evaluation of the Elder Demand Valve Resuscitator for use by first-aid personnel, ANESTRESIOLOGY 28: 623, 1967.
- Mahoney, P. D., and Colgan, F. J.: The effects of deadspace rebreathing in the treatment of atelectasis, Surg. Cynec. Obstet. 128: 1283, 1969.
- Theye, R. A.: Blood O<sub>2</sub> content measurement using the O<sub>2</sub> electrode, ANESTHESIOLOGY 28: 773, 1967.
- Anthonisen, N. R.: Effect of volume and volume history of the lungs on pulmonary shunt flow, Am. J. Physiol. 207: 235, 1964.
- Gribetz, I., Frank, N. R., and Avery, M. E.: Static volume-pressure relations of excised lung of infants with hyaline membrane dis-

- ease, newborn and stillborn infants, J. Clin. Invest. 38: 2168, 1959.
- Radford, E. P., Jr.: Static mechanical properties of mammalian lungs. In: Handbook of Physiology, Section 3: Respiration, Vol. I. Washington, D. C., American Physiology Society, 1964.
- Mead, J., and Milic-Emili, J.: Theory and methodology in respiratory mechanics with glossary of symbols. In: Handbook of Physiology, Section 3, Respiration, Vol. I. Washington, D. C., American Physiology Society, 1964.
- Finley, T. N., Hill, T. R., and Bonica, J. J.: Effect of intrapleural pressure on pulmonary shunt through atelectatic dog lung, Am. J. Physiol. 205: 1187, 1963.
- Williams, J. V., Tierney, D. F., and Parker, II. R.: Surface forces in the lung, atelectasis, and transpulmonary pressure, J. Appl. Physiol. 21: 819, 1966.
- Marshall, R., and Widdicombe, J. G.: Stress relaxation of the human lung, Clin. Sci. 20: 19, 1960.
- McIntyre, R. W., Laws, A. K., and Ramachandran, P. R.: Positive expiratory pressure plateau: Improved gas exchange during mechanical ventilation, Canad. Anaesth. Soc. J. 16: 477, 1969.
- Cheney, F. W., Hornbein, T. F., and Crawford, E. W.: The effect of expiratory resistance on the blood gas tensions of anesthetized patients, ANESTHESIOLOGY 28: 670, 1967.
- Mills, H., and Kattus, A. A., Jr.: The emphysema response to forced straining (Valsalva's maneuver), Circulation 17: 65, 1958.
- Fox, I. J., Crowley, W. P., Jr., Grace, J. B., and Wood, E. H.: Effects of the Valsalva maneuver on blood flow in the thoracic aorta in man, J. Appl. Physiol. 21: 1553, 1966.
- Elisberg, E. I.: Heart rate response to the Valsalva maneuver as a test of circulatory integrity, J.A.M.A. 186: 120, 1963.
- Levine, H. J., McInytre, K. M., and Glovsky, M. M.: Relief of angina pectoris by Valsalva maneuver, New Eng. J. Med. 275: 487, 1966.
- Mushin, N. W., Rendell-Baker, L., and Thompson, P. W.: In: Automatic Ventilation of the Lungs. Second ed. Oxford, Blackwell Scientific Publications, 1959, p. 14.
- Ferris, B. C., Jr., and Pollard, D. S.: Effect of deep and quiet breathing on pulmonary compliance in man, J. Clin. Invest. 39: 143, 1960.
- Bendixen, H. H., Hedley-Whyte, J., and Laver, M. B.: Impaired oxygenation in surgical patients during general anesthesia with controlled ventilation. A concept of atelectasis, New Eng. J. Med. 269: 991, 1963.
- Ward, R. J., Danziger, F., Bonica, J. J., Allen, G. D., and Bowes, J.: An evaluation of postoperative respiratory maneuvers, Surg. Gynec. Obstet. 123: 51, 1966.