Editorial Views

Pulmonary Compliance in Anesthetized Man

Of all the available tests of respiratory mechanics, none is less clearly understood or the changes more frequently misinterpreted than those of pulmonary compliance. Among clinicians, compliance is considered to be a measure of the degree of "stiffness" of the lungs. That is, the lower the compliance the more rigid or resistant are the lungs to inflation. This interpretation perhaps implies: (1) that reductions in pulmonary compliance are the result of abnormal changes in the architectural structure of the lungs, such as thickening or fibrosis of alveolar walls or septae; or, (2) that a lowered compliance is not readily reversible. Certainly the evidence suggests that pulmonary compliance may be reduced in diseases of the lungs or chest wall. However, this limited view of pulmonary compliance disregards the more common etiological factors, such as loss of patent alveoli resulting from collapse or closure—a reversible change.

In this issue of the Journal Nightingale and Richards treat of the compliance of infants paralyzed by *d*-tubocurarine. To evaluate this article, the reader must be familiar with both the relevant terminology and the many sources of variability attending determinations of compliance in anesthetized patients.

Both lungs and thorax possess the property of elasticity or distensibility; that is, they can be deformed from a resting state by a force and when that force is removed, they will return to their original state. During spontaneous respiration, the distorting force is the negative intrapleural pressure, whereas during controlled pressure breathing, it is the positive airway pressure. In either case the result is an increase in lung volume. The volume change per unit of pressure change, pressure-volume relation, is termed compliance. By applying increments of pressure (either positive airway pressure or negative intrapleural pressure) to the lung-thorax system, starting near residual lung volume, and measuring the resultant incremental increases in lung volume, one obtains a pressure-volume relation over a range of pressure and volume. This compliance curve is normally sigmoid in shape, with a near linear area in the resting tidal volume range.[1] Units of compliance may be described in terms of liter/cm. of water (0.1 liter/cm. of water) but for ease of communication, compliance is frequently expressed in milliliters per centimeter of water (100 ml./cm. of water).

Physiologists have subdivided pulmonary compliance into lung compliance and chest wall compliance, by relating the lung volume change to the distending force acting exclusively on either segment of the ventilatory system. For the lungs, the distending force is the transpulmonary pressure (mouth minus intrapleural pressure); for the chest wall, the transthoracic pressure (atmospheric minus intrapleural pressure). Intrapleural pressure is measured indirectly by means of an intraesophageal balloon. However, subdivisions of compliance measurements in anesthetized man are of doubtful validity because it has been demonstrated that esophageal pressures may not reflect accurately changes in intrapleural pressure in the supine position wherein mediastinal structures may compress the esophagus.[2,3]

Reasoning from the comparatively close values for *lung* compliance reported by others and the *total* compliance obtained in their study, Nightingale and Richards conclude that the compliance of the infant *chest wall* must be high. Although probably true, the data do not quite merit this conclusion, since the compliance values used for comparison are derived from different infants under dissimilar circumstances. There is evidence in conscious subjects that aging causes a decrease in total compliance[4] but an increase in lung compliance,[5,6] suggesting that the resistance of the chest wall to deformation is lowest in the young and increases with age. The events that occur during growth and aging to explain a progressive reduction in chest wall and concomitantly total compliance are unknown.

From the sigmoid shape of the normal pulmonary compliance curve, it is evident that compliance varies in an alinear fashion with absolute lung volume, the relation being an inverse one. In addition to absolute lung volume, one should know the volume history prior to the compliance measurement. It has been shown in both conscious and anesthetized man that pulmonary compliance tends to decrease with time at constant volume ventilation but that these reductions in compliance can be reversed by several near-maximal inflations. Maximal inflation must be achieved before reductions in compliance can be ascribed to anything but collapse of alveoli. Another manifestation of the interrelationship of compliance and the number of patent alveoli is the observation that compliance curves obtained during lung inflation are not identical to those obtained during lung deflation at the same lung volume, the deflation curve being displaced to the left. This dissimilarity in pressure-volume curves is called hysteresis. Why there is the hysteresis phenomenon is unsettled, but it appears to be related to changes in surface tension forces at the alveolar air-surface interface.[7]

Compliance, by definition a static determination, is measured during periods of no gas flow to eliminate all flow dependent variables; *i.e.*, airway resistance and frictional tissue resistance. Investigators have also measured compliance during spontaneous breathing by relating the change in lung volume to the differential in transpulmonary pressure between end-inspiration and end-expiration—assuming that flow resistive pressures are zero at these intervals. This dynamic pressure-volume relation or dynamic compliance appears to be similar to the static compliance value in most normal lungs. However, if the mechanical time constants for gas flow to various lung segments are different, such as might occur during rapid breathing or in patients with chronic pulmonary emphysema or induced bronchoconstriction, dynamic compliance may be appreciably lower than static compliance. Since tachypnea is frequent in anesthetized patients and since the effects of anesthetics on time constants of the lung are not known, interpretations of dynamic compliance values from anesthetized patients are subject to question. Although Nightengale and Richards did not measure flow, the system employed probably was static since each inflating pressure was held for 10 seconds, an adequate time to attain pressure equilibrium across nondiseased lungs.

Finally, although Nightingale and Richards noted no significant difference in total compliance between infants paralyzed with *d*-tubocurarine (present study) and succinylcholine (prior study), one cannot assume these drugs have the same impact on total compliance. A valid comparison must be done in the same patient population under identical circumstances. It is frequently stated from animal experiments that *d*-tubocurarine is a bronchoconstrictor, probably resulting from release of histamine. Change in bronchomotor tone with *d*-tubocurarine has never conclusively been proven to occur in man: the finding by Nightingale and Richards that total pulmonary compliance is no greater in infants paralyzed with *d*-tubocurarine then succinylcholine, argues against any appreciable alterations in airway size owing to this drug in this age group.

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