conventionally obtained by dividing the tidal volume by the difference between the "plateau" pressure measured at the airway opening (Pao) during an occlusion at end-inspiration and positive end-expiratory pressure (PEEP) set by the ventilator. This analysis is valid only if the elastic recoil pressure of the respiratory system is zero at the end of expiration, indicating that the system has reached its elastic equilibrium point. To test if this is always the case, in 14 mechanically ventilated patients with acute respiratory failure, measurements were made of Pao and of flow and volume changes. In only four of the patients did expiratory flow become nil before end-expiration and inspiratory flow started synchronously with the onset of the positive-pressure swing delivered by the ventilator, indicating that in these four patients the end-expiratory elastic recoil pressure was indeed zero. By contrast, in the remaining ten subjects, expiratory flow was still present when the ventilator had already begun to increase Pao, indicating that the end-expiratory elastic recoil pressure was not zero. Indeed, in all these ten patients, a positive ΔPao (as much as 7.5 cm H_2O) had to be applied by the ventilator before the actual onset of inspiratory flow. This ΔPao represents the pressure required to counterbalance the end-expiratory elastic recoil before inspiratory flow will begin, and can be termed intrinsic PEEP. Clearly, in order to obtain a valid measurement of respiratory compliance, the tidal volume has to be divided by the difference between end-inspiratory "plateau" Pao and total PEEP, which includes both externally applied and intrinsic PEEP. In the ten patients with intrinsic PEEP, the value of static respiratory compliance computed, without taking intrinsic PEEP into account, underestimated the correct value by as much as 48%. Apart from its implications on measurements of respiratory system compliance, intrinsic PEEP may have adverse effects in terms of hemodynamics and weaning.

Work of Breathing in Patients With Chronic Obstructive Pulmonary Disease in Acute Respiratory Failure. *Fleury B, Murciano D, Talamo C, et al.* Am Rev Respir Dis 131:822, 1985.

In 11 spontaneously breathing patients with chronic obstructive pulmonary disease (COPD) in acute ventilatory failure, we measured the total inspiratory (Wltot) and total resistive (Wl + Eres) work rate of breathing, together with lung mechanics (dynamic pulmonary elastance and inspiratory and expiratory pulmonary flow resistance). All variables were markedly increased compared with those in normal subjects. No significant correlation was found between Wltot and Wl + Eres with lung mechanics data. However, when Wl + Eres were expressed/L of ventilation, a significant positive correlation was found with all lung mechanics data. These results indicate that although in patients acutely ill with COPD, work rate and work/L of ventilation are increased, only the latter is related to the severity of pulmonary mechanical impairment, and it could be used as one of the criteria for extubation. In addition, our results indicate that at end-expiration the alveolar pressure was positive (range, 6 to 13 cm H_2O) in all patients (intrinsic PEEP), a fact that must necessarily affect hemodynamics; furthermore, it imposes an extra burden on the inspiratory muscles.

Aminophylline and Its Influence on Ventilatory Endurance in Humans. *Belman MJ, Sieck GC, and Mazar A.* Am Rev Respir Dis 131:226, 1985.

The purpose of this study was to evaluate whether the previously demonstrated improvement in contractile tension of diaphragmatic muscle with aminophylline results in improved ventilatory endurance. We measured the maximal sustained ventilatory levels during prolonged isocapnic hyperpnea as an index of ventilatory muscle function. This measurement was made in seven normal subjects and seven patients with chronic obstructive pulmonary disease during the intravenous administration of saline and aminophylline on two separate days. The order of administration of the infusions was randomized. Although both groups showed slightly higher sustained ventilatory levels during aminophylline infusion, the magnitude of change was small and unlikely to have a significant clinical benefit in the setting of respiratory muscle fatigue.

Inspiratory Muscle Function in Patients With Severe Kyphoscoliosis. *Lisboa C, Moreno R, Fava M, et al.* Am Rev Respir Dis 132:48, 1985.

In nine patients with severe kyphoscoliosis, we studied inspiratory muscle function by measuring transdiaphragmatic pressure (Pdi) and its components: gastric (Pga) and esophageal (Pes) pressures during quiet breathing. Maximal Pdi and maximal inspiratory mouth pressure (Pimax) were also measured. The results showed that Pimax and Pdimax were significantly lower in patients than in normal subjects. During quiet breathing, all patients had positive swings in Pga, indicating an active contraction of the diaphragm, but Pes was significantly more negative, suggesting the recruitment of intercostal and accessory inspiratory muscles. We did not find significant correlations between Pimax, Pdimax, ΔPga/ΔPes, FVC, PaO_2, or $PaCO_2$ and the degree of spinal deformity. The FVC tended to correlate with Pimax ($r = .63$) and with Pdimax ($r = .53$). The Pdi correlated with PaO_2 ($r = .66$) and with $PaCO_2$ ($r = .76$, $P < .05$). A significant correlation was also observed between Pimax and PaO_2 ($r = .785$, $P < .05$) and between Pimax and $PaCO_2$ ($r = .86$, $P < .01$). We concluded that impairment of inspiratory muscle function is related to the development of ventilatory failure in kyphoscoliosis.

Determinants of Maximal Inspiratory Pressure in Chronic Obstructive Pulmonary Disease. *Rochester DF and Braun NMT.* Am Rev Respir Dis 132:42, 1985.

Inspiratory muscle strength in COPD could be reduced either because of mechanical disadvantage consequent to increased lung volume or because respiratory muscles share in generalized muscle weakness. To assess the relative contributions of these factors, we measured maximal inspiratory and expiratory pressures (Pimax, PEmax, cm H_2O) at RV and TLC, respectively, in 32 patients with COPD. The TLC, RV, and diaphragm length index at RV (DLI, cm/cm height) were determined roentgenographically and compared with values from 22 normal subjects studied at comparable lung volume. Half the patients with COPD had normal and low values of PEmax, but both groups had similar values of TLC, RC, and DLI. In patients with COPD, Pimax corre-