Communications to the Editor

Estimation of Auto-PEEP

To the Editor:

The recent article by Hoffman and co-workers1 proposes a method to determine auto-PEEP in mechanically ventilated patients by using respiratory inductive plethysmography. Their method is based on the assumption that when the level of extrinsic PEEP is increased in a patient with auto-PEEP the auto-PEEP will be ablated before hyperinflation (a rise in FRC) occurs. Tuxen2 recently described six patients with auto-PEEP who received varying amounts of extrinsic PEEP. In most of these patients, as extrinsic PEEP was added, FRC rose before their auto-PEEP was relieved. Smith and Marin3 studied ten mechanically ventilated patients with chronic airflow obstruction, and they found that the application of extrinsic PEEP (10 cm H2O) in excess of the auto-PEEP (mean, 6.2 cm H2O) did not completely ablate the auto-PEEP.

Hoffman and co-workers have done intensivists a service by describing a method that will allow the application of some extrinsic PEEP to relieve some auto-PEEP without causing hyperinflation. However, the use of this method to determine the exact level of auto-PEEP would be misleading in many patients.

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2 Tuxen DV. Detrimental effects of positive end-expiratory pressure during controlled mechanical ventilation of patients with severe airflow obstruction. Am Rev Respir Dis 1989; 140:5-9

To the Editor:

Hoffman et al1 have studied the effects of PEEP on end-expiratory lung volume (VEE) in mechanically ventilated patients with dynamic hyperinflation and concluded that VEE increases only when PEEP at the proximal airway exceeds intrinsic PEEP (PEEP). This conclusion does not follow from the known determinants of passive expiratory flow and is not supported by their data. In Figure 1 of their paper, VEE starts to increase at PEEP levels between 4 and 6 cm H2O, ie, at pressures below PEEP, PEEP, is the recoil pressure of the respiratory system at VEE and represents a substantial portion of the driving pressure for expiratory flow, particularly in the lower tidal range. In the absence of expiratory flow limitation, any PEEP at the airway (reduction in driving pressure) must result in reduced isovolume flows and, thereby, raise VEE. In the presence of expiratory flow limitation, small levels of PEEP may not alter isovolume flow because the driving pressure for expiratory flow (elastic recoil pressure of the system minus airway pressure) still remains in excess of the pressure necessary to reach maximal flow. However, reducing it by a pressure equal to PEEP, invariably lowers it to subthreshold levels. In an accompanying editorial, Tobin et al support the hypothesis of Hoffman et al by using the waterfall analogy of expiratory flow limitation. However, Tobin et al assumed incorrectly that the height of the waterfall is equal to PEEP. In the article in which the waterfall analogy was first described, Pride et al related the height of the waterfall to a pressure difference between the flow-limiting segment in the lung and the mouth (downstream pressure), while the driving pressure for flow was given by the pressure difference between the alveolus and the flow-limiting segment (ie, upstream of the waterfall). Since PEEP represents the total driving pressure (upstream and downstream of the waterfall) that generates flow from the alveoli past the flow-limiting segment, the proximal airways, the endotracheal tube, ventilator circuit, and the PEEP valve, opposing PEEP, with an equal the pressure at the expiratory port of the ventilator must alter isovolume flow and cause further air trapping.

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To the Editor:

The comments by Drs Hubmayr and Judson are very welcome because they highlight some of the difficulties and misconceptions concerning auto and intrinsic positive end-expiratory pressure (PEEP). However, I feel that they have missed the thrust of our article which, as stated in the abstract and conclusion, was to evaluate whether changes in end-expiratory thoracic gas volume (EVTG) as measured by respiratory inductive plethysmography (RIP)
Communications

520

6 Tuxen DV Detrimental effects of positive end-expiratory pressure during controlled mechanical ventilation of patients with severe airflow obstruction. Am Rev Respir Dis 1989; 140:5-9

To the Editor:

I read with great interest the editorial by Drs Tobin and Lodato in the September 1989 issue of Chest. I thought their waterfall analogy was excellent (their Figure 1), but I would like to contribute a brief response. The lung is made of many gas exchange units, and there is no reason to suppose that all of these units might have the same auto-PEEP in the presence of airflow limitation during mechanical ventilation. In fact, it is more than likely that there is a variability of levels of auto-PEEP from one gas exchange unit to the next because of inhomogeneities in small airways within the same obstructed lungs. Drs Tobin and Lodato have defined a "critical level of external PEEP" which, in the presence of auto-PEEP, is presumably the highest external PEEP that can be applied without impeding expiratory airflow or causing hyperinflation. However, if one dissects this issue further, differences in auto-PEEP among gas exchange units would mean that in order to administer the critical PEEP to the gas exchange unit with the highest intrinsic PEEP or auto-PEEP, one would have to surpass the critical PEEPs of all of the other gas exchange units in the lungs. This could lead to hyperinflation and a decrease in trapped gas volume above FRC in these other units. More specifically, as opposed to a single waterfall, such lungs may be described as a circuit of many waterfalls of different heights situated in parallel, each waterfall representing a gas exchange unit with a different auto-PEEP. Thus, the lungs in expiration may behave more approximately like an ohmic circuit with each increment in extrinsic PEEP, some of the obstructed units are opened, and overall expiratory airway resistance with its accompanying gas trapping diminishes. Consistent with this notion, recently published data in patients revealed that the administration of increasing levels of extrinsic PEEP during airflow obstruction resulted in progressive hyperinflation while the gas volume trapped above FRC progressively decreased. Thus, although the analogy of a single waterfall has strength at the level of a single gas exchange unit, it may not be precise at a clinical level in reference to the lungs as a whole.

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To the Editor:

We are grateful for the interest expressed by Dr Schnader and by Dr Hubmayr and his colleagues in our editorial and are pleased to have the opportunity to respond to their letters.

It has not escaped our attention that the lungs of patients with airflow limitation most likely consist of a heterogeneous distribution of units, each with its own critical level of external positive end-expiratory pressure (PEEP), which we shall refer to as "waterfall pressure." Of course, analysis of such a system is indeed more complex than we presented in our editorial, but it is not inapproachable. For example, we offer the following solution to the problem

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6 Tuxen DV Detrimental effects of positive end-expiratory pressure during controlled mechanical ventilation of patients with severe airflow obstruction. Am Rev Respir Dis 1989; 140:5-9

520