Communications for this section will be published as space and priorities permit. The comments should not exceed 350 words in length, with a maximum of five references; one figure or table can be printed. Exceptions may occur under particular circumstances. Contributions may include comments on articles published in this periodical, or they may be reports of unique educational character. Specific permission to publish should be cited in a covering letter or appended as a postscript.

Estimation of Auto-PEEP

To the Editor:

The recent article by Hoffman and co-workers 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. Tuxen 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 Marin 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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To the Editor:

Hoffman et al. 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, i.e., 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 (TVG) as measured by respiratory inductive plethysmography (RIP)
could be used as a convenient method to estimate intrinsic PEEP (PEEP). We did not attempt to prove that TCV increased only after extrinsic PEEP (PEEP), nor that PEEP, by RIP, exactly equaled PEEP, by other methods. In fact, our method required a change in TCV of ≥10 percent of tidal volume (VT) before PEEP, by RIP was determined. As Dr Hubmayr mentioned, Figure 1 in our article demonstrated a small increase in end-expiratory TCV after 4 cm H2O of PEEP, was applied. However, since this increase was <10 percent of the VT, the PEEP, by RIP was taken after the next increment of PEEP, Dr Hubmayr and his associates have demonstrated that the addition of PEEP, caused a small (0.07 to 0.22 L) change in volume at end-expiration (VEE) in seven paralyzed patients with airflow obstruction and dynamic hyperinflation. Our patient population was heterogeneous and not paralyzed, and the PEEP, that was detected by RIP or occlusion may not have always been secondary to dynamic hyperinflation. The PEEP, recorded from RIP was not identical to PEEP, by occlusion but only within 2.0 to 2.5 cm H2O, similar to the change in PEEP, that Dr Hubmayr has previously reported to occur when PEEP, equal to or slightly less than PEEP, was added to five patients.

Dr Judson referred to two articles, one by Drs Smith and Marini and the other by Dr Tuxen, in which the effects of PEEP were studied in patients with severe airflow obstruction (either asthma or chronic airway obstruction), most of whom were paralyzed, whereas our population was much more heterogeneous and not paralyzed. As Dr Marini emphasized in his article with Dr Smith and in an editorial accompanying Dr Tuxen's publication, there are at least three distinct forms of PEEP, as well as various combinations thereof. Therefore, the pathophysiology of the individual patient, the method, and the definition of PEEP, have to be considered before attaching significance to or making therapeutic decisions based on the detection of PEEP. For example, PEEP, was represented by Dr Tuxen as the volume of trapped gas above FRC (Vcompl) after a 30- to 50-s period of apnea in paralyzed patients. Examination of Figure 2 in his article shows that PEEP, did not increase significantly until the original PEEP, (ie, Vcompl at PEEP = 0) was exceeded by PEEP, Dr Judson also noted that the application of PEEP, in excess of the original PEEP, level failed to alleviate PEEP, in the article by Smith and Marini, although the levels recorded were <2.5 cm H2O and therefore within our estimation of PEEP, by RIP.

In summary, the comments from Dr Judson and Dr Hubmayr are very important because they help to point out that all auto-PEEP is not necessarily equal. What we showed in our article was that changes in end-expiratory TCV measured by RIP can be used as a convenient, clinically useful estimate of PEEP.

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To the Editor:

I read with great interest the editorial by Drs Tobin and Lodato in the September 1989 issue of Chest.1 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