

## Commentary

# Partitioning the work-sparing effects of partial ventilatory support in airflow obstruction

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### Abstract

Sweeping conclusions regarding the utility or nonutility of elevating expiratory pressure are not warranted. The effects of manipulating airway pressure in the setting of airflow obstruction depend heavily on the nature and severity of disease, as well as on the presence of airflow limitation during tidal breathing.

**Keywords** airflow obstruction, CPAP, partial ventilatory support

In this issue of *Critical Care*, Miro and colleagues [1] report the results of an experiment designed to determine the relative effects on work of breathing of the inspiratory and expiratory components of applied airway pressure. After a methacholine aerosol was used to induce bronchoconstriction in spontaneously breathing, anesthetized dogs, equal levels of inspiratory positive airway pressure (IPAP), expiratory positive airway pressure (EPAP), and continuous positive airway pressure (CPAP) were applied, and the pressures that developed across the lungs and diaphragm were measured. The authors concluded that any reduction in inspiratory effort attributable to positive pressure during acute bronchospasm is caused primarily by the IPAP component of the airway pressure profile. Although I heartily agree that this interpretation seems correct for the model and conditions they examined, I suspect that the implications of this work extend only to a small subset of patients with acute airflow obstruction.

Fifteen years have passed since I was asked to write an editorial [2], which was in response to a research paper by Tuxen [3] that failed to show benefit from using positive end-expiratory pressure (PEEP) in acute asthma. My point then – as it is now – is that sweeping conclusions regarding the utility or nonutility of elevating expiratory pressure are not warranted. The effects of manipulating airway pressure in the

setting of airflow obstruction depend heavily on the nature and severity of disease, as well as on the presence of airflow limitation during tidal breathing.

Surely it comes as no surprise that a phasic boost of airway pressure during inspiration would assist the inspiratory muscles to accomplish their task of drawing fresh gas into the lung. Because the lung is a passive structure, this expectation follows directly from its simplified equation of motion –  $P = FR_i + V_t/C_L + PEEP_i$  – and from the fact that the work performed on the lung during inspiration is the integral of pressure and flow over the inspiratory period. In this equation,  $P$  is the transpulmonary pressure (the difference between the pressures at the airway opening and pleural space),  $F$  is flow rate,  $R_i$  is inspiratory resistance,  $V_t$  is tidal volume,  $C_L$  is lung compliance, and  $PEEP_i$  is intrinsic PEEP or auto-PEEP. With unchanging impedance properties of  $R_i$  and  $C_L$ , the job is set – at least for the same flow profile and tidal volume.

Despite the mathematical certainty implied by this relationship, in a sense I do agree with the authors' statement that '... neither the relative contribution of either EPAP or IPAP, nor the mechanisms by which they exert their effect on the *work of breathing* [emphasis added] are currently understood' [1]. The changes in breathing depth, breathing

rhythm, and muscle activation pattern evoked by these modalities are not so easily predictable and have not been well elucidated in patients or in normal human subjects, let alone in spontaneously breathing animals.

Nonetheless, we do have some solid research observations to guide us in this area. Many normal persons activate the expiratory muscles to oppose end-expiratory pressure in relation to the magnitude of the pressure applied and the minute ventilation [4]. This attempt to reduce the lung's end-expiratory set-point is a wise strategy if the objective is to avoid mechanically disadvantaging the inspiratory muscles by hyperinflation and to allow the expiratory muscles to leverage the applied airway pressure to aid their inspiratory counterparts. A similar 'work-sharing' strategy is employed routinely during exercise so as to avoid dynamic hyperinflation under conditions of strenuous effort and high minute ventilation [5].

A previous comparison of methods to raise end-expiratory pressure conducted a quarter century ago by Katz and colleagues [6] concluded that EPAP (e.g. inspiring at ambient airway pressure but expiring through a hose placed under water) was associated with greater inspiratory effort ('work of breathing') than was CPAP, but less rise in cardiac output-impeding pleural pressure. It is important to stress, however, that those observations were not in patients with airflow obstruction. In modern practice, there seems to be little place for EPAP – CPAP fulfills the beneficial functions of elevating expiratory pressure with less inspiratory workload, less tendency to afterload the left ventricle, and only modest effects on venous return. (Parenthetically, I cannot remember encountering a spontaneously breathing patient whose venous return was seriously affected by modest levels of CPAP, but perhaps my memory fails me.)

With passive inflation, the work performed in moving the integrated respiratory system (lungs and chest wall) can both be measured and partitioned; during spontaneous or partially assisted breathing, however, only the mechanical work of inflating the lung can be assessed because the pressures developed by the muscles in moving the chest wall cannot be determined. At this point it is appropriate to note that although 'work' is an emotive expression that suggests 'effort' or 'exertion', these two words are not synonymous. Isometric effort generates pressure and raises oxygen consumption without measurable work, and therefore the pressure–time product correlates better with oxygen consumption. Moreover, for the same externally measured workload, the efficiency of the muscles in generating pleural pressure varies with the configuration of the chest wall and the extent to which the muscles are preloaded.

In the specific setting of airflow obstruction, raising end-expiratory pressure has a variable effect, depending on the degree to which it counterbalances auto-PEEP, evens the

distribution of ventilation, or leads to further hyperinflation. In the study conducted by Miro and colleagues [1], the extent of bronchoconstriction was very modest and no auto-PEEP was generated. It follows that because no flow limitation occurred during tidal breathing, EPAP would be unlikely to help. Moreover, these anesthetized animals did not activate their expiratory muscles sufficiently to raise intra-abdominal pressure, limiting any potential 'work-sharing' benefit. In other experimental or clinical circumstances, however, quite the opposite conclusion might be justified. Reasoning from basic principles, I would predict that a patient with dynamic hyperinflation and flow limitation might obtain significant benefit from the expiratory component of elevated airway pressure. A similar comment applies to the patient making forceful expiratory efforts. (Raised expiratory pressure might also help even the distribution of ventilation among units with varying levels of auto-PEEP.) Conversely, in a patient lacking either expiratory flow limitation or activated expiratory musculature, any increase in end-expiratory lung volume could prove counterproductive, disadvantaging the inspiratory muscles and perhaps reducing the compliance of the respiratory system.

In the end, we cannot conclude on the basis of this experimental work that raising expiratory airway pressure does not make a major contribution to the work benefit accruing to positive airway pressure in the setting of airflow obstruction. However, although the implications of the study are limited, I commend the authors for conducting an interesting study of integrative physiology that directly addresses a 'real world' medical question. Although not trendy or fashionable in these days of exciting molecular biology and influential clinical trials, such mechanism probing, systems level physiologic investigations inform clinical practice and deserve to be encouraged.

## Competing interests

None declared.

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