Invited Editorial on “Detection of expiratory flow limitation during exercise in COPD patients”

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In humans, expiration during quiet breathing is usually considered passive, since expiratory muscles are not active.Expiration is driven by elastic energy stored in the respiratory system at end inspiration. Inspiratory muscles relax gradually, reducing flow in the early part of expiration so that lung volume does not reach functional residual capacity (FRC) before time to initiate the next breath (12). In humans at rest, FRC is the elastic equilibrium volume between the recoils of the lung and chest wall. Dogs and most other animals utilize active expiration while awake (4) so that FRC is lower than the relaxation volume of the respiratory system. During exercise, normal humans increase ventilation in part by recruitment of expiratory muscles (7). Tidal volume increases initially as much by reduction of FRC as increase of end-inspiratory volume (1, 15). Both tidal volume and frequency increase, although over most of the exercise range increased ventilation is produced predominantly by increases in tidal volume. The reduction in FRC can exceed 50% of the expiratory reserve volume (1, 15). In individuals who never reach maximal expiratory flow, the decrease in FRC presumably is limited by nonlinearities of the chest wall pressure-volume relationship.

Recent data support the following scenario of the effects of reduction of maximal flow on ventilation during exercise. With aging, there is a reduction in maximal expiratory flow so that increasing ventilation with FRC below the resting volume soon produces maximal flows (2, 6, 8, 15). Once this occurs, further increases in ventilation are produced by preserving the normal relationship between mean inspiratory and expiratory flow rates and increasing FRC rather than increasing expiratory effort to utilize maximal flows throughout expiration. Extremely fit elderly individuals may return to or exceed resting FRC at maximal exercise (8). When end inspiration approaches total lung capacity (TLC), end-inspiratory volume does not increase further.

If respiratory timing (inspiratory-to-total time) is preserved, increased ventilation requires an increased mean expiratory flow rate. When expiratory flow is maximal, mean lung volume must be increased to increase mean expiratory flow. Once end-inspiratory volume approaches TLC, mean lung volume can only be increased by increasing FRC. Therefore, at high levels of ventilation, tidal volume plateaus and then decreases (6). Ventilation increases only by increased frequency. Although the mechanisms of this response are not known, it appears that the respiratory controller is programmed to maintain normal timing if possible and that achievement of maximal flow is a powerful stimulus to terminate expiration and initiate the next breath (10).

With airway obstruction, more inspiratory muscle activation is required during inspiration to overcome the increased airway resistance, but less inspiratory muscle braking is required to retard expiratory flow. Peak expiratory flow occurs earlier during expiration, and the tidal flow-volume loop resembles a miniature of the maximal flow-volume curve. Expiration may be slowed to the point that the relaxation point is not reached before the next inspiration, and FRC becomes dynamically rather than statically determined (14). With more severe obstruction, elastic recoil of the respiratory system may be sufficient to generate maximal flow without expiratory effort. Whether or not the subjects use maximal expiratory flow at rest, maximal flow below FRC is so low that patients cannot reduce FRC with exercise, and both FRC and end-inspiratory volume increase with increasing ventilation. This reduces the ability of subjects to increase tidal volume and thus limits maximal exercise ventilation (2, 14). When end inspiration is near TLC, each inspiration requires a near-maximal inspiratory effort (11).

These findings suggest that lung mechanics may be a much more important determinant of exercise capacity than has previously been appreciated. There is considerable variability in these general relationships. Some subjects use maximal flow over most of the tidal volume and others only at FRC. Maximal inspiratory volume occurs at different fractions of TLC. These differences may contribute to the variability of the relationship between maximal exercise capacity and forced expiratory volume in 1 s (FEV1) in patients in whom lung mechanics may be an important determinant of maximal oxygen uptake (VO2max). Reaching maximal expiratory flow over even part of the tidal volume range appears to play an important role in both the mechanics and neural control of respiration, which have not been studied adequately.

The paper by Koulouris et al. (9) in this issue of the Journal of Applied Physiology describes a useful technique for determining when subjects are utilizing max-
mum tidal volume was a better predictor of $V\dot{O}_2\text{max}$ than was FEV$_1$. However, this result may be due, in part, to the fact that the normal subjects were younger and taller and the tidal volume data were not normalized for age or size. These data confirm that the presence of flow limitation affects the ventilatory response to exercise and maximum exercise capacity. It remains to be demonstrated how much this contributes to the variability in $V\dot{O}_2\text{max}$ for an FEV$_1$ low enough that $V\dot{O}_2\text{max}$ should be determined by lung disease.

REFERENCES