

# Expiratory flow limitation and regulation of end-expiratory lung volume during exercise

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PELLEGRINO, R., V. BRUSASCO, J. R. RODARTE, AND T. G. BABB. Expiratory flow limitation and regulation of end-expiratory lung volume during exercise. *J. Appl. Physiol.* 74(5): 2552–2558, 1993.—To investigate the impact of expiratory flow limitation (FL) on breathing pattern and end-expiratory lung volume (EELV), we imposed a small expiratory threshold load for a few breaths during exercise in nine volunteers (29–62 yr): six were healthy and three had mild-to-moderate airflow obstruction (67–71% predicted forced expiratory volume in 1 s). Six subjects showed evidence of FL, i.e., tidal expiratory flow impinging on maximal forced expiratory flow, at one or more exercise levels. Whenever an expiratory threshold load was imposed, mean expiratory flow decreased ( $P < 0.02$ ) in association with an increased expiratory time ( $T_E$ ;  $P < 0.05$ ). When the load was imposed during non-FL conditions,  $T_E$  increased less than expiratory flow decreased and EELV tended to increase. In contrast, during FL, with the load,  $T_E$  increased more than expiratory flow decreased, subjects did not achieve maximal expiratory flow until a lower volume, and EELV decreased ( $P < 0.001$ ). Under both FL and no-FL conditions, unloading reversed the changes associated with loading. These data indicate that the increase in EELV during exercise is linked to the occurrence of FL. We suggest that compression of airways downstream from the flow-limiting segment may elicit a reflex mechanism that influences breathing pattern by terminating expiration prematurely, thus increasing EELV.

exercise hyperpnea; breathing pattern; dynamic airway compression; transpulmonary pressure; expiratory threshold load

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THE INCREASE in minute ventilation ( $\dot{V}_E$ ) during exercise is obtained in normal subjects through an increase of both respiratory rate ( $f$ ) and tidal volume ( $V_T$ ). The increase in  $V_T$  is generally achieved by increasing end-inspiratory lung volume (EILV) and reducing end-expiratory lung volume (EELV), so that subjects breathe around resting functional residual capacity (FRC) (1, 8, 13, 25). In contrast, an increase of EELV has been observed occasionally in healthy subjects at exceptionally high levels of exercise when maximal expiratory flow is approached (2, 6, 9, 19) and in highly fit older individuals (10, 21). A marked increase in EELV is found in subjects with chronic obstructive pulmonary disease, even during low-intensity exercise (3, 5, 7, 12, 20, 22, 24). Even though the available data suggest that the increase in EELV during exercise is associated with achieving maximal expiratory flow over a large portion of  $V_T$  (2, 9, 10), the underlying mechanisms have not been clarified.

When expiratory flow during tidal breathing is increased to levels at which flow limitation (FL) occurs and if the expiratory muscles generate a transpulmonary pressure ( $P_{tp}$ , the difference between esophageal and airway opening pressure) that exceeds the minimal pressure necessary to produce maximal flow ( $P_{crit}$ ), then the airways downstream from the flow-limiting segment undergo dynamic compression (19). This dynamic compression, which may cause discomfort and dyspnea (17, 18, 23), may elicit reflexes that change breathing pattern and terminate expiration prematurely (11), thus increasing EELV.

To explore whether dynamic compression has an effect on breathing pattern and regulation of EELV during exercise, we attempted to change  $P_{tp}$  and expiratory flow by using an expiratory threshold load in subjects with and without FL. We reasoned that if achieving maximal flow had important effects on ventilatory patterns, then the response of subjects with FL to an expiratory threshold load would be different from the response of subjects without FL. For example, in subjects without FL, if expiratory effort is not increased to completely compensate for the sudden imposition of an expiratory threshold load, then the load will reduce expiratory flow and  $P_{tp}$ . The lower flow rate would allow less volume to be expired before the initiation of inspiration, and EELV might be shifted to a higher lung volume. Subjects who achieve maximal flow during expiration would already be at a higher EELV than they would have been in the absence of FL and an expiratory threshold load that reduces flow would delay the onset of achieving maximal expiratory flow and shift EELV to a lower lung volume.

## METHODS

**Subjects.** Nine male volunteers, whose anthropometric and lung function data are presented in Table 1, participated in the study. Six subjects had normal lung function and considered themselves to be healthy. Four of these subjects were nonsmokers, and two were mild smokers; five subjects were physically active in recreational activities, and one subject was sedentary. Three subjects had mild-to-moderate airflow obstruction with a forced expiratory volume in 1 s ( $FEV_1$ ) between 67 and 71% of predicted or a percentage of forced vital capacity expired in 1 s of  $<70$  ( $FEV_1/FVC$ ). These subjects were current smokers. The project was approved by the Institutional

TABLE 1. Subjects' physical characteristics, pulmonary function, and maximal exercise performance

	Subj No.								
	1	2	3	4	5	6	7	8	9
Age, yr	61	47	62	38	52	29	56	36	49
Height, cm	178	178	178	183	188	178	183	182	178
VC									
liters	3.7	4.2	4.4	6.0	5.7	5.6	6.2	6.5	5.2
%pred	84	84	93	109	106	104	126	115	109
TLC									
liters	7.1	6.8	8.5	8.0	7.8	7.0	9.6	8.8	7.6
%pred	104	100	124	110	103	101	133	129	112
RV/TLC, %	40	33	38	26	27	14	29	26	25
FEV <sub>1</sub>									
liters	2.3	2.8	2.6	4.2	4.0	4.5	4.3	5.4	5.2
%pred	67	69	71	93	97	101	108	116	109
FEV <sub>1</sub> /FVC, %	63	66	59	70	72	80	70	83	86
Ptp <sub>100 TLC</sub> , cmH <sub>2</sub> O	18	23	16	38	26	32	18	28	30
MVV, l/min	122	140	145	196	159	193	163	217	208
$\dot{V}O_{2 \max}$ , ml · kg <sup>-1</sup> · min <sup>-1</sup>	20	24	21	26	33		35	36	23
$\dot{V}E_{\max}$ , l/min	59	125	69	85	114		114	113	113

VC, vital capacity; TLC, total lung capacity; RV, residual volume; FEV<sub>1</sub>, forced expiratory volume in 1 s; FVC, forced vital capacity; Ptp<sub>100 TLC</sub>, transpulmonary pressure at TLC; MVV, maximal voluntary ventilation;  $\dot{V}O_{2 \max}$ , maximal O<sub>2</sub> uptake;  $\dot{V}E_{\max}$ , maximal minute ventilation.

Review Board, and written consent was obtained from each subject before participation.

*Measurements of lung function and exercise capacity.* Standard spirometry and electrocardiograms were obtained before subjects were admitted to the study. Static lung volumes were measured while the subjects were sitting in a pressure-corrected volume-displacement body plethysmograph. Flow was measured by a pneumotachograph (Fleisch no. 3) placed distal to the mouthpiece (Validyne transducer MP45 ±2 cmH<sub>2</sub>O). Ptp was estimated as the difference between mouth and esophageal pressure (Statham 131 ±5 psi). Esophageal pressure was obtained through a 10-cm-long thin latex balloon positioned 45 cm from the nostril and filled with ~1.3 ml of air. The balloon was connected through a polyethylene catheter to the pressure transducer. Validity of the balloon pressure was checked by having the subjects blow against an occluded airway. If Ptp remained constant while oral pressure increased, placement was considered appropriate. Flow, volume, and Ptp were displayed on a strip chart recorder (HP-7758A) and sampled in real time (66 Hz) on a computer (DEC11/73) for subsequent analysis.

A maximum incremental exercise test was performed on an electronically braked cycle ergometer (Medical Graphics) by increasing the work rate by 30 W/min. Subjects breathed through a mouthpiece and a low-resistance Hans Rudolph valve; the dead space was ~150 ml. Inspiratory and expiratory flows were measured separately by two Fleisch no. 3 pneumotachographs coupled to Celesco ±2-cmH<sub>2</sub>O pressure transducers. Volume was derived by integration of the summed flow signals. Ptp was determined using the same methodology as described above. Breath-by-breath gas exchange was determined using a Medical Graphics System model 2001.

*Experimental protocol.* After the screening, subjects visited the laboratory on a separate day to be studied during submaximal exercise at various work rates. After an esophageal balloon was placed to determine Ptp and two or three maximal forced flow-volume curves were re-

corded, the subjects were asked to cycle at 60 W for 4–6 min until  $\dot{V}E$  reached a steady state. The work load was then increased by using steps equal to 30 W or multiples of this quantity to achieve various levels of  $\dot{V}E$ . Each step was maintained for 2 min. A 2-min “recovery” at 60 W was allowed between each step.

During the last part of each step, a three-way stopcock, positioned in the expiratory line, was opened to a second expiratory circuit for 15–25 s, by which we imposed a 5-cmH<sub>2</sub>O threshold load on expiration. This expiratory circuit consisted of a 5-cm-diam latex tube that could be collapsed by generating a positive pressure surrounding it. The latex tube was sealed inside a rigid plastic chamber where pressure could be controlled by the inflow of compressed air. The addition of a connecting reservoir volume and a positive end-expiratory pressure outlet valve to the plastic chamber kept pressure constant in the chamber during a breathing cycle. Pressure in the chamber was monitored by a water manometer. The total resistance of the circuit was 1.2, 2.2, and 3.8 cmH<sub>2</sub>O at flows of 1, 2, and 3 l/s, respectively, and was not altered by the 5-cmH<sub>2</sub>O load. The chamber and the stopcock were hidden from view of the subjects, so that they could not anticipate when the expiratory load was to be applied or removed. After unloading, the subjects breathed normally for another 15–25 s and then were asked to perform a partial forced expiratory maneuver from EILV to below EELV and then to inhale to total lung capacity. These partial forced expiratory maneuvers were used to confirm whether subjects were using maximal expiratory flow during tidal breathing. Upon completion of a series of steps, the test was ended and two maximal expiratory flow-volume curves were recorded to ascertain whether bronchial caliber had changed with exercise.

*Data analysis.* Volume, flow, and Ptp were continuously recorded during exercise, and the data were stored in a computer. Irregular breaths, sighs, and swallows were discarded. The computer-stored data were screened by the investigators and played back on a graphics termi-

nal to generate exercise flow-volume and pressure-volume loops. The analysis of the ventilatory pattern was based on 6–10 breaths sampled before loading (C), during loading (L), and after unloading (UL). The two control periods, C and UL, allowed the drift of spirometric output to be corrected before EELV was estimated. This technique has been shown to accurately determine changes in EELV during exercise (1).  $\dot{V}_E$ ,  $V_T$ ,  $f$ , expiratory time ( $T_E$ ), and mean expiratory flow ( $V_T/T_E$ ) were calculated breath by breath and averaged over each sampling period, i.e., C, L, and UL. For each of these variables, the ratio of L and UL to C was calculated. For EELV, the differences between L and C and between UL and C were calculated. Initially, we looked at the first loaded and unloaded breaths. However, the patterns were stable over several breaths, and data were more consistent by averaging over 6–10 breaths. We used analysis techniques similar to those used by Goldstein et al. (4).

The flow and volume signals recorded during loaded and unloaded breathing were superimposed on the flow and volume signals obtained during the partial forced expiratory maneuvers. The impingement of tidal expiratory flow on the forced expiratory flow was considered as evidence of FL.

**Statistics.** To avoid biasing by the different number of observations in individual subjects, the data observed in each subject at all levels of ventilation during FL and during no-FL conditions were averaged. Significance testing across subjects was accomplished by using paired Student's *t* tests. For changes in  $\dot{V}_E$ ,  $V_T$ ,  $f$ ,  $T_E$ , and  $V_T/T_E$ , the ratio of loaded to unloaded measurements was compared with unity. For changes in EELV, the volume change was compared with zero.  $P < 0.05$  was considered to be statistically significant. Data are expressed as means  $\pm$  SD.

## RESULTS

**Occurrence of FL during exercise.** Before loading, tidal expiratory flow impinged in part on maximal expiratory flow in subjects 2 and 3 at all levels of exercise and in subjects 1, 5, 6, and 7 only at the highest levels of exercise. In subjects 4, 8, and 9, tidal expiratory flow never impinged on maximal expiratory flow.

Forced partial and tidal flow-volume curves before, during, and after expiratory threshold loading are shown in Fig. 1 for a representative subject. Figure 1, A and C, was obtained at a  $\dot{V}_E$  of  $\sim 35$  l/min, and Fig. 1, B and D, was obtained at a  $\dot{V}_E$  of  $\sim 75$  l/min. Figure 1, C and D, shows Ptp-volume relationships for the tidal breaths. At the lower levels of  $\dot{V}_E$  (A and C), flow was always submaximal. The response to a threshold load was a reduced expiratory flow rate and an increased EELV. At the higher levels of  $\dot{V}_E$  (B and D),  $\dot{V}_E$  approximately doubled because of a marked increase in flow with little change in  $V_T$ . At end expiration, tidal expiratory flow equaled maximum expiratory flow and EELV was slightly larger than at the lower levels of  $\dot{V}_E$ . This pattern of increased EELV without a change in  $V_T$  is characteristic of individuals who impinge on their maximal expiratory flow-volume curve at high levels of exercise. With the imposition

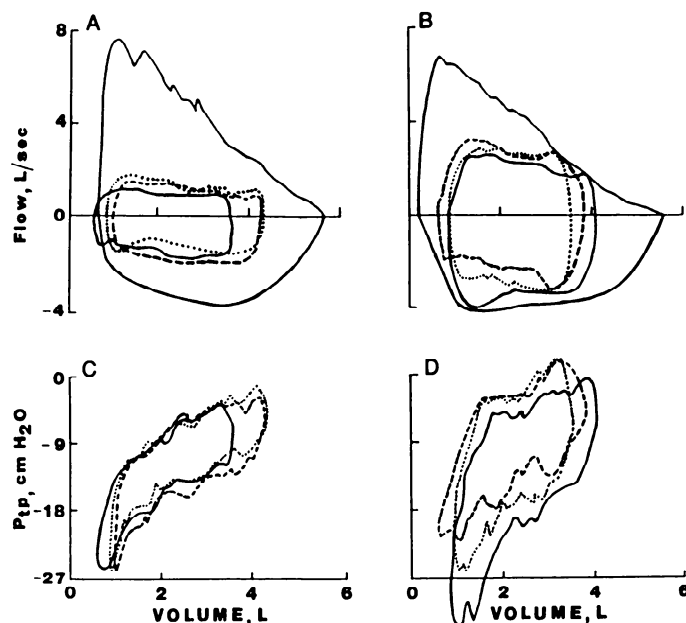


FIG. 1. Composite flow-volume (F-V, A and B) and pressure-volume (P-V, C and D) plots for subj 8 without (A and C) and with flow limitation (B and D). On F-V plots, large loops (heavy lines) were recorded during partial forced expiratory maneuvers. On F-V and P-V plots, dashed, continuous, and dotted loops were recorded before, during, and after expiratory threshold loading, respectively.

of the threshold load, expiratory flow was decreased and EELV decreased to a volume that was similar to that observed at the lower levels of ventilation. Maximal flow occurred only near end expiration in all breaths.

**Effects of loading and unloading under conditions of no FL.** The individual changes in ventilatory pattern and EELV observed when the expiratory threshold load was imposed and removed are shown in Table 2 for those individuals with no FL. Mean changes for loading and unloading are shown in Fig. 2.

During loading,  $\dot{V}_E$  decreased ( $P < 0.05$ ) because  $f$  decreased ( $P < 0.02$ ) while  $V_T$  was unchanged.  $T_E$  increased 30% ( $P < 0.02$ ). There was a nonsignificant trend to increase EELV.

When the load was removed,  $f$  and  $T_E$  returned to values that were not significantly different from those observed before loading.  $\dot{V}_E$  and  $V_T$  were slightly increased. EELV remained at control level.

**Effects of loading and unloading under conditions of FL.** The individual changes in ventilatory pattern and EELV observed when the expiratory threshold load was imposed and removed during FL are shown in Table 3. Mean changes are shown in Fig. 2.

The response to loading during FL was similar to that during no FL, in that there were similar decreases in  $f$  ( $P < 0.05$ ) and  $\dot{V}_E$  ( $P < 0.05$ ). The increase in  $T_E$  was twice as great (60 vs. 30%,  $P < 0.05$ ). The most notable difference was the highly significant decrease in EELV ( $P < 0.001$ ).

When the load was removed in both conditions (no FL and FL),  $\dot{V}_E$  was increased to values that were slightly but significantly ( $P = 0.023$ ) greater than those observed before loading.  $V_T$ ,  $f$ , and  $T_E$  were restored to values that

TABLE 2. Effects of imposing and removing expiratory threshold load on ventilatory variables in subjects without flow limitation

Subj No.	Observation No.	V <sub>E</sub> , l/min	V <sub>E</sub>		V <sub>T</sub>		f		T <sub>E</sub>		V <sub>T</sub> /T <sub>E</sub>		EELV, ml	
			L/C	U/C	L/C	U/C	L/C	U/C	L/C	U/C	L/C	U/C	L - C	U - C
1	1	37.0	1.0	1.2	1.1	1.1	1.1	1.1	0.8	1.0	1.3	27	-7	
4	5	46-105	0.9	1.1	1.0	1.0	0.9	1.3	1.0	0.8	1.1	717	40	
5	3	39-63	0.7	0.9	0.9	1.0	0.7	1.7	1.1	0.6	0.9	106	-46	
6	5	24-51	0.9	1.1	1.1	1.1	0.8	1.4	1.1	0.8	1.0	-61	-43	
7	6	34-74	0.9	1.1	1.0	1.1	0.9	1.1	1.0	0.8	1.1	221	-38	
8	4	40-64	1.0	1.1	1.1	1.1	0.9	1.3	1.1	0.9	1.1	-6	-37	
9	5	42-101	0.9	1.1	0.9	1.0	1.2	1.1	0.8	0.9	1.1	65	1	
Grand mean ± SD		54.5±15.3	0.9±0.1*	1.1±0.1*	1.0±0.1	1.1±0.1†	0.9±0.1†	1.0±0.1	1.3±0.2†	1.0±0.1	0.8±0.1†	153±264	-19±32	

C, control phase; L, expiratory threshold loading phase; U, expiratory threshold unloading phase; V<sub>E</sub>, minute ventilation; V<sub>T</sub>, tidal volume; f, respiratory rate; T<sub>E</sub>, expiratory time; V<sub>T</sub>/T<sub>E</sub>, mean expiratory flow rate; EELV, end-expiratory lung volume. \* P < 0.05; † P < 0.02.

were not significantly different from those before loading. EELV returned to the unloaded level.

DISCUSSION

The main finding of this study is that imposing an expiratory threshold load in subjects who achieve FL during exercise causes them to decrease EELV in marked contrast to subjects who do not achieve FL during exercise. Subjects who do not achieve FL tend to increase EELV, as might be expected from incomplete compensation to a load that impedes expiration. A previous study from this laboratory showed that patients increased EELV with progressive exercise and that this increase in EELV was associated with utilization of maximal expiratory flow near end expiration (2). Other investigators have also noted an increased EELV associated with FL (9, 10, 21). Sharratt et al. (21) reported that EELV had a tendency to increase during exercise in older subjects who had scooping of their flow-volume curves at low lung volumes and achieved FL during exercise. Johnson et al. (10) observed similar shifts in older fit subjects whose EELV increased with the onset of FL. Similar findings were also reported by Jensen et al. (9).

An increase in EELV during exercise theoretically could occur from derecruitment of expiratory muscles, which were responsible for the initial reduction in EELV and/or an increase in laryngeal resistance retarding expiration. However, increasing EELV is associated with increasing minute ventilation and increasing expiratory flow over the volume range common to the breaths. Increased flow is inconsistent with decreased expiratory effort or increased resistance. Tonic activity of the inspiratory muscles during inspiration has been invoked to explain hyperinflation during acute asthma (14, 16). This inspiratory tone increases mean lung volume into a region with greater maximal expiratory flow. Clearly, during acute asthmatic attacks, patients utilize maximal expiratory flow over all the V<sub>T</sub>, although it is not known whether they are generating pressures in excess of the minimum required to achieve maximal flow. This mechanism is unlikely to account for the increase in EELV during progressive exercise, unless it is counterbalanced by a greater recruitment of expiratory muscles, because the increase in EELV, as noted above, is associated with an increase in expiratory flow. Therefore we conclude that the increase in EELV is associated with some event that terminates expiration (11, 15) rather than factors that reduce expiratory flow during the T<sub>E</sub>.

Our hypothesis is that the increase in EELV, which occurs with progressive increases in V<sub>E</sub> and is associated with achieving maximal expiratory flow near end expiration, is caused by FL. Wood and Bryan (23) showed that FL induced by increased ambient pressure causes breathing discomfort in subjects with normal lung function during maximal exercise. O'Donnell et al. (17, 18) demonstrated a relationship between FL and unpleasant respiratory sensation in patients with reduced maximal expiratory flows. Therefore we hypothesize that as V<sub>E</sub> increases with exercise, subjects progressively recruit expiratory muscles (8), which increase flow rate and reduce EELV below the normal relaxation lung volume. Once

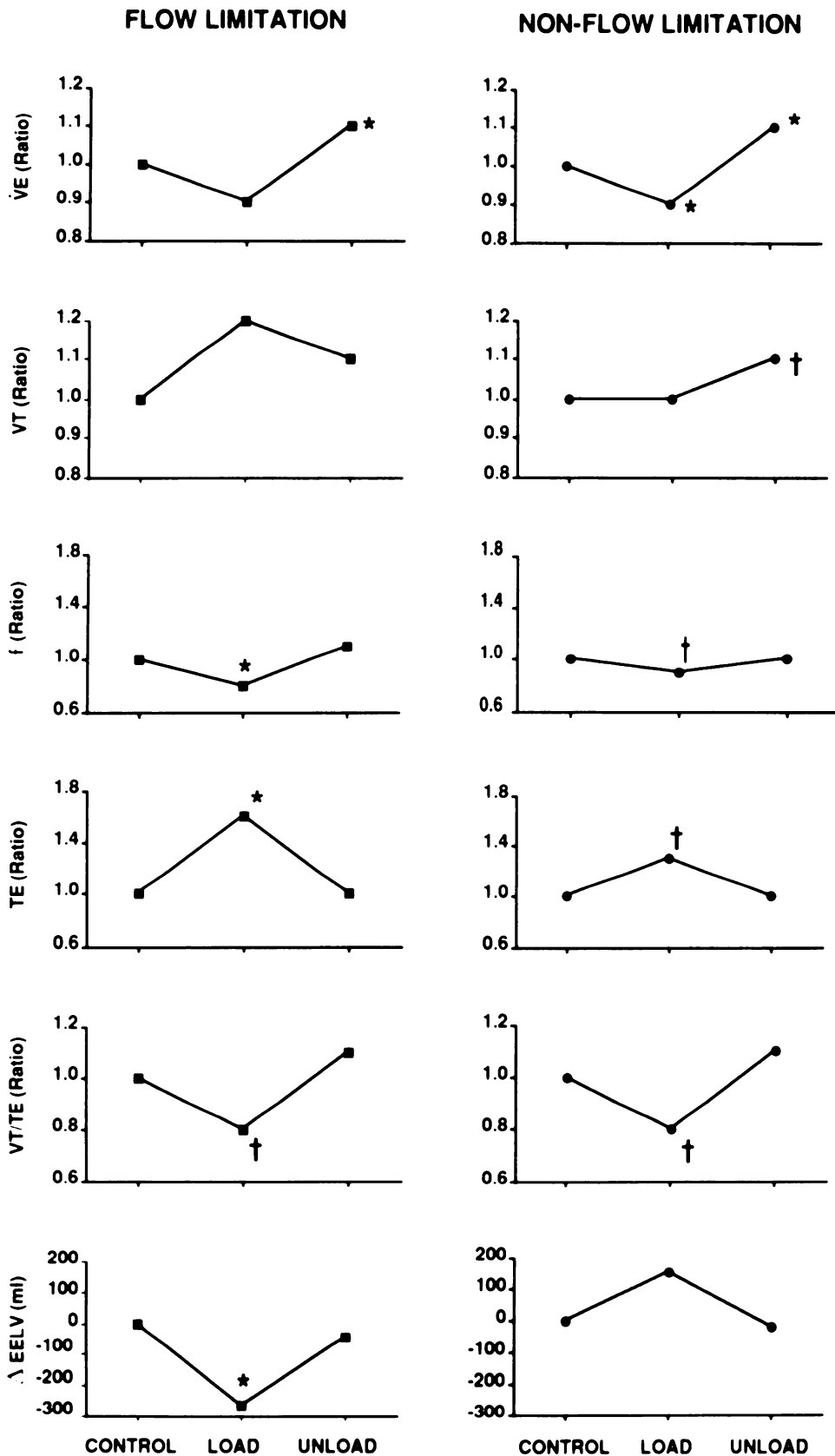


FIG. 2. Mean effects of imposing and removing an expiratory threshold load on ventilatory variables in subjects with and without flow limitation. Control, control phase; load, expiratory threshold loading phase; unload, expiratory threshold unloading phase. Data are means as a ratio of control; SDs are reported in Tables 2 and 3.  $\dot{V}_E$ , minute ventilation;  $V_T$ , tidal volume;  $f$ , respiratory rate;  $T_E$ , expiratory time;  $V_T/T_E$ , mean expiratory flow rate; EELV, end-expiratory lung volume. \*  $P < 0.05$ ; †  $P < 0.02$ .

maximal expiratory flow is reached, which will occur near end expiration because of the configuration of the maximal flow-volume curve, further increases in mean expiratory flow over the same volume range would require an increased expiratory effort, which would cause

the onset of maximal flow at a higher lung volume and generation of pressures in excess of that required to reach maximal flow over the lung volume range near the previous EELV. If the dynamic compression produced by  $P_{tp}$  greater than  $P_{crit}$  initiates a reflex termination of

TABLE 3. Effects of imposing and removing expiratory threshold load on ventilatory variables in subjects with flow limitation

Subj No.	Observation No.	V <sub>E</sub> , l/min		V <sub>T</sub>		f		T <sub>E</sub>		V <sub>T</sub> /T <sub>E</sub>		EELV, ml	
		L/C	U/C	L/C	U/C	L/C	U/C	L/C	U/C	L/C	U/C	L - C	U - C
1	4	1.0	1.1	1.2	1.0	0.8	1.1	1.3	0.9	0.9	1.2	-281	-22
2	4	0.8	1.0	1.1	1.1	0.7	0.9	1.6	1.3	0.7	0.9	-369	-54
3	5	0.9	1.1	1.5	1.2	0.6	0.9	2.1	1.2	0.7	1.1	-288	-80
5	4	0.8	1.1	1.2	1.2	0.7	0.9	1.8	1.2	0.6	1.0	-213	-67
6	2	1.1	1.3	1.0	1.0	1.0	1.3	1.1	0.8	1.0	1.2	-171	50
7	1	0.9	1.2	1.0	0.9	0.9	1.3	1.1	0.8	0.9	1.2	-259	91
Grand mean±SD		57.3±13.8	0.9±0.1	1.1±0.1*	1.1±0.1	0.8±0.2*	1.1±0.2	1.6±0.4*	1.0±0.2	0.8±0.1†	1.1±0.1	-263±68‡	-44±52

\* P < 0.05; † P < 0.02; ‡ P < 0.001.

expiration, then EELV would increase. That is, we assume that subjects who achieve FL near end expiration are breathing at a higher EELV than they would be in the absence of FL. For example, note the increase in EELV with the increased ventilation demonstrated in Fig. 1. Ideally this hypothesis would be tested by changing the maximal flow-volume relationship during a single expiration. If maximal flow decreased, the subject would achieve FL at a higher volume, terminate expiration earlier, and increase EELV. Conversely, if maximal flow were increased, the subject with the same breathing pattern would achieve FL at a lower volume and EELV would decrease. Because we could not change the maximal expiratory flow-volume curve of a subject for a single breath, we elected to manipulate the tidal flow-volume relationship by the addition or removal of an expiratory threshold load. The response to loading in the absence of any compensatory response on the part of the subject depends on whether the neural control of expiration is predominantly modulated by volume or timing. The decreased expiratory flow would be associated with a decreased V<sub>T</sub> and increased EELV if timing was constant or an increased T<sub>E</sub> if expiration was modulated by V<sub>T</sub>. Unloading would produce the reverse effect. Our subjects who did not achieve FL during tidal breathing had partial compensation in that the P<sub>tp</sub> was usually reduced by somewhat less than the 5-cmH<sub>2</sub>O expiratory threshold load. However, they all had a decrease in expiratory flow over the same volumes. T<sub>E</sub> was significantly increased, and there was a nonsignificant tendency for V<sub>T</sub> to decrease and EELV to increase. These results are consistent with that reported in the literature by Goldstein et al. (4), who used a larger threshold load and found an increase in EELV.

In contrast, our subjects who achieved FL during tidal breathing behaved quite differently, as predicted by our hypothesis. When expiratory flow rate was reduced by the expiratory threshold load, T<sub>E</sub> was increased not only until the subjects achieved normal EELV and V<sub>T</sub> but expiration continued until they achieved FL at a lower lung volume, resulting in an EELV response qualitatively different from that in the subjects without FL (Fig. 1). With a sudden removal of an expiratory load, the reverse occurred with higher expiratory flows, development of FL at a higher lung volume, and termination of expiration with a smaller V<sub>T</sub>, higher EELV, and shorter T<sub>E</sub> than during the loaded breaths. It is not possible to precisely determine the volume at which a subject first reaches maximal flow, so it is not possible to prove that with loading the subjects with FL expired until the instant they achieved maximal flow. However, the partial flow-volume curves after unloading clearly demonstrates when subjects were using maximal flow and when they were not. The decreased EELV, which occurs with the reduced flow only in subjects with FL, is strongly supportive of our hypothesis. We do not believe that these different responses to loading are unique properties of individuals, because subjects who were not flow limited at low levels of exercise but were flow limited at high levels of exercise manifested the behavior predicted from the presence or absence of FL. Also the loading response was not determined by whether ventilation was high or low. Sub-

jects with reduced maximal flow who had FL at low levels of ventilation and subjects with high levels of maximal flow who did not achieve FL at very high levels of ventilation responded to the threshold load as determined by the presence or absence of FL and not the level of ventilation.

The results of this study are consistent with our hypothesis and support, but do not prove, the contention that the increase in EELV results from premature termination of expiration produced by the onset of FL. However, we can think of no other potential explanation for why an expiratory threshold load, which reduces the driving pressure for expiration and increases the relaxation volume of the respiratory system, should have the paradoxical effect of decreasing EELV only in subjects with FL. Our study gives no insight into what receptors or what neural pathways may be involved.

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