

Mechanism of reduced maximal expiratory flow with aging

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Babb, T. G. and J. R. Rodarte. Mechanism of reduced maximal expiratory flow with aging. *J Appl Physiol* 89: 505–511, 2000.— To investigate the determinants of maximal expiratory flow (MEF) with aging, 17 younger (7 men and 10 women, 39 ± 4 yr, mean \pm SD) and 19 older (11 men and 8 women, 69 ± 3 yr) subjects with normal pulmonary function were studied. For further comparison, we also studied 10 middle-aged men with normal lung function (54 ± 6 yr) and 15 middle-aged men (54 ± 7 yr) with mild chronic airflow limitation (CAL; i.e., forced expiratory volume in 1 s/forced vital capacity = $63 \pm 8\%$). MEF, static lung elastic recoil pressure (Pst), and the minimal pressure for maximal flow (Pcrit) were determined in a pressure-compensated, volume-displacement body plethysmograph. Values were compared at 60, 70, and 80% of total lung capacity. In the older subjects, decreases in MEF ($P < 0.01$) and Pcrit ($P < 0.05$), compared with the younger subjects, were explained mainly by loss of Pst ($P < 0.05$). In the CAL subjects, MEF and Pcrit were lower ($P < 0.05$) than in the older subjects, but Pst was similar. Thus decreases in MEF and Pcrit were greater than could be explained by the loss of Pst and appeared to be related to increased upstream resistance. These data indicate that the loss of lung recoil explains the decrease in MEF with aging subjects, but not in the mild CAL patients that we studied.

flow limitation; respiratory mechanics; pulmonary function

MAXIMAL EXPIRATORY FLOW (MEF) at a given lung volume is determined by static lung elastic recoil (Pst) and the length and pressure diameter behavior of the airways upstream of the point at which wave speed is reached in the airway (8, 13, 14, 18). The total pressure drop from the pleural surface to the airway opening, at the minimum pressure that produces maximal flow (Pcrit), is a reflection of the geometry of the entire airway tree, including the extrathoracic upper airway. Both MEF and Pst at constant lung volume decrease with age (10, 12, 16, 19). It is uncertain whether there are changes in the intrathoracic airways that contribute to the observed changes in MEF. Therefore, we performed lung mechanics measurements on volunteers, ranging in age from young adults to the elderly, with normal pulmonary function and no diagnoses or symptoms of cardiopulmonary disease. We also studied a small number of patients with mild-to-moderate chronic air-

flow limitation (CAL), as determined from spirometry screening.

METHODS

Subjects. Younger ($n = 17$, 35–45 yr) and older ($n = 19$, 65–75 yr) volunteers (both men and women) with normal pulmonary function were recruited through local advertisements. Subjects had no history of asthma, respiratory symptoms, pulmonary disease, overt cardiovascular disease, or had participated in regular vigorous exercise for the last 6 mo. None of the subjects had a significant change in spirometry with inhaled bronchodilators. In accordance with the Institutional Review Board, all details of the study were discussed with the volunteers, and informed consent was obtained. Two groups of middle-aged men ($\sim 54 \pm 7$ yr), with pulmonary function ranging from moderate CAL ($n = 15$) to above normal ($n = 10$) and with a TLC $\geq 90\%$ of predicted, were selected from previous studies (3–5).

Pulmonary function. All subjects had standard spirometry, lung volume, and diffusing capacity determinations performed. Pulmonary function was determined according to guidelines of the American Thoracic Society (2). Also, American Thoracic Society standards were used for the evaluation of respiratory impairment (1).

Respiratory mechanics. Resting maximal flow-volume loops and pressure-volume loops were measured in pressure-corrected, volume-displacement body plethysmographs to eliminate gas-compression artifacts. Transpulmonary pressure (Ptp) was estimated as the differential pressure between airway opening pressure and esophageal pressure, which was measured using an esophageal balloon placed ~ 45 cm from the nostril (20). Flow, volume, and Ptp were displayed and sampled in real time on a computer for subsequent analysis. All procedures were performed in the seated position.

Static relaxation pressure-volume curves were obtained by having the subject inspire to total lung capacity (TLC), relax, inspire to TLC again, and relax against an occluded mouthpiece. The subject was then asked to expire passively with intermittent occlusions of the mouthpiece. Isovolumetric pressure flow (IVPF) curves were constructed from data collected from multiple vital capacities of various efforts (graded flow-volume curves; Ref. 21).

Data analysis. An interactive computer program developed in this laboratory was used to analyze the respiratory mechanics data. The pressure-volume curves were plotted, and a line of best fit was drawn by eye (16). From the best fit

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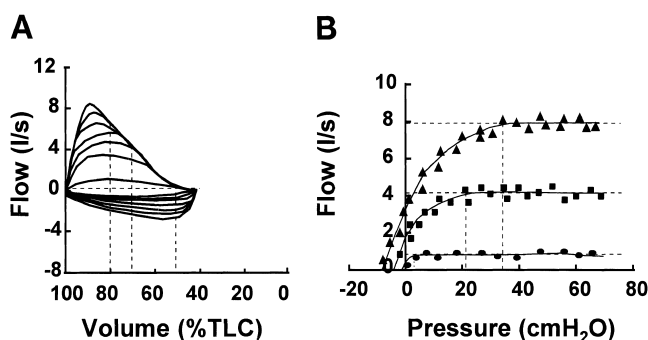


Fig. 1. Graded flow-volume loops (A) and isovolume pressure-flow curves (B) for a typical older subject. Isovolume pressure-flow curves were constructed from three trials of graded loops. Dashed lines indicate the critical pressure (P_{crit}), as defined as the minimal pressure for maximal flow at 80, 70, and 50% of total lung capacity (TLC).

curve, P_{st} was determined at 50, 60, 70, and 80% of TLC in each subject. P_{st} at TLC was determined as the highest pressure obtained at maximal inspiration when inspiratory flow and oral pressure were zero (12). P_{st} at 90% of TLC was also determined. IVPF curves were plotted, and a line of best fit was drawn by eye; P_{crit} was estimated as the lowest pressure where flow no longer increased but pressure continued to rise (21). The data for a typical older subject is shown in Fig. 1. P_{crit} was determined at 60, 70, and 80% of TLC in each subject and at 50% of TLC when possible.

t-Tests were used to test the significance of differences with aging between the younger and older subjects (men and women separately) and to test differences between the normal, middle-aged men and those with CAL. A one-way ANOVA was used to test for significant differences among the four groups of men, and multiple contrasts were used to test among the four groups when a significant *F* ratio was detected with the one-way ANOVA.

RESULTS

Subjects. Subject characteristics are presented in Table 1. Except for differences in age, the younger and older subjects were quite similar. The younger and older subjects were selected with no or minimal smoking history. The middle-aged normal and CAL subjects were also similar, including their ages. Smoking history was larger in the middle-aged normal and CAL subjects compared with the younger and older subjects.

The middle-aged subjects were allowed to participate if they had normal pulmonary function, regardless of smoking history.

Pulmonary function data are presented in Table 2. As expected, there was a consistent and significant decline in pulmonary function with age. However, of the younger and older subjects, only the older women had any percent predicted values that were less than those of their younger counterparts. Other measures of respiratory function are shown in Table 3. TLC was not significantly different with aging in absolute terms. As percent predicted, the older women had a larger TLC than the younger women, although mean height was not different between the two groups. Also, in the women, unlike the men, P_{st} at TLC was significantly ($P < 0.001$) reduced with aging. However, P_{st} at 90% of TLC was significantly ($P < 0.05$) lower with aging in both the men and women. The maximal voluntary ventilation was similarly reduced with aging. Data for the middle-aged normal and CAL men are also shown in Tables 1–3.

Presentation of flow-volume data. It is customary to present flow-volume curves with absolute flow as a function of percent vital capacity. This minimizes the effects of aging on lung function, as shown in Fig. 2, which presents flow-volume curves for both a younger and older man as percent vital capacity in A and as percent TLC in B. The differences in the apparent magnitude and the volume dependence of the decrease in flow with age is underestimated at all lung volumes in Fig. 2A. Therefore, we present flow in relation to lung volume as a percentage of TLC.

Effects of aging: relationships among flow, pressure, P_{crit} , and lung volume. Flow, P_{st} , and P_{crit} are plotted against %TLC in Fig. 3. All were significantly ($P < 0.05$) reduced with age, although the linear relationship with volume was maintained with aging in both men and women. In Fig. 4, flow and P_{crit} are plotted against P_{st} , and flow is also plotted against P_{crit} . In all cases, the relationship was similar in the younger and older women and men, although the curve for the older subjects was shifted down and to the left. The graphs indicate that the decrease in MEF with aging is proportional to the loss of P_{st} .

Table 1. Subject characteristics

Subjects	<i>n</i>	Age, yr	Height, cm	Weight, kg	Subjects with Smoking History, no.	Years Smoked	Pack Years
Women							
Younger	10	39.7 ± 4.1	163 ± 9	63.5 ± 10.6	2	7 ± 6‡	11 ± 15‡
Older	8	69.5 ± 3.6*	162 ± 6	58.7 ± 11.9	2	18 ± 23	21 ± 30
Men							
Younger	7	38.3 ± 3.2	179 ± 7	81.8 ± 7.4	2	10 ± 4‡	1 ± 1‡
Older	11	68.7 ± 2.3*	177 ± 6	80.9 ± 10.2	8	23 ± 15	17 ± 12
Middle-aged	10	54.0 ± 6.2	178 ± 8	90.2 ± 13.2	8	27 ± 12‡	44 ± 30‡
CAL	15	54.4 ± 7.0	178 ± 5	88.0 ± 13.4	15	32 ± 11†	46 ± 24†

Values are means ± SD. CAL, chronic airflow limitation; *n*, no. of subjects. * $P < 0.001$, significantly different from age-matched controls; †smoking history unavailable on 3 subjects; ‡not compared statistically between groups.

Table 2. *Pulmonary function*

Subjects	<i>n</i>	FVC, liters (%pred)	FEV ₁ , liters (%pred)	FEV ₁ /FVC, % (%pred)	PEF, l/s (%pred)	FEF ₅₀ , liters (%pred)	MVV, l/min (%pred)
Women							
Younger	10	3.71 ± 0.34 (110 ± 10)	2.93 ± 0.33 (104 ± 9)	79 ± 4 (93 ± 5)	7.51 ± 0.94 (120 ± 12)	3.59 ± 0.86 (93 ± 21)	126 ± 17 (117 ± 15)
Older	8	3.34 ± 0.44 (118 ± 12)	2.34 ± 0.35† (106 ± 12)	70 ± 3‡ (87 ± 5)*	5.75 ± 0.88‡ (105 ± 13)*	2.16 ± 0.55‡ (74 ± 17)	91 ± 14‡ (108 ± 14)
Men							
Younger	7	5.54 ± 0.53 (108 ± 7)	4.37 ± 0.36 (104 ± 6)	79 ± 4 (96 ± 5)	10.82 ± 1.01 (114 ± 10)	4.95 ± 0.73 (92 ± 18)	194 ± 20 (115 ± 11)
Older	11	4.76 ± 0.84* (114 ± 17)	3.45 ± 0.51‡ (106 ± 14)	74 ± 5* (96 ± 8)	9.75 ± 1.60 (117 ± 14)	3.78 ± 0.75† (88 ± 20)	140 ± 20‡ (108 ± 10)
Middle-aged	10	5.06 ± 0.72 (109 ± 9)	4.04 ± 0.48 (106 ± 7)	82 ± 8 (101 ± 10)	10.09 ± 1.07 (112 ± 17)	4.50 ± 1.30 (97 ± 28)	168 ± 24 (113 ± 13)
CAL	15	4.46 ± 0.60* (94 ± 11)†	2.83 ± 0.54‡ (75 ± 11)‡	63 ± 8‡ (78 ± 9)‡	8.80 ± 2.06* (100 ± 21)	1.87 ± 0.69‡ (40 ± 14)‡	133 ± 25† (90 ± 15)‡

Values are means ± SD. FVC, forced vital capacity; FEV₁, forced expiratory volume in 1 s; PEF, peak expiratory flow; FEF₅₀, forced expiratory flow at 50% of FVC; MVV, maximal voluntary ventilation; %pred, %predicted. **P* < 0.05, †*P* < 0.01, ‡*P* < 0.001: significantly different from age-matched controls.

Effects of CAL: relationships among flow, pressure, Pcrit, and lung volume. In Fig. 5, flow, Pst, and Pcrit are plotted against %TLC for the younger and older men and the middle-aged normal and CAL subjects. The CAL subjects had significant (*P* < 0.05) reductions in flow, Pst, and Pcrit compared with the middle-aged subjects with normal lung function. Flow was also significantly less than in the older subjects, and, at the lower lung volumes, so was Pcrit. Interestingly, Pst was not significantly less in the CAL subjects compared with the middle-aged or older normal subjects. In Fig. 6, flow and Pcrit are plotted against Pst, and flow is plotted against Pcrit. In the CAL subjects, flow was lower for a given level of Pst than in the subjects with normal pulmonary function, regardless of age. To test this relationship, flow was tested at a constant pressure of 5 cmH₂O (*P* < 0.01). The same relationship held for Pcrit and Pst (*P* < 0.01 at 5 cmH₂O). That is, Pcrit was less per Pst in the CAL subjects than in subjects with normal pulmonary function, regardless of age. However, the relationship between flow and

Pcrit was similar in all men, regardless of age or MEF. The graphs in Figs. 5 and 6 indicate that the decreases in MEF and Pcrit with CAL are greater than loss of Pst would predict.

DISCUSSION

The major finding of this study was that the decrease in MEF with aging can be explained almost entirely by a loss of Pst. Thus, with normal aging, flow as a function of pressure remained constant. Also, Pcrit with age had the same relationship to pressure as did flow. To the authors' knowledge, this is the first time the interrelationships between MEF, Pst, and Pcrit with aging have been analyzed together in both men and women.

The decrease in MEF observed in our older subjects was similar to that reported in the literature (16, 24). The decrease in Pst was also similar to that reported in the literature for older men and women (7, 12, 19, 22, 24), although the mean ages were not exactly the same.

Table 3. *Lung volumes and respiratory mechanics*

Subjects	<i>n</i>	TLC, liters (%pred)	RV/TLC, %	Pst ₁₀₀ , cmH ₂ O (%pred)	Pst ₉₀ , cmH ₂ O (%pred)	Cst, l/cmH ₂ O	RL cmH ₂ O · l ⁻¹ · s
Women							
Younger	10	5.04 ± 0.63 (99 ± 13)	26 ± 4	32 ± 7 (101 ± 18)	12 ± 2 (65 ± 14)	0.22 ± 0.08	2.1 ± 0.4
Older	8	5.48 ± 0.51 (116 ± 6)†	39 ± 5‡	18 ± 3‡ (84 ± 15)*	6 ± 2‡ (56 ± 23)	0.30 ± 0.03*	2.3 ± 0.8
Men							
Younger	7	7.11 ± 0.76 (100 ± 7)	22 ± 2	31 ± 5 (96 ± 17)	14 ± 3 (77 ± 20)	0.28 ± 0.06	1.5 ± 0.4
Older	11	7.23 ± 1.05 (112 ± 14)	34 ± 5‡	27 ± 5 (131 ± 24)†	10 ± 2† (86 ± 17)	0.31 ± 0.05	2.0 ± 0.6
Middle-aged	10	7.28 ± 1.42 (109 ± 12)	27 ± 4	34 ± 10 (132 ± 36)	13 ± 2 (100 ± 18)	0.50 ± 0.24	2.1 ± 0.7
CAL	15	7.37 ± 1.12 (108 ± 12)	34 ± 5†	28 ± 11 (108 ± 41)	11 ± 3* (85 ± 28)	0.47 ± 0.17	3.0 ± 1.5

Values are means ± SD. TLC, total lung capacity; RV, residual volume; Pst₁₀₀, static elastic recoil pressure of the lung at 100% of TLC; Pst₉₀, Pst at 90% of TLC; Cst, static compliance of the lung over 1 liter above functional residual capacity; RL, pulmonary resistance. **P* < 0.05, †*P* < 0.01, ‡*P* < 0.001: significantly different from age-matched controls.

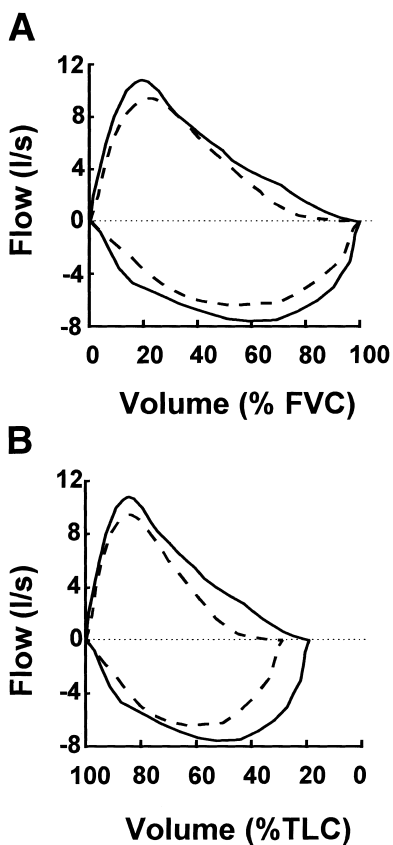


Fig. 2. Effects of aging on flow plotted against lung volume as percentage of forced vital capacity (FVC; A), and as percentage of TLC (B). Data are for a typical younger (solid line) and older man (dashed line).

However, for similarly aged older subjects, our Pst values for both men and women were lower than those reported by Knudson et al. (16). This could be related to the fact that we used a slightly greater balloon volume than Knudson and colleagues, who used a minimal balloon volume to measure static pressure-volume curves, thus shifting their pressure-volume curves to the right of ours. Another possibility is that the subjects in that study were screened for disease in much greater detail than were our subjects, some of whom had a smoking history. This also could have led to some of the differences between our pressure-volume curves and those of Knudson et al. (16). However, our subjects had normal pulmonary function, no history of lung disease, and no other indication of lung injury in their other respiratory mechanics data, which is consistent with the findings of a study that compared smokers and nonsmokers with normal lung function (24).

One notable difference between the older men and women was the small change in Pst in the men at 90 and 100% of TLC, which is in contrast to the marked decrease in Pst in the women at the same TLC percentages (23). This observation was also noted by Knudson et al. (16); however, the reason for the difference remains unclear and may be related to respiratory muscle strength (12). Pcrit values for the older men were similar to those reported by Johnson et al. (15) for

similarly aged older men. Pcrit for the younger men has not been reported before, although Olafsson et al. (21) reported values for men with a similar mean age, although their subjects ranged in age from 24 to 54 yr. Thus their values fell between our means for the younger and older men, as one would expect. The difference in Pcrit between the younger and older men was not significant at the highest lung volumes, unlike the older women, whose values are reported for the first time in this study. This is probably related to the differences observed in Pst at TLC between the men and women.

Also shown for the first time are the interrelationships among MEF, Pst, and Pcrit for both older and younger men and women. It was observed that, with aging, both flow and Pcrit drop in proportion to Pst for both the older men and women. This indicates that the decrease in MEF with aging is due mostly to a loss of Pst, as previously suggested by others (16, 19, 24). However, in contrast to the findings of Krumpke et al. (17), which suggest that flow is well maintained at high lung volumes despite large losses in Pst, we observed flow to be directly proportional to Pst over 60, 70, and 80% of TLC in both men and women (Fig. 4A). Peak flow was significantly reduced in both absolute and relative (percent predicted) terms in the older women. This was not the case in the older men. It is possible

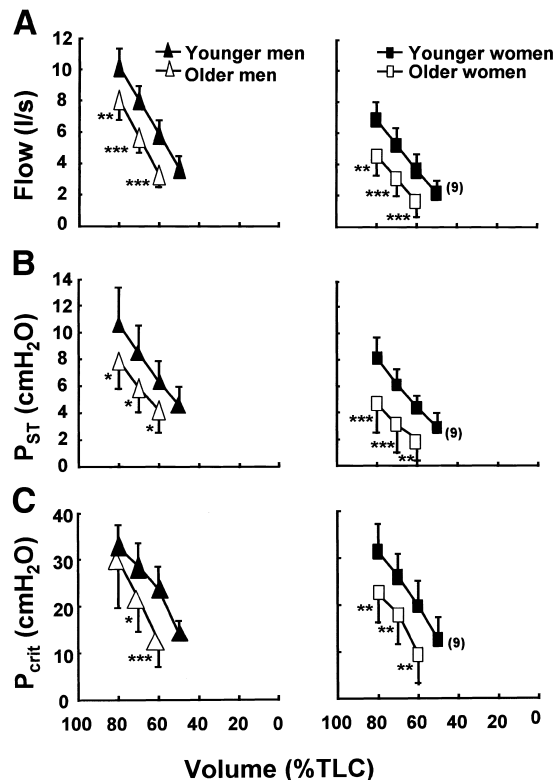


Fig. 3. Effects of aging on flow (A), lung static elastic recoil pressure (Pst; B), and Pcrit (C) in younger and older men (left) and younger and older women (right) in relation to volume. * $P < 0.05$, ** $P < 0.01$, and *** $P < 0.001$, significant differences between ages. See Table 1 for n values. Numbers in parentheses indicate a change in no. of subjects.

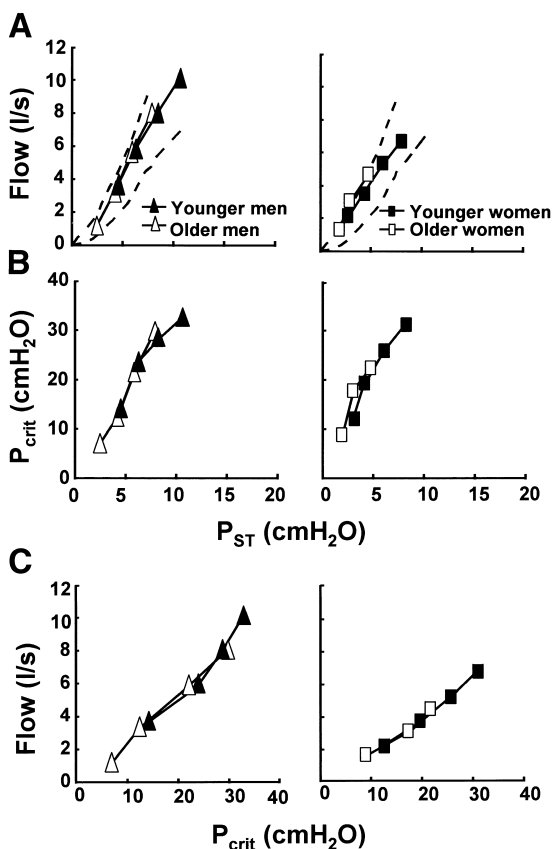


Fig. 4. Effects of aging on flow-P_{ST} (A), P_{crit}-P_{ST} (B), and flow-P_{crit} (C) relationships in older and younger men (left) and women (right).

that the decrease in peak flow in the older women was due to the large decrease in P_{ST} at higher lung volumes. Nevertheless, in both men and women, upstream resistance (R_{us}), calculated from the data presented in Fig. 4A ($R_{us} = P_{ST}/\text{maximal flow}$), was not different with aging at 60, 70, or 80% of TLC. This would indicate that R_{us} did not contribute to the decrease in MEF with aging over the lung volumes tested in this study.

Changes in P_{crit} with aging have not been previously reported. P_{crit} was lower at the lower lung volumes with aging, and P_{crit} decreased in proportion to the decline in P_{ST} in both the men and women in the present study. If flow limitation occurs at the equal pressure point, then P_{crit} is the measure of the airway resistance from the flow-limiting segment to the airway opening (down stream resistance, R_{ds}). It reflects a resistance of the downstream intra- and extrathoracic airways under conditions of just maximal flow. In normal men and women, P_{crit} is a monotonically decreasing function of both flow and P_{ST} that is independent of age. The constancy of the P_{crit}-MEF curves with aging in normal individuals further supports the contention that all age-related changes in flow are produced by reduced P_{ST}. Because the relationship between flow and P_{ST} does not vary with age, it cannot be determined from these results whether R_{ds} , as evidenced by P_{crit}, is determined by flow or P_{ST}. This

suggests that, with aging, the P_{crit}-flow relationship of the downstream airways (presumably the tracheal and extrathoracic airways) is constant in both genders and is independent of age. In subjects of either gender, the pressure drop is determined by the flow, which varies with lung volume, age, and peripheral airway disease.

Theoretically, MEF is determined by the coupled equations of airway geometry and elastic properties and pressure losses with flow (18). Decreases in pressure in the airway at a given flow and a constant P_{ST} are determined by frictional and convective pressure losses along each airway generation and coupled with the pressure diameter behavior of the airway, which decreases cross-sectional area and, therefore, increases resistance. Additional losses occur due to convective acceleration at each junction of two daughter branches with the more proximal airway, which further decreases airway lumen. Therefore, the transmural pressure at any given airway in the tree is determined by the resistance of the upstream airways, which is a

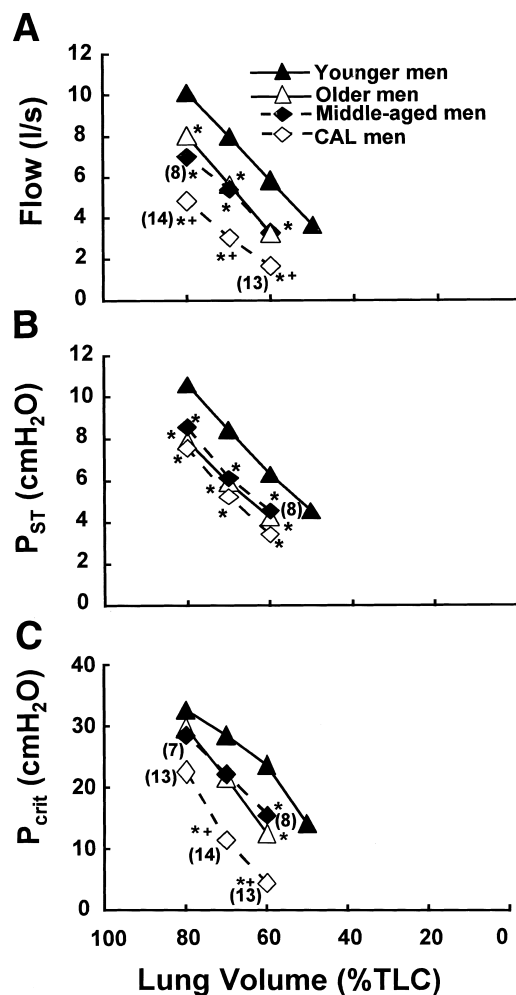


Fig. 5. Effects of chronic airflow limitation (CAL) on flow (A), P_{ST} (B), and P_{crit} (C) compared with younger, middle-aged, and older men with normal lung function. * $P < 0.05$, significant difference from younger men; + $P < 0.05$, significant difference from middle-aged men. See Table 1 for n values. Nos. in parentheses indicate where n has changed.

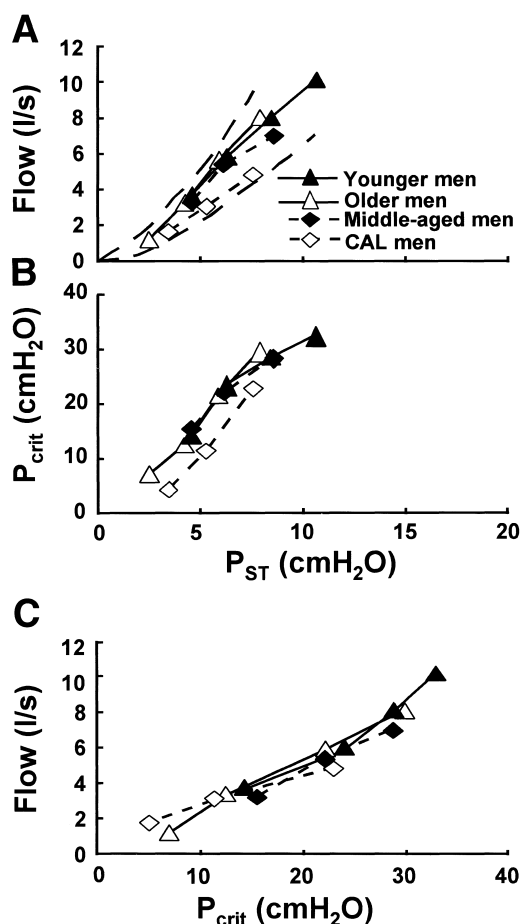


Fig. 6. Effects of CAL on relationships between flow (A), P_{st} (B), and P_{crit} (C) compared with younger, middle-aged, and older men with normal lung function. Flow and P_{crit} were significantly ($P < 0.05$) less for the CAL men at a constant P_{st} of 5 cmH₂O.

function not only of their static anatomy but also of their compliance. Because the resistance is inversely proportional to the fourth power of the radius (and only linearly related to airway length, which is proportional to the cube root of lung volume), changes in volume at a given P_{st} have little effect. MEF occurs when wave speed is achieved at some point in the airway. Wave speed of a given airway is proportional to the area at that transmural pressure, to the 3/2 power, and is inversely proportional to the square root of the compliance. Therefore, the major effect of airway compliance is the change in area that occurs because of pressure losses between the alveoli and the flow-limiting site rather than a change in the slope of the airway pressure area curve under wave-speed conditions.

Although it is theoretically possible for combinations of airway dilatation and increased compliance, or airway narrowing and stiffening, to produce the same MEF at a given P_{st} (14), it seems unlikely that this would occur over a range of recoils. If MEF at a given P_{st} over the entire vital capacity did not change with age, it is highly suggestive that the changes of flow at a given lung volume are entirely produced by observed changes in P_{st}.

Wave speed can occur at positive or negative transmural pressures, and, therefore, the flow-limiting region of the lung or "choke point" does not necessarily occur at the equal-pressure point that has a transmural pressure of zero. However, the shape of normal airway transmural pressure area curves, theoretical modeling, and measurements in experimental animals are all consistent with the premise that wave speed occurs near zero transmural pressure (18). Therefore, P_{crit} is a reasonable measure of the pressure losses from the choke point to the airway opening. If flow at a given P_{st} is unchanged with aging, the location of the choke point is unlikely to change, and, therefore, R_{ds}, the P_{crit}-to-MEF ratio (Fig. 4), is estimated over the same anatomic region in the airway. In fact, this so-called R_{ds} may be dominated by the extrathoracic airway.

In regards to the normal subjects in the present study, it is difficult to identify large numbers of healthy elderly who have never smoked. Therefore, we included smokers in our normal population if they had no symptoms suggestive of lung or cardiac disease and if their baseline pulmonary function was normal. Of course, it is impossible to exclude the possibility that smoking-related diseases may have contributed to this work. However, in support, others have observed no significant differences in respiratory mechanics between older smokers and nonsmokers with normal pulmonary function (24). In contrast, the patients with mild CAL in the present study had a decrease in flow at a P_{st}. Black and colleagues (6) showed that, in patients with α_1 -antitrypsin deficiency-associated emphysema, and only in those with no symptoms of bronchitis, MEF was normal as a function of P_{st}.

In contrast to the P_{st} in normal aging, the P_{st} of the mild CAL men decreased, but the decrease in MEF with CAL was greater than the loss of P_{st}. Furthermore, P_{crit} was less at constant P_{st} but the same as a function of flow in CAL. To the author's knowledge, this is also the first time that the interrelationships among MEF, P_{st}, and P_{crit} have been presented and analyzed in subjects with mild-to-moderate CAL.

MEF in the middle-aged CAL men was significantly lower than in both the middle-aged and older men with normal pulmonary function, despite a 15-yr difference in age for the older men (Fig. 4A); however, P_{st} was not significantly less than that in the middle-aged or older men with normal pulmonary function. The P_{st} values in the CAL subjects were consistent with those reported in patients with mild reductions in lung function (9, 11, 25). Therefore, in contrast to the effects of aging, there was a reduction in flow in the CAL patients of the present study that was not explained by a reduction of P_{st}, which is consistent with a reduction in flow due to airway disease. The relationship between MEF and P_{st} was much less steep than in the younger, middle-aged, and older men with normal pulmonary function and indicated a much larger R_{us} in the CAL men.

In the CAL subjects, P_{crit} was significantly less at 60 and 70% of TLC, but, unlike that of normal aging, the

relationship between Pcrit and Pst was shifted to the right. Thus flow was reduced as a function of Pst, but Pcrit was the same as in the normal subjects and was independent of age as a function of flow. This suggests that, with mild CAL, the Pcrit-flow relationship of the downstream airways is constant, and the pressure drop is determined by the flow. Considering that the most important lesions in smoking-related CAL are felt to be in the small airways and the Rds includes the extrathoracic upper airways, the constancy of downstream pressure-flow curves not only with aging, but also with disease, seems plausible.

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