Breathing at low lung volumes and chest strapping: a comparison of lung mechanics

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DOUGLAS, N. J., G. B. DRUMMOND, AND M. F. SUDLOW. *Breathing at low lung volumes and chest strapping: a comparison of lung mechanics.* J. Appl. Physiol.: Respirat. Environ. Exercise Physiol. 50(3): 650–657, 1981.—In six normal subjects forced expiratory flow rates increased progressively with increasing degrees of chest strapping. In nine normal subjects forced expiratory flow rates increased with the time spent breathing with expiratory reserve volume 0.5 liters above residual volume, the increase being significant by 30 s ($P < 0.01$), and flow rates were still increasing at 2 min, the longest time the subjects could breathe at this lung volume. The increase in flow after low lung volume breathing (LLVB) was similar to that produced by strapping. The effect of LLVB was diminished by the inhalation of the atropinelike drug ipratropium. Quasi-static recoil pressures were higher following strapping and LLVB than on partial or maximal expiration, but the rise in recoil pressure was insufficient to account for all the observed increase in maximum flow. We suggest that the effects of chest strapping are due to LLVB and that both cause bronchodilatation.

lung compliance; flow-volume curves; airway hysteresis; humans

CONSTRICTION OF THE THORAX by strapping applied around the chest wall has been shown to increase the static recoil pressures of the lung (5, 12, 17, 18) and forced expiratory flow rates (17, 18), but the cause of these changes is not clear. We have tried to determine whether the effect is related to the chest strap, or whether similar alterations occur following voluntary breathing at a low lung volume. The influence of breathing at a low lung volume particularly interested us because functional residual capacity is reduced during general anesthesia in humans (2, 8, 11, 21) and following abdominal operations (1).

The time course of the changes in lung mechanics resulting from chest strapping has not been clearly defined, probably because a chest strap cannot be applied rapidly in a body plethysmograph to allow measurement soon after the onset of strapping. We hoped that by studying voluntary breathing at low lung volumes, we might show the speed of development of these changes.

METHODS

We studied nine normal subjects, seven male and two female, aged 23–38 yr (Table 1). All were nonsmokers who were free of pulmonary disease. Lung volumes were measured in a pressure-compensated flow body plethysmograph (Pulmorex, Fenyves and Gut, Basel, Switzerland), and forced expiratory maneuvers were performed in a pressure-compensated volume-displacement body plethysmograph linked on-line to a PDP 11/40 computer to record and analyze mouth flow and thoracic gas volume.

In six males (*subj 1–6*) lung volumes and forced expiratory flow rates were measured before and after chest strapping of varying severity. The strapping was produced using a rigid moulded acrylic split corset that extended from clavicle to lower abdomen with the constriction controlled by three strong rope-and-pulley systems. Before strapping, each subject took three vital capacity breaths and then inhaled to total lung capacity before performing maximal forced expiratory maneuvers (Fig. 1*A*). For all maneuvers, three satisfactory flow-volume curves were obtained on each occasion, and the mean data from the three curves were used. The vital capacities of the three maximal curves had to be within 150 ml. After a further three vital capacity breaths, the subjects inhaled from residual volume to 50% of vital capacity (VC) followed by a partial forced expiratory maneuver (Fig. 1*B*). At varying degrees of chest strapping, the subjects first took three vital capacity breaths, within the limits of the chest strap, and then breathed normally for 1 min before inhaling as far as possible and performing a forced expiration (Fig. 1*C*).

In all nine subjects we studied forced expiratory maneuvers after breathing with expiratory reserve volume held at 500 ml above residual volume. Each maneuver in this study was also preceded by three vital capacity breaths and then inhalation from residual volume to *1*) total lung capacity (TLC) followed by a maximal expiratory maneuver (Fig. 1*A*); *2*) 50% VC followed by a partial expiratory maneuver (Fig. 1*B*); *3*) breathing at functional residual capacity (FRC) for 2 min followed by inhalation to 50% VC and then forced expiration (FRC partial) (Fig. 1*D*); and *4*) breathing with expiratory reserve volume (ERV) 500 ml above residual volume (RV) and with a tidal volume of less than 700 ml—low lung volume breathing (LLVB) for 30, 60, and 120 s, thereafter inhaling to 50% VC and performing a forced expiratory maneuver (Fig. 1*E*).

To investigate whether the tidal cycling of air was a factor governing flow rates after LLVB, we compared flow rates on partial expiration in six subjects (*subj 1, 2, 4,* and *7–9*) who followed three vital capacity breaths by

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holding their breath either at 50% VC or at residual volume + 0.5 liters for 1, 30, and 60 s.

Quasistatic expiratory lung recoil pressures were measured using a 10-cm thin-walled latex esophageal balloon and catheter system (13), with the balloon inflated to 0.5 ml, and a Statham PM131TC transducer. The balloon was placed at 40 cm from the nostril and adjusted to minimize cardiac artifacts. Expiratory flow rate was kept below 0.25 l/s. In five subjects (*subj 1–4* and *8*) compliance was measured after volume histories *A* and *B* and also after *E* with 60 s of LLVB (Fig. 1). For each maneuver at least three satisfactory recordings were made, and the mean values are quoted.

In three of these five subjects partial forced expiratory maneuvers had previously been performed after a volume history of three vital capacity breaths, LLVB for 1 min, inhalation to 50% VC, and exhalation to RV over 20 s followed by rapid inhalation to 50% VC to perform a forced expiratory maneuver. This was an attempt to simulate the volume history of the exhalation required to measure quasistatic compliance to determine whether the effects of LLVB decayed during a QPV maneuver.

TABLE 1. *Physical characteristics of subjects*

Subj No.	Sex	Age, yr	Ht, m	Wt, kg	$FEV_{1.0}$, liters	FVC, liters
1	M	28	1.80	75	5.0	5.7
2	M	38	1.78	70	4.1	5.4
3	M	23	1.83	68	4.0	5.3
4	M	25	1.78	68	4.0	4.7
5	M	25	1.85	70	4.0	5.3
6	M	33	1.83	73	4.5	5.8
7	M	33	1.88	76	5.0	5.7
8	F	29	1.63	79	3.2	4.1
9	F	24	1.73	55	4.0	4.6

FEV, forced expiratory volume in 1.0 s; FVC, forced vital capacity.

In four subjects (*subj 1–4*) flow-volume curves and quasistatic recoil pressures were measured at the same session for volume histories *A* and *B* (Fig. 1) and also *C*, with sufficient severity of chest strapping to reduce TLC to 50% VC. After the strapping had been removed, each subject then breathed normally for 10 min, taking several vital capacity breaths to abolish the effects of strapping. The subjects then followed the same volume history recorded during their chest strapping, and flow-volume curves and quasistatic recoil pressures were obtained. Thus the unrestrained subjects took three breaths to the "strapped TLC," then breathed for as long as possible, 1–2 min, at the "strapped FRC" before inhaling to the "strapped TLC" to performed forced or quasistatic expiratory maneuvers.

In two subjects (*subj 3* and *4*) the effect of chest strapping was compared with that of 120 s LLVB at the same session.

In four subjects we measured the effect of LLVB on flow-volume curves before and 90 min after inhalation of an aerosol containing 160 μg of the atropinelike agent ipratropium and in two the effect of LLVB was studied on another occasion after four doses of propranolol 80 mg orally taken at 8-h intervals.

RESULTS

Mild chest restriction, with VC reduced by less than 25%, reduced TLC, but had no effect on forced expiratory flow rates (Fig. 2). Tighter strapping increased flow rates at all lung volumes. Residual volumes did not change consistently after chest strapping or after low lung volume breathing. Partial and maximal flow-volume curves are often compared by using the flow rates at specific percentages of vital capacity. However, because small changes in residual volume after chest strapping were reported in some series (17, 18), we were concerned that

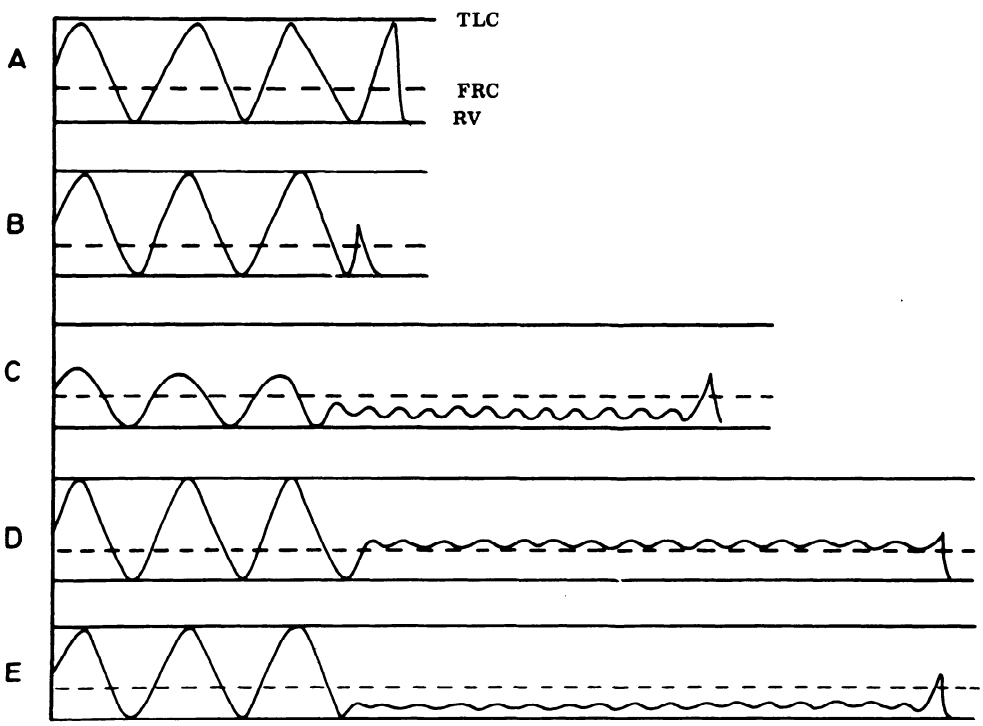

FIG. 1. Volume histories studied (for explanation, see test). TLC, total lung capacity; FRC, functional residual capacity; RV, residual volume.

these could make estimation of absolute lung volume inaccurate and cause errors in the volumes at which flow rates were compared. We observed that the tail of the flow-volume curve appeared linear, both before and after chest strapping. Therefore we used the slope of the flow-volume curve from 30 to 10% of the unrestrained VC (calculated as liters per second per liter) as our index of changes in flow as we felt that this was less dependent on small changes in residual volume which might have passed undetected. To permit comparison with other studies, we also show results for flow rates at 30% VC (Table 2). More than 10 (range 11–19) flow-volume curves with identical volume history were obtained at the same session in each of five subjects, and the mean coefficient of variation of the 30–10% VC slope was 7.5% (range 6.0–9.8%). All of the results quoted are the mean slope for three satisfactory flow-volume curves for each volume history. For the six subjects who were strapped, we have plotted the relation between this slope and the degree of chest strapping as measured by reduction of ERV (Fig. 3).

This slope was also increased by LLVB (Fig. 4). The effect was seen within 30 s ($P < 0.01$) and appeared to be still increasing at 2 min, which was the longest time the subjects could breathe at this lung volume (between 60 and 120 s, $P < 0.05$). Taking all six subjects who were

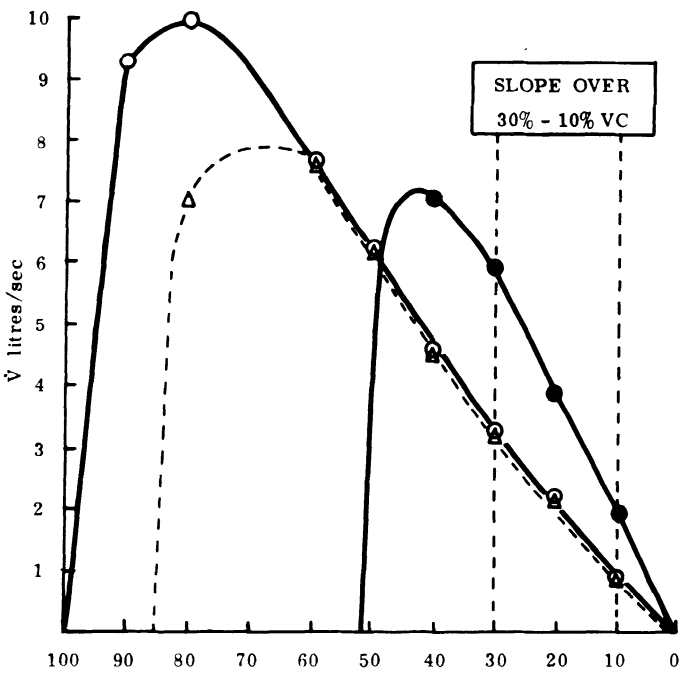

FIG. 2. Effect on forced expiratory flow rates of mild (△) and severe (●) chest strapping compared with maximal flow-volume curve (○) in *subject 2*.

TABLE 2. *Forced expiratory flow rates at 30% VC*

Subj No.	Maximal	Partial	Strapping	LLVB
1	4.62	3.87	6.22	6.55
2	3.58	3.47	4.06	5.10
3	3.29	3.68	4.76	4.69
4	3.04	3.01	4.83	3.82
5	2.82	2.75	3.93	3.71
6	3.63	3.15	6.06	5.22
7	3.10	3.28	4.60	3.44
8	2.09	2.15		3.37
9	3.42	2.67		3.80

Flow rates (l/s) at 30% vital capacity (VC) in each subject on maximum and partial flow-volume curves, at the most severe degree of chest strapping and following 120 s low lung volume breathing (LLVB). Each result is the mean of 3 values.

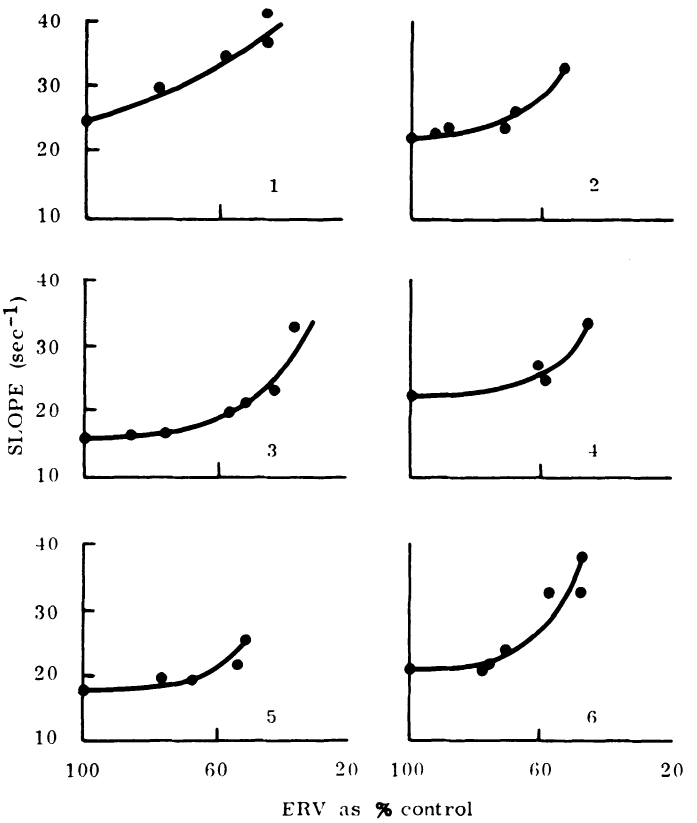

FIG. 3. Relation between severity of chest strapping, measured by decrease in expiratory reserve volume (ERV), and slope of 30–10% portion of flow-volume curve in *subjects 1–6*.

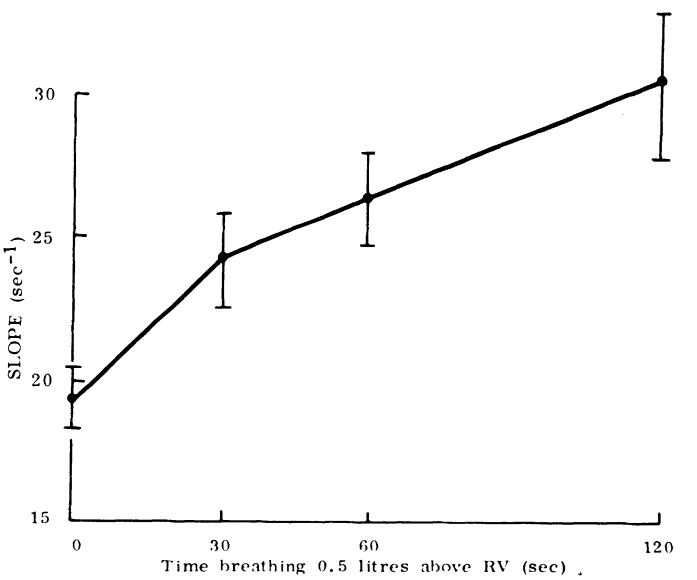

FIG. 4. Time courses of changes in slope of 30-10% portion of flow curve following low lung volume breathing. Mean ± SE in 9 subjects.

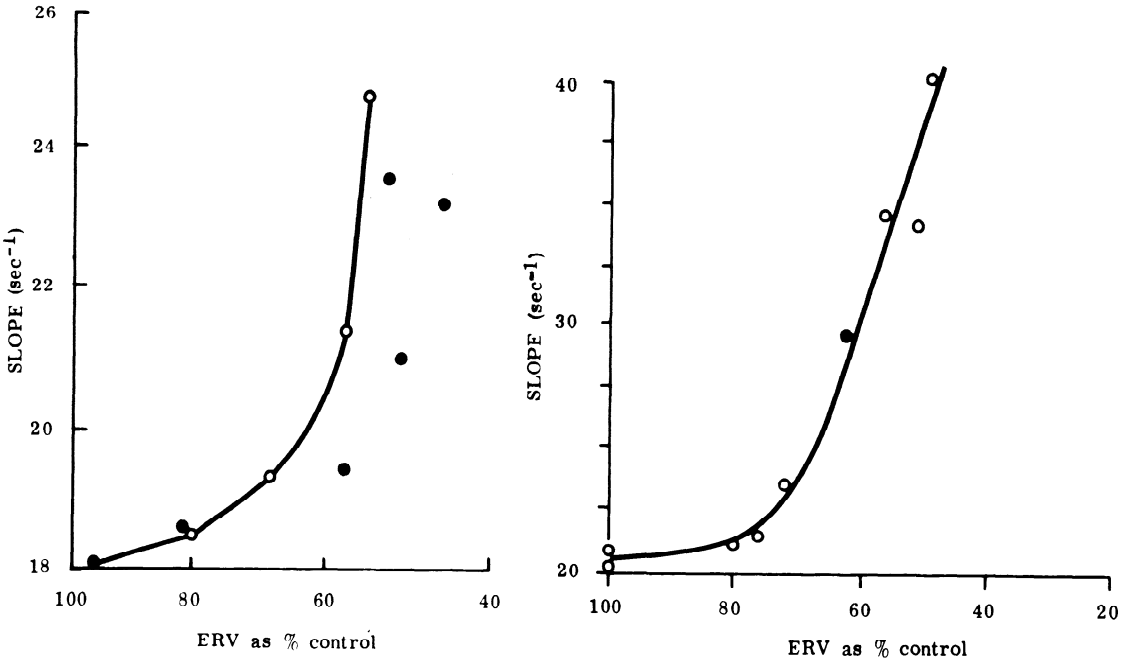

FIG. 5. Comparison of effect on slope of 30-10% portion of flow-volume curve of progressive chest strapping (○——○) with low lung volume breathing (LLVB) for 2 min (●). *Left: subject 3* in whom LLVB was repeated at different levels of expiratory reserve volume (ERV); *right: subject 6* in whom LLVB was performed at one level of ERV.

strapped, there was no significant difference between the greatest slope of the flow-volume curve after strapping as compared with after 2 min LLVB. In all four subjects in whom strapping, simulated strapping, and partial maneuvers were performed at the same session, slopes on strapping and simulated strapping were greater than on partial maneuvers; the slope was greatest after strapping in three and after simulated strapping in one [slope, second^{-1}: partial 2.5 ± 0.7 (SD), strapped 3.7 ± 0.7 (SD), simulated strapping 3.4 ± 0.9 (SD). In the two subjects in whom LLVB and progressive strapping were directly compared, the magnitude of the effect of LLVB was similar to that seen after strapping (Fig. 5).

The slope of the flow-volume curve was greater during maximal than partial maneuvers, when both were immediately preceded by three VC breaths [21.9 ± 4.3 (SD), 19.4 ± 3.5 (SD) s^{-1}, $P < 0.01$]. Similarly, the slope during maximal maneuvers was greater than the during FRC partial maneuvers [21.9 ± 4.3 (SD), 20.5 ± 3.5 (SD) s^{-1}, $P < 0.01$], but there was no significant difference between the slopes in the above two forms of partial maneuvers. There was a significant difference ($P < 0.01$) in flow rates between partials following inhalation from RV depending on whether a 1-s breath hold occurred at RV + 0.5 liters or at 50% VC (Fig. 6). Breath holding at 50% for 30 s reduced the slope of the flow-volume curve ($P < 0.05$), but breath holding at RV + 0.5 liters for 30 s increased the slope ($P < 0.05$). Breath holding at RV + 0.5 liters for 30 s increased the slope by 26% over normal partial flow-volume curves (breath holding for 1 s at about 50% VC), whereas LLVB for 30 s, in the same subjects, increased the slope by 28%.

After inhalation of 160 μg of ipratropium the change produced by LLVB was abolished in two subjects and markedly diminished in the other two (Fig. 7). Oral propranolol produced profound β-blockade as indicated by resting heart rates of 42 and 44 beats/min in *subjects 1* and *3*, respectively, but did not alter the effects of LLVB: the slope (second^{-1}) in *subject 1* before LLVB was 29 and after LLVB 43, and in *subject 3*, 12 before and 22 after.

Quasistatic lung recoil pressure (Fig. 8*A*) during expiration from 55% VC was greater than during expiration from TLC in all five subjects, the mean difference being 1.1 cmH_2O at 50% VC. Similarly, recoil pressure was higher in all five subjects after LLVB than during expiration from TLC, the mean difference being 1.6 cmH_2O at 50% VC. In four subjects the recoil pressure at 50% VC was higher after LLVB than on a partial maneuver, but there was no difference in this pressure in the fifth subject. Overall there was no significant difference between recoil pressures following LLVB than on partial maneuvers, even on paired-*t* testing. In the four subjects in whom recoil pressures were measured following maximal and partial maneuvers, strapping, and simulated strapping, all four showed higher recoil pressures after strapping, simulated strapping, and partial maneuvers than after maximal maneuvers (Fig. 8*B*). There was no such clear trend when comparisons were made between recoil pressures after partial simulated strapping and strapping maneuvers [mean recoil pressure at 40% VC: maximum 5.1 ± 2.4, (SD), partial 6.3 ± 1.7 (SD), simulated strapping 6.8 ± 2.9 (SD), strapping 7.1 ± 3.7 (SD) cmH_2O].

In all four subjects the resistance of the upstream segment (Rus) on strapping and simulated strapping was less than on partial expiration, but there was no consistent difference between Rus during strapping and simulated strapping [Rus, $cmH_2O \cdot l^{-1} \cdot s$: partial 1.2 ± 0.7 (SD), strapped 0.9 ± 0.6 (SD), simulated strapping 0.9 ± 0.6 (SD)]. In the three subjects in whom simulated quasistatic expirations were performed after LLVB, forced

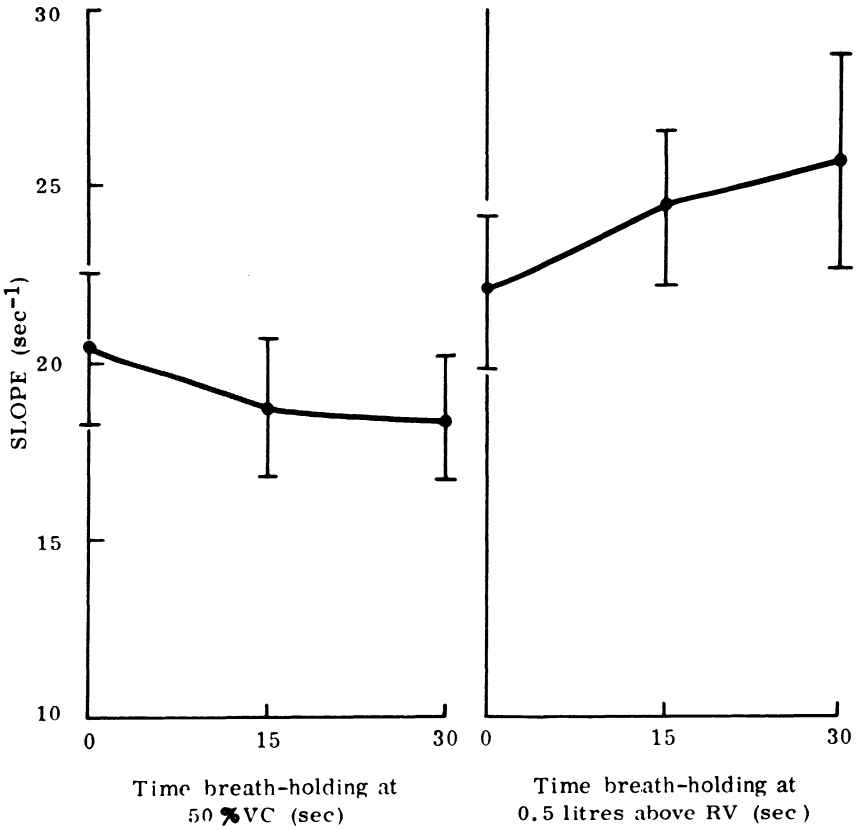

FIG. 6. Time course of changes in slope of 30–10% portion of flow-volume curve following breath holding at 50% vital capacity (VC) (*left*) and at residual volume (RV) plus 0.5 liters (*right*). Mean ± SE in 6 subjects.

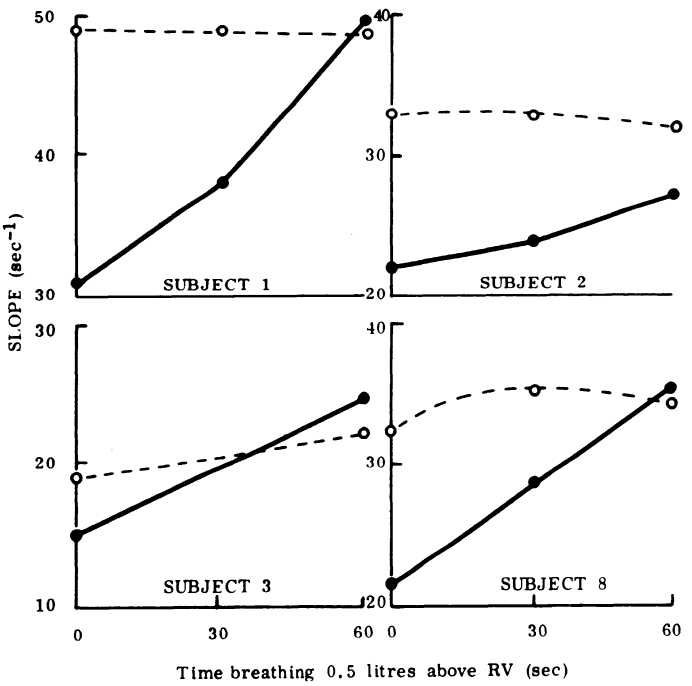

FIG. 7. Time course of changes in slope of 30-10% portion of flow-volume curve with low lung volume breathing before (●—●) and after (○---○) ipratropium in 4 subjects.

expiratory flow-volume curve slope after this maneuver was unchanged from that after LLVB alone [mean slope, second^{-1}: 10.8 for control partial, 15.1 after LLVB, 15.3 after LLVB and quasistatic maneuver].

DISCUSSION

We confirmed earlier observations (17, 18) that chest strapping increased forced expiratory flow rates and also showed the relation between the degree of chest strapping and the increase in flow rates, which had not been demonstrated before. Our analysis of the flow-volume curves depends partly on assuming that there were no changes in residual volume. However, our direct observations that residual volume did not change are supported by the two large series of chest strapping experiments (5, 12), but not by two smaller studies (17, 18).

Breathing at low lung volumes increased forced expiratory flow rates over the range 30–10% VC. Prefaut et al. (15) have previously studied the effect of functional residual capacity reduction on forced expiratory flow rates. They achieved a 27% drop in functional residual capacity by water immersion and found, in five subjects, small increases in flow rates at lung volumes above 40% VC and small decreases in flow rates at lower lung volumes. Our finding of significant increases in flow rates at lower lung volumes might reflect our greater reduction in functional residual capacity.

The effects of strapping and low lung volume breathing on flow rates and recoil pressures were similar; so we conclude that the effects of strapping described previously are caused solely by breathing at a low lung volume. This conclusion is not surprising in view of the earlier demonstration that removal of the chest strap did not affect the changes in forced expiratory flow rates until a deep breath was taken (18).

Increased flow rates after low lung volume breathing

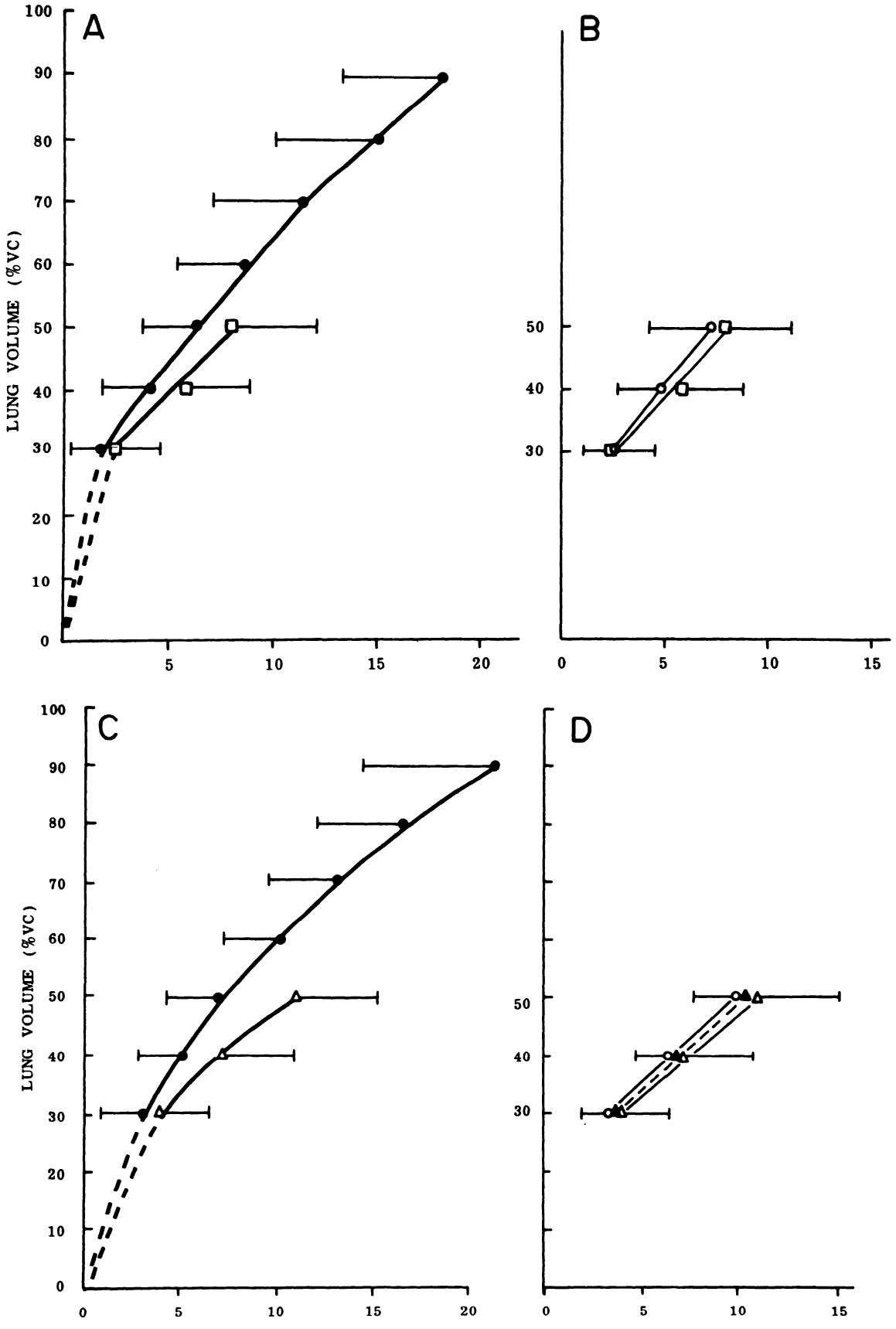

FIG. 8. Quasistatic recoil pressure plotted against lung volume for *A*: maximal (●) compared with low lung volume breathing (LLVB) (□); *B*: partial (○) compared with LLVB (□); *C*: maximal (●) compared with strapped (△); *D*: partial (○) compared with simulated strapping (▲) and strapped (△). Mean ± SD in 5 subjects for *A* and *B* and 4 subjects for *C* and *D*.

could be caused by a decrease in airflow resistance, an increase in lung recoil pressures, or to a combination of both factors. Our study confirms previous observations that chest strapping (5, 12, 17, 18) and low lung volume breathing (16) increase recoil pressure when compared with recoil pressure measured on expiration from a total lung capacity. However, these values of static recoil pressure on deflation from total lung capacity are not appropriate values to compare with measurements made during chest strapping or low lung volume breathing because of the different volume history involved. For example, at any lung volume static recoil pressure on exhalation from 50% VC is greater than recoil pressure on exhalation from total lung capacity (9, 19). Indeed, although our control maneuvers were more appropriate than those in other studies, they were not perfect. Although the lung volume at which forced expiration commenced was standardized to 50% VC for control partials and low lung volume breathing, and also for strapping and simulated strapping in the comparative study of static recoil pressure and flow rates, this volume was, of course, variable in the studies of progressive strapping. Further, the lung volume at which the preceding inhalation started was residual volume for control partials, RV + 0.5 liters for low lung volume breathing and the resulting functional residual capacity for strapping and simulated strapping. However, we think any errors due to these discrepancies would be small. Our finding of only small increases in static recoil pressure after low lung volume breathing or chest strapping when compared with recoil pressure during partial maneuvers could mean that the affect of low lung volume breathing or strapping is extremely transient and partially wears off during the quasistatic maneuver. This is unlikely because the flow-volume curves performed after a quasistatic maneuver showed that the effect of low lung volume breathing on flow rate was not altered by this maneuver.

The increase in recoil pressure following low lung volume breathing compared with partial maneuvers was of the order of 10–15%. However, the increases in flow rates observed following low lung volume breathing were in the order of 50–60%. Thus, although we did not directly measure the resistance of the upstream segment following low lung volume breathing, we would conclude that an important factor resulting in the increased forced expiratory flow rate following low lung volume breathing was a decrease in the resistance to airflow. This conclusion is supported by the finding of a marked decrease in the effect on flow rates following low lung volume breathing by giving ipratropium, as ipratropium results in bronchodilatation but has no effect on static recoil pressure (7, 9). In the four subjects in whom recoil pressures and flow rates were measured at the same session, the resistance of the upstream segment was in all cases greater on partial than on maximal maneuvers, as previously observed (9, 19). In all four subjects there was a fall in upstream resistance after simulated strapping or strapping, when compared with upstream resistance on a partial maneuver, but there was no consistent difference between upstream resistance on simulated strapping and on strapping, or between upstream resistance on either of these maneuvers and during maximal maneuvers. These results support our contention that the effects of low lung volume breathing and strapping on airflow are at least in part caused by a decrease in the resistance to airflow.

The resistance to airflow could be changed by bronchodilatation, upstream movement of the equal pressure point, or variation in the collapsibility of the airways. It seems unlikely that the position of the equal pressure point will be altered when neither static recoil pressure nor exhaled gas composition has changed, unless airway caliber has also changed. Alterations in airway compliance could affect forced expiratory flow rates, but it has been previously argued (9) that the sense of such changes cannot be predicted with the current understanding of gas flow in the lungs. We think that breathing or breath holding at low lung volumes and chest strapping all cause bronchodilatation. This is supported by previous evidence that chest strapping increases lung conductance and dead space (17) and that chest strapping or breathing at low lung volume decreases airways resistance (5), although Butler, Caro, and co-workers (4, 5) felt that the changes in airways resistance could be explained by the altered lung recoil pressures alone. However, their comparison was with the lung recoil pressures on maximal expiration and not with the more directly applicable recoil pressure on partial expiration. This bronchodilatation following low lung volume breathing is not the same as the normal airway hysteresis that results in narrower airways at a given lung volume on inhalation after transient exhalation to residual volume, as compared with airway caliber on exhalation from total lung capacity (10). This finding is supported by our observations that flow rates on partial expiratory maneuvers are lower than on maximal respiratory maneuvers despite the higher recoil pressure during partial maneuvers.

Sybrecht et al. (18) calculated that strapping increased the upstream resistance of the airways. However, these workers compared upstream resistance on a maximal maneuver before strapping with upstream resistance after strapping when the appropriate comparison would have been with a partial maneuver. Our study confirmed that upstream resistance during partial maneuvers is greater than on maximal maneuvers (9, 19), and thus Sybrecht's observations (18) need not be at variance with our suggestion that strapping and low lung volume breathing both cause bronchodilatation. However, we found no consistent changes in upstream resistance during chest strapping or simulated chest strapping when compared with upstream resistance during maximal maneuvers.

The cause of bronchodilatation following low lung volume breathing is not clear. The time course of the phenomenon and its diminution by the atropinelike drug ipratropium is compatible with, but by no means proof of, a vagally mediated reflex. Bronchodilatation could result alternatively from widespread small areas of atelectasis producing traction dilatation of the remaining patent conducting airways. However, despite controversy (3, 12, 16–18) there is no conclusive direct evidence that such atelectasis occurs following low lung volume breathing (23), nor was there any change in residual volume in this study. We wondered whether the effort of low lung

volume breathing might stimulate catecholamine release producing bronchodilatation, but β-blockade did not influence the effect of low lung volume breathing. End-tidal carbon dioxide was measured in two subjects and was not change by low lung volume breathing; so carbon dioxide is unlikely to be a factor. Breathing at low lung volume could have a more marked effect on the surface properties of the airways than on alveolar surface properties, thus altering airway caliber without producing such a marked effect on overall lung compliance.

This study emphasizes further (20) the importance of standardizing volume history before measuring pulmonary mechanics, even in normal subjects. The slope of the flow-volume curve was greater during maximal maneuvers than in either partial of functional residual capacity partial maneuvers, but there was no significant difference between the slopes in these two forms of partial maneuvers. There was a difference in flow rates between partial maneuvers following inhalation from residual volume (Fig. 5), depending on whether a 1-s breath hold occurred at 50% VC or at RV + 0.5 liters, and the longer the breath hold continued, the larger this difference became. We suggest that breath holding at RV + 0.5 liters results in progressive bronchodilatation, accounting for the initial disparity between these two forms of partial and the subsequent increase in flow the longer breath holding occurs at 0.5 liters above RV. The decline in flow rates following breath holding at 50% VC may be accounted for by the wearing off of bronchodilatation produced by the preceding three inspirations to total lung capacity.

Functional residual capacity decreases by about 25% from sitting to supine (22) and by a further 25% during anesthesia (14, 21). For the mean age and size of our subjects, such a 40% reduction of functional residual capacity would represent a decrease from 3.8 to 2.3 liters (6). If it is assumed that residual volume decreases by 300 ml when supine posture is adopted (22), this represents a reduction in expiratory reserve volume from 1.8 liters seated to about 0.6 liters during anesthesia. Thus it seems probable that the mechanical changes we have observed during low lung volume breathing would also occur during anesthesia.

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