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Research Article

SOME EFFECTS OF RESTRICTION OF CHEST CAGE EXPANSION ON PULMONARY FUNCTION IN MAN: AN EXPERIMENTAL STUDY*†

By COLIN G. CARO, JOHN BUTLER‡ AND ARTHUR B. DuBOIS§

(From the Department of Physiology of the Graduate School of Medicine, University of Pennsylvania, Philadelphia, Pa.)

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Certain alterations in pulmonary mechanics occur in man when an inspiration is taken from below the resting lung volume (1) and during shallow breathing (2). Some effects of a sustained reduction of the total lung capacity on respiration have been studied previously (3–5), yet the effects of chest strapping on pulmonary mechanics have received relatively little attention.

In the preliminary experiments we tightly bound the chest in the expiratory position with either adhesive tape or a long strip of rubber. Subsequently we designed a special chest corset that permitted us to reduce the resting lung volume and inspiratory capacity at will. The entire pressure-volume curve of the lung was altered by these procedures, but was restored to normal when a deep inspiration was taken after release of the restricting apparatus. Simultaneously, the escape of gas from poorly ventilated regions of the lung was detected. The effect of these changes on other aspects of pulmonary and circulatory function was examined and our findings, together with a consideration of their physiological and clinical significance, form the basis of this report.

METHOD

Twenty-five normal subjects were studied, all but two of them males. They ranged in age from 19 to 40 years. The thoracic cage was tightly strapped in the position of full expiration with one of the following: *1*) two inch inextensible adhesive tape (Figure 1A); *2*) a long strip, one inch wide, of rubber inner tubing wound several times about the chest, referred to as the extensible method (Figure 1B); or *3*) an adjustable chest corset (made by Henry Saur Co., Inc., Philadelphia, Pa.; Figure 2). In a few subjects the abdomen alone was restricted with the rubber strip. All measurements were made with the subject in the seated position.

The vital capacity (VC) and its subdivisions were measured with a recording spirometer, while the functional residual capacity (FRC) was determined by means of a body plethysmograph (6). A Tissot spirometer and kymograph was used to record the minute volume of ventilation, respiratory frequency and tidal volume. Volumes were corrected to BTPS.

A nitrogen gas analyzer was used to detect uneven alveolar ventilation following a single breath of oxygen (7). To measure the peripheral venous pressure, a glass tube manometer filled with 3.8 per cent sodium citrate solution was attached to an 18 gauge needle inserted into a subject's median antecubital vein. The initial reading made with reference to the manubrial angle was compared with a measurement, again made sitting, after the chest was strapped.

The pressure-volume relationship of the lung was studied with an air-containing esophageal balloon (8), attached via 1 mm ID polyethylene tubing to a capacitance manometer. The esophageal pressure was registered on a direct-writing Brush recorder and on a cathode ray oscillograph.

Several methods were used to measure the lung volume. In studying the *static* pressure-volume relationship of the lung, the thoracic gas volume was determined

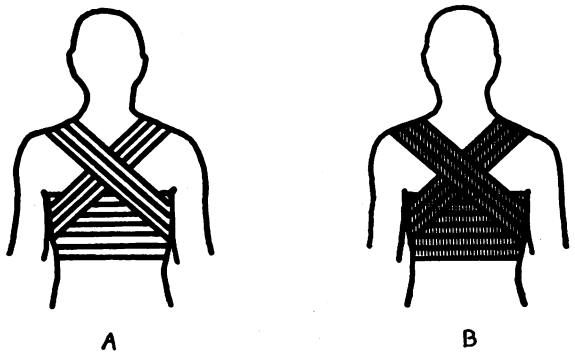

FIG. 1. METHODS OF CHEST RESTRICTION: A, ADHESIVE TAPE; B, STRIP OF RUBBER INNER TUBING.

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‡ This work was done during the tenure of a Rockefeller Traveling Fellowship in Medicine. Present address: Department of Medicine, University of Manchester, Manchester, England.

§ Established Investigator of the American Heart Association.

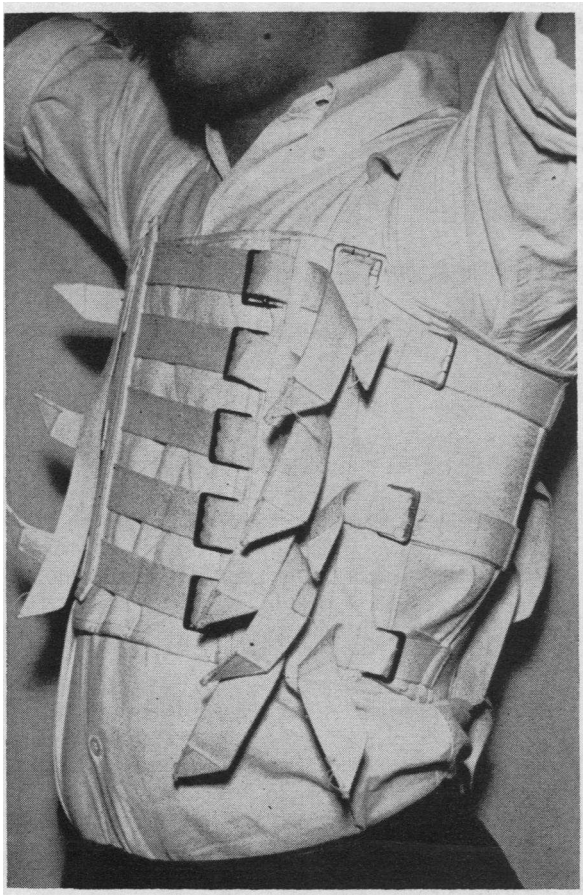

FIG. 2. PHOTOGRAPH OF A SUBJECT WEARING CHEST CORSET. The corset was made of coutil with 26 gauge rubber side panels. One inch web straps with prong buckles were used to join the two sections back and front, as shoulder straps, and to bridge over the rubber side panels.

by means of the body plethysmograph (6) at different respiratory levels over the range of the vital capacity. The corresponding mean esophageal pressure during shallow panting was recorded simultaneously. Since in normal subjects during shallow panting the inspiratory and expiratory nonelastic resistances are approximately equal, mean esophageal pressure was taken to indicate the elastic component of esophageal pressure at each lung volume. The fluctuations in esophageal pressure were approximately 2 cm H_2O. In measuring lung compliance during *spontaneous* breathing (8) the tidal volume was determined by electrical integration of the signal from the pneumotachygraph and recorded on the Y-axis of the oscillograph. The esophageal pressure, minus a voltage proportional to the airflow (9) was recorded on the X-axis and the resulting slope was measured as lung compliance. The tidal volume during *nitrogen dilution procedures* was measured from the pressure changes registered by a capacitance manometer attached to a box containing a breathing bag (i.e. a Donald-Christie box). This system had no detectable phase lag at the breathing frequencies encountered, and lung compliance was calculated from the tidal volume and esophageal pressure tracings. A recording spirometer was connected to the Donald-Christie box via a stopcock and used to measure the volume of deep breaths, thus avoiding large pressure changes within the system. In studying the *time course* of the onset of and recovery from lung compliance changes, the tidal volume and changes in resting lung volume were recorded with a closed circuit spirometer system. Esophageal pressure was measured at points of no airflow indicated by the pneumotachygraph recording.

The airway conductance is defined as the rate of airflow at the mouth per unit of alveolar-mouth pressure gradient. Airway conductance was measured during shallow panting by means of a body plethysmograph and compared with thoracic gas volume (10, 11). Observations were made at the resting lung volume and at other lung volumes over the range of the vital capacity.

To test for the presence of underventilated gas within the lungs during chest restriction, nitrogen dilution curves were plotted and analyzed for the release of nitrogen after removal of the restriction about the chest (12) and after a deep inspiration of oxygen. Additional details of these procedures are given with the results.

The oxygen tension of samples of brachial arterial blood was measured in control experiments in subjects who had breathed medical oxygen from a nonreturn valve system for periods of from 15 to 60 minutes. While the subjects continued to breathe oxygen, the chest cage was strapped and samples were drawn at intervals for as long as 45 minutes after the onset of chest restriction and after the restriction was released. The partial pressure of oxygen in the arterial samples was measured by means of a modification (13) of the Clark electrode. Measurement of arterial oxygen tension, for further investigating the previously reported effects of chest cage restriction on pulmonary mechanics (14), was suggested by other workers (15).

RESULTS

Lung volumes. The changes in the subdivisions of the total lung capacity produced by chest strapping are recorded in Table I. The average residual volume was not significantly changed; however, both the inextensible and extensible methods of chest strapping reduced the functional residual capacity and hence reduced the expiratory reserve volume. The inspiratory capacity was most markedly decreased by the inextensible strapping and it follows that the greatest reduction of the vital capacity and total lung capacity occurred with this latter method. Similar but less marked changes followed abdominal strapping (Table I).

TABLE I

Effect of chest restriction on subdivisions of total lung capacity *

Subject	Method of restriction	Total lung capacity		Subdivisions of total lung capacity									
				Vital capacity		Inspiratory capacity		Expiratory reserve volume		Functional residual capacity		Residual volume	
	Chest restricted	Control	Restricted	Control	Restricted	Control	Restricted	Control	Restricted	Control	Restricted	Control	Restricted
		L	*L*	*L*	*L*	*L*	*L*	*L*	*L*	*L*	*L*	*L*	*L*
AD†	Tape	8.20	2.80	5.50	0.80	3.65	0.20	1.85	0.60	4.55	2.60	2.70	2.00
CC	Tape	6.33	3.18	5.18	1.70	2.96	1.03	2.44	0.51	3.37	2.15	0.93	1.64
SS	Tape	6.69	3.28	6.00	1.62	2.91	0.66	2.50	0.96	3.78	2.62	1.28	1.62
NS	Tape left hemithorax	6.75	4.28	5.15	2.85	3.50	1.70	1.60	1.15	3.25	2.58	1.65	1.43
Mean		6.99	3.39	5.46	1.74	3.26	0.90	2.10	0.81	3.74	2.49	1.64	1.67
SE		0.41	0.32	0.20	0.42	0.19	0.32	0.22	0.15	0.29	0.11	0.38	0.12
PB	Rubber	6.57	5.52	5.37	4.43	3.56	3.57	1.83	0.87	3.01	1.95	1.18	1.08
GL	Rubber	6.12	3.77	4.40	2.04	2.70	1.42	1.69	0.58	3.42	2.35	1.73	1.77
GP	Rubber	4.91	4.19	4.03	3.08	2.71	2.67	1.26	0.39	2.20	1.52	0.94	1.13
DM	Rubber	6.13	4.55	4.68	3.31	3.61	2.81	1.02	0.50	2.52	1.74	1.50	1.24
JB	Rubber	8.08	6.33	5.70	4.80	4.18	4.23	1.52	0.57	3.90	2.10	2.38	1.53
TS	Corset	6.60	5.90	4.90	4.10	3.05	3.15	1.85	0.95	3.55	2.75	1.70	1.80
ST†	Rubber	4.20	2.60	3.00	1.45	1.86	1.04	1.14	0.41	2.34	1.56	1.20	1.15
CC†	Rubber	6.90	4.20	4.90	2.70	3.36	1.92	1.54	0.78	3.54	2.28	2.00	1.50
HL†	Rubber (very tight)	7.70	1.90	5.60	0.65	3.75	0.00	1.85	0.82	3.95	2.07	2.10	1.25
AD†	Rubber	7.50	6.60	5.40	4.70	3.28	4.02	2.12	0.68	4.22	2.58	2.10	1.90
Mean		6.47	4.56	4.80	3.13	3.21	2.48	1.58	0.66	3.27	2.09	1.68	1.44
SE		0.38	0.49	0.26	0.45	0.21	0.43	0.11	0.06	0.23	0.13	0.15	0.10
Mean, all subjects (chest restricted)		6.62	4.22	4.99	2.73	3.22	2.03	1.73	0.70	3.40	2.20	1.67	1.50
SE		0.28	0.38	0.21	0.38	0.15	0.37	0.17	0.06	0.19	0.11	0.15	0.08
	Abdomen restricted												
CC†	Rubber	7.20	5.50	5.00	3.30	3.47	2.53	1.53	0.77	3.73	2.97	2.20	2.20
JB†	Rubber	5.50	4.20	3.50	2.80	2.37	2.40	1.13	0.40	3.13	1.80	2.00	1.40
KA†	Rubber	6.20	5.80	4.90	4.40	3.23	3.59	1.67	0.81	2.97	2.21	1.30	1.40
Mean		6.30	5.17	4.47	3.50	3.02	2.84	1.44	0.66	3.28	2.33	1.83	1.67
SE		0.49	0.49	0.49	0.47	0.33	0.38	0.16	0.13	0.23	0.35	0.27	0.27

* Pressure-volume relationships of the lung, obtained during these experiments, are listed in Table III.
† Subdivisions of total lung capacity were measured plethysmographically.

TABLE II

Effect of chest restriction on minute volume, respiratory frequency and tidal volume

Subject	Method of restriction	Minute volume		Respiratory frequency		Tidal volume	
		Control	Restricted	Control	Restricted	Control	Restricted
		L/min		*rpm*		*L*	
SSa*	Rubber	7.9	8.8	12	17	0.66	0.52
JB*	Rubber	12.0	13.3	19	20	0.63	0.67
ER†	Corset	10.8	10.6	14	22	0.77	0.48
RS†	Corset	10.3	9.5	27	32	0.38	0.30
JJ†	Corset	10.6	10.1	11	17	0.96	0.59
EL†	Corset	12.4	11.3	25	29	0.50	0.39
GJ†	Corset	8.8	9.7	18	23	0.49	0.42
LM†	Corset	12.1	14.0	20	29	0.60	0.48
LU†	Corset	9.6	8.8	18	21	0.53	0.42
Mean		10.5	10.7	18	23	0.61	0.47
SE		0.51	0.62	1.8	1.8	0.06	0.04

* Breathing air.
† Breathing oxygen during measurement of arterial oxygen tension.

Chest strapping caused an increase of respiratory frequency and a decrease of tidal volume. The minute volume of ventilation was, however, not significantly altered (Table II).

Pressure-volume relationship of the lung. The data obtained on a single subject (PB, Table III) have been plotted in Figures 3A and 3B to illustrate the effect of chest strapping on the static

TABLE III

Effect of chest restriction on pressure-volume relationship of the lung *

Subject	Method of restriction Chest restricted	Lung compliance†			Trans-pulmonary pressure‡ at lung volume = control FRC			Maximal trans-pulmonary pressure‡	
		Control	Restricted: At control FRC	Restricted: At reduced FRC	Control	Restricted	After release of restriction, but prior to full inspiration	Control	Restricted
		L/cm H_2O			*cm H_2O*			*cm H_2O*	
AD	Tape	0.36		0.11	5.1		6.2	13	7
CC	Tape	0.22§		0.08‡					
SS	Tape	0.25		0.10	11.9			19	11
NS	Tape Left Hemithorax	0.19§		0.17‡					
Mean		0.25		0.11	8.5		6.2	16	9
SE		0.04		0.02					
PB	Rubber	0.33	0.19	0.15	0.0	6.7	12.3	20	17
GL	Rubber	0.34		0.22	4.2			10	7
GP	Rubber	0.16	0.10	0.13	4.0	9.2	4.6	25	17
DM	Rubber	0.23	0.18	0.18	6.4	7.3	10.4	27	11
JB	Rubber	0.34	0.30	0.24	2.4	9.2	4.8	20	12
TS	Corset	0.28	0.43	0.19	1.6	7.2		11	11
ST	Rubber	0.12	0.04	0.09	7.3	15.0		26	20
CC	Rubber	0.22	0.18	0.10	3.9	10.2		25	15
HL	Rubber (very tight)	0.28		0.08	4.1		7.2	20	8
AD	Rubber	0.48	0.48	0.29	6.3	7.5		26	16
Mean		0.28	0.24	0.17	4.0	9.0	7.9	21	13
SE		0.03	0.05	0.02	0.72	0.96	1.53	1.6	1.3
Mean, all subjects (chest restricted)		0.27	0.24	0.15	4.8	9.0	7.6	20	13
SE		0.025	0.054	0.055	0.88	0.96	1.28	1.7	1.2
	Abdomen restricted								
CC	Rubber	0.22	0.28	0.23	1.7	3.3		17	10
JB	Rubber	0.23	0.29	0.09	4.1	12.8		23	16
KA	Rubber	0.37	0.82	0.18	4.6	4.6		23	14
Mean		0.30	0.46	0.17	3.5	6.9		21	13
SE		0.03	0.18	0.04	0.9	3.0		1.9	1.9

* For subdivisions of total lung capacity corresponding experiments see Table I.
† Measured in range to tidal volume from P-V curves.
‡ Transpulmonary pressure = mouth pressure (atmospheric pressure) minus esophageal pressure.
§ Lung compliance measured during spontaneous breathing.

pressure-volume relationship of the lung. An S-shaped line (——) has been drawn by inspection through points (●) obtained in random order over the range of the vital capacity during the control period. After the chest was strapped new points (○) were obtained, again in random order, over the diminished ranges of the vital capacity and of transpulmonary pressure (mouth pressure minus esophageal pressure) and a curve (- - - -) has been drawn through them. The latter points were displaced downward and to the right of the former. When the strapping was released the subject permitted his resting lung volume to return to normal but breathed quietly and without taking in a deep breath. New points (×) were then obtained at progressively increasing values of lung volume until the fullest possible inspiratory volume was reached and further points (×) were thereafter obtained at values of progressively decreasing lung volume. The direction of the curves (·——·) drawn through these points is indicated by the arrows on the diagram. They form a loop and the descending curve approaches and approximates the control curve.

The essential features were a shift in the pressure-volume curve of the lung when the chest was strapped and failure of the pressure-volume relationship of the lung to return to normal until a high inspiratory volume was reached after release of the strapping. Similar data were obtained on several additional subjects whose chests or abdomens were strapped (Table III). Because of the sigmoid shape of the pressure-volume curve, the lung compliance slope varied after strapping depending upon whether it was measured on a steep or a flat part of the S (Table III). However, transpulmonary pressure measured at a lung volume corresponding to control functional residual capacity was significantly increased. The mean transpulmonary pressure in eight subjects was 4.0 cm H_2O, SE 0.72, before chest strapping and 9.0 cm H_2O, SE 0.96, while strapped ($p = 0.006$). Both measurements were made at a lung volume corresponding to control FRC (Table III). This indicated that there had been a significant separation of the curves.

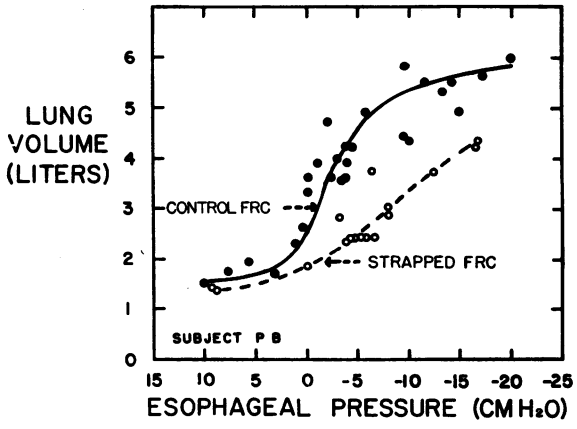

FIG. 3A. GRAPH OF PRESSURE-VOLUME RELATIONSHIP OF THE LUNG, SUBJECT PB. Curve (——) was obtained prior to chest restriction. The pressure-volume relationship was displaced to the right and downward during restriction, curve (- -), i.e., transpulmonary pressure was greater than at equivalent lung volume in the control state.

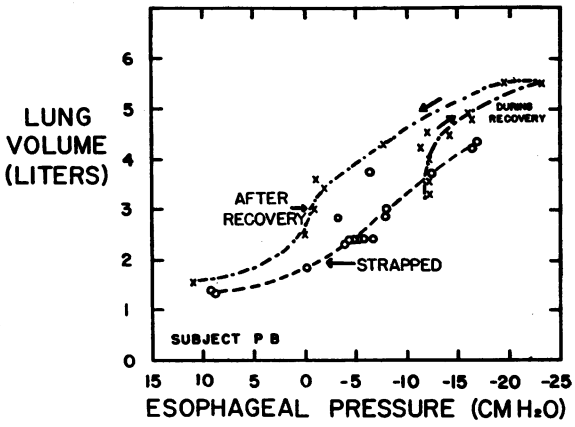

FIG. 3B. CURVE (-·-) WAS OBTAINED AFTER RELEASE OF RESTRICTION. Normal lung pressure-volume relationships were restored via a hysteresis-like pathway (see arrows) following a deep inspiration.

It was also shown in several subjects that the transpulmonary pressure, measured at a lung volume corresponding to control FRC, remained increased after the chest strapping was released. Thus the mean transpulmonary pressure in six subjects at a lung volume corresponding to the control FRC was 3.7 cm H_2O, SE 0.91, prior to chest strapping and 7.6 cm H_2O, SE 1.28 ($p = 0.006$), at the same volume after the restriction was released (Table III). The lung compliance was measured during spontaneous breathing and during breathing at 40 to 50 respirations per minute in four subjects in the control period and then when their chests were strapped; the

lung compliance did not appear to alter with the change in breathing frequency during either state. A reduction of lung compliance, measured at a resting lung volume corresponding to control FRC, was detectable in most subjects immediately after the chest corset was tightened, and this procedure occupied only about 1 minute (Table IV). A single deep inspiration, taken after the chest corset was released usually restored the lung compliance to normal (Table IV).

Venous pressure. The venous pressure showed a mean rise of 1.2 cm H_2O in three subjects, 1 minute after the chest corset was tightened.

TABLE IV

Onset of and recovery from changes in lung compliance

Subject	Lung compliance*				
	Control	Chest restricted			
		At reduced FRC	In inspiratory position†	After restriction released	After deep inspiration
	L/cm H_2O				
TS	0.31	0.16	0.17	0.17	0.22
GP	0.12	0.09	0.08	0.09	0.13
AD	0.21	0.13	0.12	0.22	0.29
EL	0.20		0.15	0.16	0.22
ST	0.13		0.15	0.11	0.13
GL	0.22		0.19	0.20	0.29
Mean	0.198		0.143	0.158	0.213
SE	0.028		0.016	0.020	0.029

* Measured during spontaneous breathing.
† At about control FRC immediately after tightening chest corset.

Airway conductance. In Figure 4 the curve, together with the standard error of the estimate, relates airway conductance to thoracic gas volume over the range of the vital capacity for ten subjects in the control state. The mean airway conductance for these subjects at resting lung volume is also shown and is designated as "control." It was significant that the average airway conductance in these subjects was not decreased at the reduced resting lung volume caused by chest strapping (see point designated as "strapped," Figure 4, and also Table V), whereas it was decreased during voluntary panting at the same reduced lung volume. This finding was further investigated by making repeated measurements of airway conductance and thoracic gas volume in three subjects who panted continuously for as long as possible without chest strapping, at a lung volume close to residual volume. In the course of about 1 minute the initially low airway conductance in these subjects had risen to about normal.

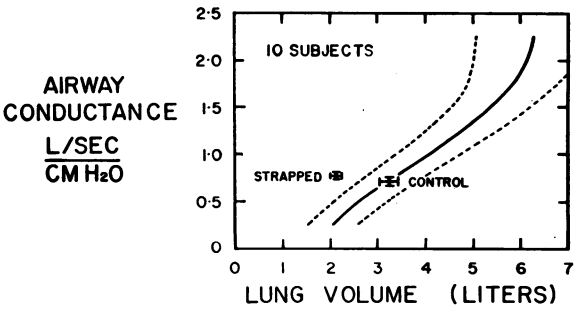

FIG. 4. GRAPH OF AIRWAY CONDUCTANCE AND LUNG VOLUME. Airway conductance was measured in 10 subjects at resting respiratory level prior to chest restriction (control) and at reduced lung volume during chest restriction (strapped). The average value for airway conductance was not significantly changed (Table V); but an equivalent voluntary reduction of lung volume in the same subjects without chest restriction (curve with standard error of estimate) lowered the airway conductance.

Distribution of inspired gas. Chest strapping caused a slight but statistically significant increase in the non-uniformity of alveolar ventilation as measured by the nitrogen meter single breath test (Table VI).

Underventilated gas within the lungs. When a deep inspiration of oxygen was taken by Subjects TS, PS, GL and AD during the measurement of

TABLE V

Effect of chest restriction on airway conductance

Subject	FRC		Airway conductance	
	Control	Restricted	Control	Restricted
	L		*L/sec/cm H_2O*	
SS	3.78	2.62	1.43	1.17
NS	3.25	2.74	0.43	0.68
CC	3.37	2.15	0.54	0.53
AD	4.55	2.60	0.78	0.87
DM*	2.40	1.61	0.72	1.14
GL	3.42	2.35	0.74	0.79
GP	2.20	1.52	0.45	0.60
HL	3.95	2.07	0.74	0.43
PB	3.01	1.95	0.65	0.60
DM*	2.52	1.74	0.84	1.02
KA	3.20	1.98	0.49	0.67
Mean	3.24	2.21	0.71	0.77
SE	0.21	0.13	0.08	0.08

* Studies performed on two occasions on this subject.

TABLE VI

Chest restriction and uniformity of alveolar ventilation

Subject	Alveolar gas uniformity* index	
	Control	Restricted
SS	0.4	1.7
GL	0.1	2.3
GP	1.1	1.7
HL	0.7	0.7
DM†	0.5	0.7
DM†	0.0	1.1
PB	0.3	1.1
AD	1.1	2.8
NS	0.0	0.7
JB	0.4	1.4
Mean	0.5	1.4
SE	0.13	0.26
		p = 0.001

* Per cent N_2 increase during expiration of 750 to 1,250 ml following a single breath of O_2. Mean of 3 determinations.

† Studies on same subject, 2 occasions.

a nitrogen dilution curve in the control state, the concentration of nitrogen fell in the succeeding expiration to a predictable level when the end-expiratory nitrogen concentration was plotted against the cumulative volume of oxygen inspired. However, when this experiment was repeated in Subjects TS, PS, GL, AD and CC, whose chests were strapped, the concentration of nitrogen in the succeeding expiration was elevated (Figure 5). The additional nitrogen had apparently been released from poorly ventilated regions of the lung and its volume was calculated by measuring the area beneath the elevation in the nitrogen dilution curve. The mean volume of nitrogen released was 6 ml, range 0 to 15 ml.

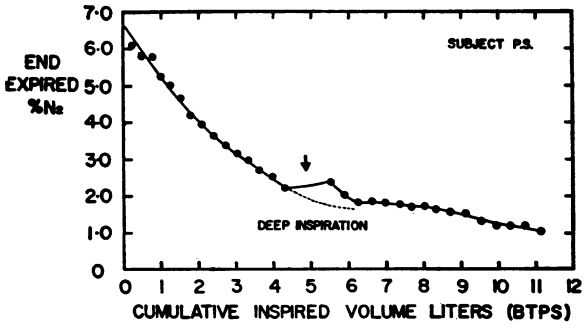

FIG. 5. GRAPH OF PORTION OF N_2 DILUTION CURVE DURING CHEST RESTRICTION (ARBITRARY ZERO ON ABSCISSA). A deep inspiration of O_2 with the chest restricted caused an elevation of end-expired per cent N_2.

In other experiments the chest corset was released during the course of the nitrogen dilution procedure (Figure 6). This allowed the subject's FRC to return to normal and the end-expiratory nitrogen concentration rose during several subsequent tidal breaths with the elimination of additional nitrogen. The mean volume released was 37 ml, range 7 to 90 ml. During succeeding tidal breaths (Figure 6) the expired nitrogen concentration again fell in a predictable manner. However, a deep inspiration caused a further elevation in the end-expired nitrogen concentration, indicating the elimination of a further volume of nitrogen (mean volume 8 ml, range 4 to 12 ml).

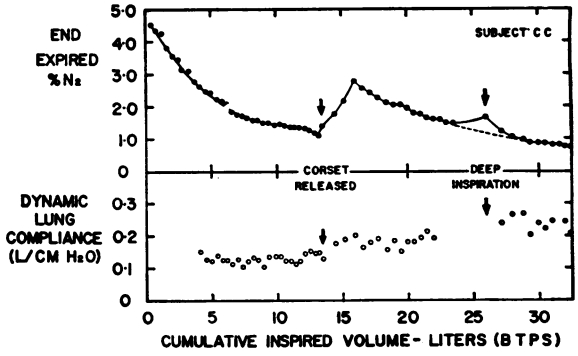

FIG. 6. UPPER DIAGRAM: GRAPH OF PORTION OF A NITROGEN DILUTION CURVE. End-expired per cent N_2 and cumulative inspired volume of O_2 (arbitrary zero) during and after release of chest restriction. End-expired per cent N_2 rose following release of corset and again following a deep inspiration of O_2. LOWER DIAGRAM: Lung compliance during spontaneous breathing, same experiment. Lung compliance rose after release of corset, permitting return of lung volume to about control functional residual capacity. Lung compliance was further increased (to normal value for this subject) by deep inspiration of O_2.

It has previously been shown that the altered pressure-volume relationship of the lung was restored to normal by a deep inspiration once the chest strapping was released (Figure 3 and Table IV). The lower diagram in Figure 6 shows that normal lung compliance was restored at the same time that nitrogen was released by the deep inspiration. Similar findings were obtained in the other subjects. In a final series of experiments the deep inspiration was delayed until the three subjects, TS, GL and CC had continued to

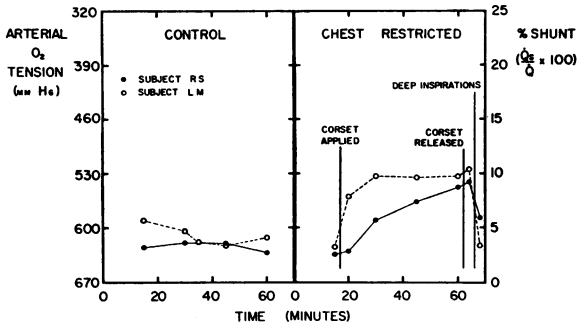

FIG. 7. ARTERIAL OXYGEN TENSION DURING A ONE HOUR PERIOD BREATHING 100 PER CENT OXYGEN. Control (left) and alterations following tight application of a chest corset (right) showing increased percentage of venous admixture. Recovery on release of chest corset.

breathe oxygen for 7 minutes after the chest corset was released. No additional nitrogen was then detectable in any of the three.

As already mentioned, the quantity of nitrogen released in these experiments was measured from the curve relating the end-expiratory nitrogen concentration and minute volume of ventilation. To determine the accuracy of this method of calculation the measurements were checked in one subject by plotting mean expired nitrogen concentration, obtained by graphic integration of the instantaneous expired nitrogen concentration and volume, against minute volume. Using this more laborious method of calculation, reasonable agreement was found.

Systemic arterial oxygen tension. The arterial oxygen tension of two subjects, LM and RS, was relatively constant when drawn at intervals during a 1 hour control period of oxygen breathing. However, when the chest corset was tightened in subjects who had already breathed oxygen for 15 minutes, a fall of arterial oxygen tension was detectable 5 to 45 minutes later in six out of eight subjects (Figure 7). Following release of the chest corset the arterial oxygen tension continued to be decreased until one or more deep inspirations of oxygen were taken. One subject became faint after 15 minutes of chest restriction. His arterial oxygen tension had not decreased so that his symptoms could not be attributed to arterial hypoxemia. The arterial P_{CO_2} was measured in one of the subjects (DJ, Table VII) and found to be unchanged from the control value of 44 mm Hg, after chest strapping. In two other subjects the end-expired alveolar CO_2 concentration was measured with a CO_2 analyzer and was not altered by chest restriction.

DISCUSSION

In these experiments, chest restriction led to a change in the pressure-volume relationship of the lungs in normal man. There was slightly uneven distribution of inspired gas, measured by the single breath test, and a small quantity of very poorly ventilated gas measured by nitrogen washout procedures. Airway resistance was not increased.

TABLE VII

Systemic arterial oxygen tension in subjects breathing 100 per cent oxygen with chest restriction

	Arterial oxygen tension (mm Hg)											
		During chest restriction (minutes after chest restricted)										
Subject	Before chest restriction (after 15 min O_2 breathing)	1	5	10	15	30	40	45	Immediately pre-release of corset	1 Min after release but before deep inspirations	After 1 deep inspiration	After several deep inspirations
GJ	605* (606, 604)	585	586		591	586	569		569	554	578	608
JJ	605* (605, 605)	573	556		546	532		536	536	517	593	605
EL	605* (603, 606)	603		606	613	626		623	623	623	611	611
ER†	592* (592, 592)	598	590		591				591	590	584	572
LM	627		564		537	538		537	537	529		625
RS	635* (634, 636)	639	631		594	570		551	551	543		591
LU	607	593	604		605	575			575	559	568	
DJ‡	597					551			551			616
Mean	609								569			
SE	5.1								11.8			

* Mean of duplicate arterial samples.
† Corset was released after 15 min, because of faintness of subject.
‡ Measured supine during cardiac catheterization. Normal heart and lungs by physiological study.

A slight increase in venous admixture to arterial blood was detected. These effects were reversed when the subjects took a deep breath after the release of chest restriction.

We believe we have shown that forces external to the chest cage, by reducing thoracic gas volume, can affect the function of the lungs. We speculate that this effect may occur in other conditions in which there is a reduction of thoracic gas volume, as in individuals wearing tight garments, or perhaps aviation suits designed to counteract gravitational stress. Reduced thoracic gas volume may occur if the chest is strapped for rib fracture, encased in a body cast, or is subjected to hydrostatic pressure under water or to increased gravitational force. The total lung capacity may be reduced in clinical disorders that affect the expansion of the chest wall or pleura: *1*) paralysis of the respiratory muscles; *2*) flail chest resulting from multiple rib fractures; *3*) thoracic deformity in kyphosis or scoliosis; *4*) funnel chest; *5*) thoracoplasty; *6*) ankylosing spondylitis; *7*) obesity; *8*) fibrothorax or calcification of the pleura. A complete discussion of the alterations of pulmonary function in these conditions is beyond the scope of this paper. The reader is referred to other articles on the subject (16–21).

In young patients with scoliosis (19, 21), one frequently finds that the static lung volumes are decreased, lung compliance is reduced, there is slight abnormality of the distribution of inspired gas and, unexpectedly (21), the airway conductance may be normal despite the reduction of thoracic gas volume. There is similarity between these findings and those that we produced in normal subjects by acute experimental restriction of chest cage expansion. We suggest that the pulmonary changes found in young patients with scoliosis may be secondary to a reduction of thoracic gas volume.

In searching for an explanation for the altered mechanical properties of the lung that followed chest strapping several possibilities were considered. It seemed unlikely that the changes were the result of pulmonary vascular engorgement, because the increase in venous pressure was slight in contrast to that obtained under different conditions (22). It seemed more likely that there had been an alteration of the number of terminal lung units (a terminal lung unit being thought to consist of an airway, small airspace, alveolar walls and capillaries) participating in expansion and ventilation, or an alteration of the elastic characteristics of the lung tissue or surface lining film. The following part of the discussion is a further attempt to analyze these possibilities in the light of our experimental data, compared with the findings of other investigators.

An observation which pointed toward a change in the number of ventilated lung units was the release of nitrogen in association with the recovery of the normal pressure-volume relationship of the lung.

After chest strapping the single breath test for distribution of gas was almost unchanged, and lung compliance was not diminished by increasing the breathing frequency. We concluded that the accessible portions of the lung were evenly ventilated, and time constants were not uneven throughout this region (23). Hence the release of nitrogen from the lung following an increase of resting lung volume or with a deep inspiration seemingly indicated that the gas had been present in virtually nonventilated regions. The smallness of the volume of nitrogen released makes it unlikely that closure of major (e.g., lobar or segmental) airways occurred, and we are inclined to believe that closure had occurred in terminal lung units. A mechanical basis for the closure and opening of such units has been described (1, 24) and the phenomenon of their sequential opening has been observed in isolated animal lungs (25). Our data do not permit conclusions to be drawn on the volume of gas that might be trapped in an occluded lung unit (24, 26) or on the number of units that were occluded.

Despite the reduction of the resting lung volume (11) and the apparent closure of lung units and therefore reduction of the total number of conducting airways, mean airway conductance was not decreased with chest restriction. Furthermore, it rose toward normal during continued voluntary panting in the expiratory position. The explanation of these apparent paradoxes appears to be enlargement of the caliber of the patent airways by the increased lung elastic pressure (27).

It would seem that some of the lung units that had become occluded at small lung volume temporarily remained so during tidal breathing after return, unstrapped, to resting lung volume. We

failed, however, to detect any evidence (nitrogen release) of the existence of occluded units in subjects at normal resting lung volume without previous restriction. This is consistent with the findings of others (28) and with the observation that the volume of the functional residual capacity was no different in normal subjects, whether measured plethysmographically or by a nitrogen dilution technique (29). The hysteresis, i.e., failure of the lung to retrace the same pressure-volume curve on inflation and deflation, normally detectable on taking a deep inspiration starting from resting lung volume (1, 30), may therefore be due to mechanisms other than the recruitment of lung units, such as changes in the surface lining film or in tissues of the lung.

The fall in systemic arterial oxygen tension during chest restriction is of theoretical interest. It could be due to the shunting of blood through nonventilated regions of the lung, to a fall in the oxygen tension of the mixed venous blood, or to a decrease in alveolar oxygen tension. Since the subjects had been breathing oxygen for 15 minutes prior to chest restriction, the alveolar oxygen tension would be equal to the atmospheric pressure minus the partial pressures of water vapor and of carbon dioxide. Our evidence suggests that there was no alteration in the arterial or alveolar P_{CO_2} despite the slight increase of respiratory frequency and decrease of tidal volume that occurred during chest restriction. Further studies will be needed, however, to distinguish between the two remaining possibilities.

As a physiological technique, chest restriction permits measurements to be made at equal lung volume but at different transpulmonary pressures, thus allowing separation of the effects of volume and pressure. This principle has been applied to the study of pulmonary hemodynamics in anesthetized dogs (31) and may be applicable in unanesthetized man.

SUMMARY

1. The total lung capacity and its subdivisions were reduced by restricting chest expansion in normal man.

2. The lung pressure-volume relationship was altered (smaller volume resulting from unit pressure) over much of the vital capacity. Respiratory frequency was increased and tidal volume was reduced. Airway conductance at resting lung volume was not changed despite reduced functional residual capacity. There was slight unevenness of alveolar ventilation with evidence of non- or poorly ventilated lung units. Systemic arterial O_2 tension fell during oxygen breathing, and there was a slight increase of peripheral venous pressure.

3. Following release of chest restriction, the mechanical changes in the lungs were reversed via a hysteresis-like pathway. They appear to be explained in part by the opening of lung units which had become closed during chest restriction.

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REFERENCES

1. Mead, J., Whittenberger, J. L., and Radford, E. P., Jr. Surface tension as a factor in pulmonary volume-pressure hysteresis. J. appl. Physiol. 1957, **10**, 191.
2. Ferris, B. G., Jr., and Pollard, D. S. Effect of deep and quiet breathing on pulmonary compliance in man. J. clin. Invest. 1960, **39**, 143.
3. Haldane, J. S., Meakins, J. C., and Priestley, J. G. The effects of shallow breathing. J. Physiol. (Lond.) 1919, **52**, 433.
4. D'Silva, J. L., Freeland, D. E., and Kazantzis, G. The performance of patients with ankylosing spondylitis in the maximum ventilatory capacity test. Thorax 1953, **8**, 303.
5. Carton, R. W., and Sepp, E. On taping the chest. Amer. Rev. Tuberc. 1957, **76**, 167.
6. DuBois, A. B., Botelho, S. Y., Bedell, G. N., Marshall, R., and Comroe, J. H., Jr. A rapid plethysmographic method for measuring thoracic gas volume: A comparison with a nitrogen washout method for measuring functional residual capacity in normal subjects. J. clin. Invest. 1956, **35**, 322.
7. Comroe, J. H., Jr., and Fowler, W. S. Lung function studies: VI. Detection of uneven alveolar ventilation during a single breath of oxygen; a new test of pulmonary disease. Amer. J. Med. 1951, **10**, 408.
8. Mead, J., and Whittenberger, J. L. Physical properties of human lungs measured during spontaneous respiration. J. appl. Physiol. 1953, **5**, 779.

9. Marshall, R., and DuBois, A. B. The measurement of the viscous resistance of the lung tissues in normal man. Clin. Sci. 1956, **15**, 161.
10. DuBois, A. B., Botelho, S. Y., and Comroe, J. H., Jr. A new method for measuring airway resistance in man using a body plethysmograph: Values in normal subjects and in patients with respiratory disease. J. clin. Invest. 1956, **35**, 327.
11. Briscoe, W. A., and DuBois, A. B. The relationship between airway resistance, airway conductance and lung volume in subjects of different age and body size. J. clin. Invest. 1958, **37**, 1279.
12. Lewis, B. M., Forster, R. E., and Beckman, E. L. Effect of inflation of a pressure suit on pulmonary diffusing capacity in man. J. appl. Physiol. 1958, **12**, 57.
13. Polgar, G., and Forster, R. E. Measurement of Po_2 in small unstirred blood samples with a Clark electrode covered with a relatively impermeable membrane. Fed. Proc. 1959, **18**, 433.
14. Caro, C. G., Butler, J., and DuBois, A. B. Effects of chest strapping on lung mechanics. Physiologist 1958, **1** (no. 4), 8.
15. McIlroy, M. B., Butler, J., and Finley, T. Effect of chest strapping on pulmonary function in normal subjects. Fed. Proc. 1959, **18**, 102.
16. Chapman, E. M., Dill, D. B., and Graybiel, A. The decrease in functional capacity of the lungs and heart resulting from deformities of the chest: Pulmonocardiac failure. Medicine (Baltimore) 1939, **18**, 167.
17. Ferris, B. G., Jr., Mead, J., Whittenberger, J. L., and Saxton, G. A., Jr. Pulmonary function in convalescent poliomyelitic patients, III. Compliance of the lungs and thorax. New Engl. J. Med. 1952, **247**, 390.
18. Fishman, A. P., Turino, G. M., and Bergofsky, E. H. The syndrome of alveolar hypoventilation (Editorial). Amer. J. Med. 1957, **23**, 333.
19. Bergofsky, E. H., Turino, G. M., and Fishman, A. P. Cardiorespiratory failure in kyphoscoliosis. Medicine (Baltimore) 1959, **38**, 263.
20. Hanley, T., Platts, M. M., Clifton, M., and Morris, T. L. Heart failure of the hunchback. Quart. J. Med. 1958, **27**, 155.
21. Caro, C. G., and Gucker, T., III. Effects of kyphoscoliosis upon mechanics of breathing in children (abstract). Amer. Rev. Tuberc. 1958, **78**, 326.
22. Bondurant, S., Hickam, J. B., and Isley, J. K. Pulmonary and circulatory effect of acute pulmonary effects of acute pulmonary vascular engorgement in normal subjects. J. clin. Invest. 1957, **36**, 59.
23. Otis, A. B., McKerrow, C. B., Bartlett, R. A., Mead, J., McIlroy, M. B., Selverstone, N. J., and Radford, E. P., Jr. Mechanical factors in distribution of pulmonary ventilation. J. appl. Physiol. 1956, **8**, 427.
24. Clements, J. A., Brown, E. S., and Johnson, R. P. Pulmonary surface tension and the mucus lining of the lungs: Some theoretical considerations. J. appl. Physiol. 1958, **12**, 262.
25. Radford, E. P., Jr., and McLaughlin, M. Dependence of lung mechanical properties on anatomic relationships within terminal lung units. Fed. Proc. 1956, **15**, 147.
26. Mead, J., and Collier, C. R. Pulmonary mechanical changes in supine anesthetized dogs (abstract). Amer. J. Physiol. 1956, **187**, 616.
27. Butler, J., Caro, C. G., and DuBois, A. B. Lung tissue tension and airway conductance. Physiologist 1958, **1** (no. 4) 7.
28. Armitage, G. H., and Arnott, W. M. Air distribution in lungs during hyperventilation. J. Physiol. (Lond.) 1949, **109**, 70.
29. Bedell, G. N., Marshall, R., DuBois, A. B., and Comroe, J. H., Jr. Plethysmographic determination of the volume of gas trapped in the lungs. J. clin. Invest. 1956, **35**, 664.
30. Butler, J., White, H. C., and Arnott, W. M. The pulmonary compliance in normal subjects. Clin. Sci. 1957, **16**, 709.
31. Frank, R., Borst, H., Berglund, E., Collier, C., and Whittenberger, J. L. Influence of lung volume and airway pressure on pulmonary vascular resistance in dogs' lungs. Fed. Proc. 1958, **17**, 48.