# RIB CAGE RESTRICTION BUT NOT ABDOMINAL RESTRICTION CAUSES CHANGES IN THE ELASTIC RECOIL OF THE HUMAN LUNG

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Abstract

Chest-strapping increases recoil and this has been thought to be secondary to breathing at low lung volumes. However immersion decreases lung volumes without an increase in recoil, suggesting either that reduction of lung volumes by abdominal compression does not change recoil, or that blood shifts due to immersion prevent recoil changes. Measurements of lung volumes, deflation pressure-volume curves and maximum expiratory flow rates (MEF) were made in four healthy subjects during a variety of restrictive procedures. Reductions in lung volumes were achieved by inflating a pneumatic cuff beneath an adjustable chest corset which had been tightened while the subject held his breath at residual volume. The corset was applied either to the rib cage or the abdomen. Rib cage restriction decreased total lung capacity (TLC) by 43% and significantly increased elastic recoil and MEF. Subjects with rib cage restriction showed no further change in elastic recoil or MEF with immersion; blood shifts did not reverse recoil changes due to rib cage restriction. Abdominal restriction decreased TLC by only 20% and no change in recoil or MEF was observed. No change in recoil or MEF were seen when a similar reduction in TLC was achieved by rib cage restriction. In order to produce greater volume loss a combination of modest rib cage and maximal abdominal restriction was studied. Modest rib cage restriction reduced TLC by 20% and when maximal abdominal restriction was added TLC was reduced 40%, comparable to that observed with maximal rib cage restriction. Measurements were repeated in eight healthy subjects during both rib cage and combined restriction. Both forms of restriction increased MEF

by a similar amount, but no increase in elastic recoil was observed with combined restriction. Further the resistance of the airways upstream from the equal pressure point (Rus) was greater during rib cage restriction. Measurements of the uneveness of inspired gas distribution by the single breath $N_2$ test showed an increase during rib cage restriction which was significantly greater than that during combined restriction. We conclude that lung volume restriction induces changes in lung function, but the nature of these changes depend on how the restriction is applied, and therefore cannot be ascribed to low lung volume breathing per se.

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# I. INTRODUCTION

The process of respiration requires an adequate inflow and outflow of air between the atmosphere and the alveoli, that is an adequate alveolar ventilation. The mechanical properties of the respiratory system are designed to achieve this.

At normal lung volumes, the mechanics of breathing involves changing the point of balance between the opposing elastic recoil of the lungs, tending toward collapse, and the elastic forces of the chest wall, tending toward expansion. The tendency of the chest wall to expand is caused by the natural elasticity of its bones, muscles, and connective tissue.

The tendency of the lungs to collapse and recoil inward away from the chest wall is attributed to two factors, first deduced by Von Neergaard in 1929 (1). One-third of the recoil tendency arises from the elastic properties of the lung tissue itself. The remaining two thirds can be accounted for by the surface tension of the fluid lining the alveoli. This surface tension at the air-liquid interface in the alveoli is caused by the strong intermolecular attraction within the liquid that tends to continually diminish the surface area of the alveoli. The presence of surfactant, a surface active lipoprotein secreted by special surfactant secreting cells that are components of the alveolar epithelium effectively lowers the surface tension of this interface. This combined with the hysteresis properties of surfactant tends to maintain alveolar stability and helps prevent alveolar collapse (3,4).

The total recoil tendency of the lungs can be estimated by measuring, under static (no flow) conditions, the pressure across the lung at any volume. This transpulmonary pressure ($P_L$) is defined as

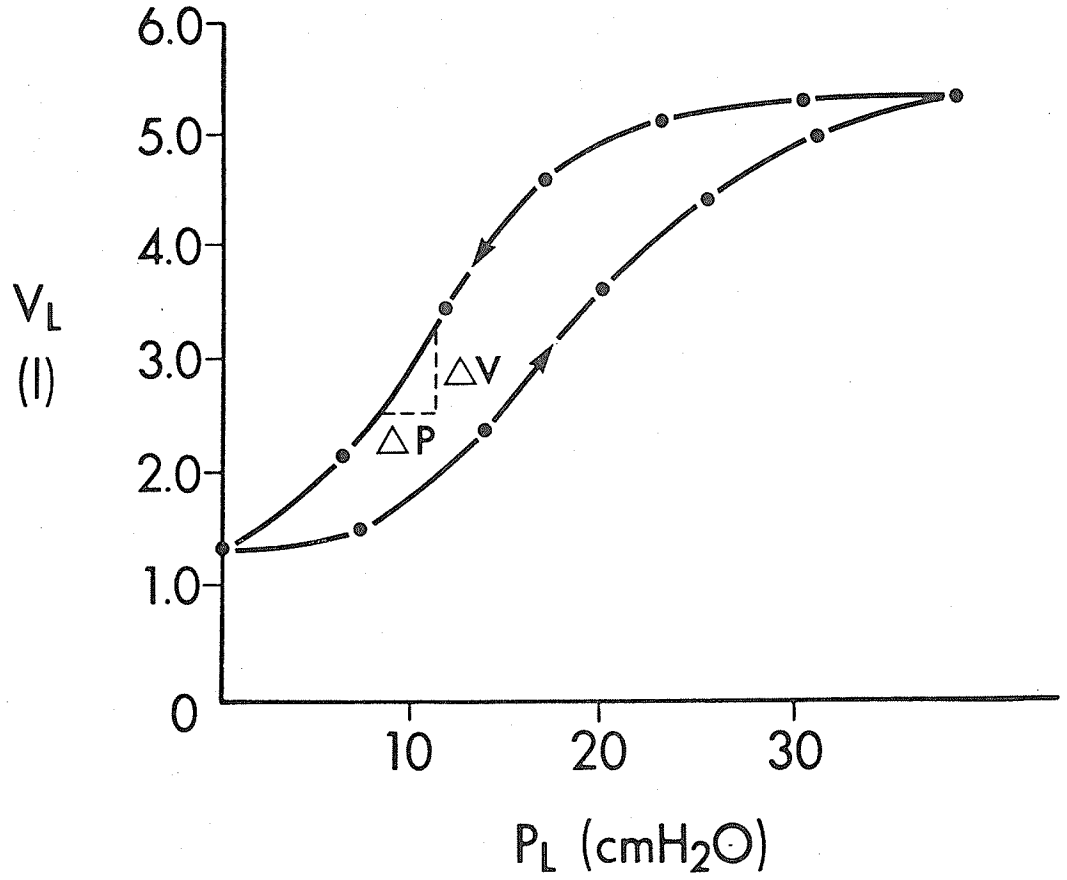


Figure 1: Static pressure-volume relationships in a normal subject. Absolute lung volume is shown on the ordinate, transpulmonary pressure on the abscissa, arrows indicate inflation and deflation limb.

the pressure at the mouth, which in the absence of airflow equals alveolar pressure ($P_A$), minus the pressure in the pleural space (Ppl). As a direct measurement of pleural pressure is both difficult and dangerous in human beings, estimations of Ppl are made using pressure recordings from a balloon placed in the subject's esophagus. Because the esophagus is located between the two pleural spaces, esophageal pressure measurements provide a close approximation of pleural pressure at the level of the balloon in the thorax (2).

The relationship between the elastic recoil of the lung and lung volume is usually demonstrated by the static pressure-volume curve (Figure 1). The transpulmonary pressure ($P_L$) is measured at a variety of lung volumes which are maintained without airflow and this pressure is plotted against the relevant lung volume. The slope of this curve, that is the change in lung volume per unit change in transpulmonary pressure ($\Delta V/\Delta P$) is the compliance (Cst) of the lungs. When describing the pressure-volume relationship of the lung it is more informative to examine the whole pressure-volume curve than to attempt to describe it in terms of compliance. This is because the pressure-volume curve is alinear, and thus values of compliance depend on the degree of lung inflation at which they were measured. Also the pressure-volume curve exhibits hysteresis, that is the paths the lung follows during inflation and deflation are different. Thus at a given transpulmonary pressure the lung volume is greater during deflation than during inflation as is the rate of change of volume with respect to pressure (i.e. Cst). Hence measurements of compliance are influenced by lung volume history as well as by lung volume.

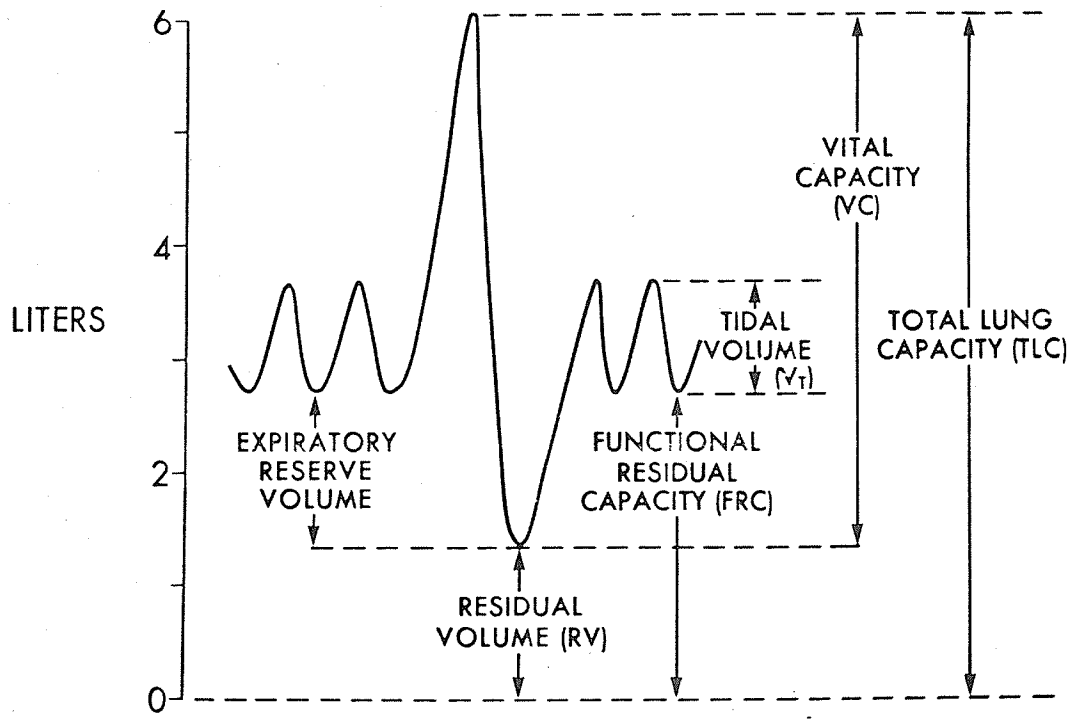


Figure 2: Subdivisions of lung volume as they appear on a spirographic tracing.

The static volumes of the lung are determined by the elastic recoil of the lung and thorax, plus respiratory muscle activity. Functional Residual Capacity (FRC) , the lung volume at normal end-expiration, is the volume at which the inward recoil of the lung is equal and opposite to the outward recoil of the chest wall, and there is no activity of the respiratory muscles. Contraction of the inspiratory muscles shifts the balance point from FRC toward expansion or inspiration. The release of this inspiratory force allows the point of balance to move back toward the original volume, FRC, thus producing an essentially passive expiration. If forceful expiration is required, the muscles of expiration contract, and the balance point now moves toward compression or active expiration. When these muscles relax, the elastic structures of the lung, chest cage and the abdomen restore the equilibrium volume.

A schematic representation of the subdivisions of lung volume is presented in Figure 2. Total lung capacity (TLC) is achieved by taking a maximal inspiration from FRC, and the volume of the inspirate is defined as inspiratory capacity (IC). After a maximal expiration, there remains a residual amount of gas in the lung, termed residual volume (RV), and the volume expired is the expiratory reserve volume (ERV). The vital capacity (VC) is the volume difference between maximal inspiration and maximal expiration, and can be equated to the sum of IC and ERV.

Changes in the subdivisions of the lung volume are due to changes in recoil of lung or chest wall or by changes in muscle power. Increased elastic recoil of the lung or chest wall, or weakness of the inspiratory muscles will reduce IC. The FRC is the sum of the RV and the ERV. Thus, with RV unchanged, a reduction of ERV is analagous to a reduction in

FRC which can be effected in two ways: by increasing the elastic recoil of the lung and/or decreasing the outward recoil of the chest wall.

The static mechanical properties of the lung and chest wall are important determinants of alveolar ventilation, but not the only ones. Ventilation is dynamic, that is air flow is involved. For reasons outlined below, expiratory air flow normally limits maximum ventilation.

Forced expiration is achieved by contraction of the expiratory muscles, raising pleural pressure (Ppl). This change in pleural pressure is transmitted to the alveoli; alveolar pressure ($P_A$) is increased and expiratory flow is achieved. The relationship between $P_A$ and Ppl is defined by the static pressure-volume curve ($P_L = P_A - Ppl$). At high lung volumes the magnitude of expiratory flow is dependent on the magnitude of effort or Ppl. At mid or low lung volumes, however, expiratory flow increases with increased effort only to a point (5, 6). Beyond this point, further increases in effort or Ppl do not result in increases in expiratory flow; flow is effort independent (5,6). This limitation of expiratory flow results from dynamic compression of intrathoracic airways by the pleural pressure surrounding them. This compression implies that pleural pressure exceeds intraluminal pressure in the compressed airway segment. It therefore follows that at some point between the compressed segment and the alveoli, intraluminal pressure equals pleural pressure. This has been defined as the equal pressure point (EPP) (7,8). The factors governing maximum expiratory flow have been analyzed by notationally dividing the airways into two segments, one upstream from EPP and one downstream; maximum flow in each segment at any given lung volume is necessarily the same. Upstream

from the EPP the pressure driving flow is $P_A$-Ppl which equals the static recoil of the lung ($P_L$) and the flow achieved equals $P_L$ divided by the resistance of the airways upstream from the equal pressure point (Rus) (7,8). Thus increases in static recoil of the lung might be expected to increase maximum expiratory flow at low lung volumes, and decreases in flow could be caused by reduced $P_L$ or increased Rus.

Restrictive lung diseases are characterized by increases of lung elastic recoil. Lung volumes, particularly TLC and FRC, are reduced, the pressure-volume curve shifted to the right and maximum expiratory flow at low lung volumes is increased (9,11,19).

An interesting analogue of restrictive lung disease was discovered by Caro, Butler and DuBois (12) who found that restrictive strapping of the chest wall of normal humans resulted in an increase of lung elastic recoil. This increase persisted after the removal of the strap, providing the subject continued to breathe at low lung volumes. It was restored to normal after a full inflation to TLC. Thus the increased recoil was thought to be due to breathing at low lung volumes per se. Stubbs and Hyatt (14) and Sybrecht et al (15) demonstrated similar changes in lung recoil as well as increased maximum expiratory flow during chest strapping. As increased maximum expiratory flow is an indirect indication of increased elastic recoil, independent of measurements of esophageal pressure, this finding eliminated the possibility that the observed increase in elastic recoil was a reflection of distortion of the esophageal balloon during chest strapping. Both groups (14,15) also found that the removal of the chest strap did not cause an immediate decrease of elastic recoil to

values observed in the unstrapped state, but that this occurred once the subject inspired to total lung capacity. This also suggested that the changes in the mechanical properties of the lung could be attributed to low lung volumes as opposed to a specific effect of the chest strap.

Large reductions in functional residual capacity are produced by water immersion to the neck, and providing that the subject does not make maximal inspirations it would be reasonable to expect increases in elastic recoil comparable to that seen during chest strapping. Prefaut et al (16) found that immersion produced only a slight increase in elastic recoil at high lung volumes, while at low lung volumes recoil was less than control. Similarly, maximum expiratory flows were slightly increased at high lung volumes and decreased at low lung volumes. When subjects refrained from taking a deep breath while immersed there was no increase of elastic recoil or increase of maximum expiratory flow. These results were similar to those obtained with immersion without volume restriction. Thus, during immersion, low lung volume breathing did not produce the mechanical changes observed during chest strapping.

The most obvious difference between the two situations is that chest strapping causes a decrease in lung volumes largely by displacement of the rib cage, whereas the volume changes during immersion can be explained by vascular engorgement and cephalad displacement of the diaphragm. In their original work, Caro et al (12) reported that abdominal strapping also increased lung recoil. Mean recoil measured at control FRC increased from 3.5 cm $H_2O$ in the unstrapped state to 6.9 cm $H_2O$ in the abdominal strapped state. These results are misleading, as in the three subjects they studied, elastic recoil was significantly increased in only one. This individual result weighted the mean in favour of in-

creased recoil.

However, Prefaut et al (16) assumed that immersion imposed the same type of restriction as abdominal strapping and on the basis that restriction of the abdomen did increase lung recoil (12) they suggested that the shift of blood into the chest with immersion prevented the increase in recoil due to volume restriction.

If Caro et al (12) were correct in their observation that restriction of the abdomen increases recoil, then, blood shifts into the chest during immersion are important. Alternatively, if this observation was incorrect, and abdominal strapping was not the same as chest strapping, the implication would be that not all forms of volume restriction are associated with an increase of elastic recoil.

This study tested both hypotheses by initially examining the effects of immersion on the lung mechanics of strapped subjects, and then further comparing the effects of strapping the abdomen with those of strapping the rib cage.

# II. EXPERIMENTAL DESIGN

A. Methods

B. Calibration

C. Statistics

A. Methods

Eight healthy male volunteers (4 smokers) aged 22-44 years were studied under three conditions: unstrapped, with rib cage restriction, and with abdomen restricted. In addition 4 subjects were studied during a combination of rib cage restriction and immersion to the neck in water.

The rib cage was restricted by a pneumatic cuff secured to the anterior rib cage by an inextensible canvas corset which was tightened manually while the subject held his breath at RV. The pneumatic cuff was then inflated and maintained at a constant pressure (not less than 40 mmHg) throughout the study (Figure 3a). Abdominal restriction was achieved initially by applying the pneumatic cuff to the abdomen. The resulting reductions in lung volumes were less than those achieved during rib cage restriction. With abdominal restriction TLC was 80% of control while rib cage restriction reduced TLC to 60% control. Further studies showed that a 20% reduction in TLC achieved by rib cage restriction was not sufficient to produce changes in the elastic properties of the lung. A detailed explanation of these preliminary experiments, together with the results, is presented in the appendix. Thus, in order to reduce lung volumes during abdominal strapping to the same level observed during chest strapping, the following method was employed. Rib cage expansion was prevented by fastening the canvas corset around the chest while the subject held his breath at FRC. The subject then breath held at RV while the pneumatic cuff was secured tightly to the anterior abdomen with two tensor bandages. The cuff was inflated and maintained at not less than 60 mmHg throughout the course of the study (Figure 3b).

In each study of each subject lung volumes, deflation static pressure-volume (P-V) curves of the lung, maximum expiratory flow volume (MEFV) curves and closing volumes were measured unstrapped (control)

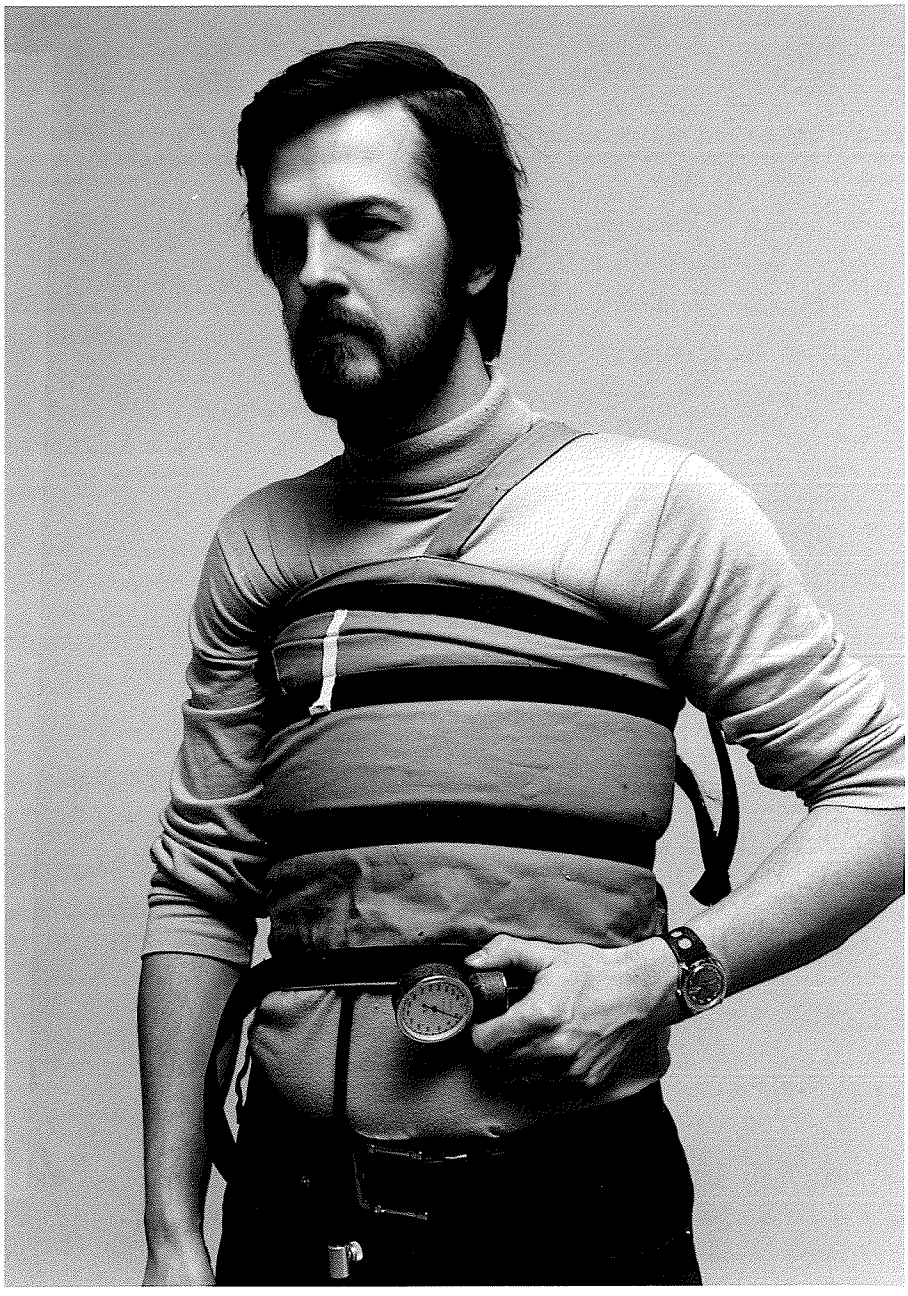

Figure 3a: Photograph of J.F. during rib cage restriction.

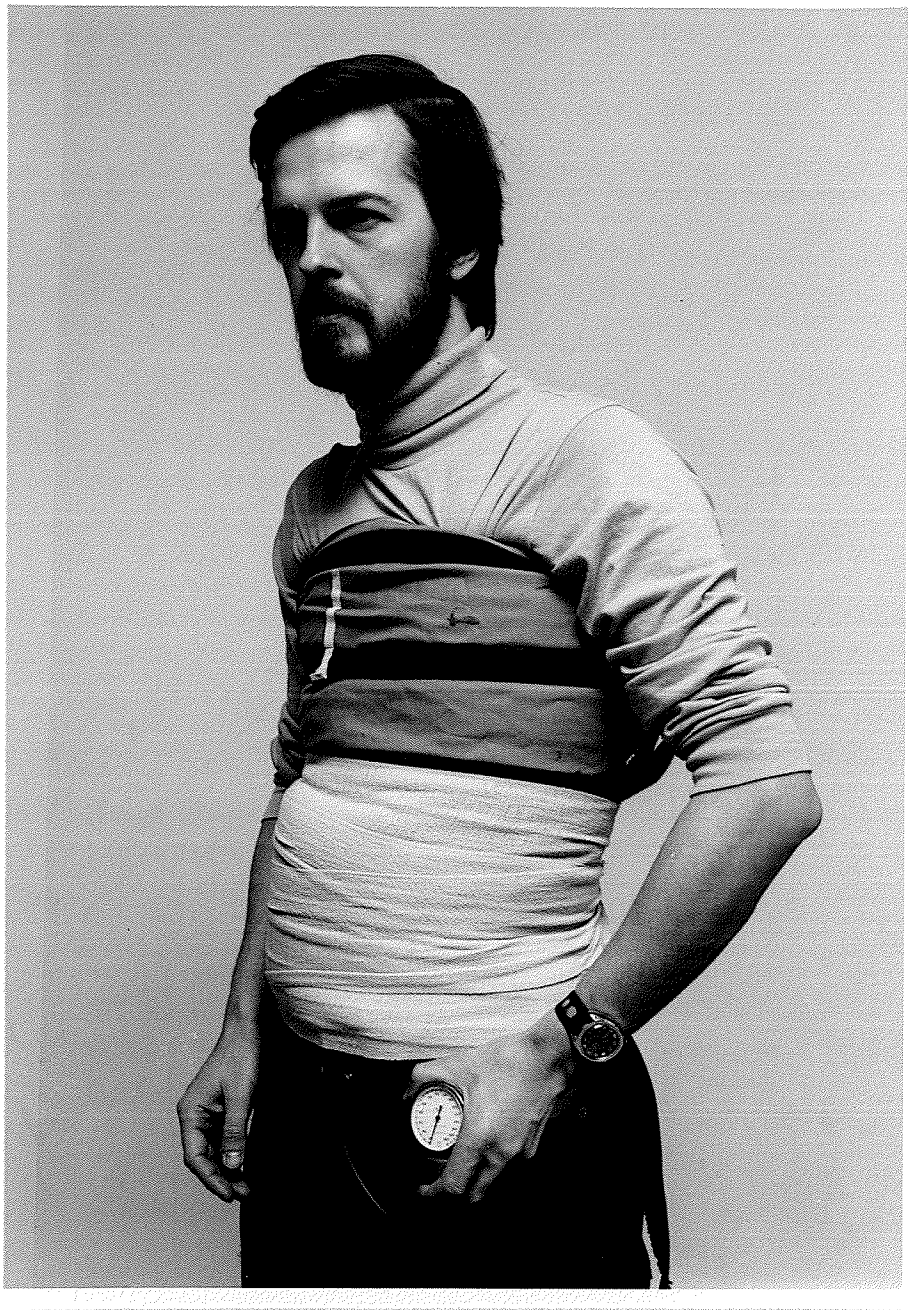

Figure 3b: Photograph of subject J.F. during abdominal restriction.

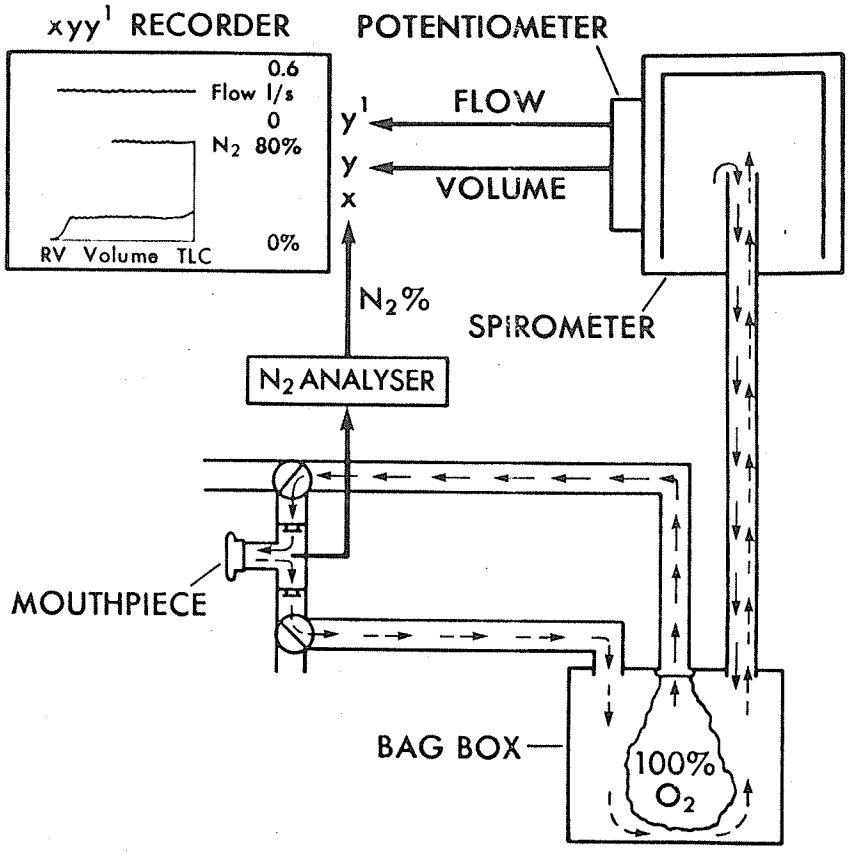


Figure 4: Schematic representation of apparatus used in the single breath $N_2$ technique. ——→ denotes inspiration, - - - - → expiration. See text for details.

and with rib cage and abdominal restriction. During the unstrapped state, maneuvers generating deflation P-V curves and MEFV curves were initiated at 60% vital capacity (partials) as well as at total lung capacity (controls). Thus, data acquired during restriction could be compared to maneuvers in the unstrapped state which began at the same lung volume. Four subjects were also immersed to the neck in water while their rib cage was restricted. While measurements were made in this restricted immersed state, the water temperature was continually maintained at comfortable levels.

Subjects were studied in the seated position. The VC and ERV were measured with a rolling-seal volume-displacement spirometer connected via a potentiator to a Hewlett Packard two-channel strip recorder. All volumes were corrected to body temperature, pressure, saturated with water vapour (BTPS). During each study, measurements were made in triplicate and the means are reported.

Lung volumes were measured by the single breath nitrogen test (17) as modified by Anthonisen and associates (18). The equipment circuit is shown in Figure 4. Subjects, in the seated position, were fitted with a nose clip and instructed to breath through a mouth piece attached to a non-rebreathing Hans Rudolph valve. Both the inspiratory and expiratory lines were connected to two-way taps. The inspiratory tap allowed the subject to be switched either to room air or to a bag containing $0_2$ which was in an air tight box. Changes in box and therefore bag volume were measured by the rolling-seal spirometer. The tap in the expiratory line enabled the subject to exhale either into the air or back into the box and thus into the spirometer. Expired $N_2$

was measured continuously at the mouth with a Hewlett Packard 47302A $N_2$ meter. Flow (volume electronically differentiated with respect to time) also was monitored. While still breathing air the subject was asked to inspired maximally twice, exhale the second breath completely to RV, during which both inspiratory and expiratory taps were turned to connect the subject to the bag box. From RV the subject then inspired 100% $0_2$ to TLC, and then expired slowly ($\leq$0.5 l/s) to RV while volume was recorded on the X axis and flow and $N_2$ concentration were recorded on the Y axis of an XYY' recorder. This procedure was repeated at least three times, with an appropriate delay between repetitions to allow re-establishment of normal alveolar gas composition. Vital capacities between runs varied less than 5%, and within each run inspired and expired vital capacities did not differ by more than 5%. RV was calculated as follows, using the method of Buist and Ross (19). Total lung capacity first is estimated using the alveolar dilution equation. This is derived using the following reasoning.

After the first expiration to residual volume (RV) the amount of nitrogen in the subject's lungs is $V_{N2}$ = RV ($\overline{F}_{AN_2}$) where $\overline{F}_{AN_2}$ is the normal alveolar nitrogen concentration when breathing room air. After the subject inspires pure oxygen to total lung capacity (TLC) this same amount of $N_2$ can be expressed in two ways.

(1) $V_{N_2} = (TLC - VC_I)\ \overline{F}_{AN_2}$

- where $VC_I$ is the inspired volume, thus RV = TLC - $VC_I$

or

(2) $V_{N_2} = (TLC - V_D)\ \overline{F}_A{}'_{N_2}$

- where $V_D$ is the dead space measured according to Fowler (20)
- TLC - $V_D$ is the effective alveolar volume
- $\overline{F}_A{}'_{N_2}$ is the mean alveolar concentration of the maximally

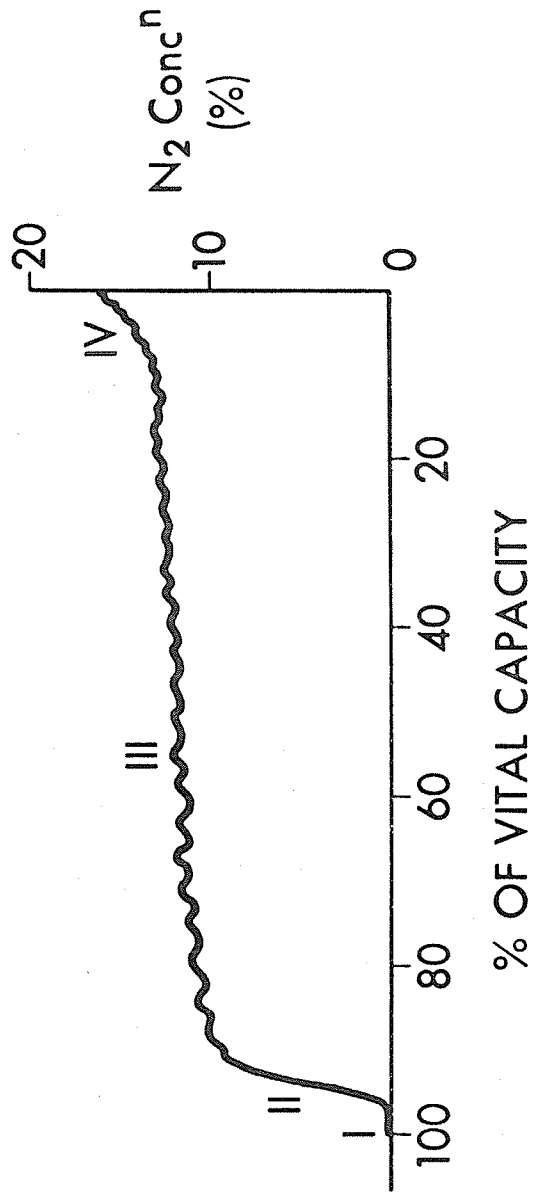


Figure 5: Concentration of $N_2$ in the expired gas, measured at the mouth, as a function of expired volume. Phases I, II, III, IV are discussed in the text.

diluted nitrogen.

The alveolar dilution equation is now obtained by equating (1) and (2) and simplifying.

$$(TLC - VC_I)\ \bar{F}_{AN_2} = (TLC - V_D)\ \bar{F}_A{}'_{N_2}$$

or

$$TLC = \frac{VC_I\ \bar{F}_{AN_2} - V_D\ \bar{F}_A{}'_{N_2}}{\bar{F}_{AN_2} - \bar{F}_A{}'_{N_2}}$$

$\bar{F}_A{}'_{N_2}$ is estimated from the plot of $N_2$ against volume during the second VC expiration. The area under this curve is equal to the amount of nitrogen expired, and correcting for dead space, the mean concentration of the alveoli is then $F_A{}'_{N_2} = \frac{\text{area}}{\overline{VC}-\overline{V}_D}$ where VC is the volume of the expired vital capacity.

Residual volume was calculated by subtracting from this estimated TLC the larger recorded vital capacity (inspired $VC_I$ or expired $VC_E$). Values for RV allowed computation of TLC and FRC.

The expiratory curve obtained during the single breath nitrogen test can be divided into four phases (Figure 5). Phase I is dead space gas, that is pure $0_2$ expired from the larger conducting airways; Phase II reflects the transitional zone or mixed alveolar and dead space gas; Phase III and Phase IV combined are representative of the alveolar gas. Phase III is commonly termed the alveolar plateau, and the junction between Phase III and IV is termed Closing Volume (21). The closing volume curves in this study were analyzed in terms of slope of Phase III, which was taken as the slope of the line of best visual fit drawn at volumes less than 70% VC. This slope reflects the variation of RV/TLC among lung units and the tendency of units with differing RV/TLC to empty sequentially (17,22). We also measured closing capacity, defined

as closing volume plus RV. The closing capacity is thought to be the volume at which dependent lung units undergo airway closure (23).

Pleural pressure was estimated by esophageal pressure which was measured by a 10 cm long balloon, inflated with 1 ml air according to the method of Milic-Emili and associates (2). This was positioned in the upper third of the esophagus in an area free from artifacts as tested by head motion (24). Mouth pressure was obtained from a lateral tap at the mouth piece. Transpulmonary pressure, expressed as mouth pressure minus pleural pressure, was monitored by connecting pleural pressure and mouth pressure to either side of a differential pressure transducer (Validyne DP15). An occluder was placed just distal to the mouth pressure tap and was used to interrupt air flow intermittently while the patient expired into the spirometer. Measurements of volume and transpulmonary pressure during these interruptions (Flow = 0) allowed computation of the static P-V curve. Transpulmonary pressure and lung volume were recorded simultaneously on a Hewlett Packard 2 channel strip recorder and records were analysed manually. Subjects were asked to breathe quietly on the mouthpiece until their FRC stablized. They then took a maximal inspiration to TLC, and slowly exhaled to RV while being interrupted, and then inspired back to TLC.

Partial P-V curves were obtained by a similar maneuver which differed only in that the deflation curve was initiated at 60% control VC rather than TLC. Four subjects, after release of rib cage and abdominal restriction voluntarily maintained their end tidal volume at less than 60% of control VC. Static pressure-volume data was thus obtained during an expiration from 60% VC, and inspiration to TLC and a second deflation from full inspiration.

To ensure that P-V characteristics of the lung returned to normal

a control P-V curve was obtained in all subjects after each restrictive procedure.

MEFV curves were determined by plotting flow (the derivative of volume with respect to time) against volume on a rapid response XY recorder. Volume was measured with the rolling-seal volume-displacement spirometer. After taking a full inspiration to TLC, subjects were asked to exhale forcibly to RV. For curves to be considered reproducible, forced vital capacities had to be within 5% of each other and peak flows within 10%. Partial MEFV curves were initiated at 60% control VC after slow inspiration from RV.

B. Calibrations

The following calibrations preceeded each study.

1. Rolling-seal volume-displacement spirometer

(i) Volume

(a) The linearity of the spirometer recording system was checked using fixed electrical signals.

(b) With the temperature compensation dial at 37°C (i.e. no compensation) a 1 liter calibration syringe was used to add 1 liter of gas to the spirometer. Adjustments were made such that a pre-determined deflection represented 1 liter on both the XYY' recorder and the Hewlett-Packard two channel strip recorder. This was confirmed by consecutive 1 liter inflations and deflations.

(c) The temperature compensation was then checked by turning the dial to 21°C (i.e. maximum compensation). The subsequent addition of 1 liter of gas should be represented by a deflection equal to 1.19 liters.

(ii) Flow

Once volume calibration had been concluded, a constant flow of gas was added to the spirometer while (i) flow was recorded on the XYY' recorder, (ii) volume was recorded on the Hewlett Packard strip recorder running at a known speed. Flow was calculated from the strip recorder record and the readout on the XYY' recorder checked. In case of discrepancy the necessary adjustments were made.

2. Validyne Pressure Transducer

(a) With no input, the zero was set on the Hewlett Packard recorder;

(b) Using a water manometer, 20 cm $H_2O$ positive pressure was added to the positive side of the transducer. The recorder was set such

that 1 cm represented 10 cm $H_2O$.

(c) Using a water manometer, 20 cm $H_2O$ negative pressure was added to the positive side of the transducer to ensure a deflection of -1 cm represented -10 cm $H_2O$.

(d) This procedure was repeated for the negative side of the transducer.

(e) The zero was checked before very run.

3. Nitrogen Meter

The linearity of this meter was checked initially to ensure that it was linear over the range being used. This was done using a proportional gas mixing pump (Wosthoff type S18/3a) which prepares gas mixtures containing precisely known concentrations of oxygen and nitrogen.

Before each study the meter was zeroed with 100% $O_2$. The needle valve was then adjusted to ensure that the meter readout was maximum. Compressed air, humidified by bubbling through water, was sampled until the nitrogen meter gave a steady readout. This was then adjusted to 80% $N_2$. The XYY' recorder was subsequently calibrated such that 2 cm represented 10% $N_2$.

Throughout the study the zero was checked repeatedly.

Validation of the Accuracy of the Residual Volume Measurements using the Single Breath $N_2$ Technique.

In one subject measurements of residual volume during rib cage restriction were made using both the single breath $N_2$ technique and the Steady-state Helium Dilution technique. No significant difference was found between the two methods.

C. Statistics

The statistical methods used to analyze the results of these experiments are outlined in the Appendix.

# III. EXPERIMENTAL RESULTS

Immersion Studies

Rib cage restriction in four subjects significantly decreased lung volumes from those observed in the control state. Mean TLC was 57% of control ($p < 0.05$) while mean FRC was 65% of control ($p < 0.05$). When restricted subjects were immersed TLC was 59% control while FRC decreased to 50% of control. The change in FRC with immersion was not statistically different. RV was not changed. Volumes are listed in Table 1.

In all subjects rib cage restriction shifted the static deflation P-V curve to the right of control and subsequent immersion showed no further systematic change. The deflation P-V curves for all subjects are shown in Figure 6.

Peak maximum expiratory flow was decreased during chest restriction in all subjects, but once flow became effort independent, maximum expiratory flow ($\dot{V}max$) was consistently greater than control. Subsequent immersion increased $\dot{V}max$ even further in one subject, but in the remaining three subjects there was no change. MEFV curves for all subjects are presented in Figure 7.

Table 2 shows measurements of elastic recoil pressure ($P_L$) at 50% control TLC, static compliance (Cst) over the interval 50% control TLC + 0.5 liters, and $\dot{V}max$ at 40% control TLC. Mean $P_L$ increased from 4.4 cm $H_2O$ during control to 12.9 cm $H_2O$ during chest restriction ($p < 0.05$). Immersion while strapped gave a mean recoil pressure of 13.0 cm $H_2O$, not significantly different from that observed during chest strapping alone. Mean Cst was 0.31 L/cm $H_2O$ in the unstrapped state, and decreased significantly to 0.11 L/cm $H_2O$ during rib cage restriction ($p < 0.05$). Subsequent immersion did not effect the reduction of Cst seen during rib cage restriction. Mean $\dot{V}max$ at 40% control TLC

TABLE 1: Effect of rib cage restriction and immersion on subdivisions of lung volume.

| | TLC, (liters) | | | FRC, (liters) | | | RV, (liters) | | |
|---|---|---|---|---|---|---|---|---|---|
| Subject | C | RC | RCI | C | RC | RCI | C | RC | RCI |
| N.A. | 8.0 | 4.3 | 4.6 | 4.2 | 2.2 | 2.1 | 1.9 | 1.5 | 1.9 |
| G.D. | 5.9 | 3.8 | 4.0 | 2.7 | 1.9 | 1.5 | 1.1 | 1.0 | 1.1 |
| G.F. | 6.3 | 3.6 | 4.1 | 2.9 | 2.1 | 1.6 | 1.2 | 1.3 | 1.2 |
| D.G. | 6.8 | 4.0 | 3.4 | 3.6 | 2.4 | 1.7 | 1.3 | 1.2 | 1.1 |
| Mean | 6.8 | 3.9 | 4.0 | 3.4 | 2.2 | 1.7 | 1.4 | 1.3 | 1.3 |
| S.D. | 0.92 | 0.31 | 0.49 | 0.69 | 0.21 | 0.26 | 0.36 | 0.21 | 0.39 |
| S.E.M. | 0.46 | 0.16 | 0.25 | 0.34 | 0.10 | 0.13 | 0.18 | 0.10 | 0.19 |

C = control, RC = rib cage restriction, RCI = immersion with rib cage restriction.

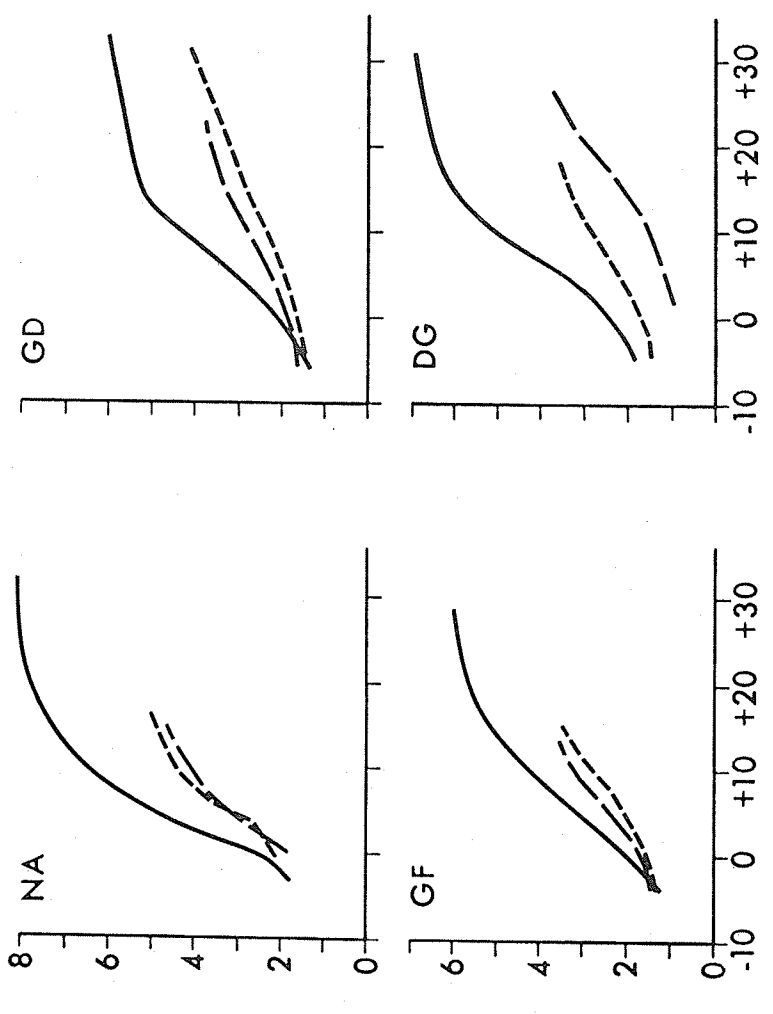


Figure 6: Static pressure-volume curves during deflation in four subjects under control conditions (——), during rib cage restriction(— — —), and during rib cage restriction while immersed (- - - - - -). Absolute lung volume is shown on the ordinate, transpulmonary pressure on the abscissa.

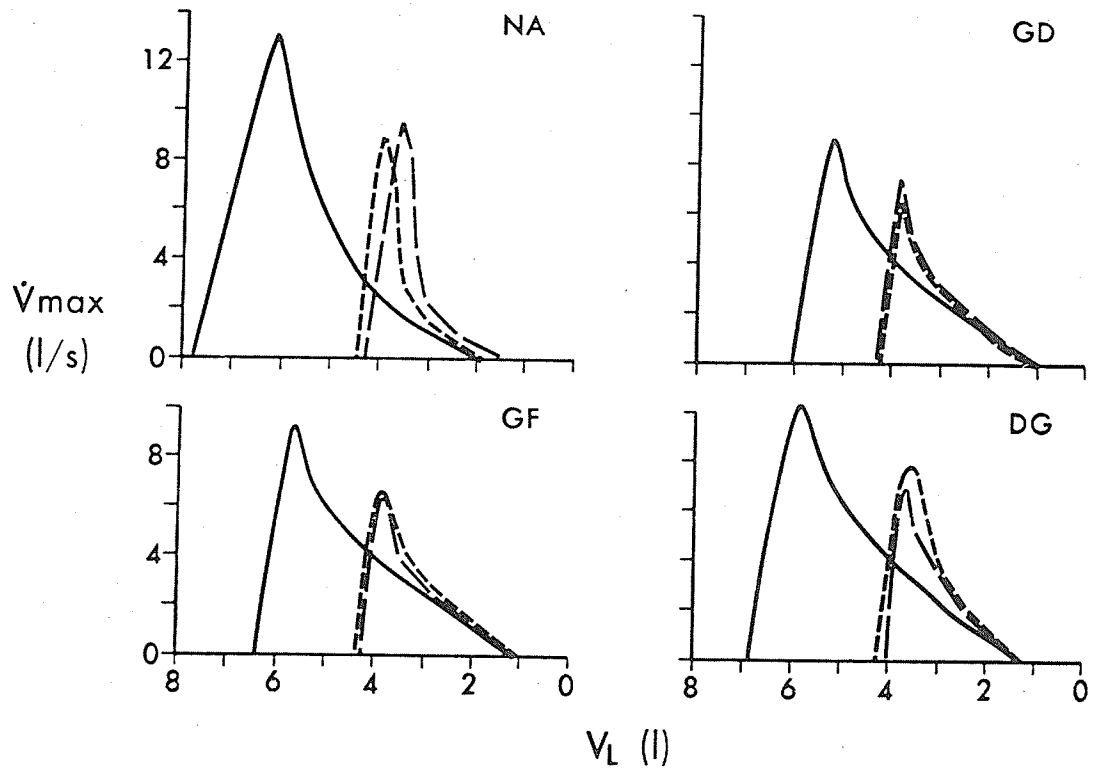


Figure 7: Maximum expiratory flow-volume curves in four subjects under control conditions (——), during rib cage restriction (— — —), and during rib cage restriction while immersed (------). Maximum expiratory flow (V̇max) is shown on the ordinate, absolute lung volume on the abscissa.

TABLE 2: Effects of rib cage restriction and immersion on transpulmonary pressure, maximum expiratory flow and static compliance.

| | $P_L$ at 50% TLC, ($cmH_2O$) | | | MEF at 40% TLC, (1/s) | | | Cst at 50% TLC, ($1/cmH_2O$) | | |
|---|---|---|---|---|---|---|---|---|---|
| Subject | C | RC | RCI | C | RC | RCI | C | RC | RCI |
| N.A. | 2.6 | 8.7 | 8.0 | 1.2 | 4.0 | 1.8 | 0.43 | 0.11 | 0.20 |
| G.D. | 4.8 | 10.7 | 16.5 | 1.8 | 2.2 | 1.9 | 0.25 | 0.09 | 0.08 |
| G.F. | 5.4 | 9.0 | 11.7 | 1.6 | 1.9 | 2.0 | 0.25 | 0.11 | 0.15 |
| D.G. | 5.0 | 23.0 | 15.6 | 1.9 | 2.6 | 2.7 | 0.31 | 0.11 | 0.07 |
| Mean | 4.4 | 12.9 | 13.0 | 1.6 | 2.7 | 2.1 | 0.31 | 0.11 | 0.13 |
| S.D. | 1.25 | 6.82 | 3.90 | 0.31 | 0.93 | 0.41 | 0.085 | 0.010 | 0.061 |
| S.E.M. | 0.63 | 3.41 | 1.95 | 0.15 | 0.46 | 0.20 | 0.042 | 0.005 | 0.031 |

C = control, RC = rib cage restriction, RCI = immersion with rib cage restriction.

TABLE 3: Effects of rib cage restriction and abdominal restriction on the subdivisions of lung volume.

| | T.L.C., (liters) | | | | F.R.C., (liters) | | | | R.V., (liters) | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Subject | C | P | RC | ABD | C | P | RC | ABD | C | P | RC | ABD |
| J.F. | 6.40 | 4.05 | 3.64 | 3.84 | 3.00 | 3.00 | 1.74 | 1.91 | 1.40 | 1.40 | 1.34 | 1.44 |
| P.W. | 7.13 | 4.33 | 3.71 | 4.32 | 3.63 | 3.63 | 2.08 | 2.22 | 1.13 | 1.13 | 1.31 | 1.42 |
| W.G. | 6.88 | 4.08 | 4.25 | 3.81 | 2.45 | 2.45 | 1.85 | 1.51 | 1.08 | 1.08 | 1.05 | 1.01 |
| B.M. | 7.30 | 4.32 | 3.97 | 4.50 | 3.37 | 3.37 | 1.77 | 1.83 | 1.22 | 1.22 | 1.17 | 1.10 |
| N.A. | 8.03 | 4.58 | 4.31 | 4.00 | 4.20 | 4.20 | 2.20 | 1.80 | 1.90 | 1.90 | 1.50 | 1.30 |
| G.D. | 5.90 | 3.55 | 3.75 | 3.70 | 2.70 | 2.70 | 1.90 | 1.50 | 1.10 | 1.10 | 1.00 | 1.00 |
| G.F. | 6.32 | 3.33 | 3.60 | 4.50 | 2.90 | 2.90 | 2.10 | 1.70 | 1.20 | 1.20 | 1.30 | 1.10 |
| D.G. | 6.77 | 4.00 | 4.00 | 4.10 | 3.60 | 3.60 | 2.40 | 1.80 | 1.30 | 1.30 | 1.20 | 1.10 |
| Mean | 6.84 | 4.03 | 3.90 | 4.09 | 3.23 | 3.23 | 2.01 | 1.78 | 1.29 | 1.29 | 1.23 | 1.18 |
| S.D. | 0.65 | 0.41 | 0.27 | 0.31 | 0.57 | 0.57 | 0.23 | 0.23 | 0.27 | 0.27 | 0.16 | 0.18 |
| S.E.M. | 0.23 | 0.15 | 0.10 | 0.11 | 0.20 | 0.20 | 0.08 | 0.08 | 0.09 | 0.09 | 0.06 | 0.06 |

C = control, P = partial, RC = rib cage restriction, ABD = abdominal restriction.

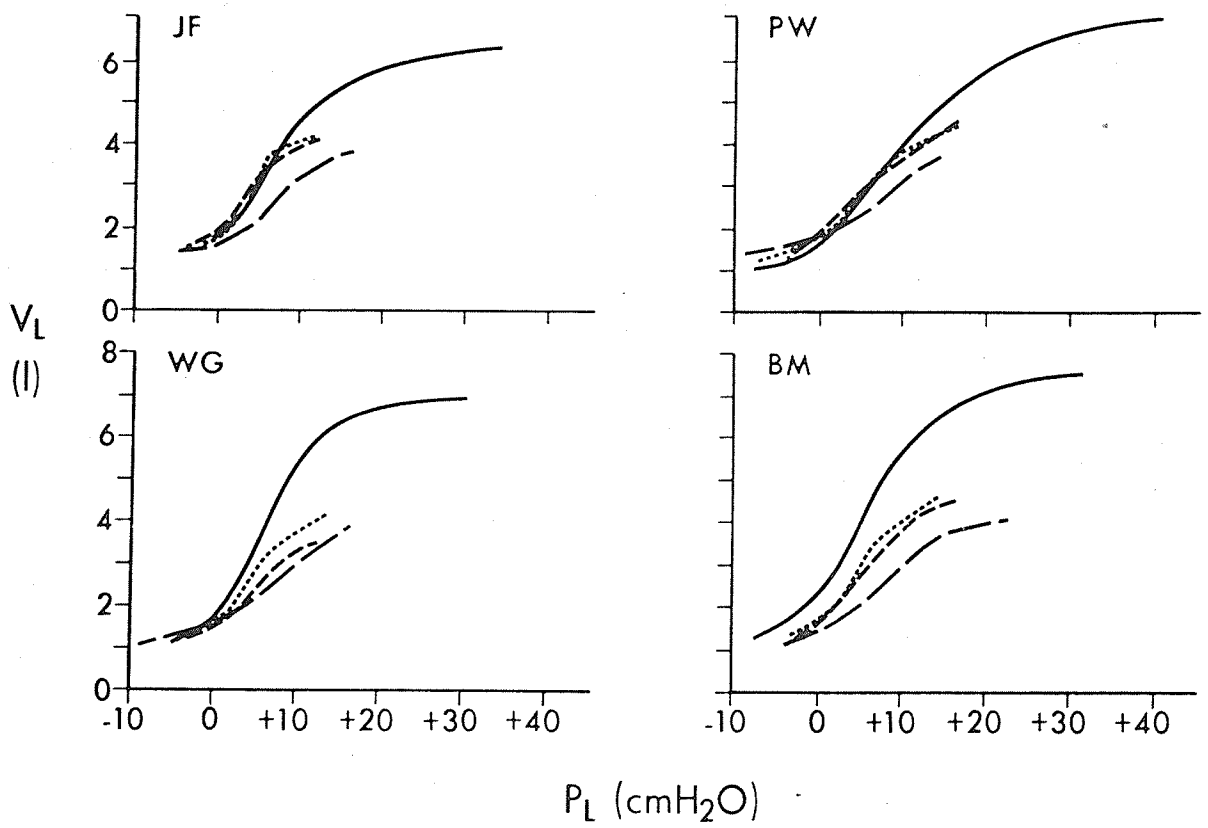


Figure 8a: Static pressure-volume curves during deflation in four subjects under control conditions (——), a partial maneuver (-------), during rib cage restriction (— — —) and during abdominal restriction (– – –). Absolute lung volume is shown on the ordinate, transpulmonary pressure on the abscissa.

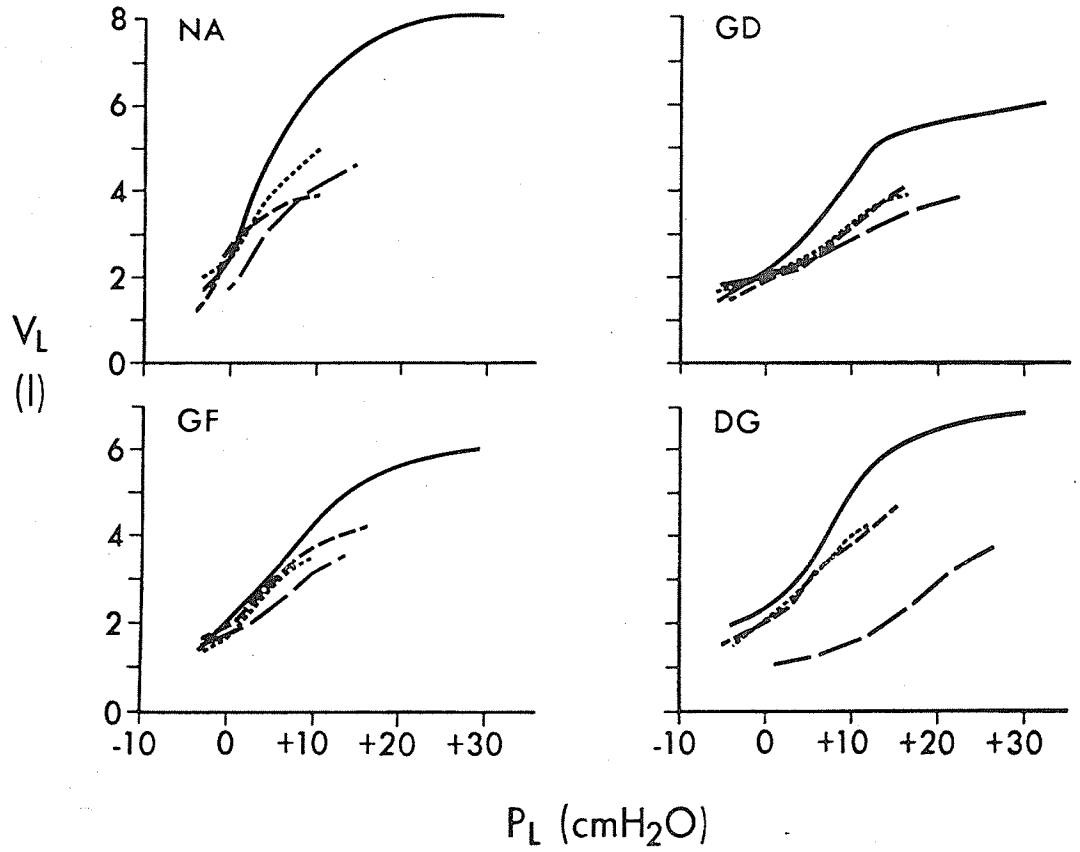


Figure 8b: Static pressure-volume curves during deflation in four subjects under control conditions (———), a partial maneuver (-------), during rib cage restriction (— — —) and during abdominal restriction (— — —). Absolute lung volume is shown on the ordinate, transpulmonary pressure on the abscissa.

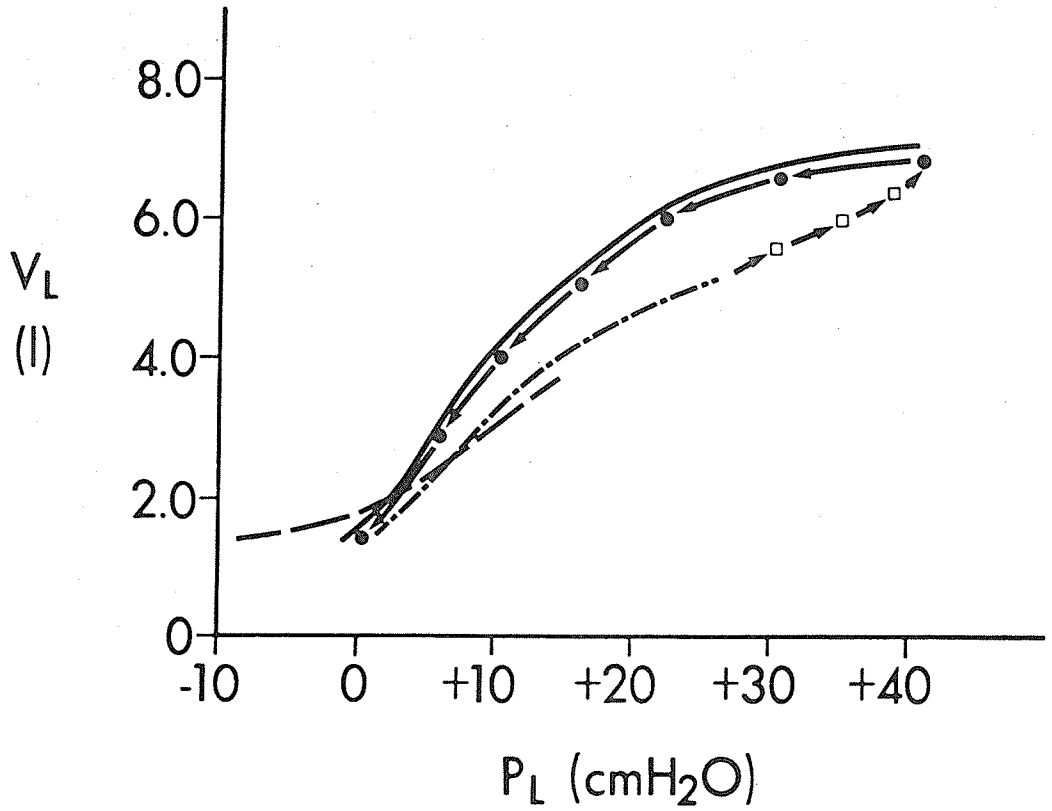


Figure 9a: Static pressure-volume curves during deflation in subject P.W. under control conditions (———), and during rib cage restriction (— — —). Following release of restriction, a partial static deflation pressure-volume curve (— - — - —) was obtained. After a full inspiration to TLC (→□ →□ ) the static deflation pressure-volume curve was returned to normal (→● →● ). Absolute lung volume was plotted on the ordinate, transpulmonary pressure on the abscissa.

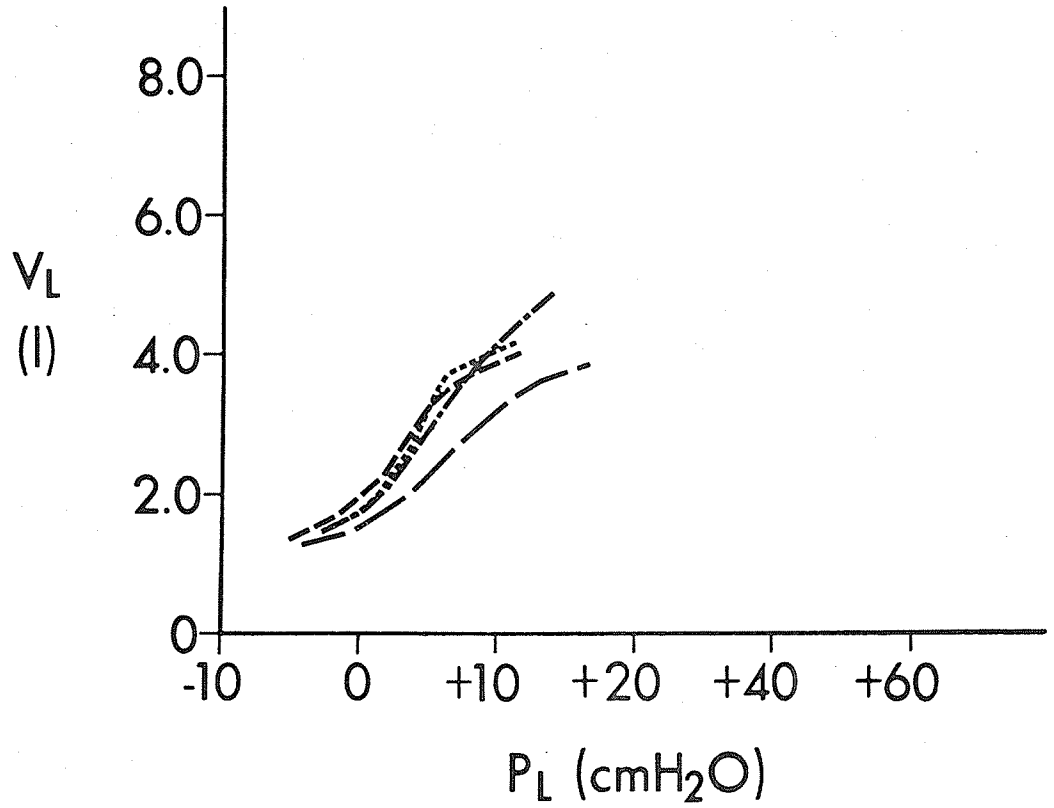


Figure 9b: Static pressure-volume curves during deflation in subject J.F. during a partial maneuver prior to restriction (----------), and during a partial maneuver immediately following the removal of the abdominal restriction (— - — - —). Static deflation pressure-volume curves during rib cage restriction (— — —) and during abdominal restriction (— — — —) are shown for the same subject. Absolute lung volume is shown on the ordinate, transpulmonary pressure on the abscissa.

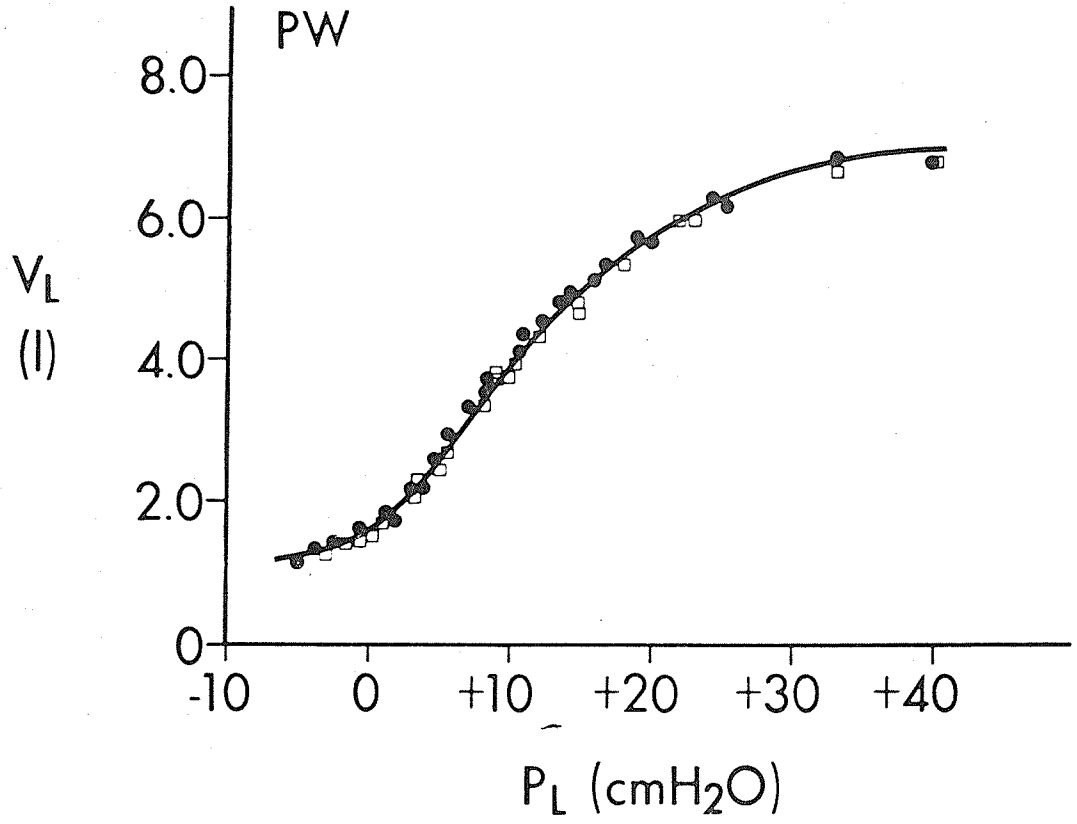


Figure 10: Static pressure-volume curves during deflation in subject P.W. under control, unstrapped conditions. The solid line was obtained prior to any form of restriction. Following release of either form of restriction and a full inspiration to TLC, the static deflation pressure-volume relationship returned to normal. Solid circles represent points obtained following release of rib cage restriction; open squares represent points obtained following release of abdominal restriction.

increased over control during rib cage restriction and was not changed by subsequent immersion.

Abdominal Restriction compared to Rib Cage Restriction

The effects of both forms of restriction on the subdivisions of lung volume are listed in Table 3. Rib cage restriction produced an overall reduction in TLC of 43% and in FRC of 38%. Similar decreases were seen with abdominal restriction (40% and 45% respectively). The TLC and FRC during both forms of restriction were significantly less than the control values ($p < 0.05$). Neither form of restriction significantly altered RV.

In all subjects rib cage restriction shifted the static deflation P-V curve to the right of both the control and partial curves (Figures 8a and 8b). In six of the eight subjects static deflation curves during abdominal restriction were indistinguishable from those obtained during a partial maneuver (Figures 8a and 8b). The remaining two did show a right shift of their deflation curve during abdominal strapping, but this was less than that observed during rib cage restriction.

As reported by previous authors (12,14) the increase in elastic recoil persisted after removal of either the chest corset or the abdominal binder provided the subject refrained from taking a large inspiration (Figure 9), but following a full inspiration to TLC, elastic recoil was restored to normal. Indeed, following either form of restriction, in all subjects an inspiration to TLC produced a return of the static deflation curve to that observed before restriction (Figure 10).

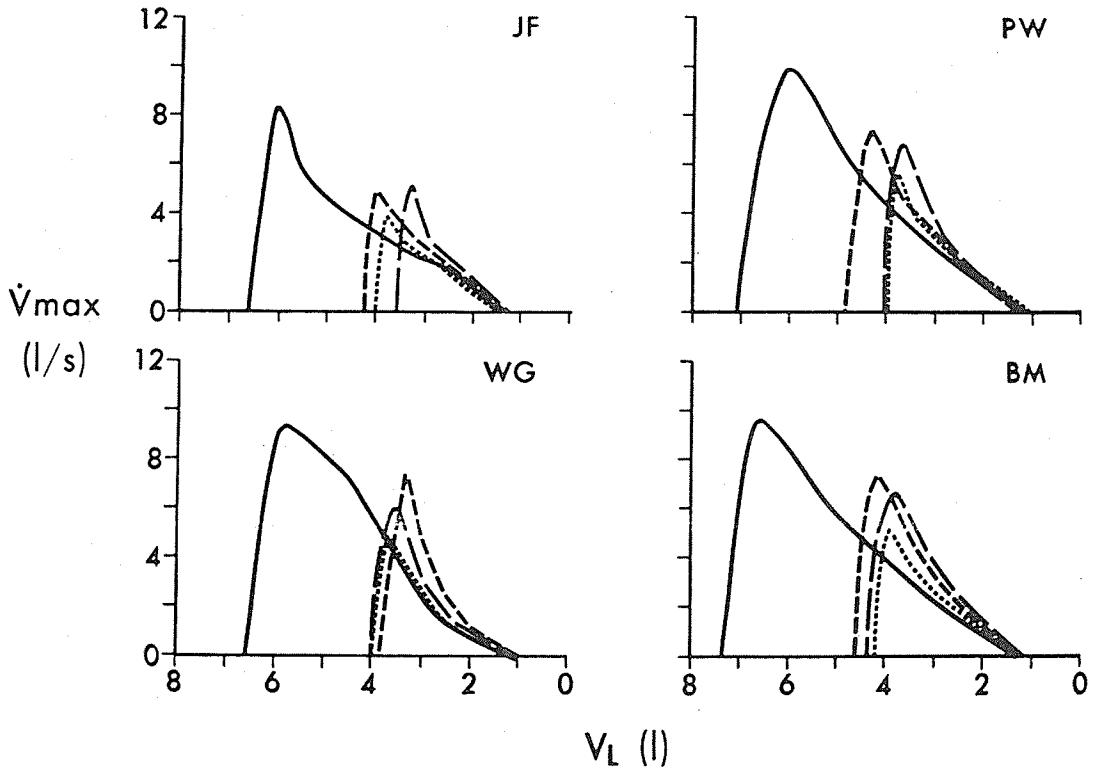


Figure 11a: Maximal expiratory flow-volume curves in four subjects under control conditions (———), a partial maneuver (------), during rib cage restriction (— — —) and during abdominal restriction (— — — —). Maximum expiratory flow is shown on the ordinate, absolute lung volume on the abscissa.

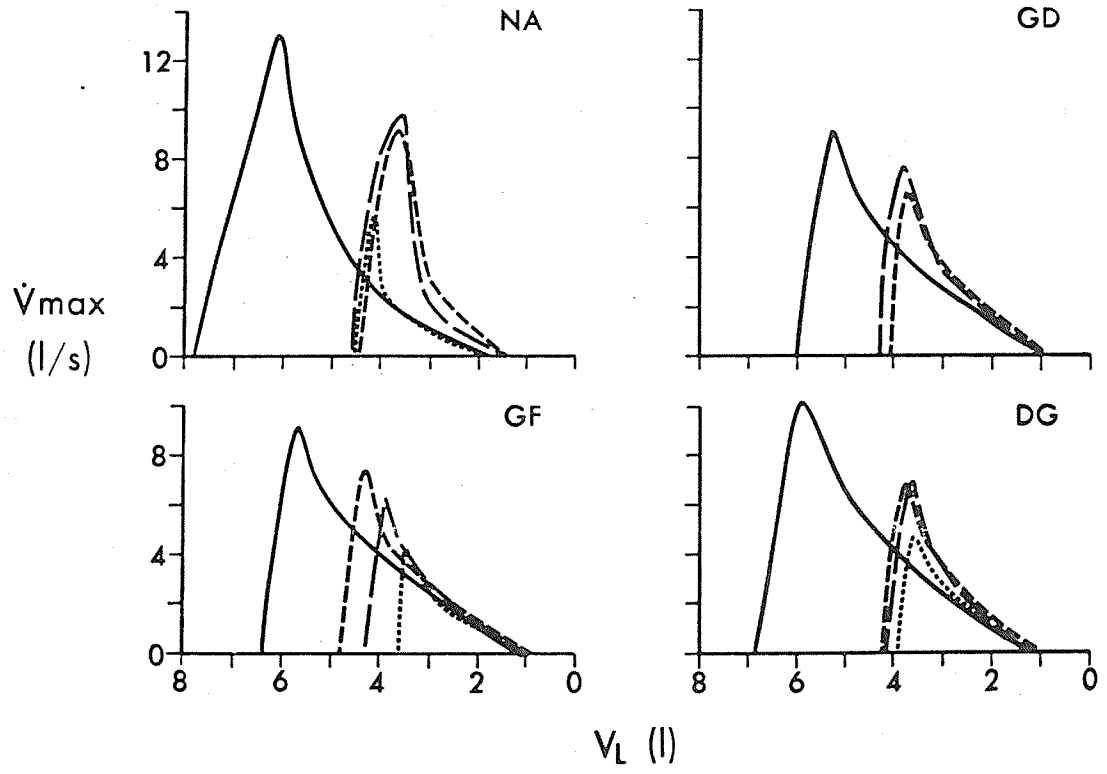


Figure 11b: Maximal expiratory flow-volume curves in four subjects under control conditions (———), a partial maneuver (-------), during rib cage restriction (— — —), and during abdominal restriction (— — — —). Maximum expiratory flow is shown on the ordinate, absolute lung volume on the abscissa.

TABLE 4: Effects of rib cage restriction and abdominal restriction on transpulmonary pressure, maximum expiratory flow and static compliance.

| | $P_L$ at 50% TLC, ($cmH_2O$) | | | | MEF at 40% TLC, (1/s) | | | | Cst at 50% TLC, ($1/cmH_2O$) | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | C | P | RC | ABD | C | P | RC | ABD | C | P | RC | ABD |
| J.F. | 5.8 | 5.5 | 10.3 | 5.5 | 1.5 | 1.5 | 2.0 | 1.8 | 0.22 | 0.25 | 0.11 | 0.19 |
| P.W. | 8.3 | 8.9 | 13.7 | 9.5 | 2.3 | 2.8 | 2.9 | 2.6 | 0.20 | 0.14 | 0.12 | 0.13 |
| W.G. | *3.5 | *4.5 | *8.8 | *7.0 | 1.9 | 1.7 | 2.5 | 3.3 | *0.42 | *0.20 | *0.18 | *0.14 |
| B.M. | 3.7 | 6.5 | 11.7 | 7.7 | 2.1 | 2.5 | 3.3 | 3.0 | 0.33 | 0.25 | 0.14 | 0.20 |
| N.A. | *1.2 | *2.1 | *4.7 | *3.0 | 1.2 | 1.1 | 4.0 | 4.4 | *0.56 | *0.31 | *0.20 | *0.14 |
| G.D. | 4.8 | 8.3 | 10.7 | 9.2 | 1.8 | | 2.2 | 2.1 | 0.25 | 0.14 | 0.09 | 0.16 |
| G.F. | 5.4 | 6.7 | 9.0 | 6.7 | 1.6 | 1.4 | 1.9 | 1.8 | 0.25 | 0.15 | 0.11 | 0.14 |
| D.G. | 5.0 | 7.3 | 23.0 | 7.4 | 1.9 | 2.0 | 2.6 | 2.8 | 0.31 | 0.22 | 0.11 | 0.17 |
| Mean | 4.7 | 6.2 | 11.5 | 7.0 | 1.8 | 1.9 | 2.7 | 2.7 | 0.32 | 0.21 | 0.13 | 0.16 |
| S.D. | 2.06 | 2.19 | 5.34 | 2.07 | 0.35 | 0.60 | 0.71 | 0.87 | 0.120 | 0.061 | 0.038 | 0.028 |
| S.E.M. | 0.73 | 0.77 | 1.89 | 0.73 | 0.12 | 0.23 | 0.25 | 0.31 | 0.042 | 0.022 | 0.013 | 0.010 |

C = control, P = partial, RC = rib cage restriction, ABD = abdominal restriction

* Measurements were made at 40% TLC, see text for details.

MEFV curves for all subjects are presented in Figure 11a and 11b. Peak flow was decreased from control during both forms of restriction, as well as during the partial maneuver. However, once peak flow had been obtained, $\dot{V}max$ was consistently higher during both rib cage and abdominal restriction than it was during either control or partial maneuvers. There was no consistent difference between $\dot{V}max$ during rib cage restriction and that during abdominal restriction. At low lung volumes $\dot{V}max$ during a partial maneuver was greater than control in 2 out of 7 subjects; in all others $\dot{V}max$ did not differ from the control curve.

Examples of Cst, $P_L$ and $\dot{V}max$ for all subjects during rib cage restriction and abdominal restriction are shown in Table 4. Cst and $P_L$ are compared at 50% of control TLC except for two subjects in whom restriction was so severe that 50% control TLC approximated TLC during restriction. $\dot{V}max$ is compared at 40% control TLC in all subjects.

There was a statistically significant increase in mean recoil pressure from 6.2 cm $H_2O$ during the partial maneuver to 11.5 cm $H_2O$ during chest restriction ($p < 0.05$). During abdominal restriction however mean recoil pressure was 7.0 cm $H_2O$, which was not significantly different from that of a partial maneuver (6.2 cm $H_2O$). Rib cage restriction reduced mean static compliance from 0.21 L/cm $H_2O$ during a partial maneuver to 0.13 L/cm $H_2O$ ($p < 0.05$). A similar reduction was seen with abdominal restriction in which mean static compliance decreased from 0.21 L/cm $H_2O$ to 0.16 L/cm $H_2O$ ($p < 0.05$).

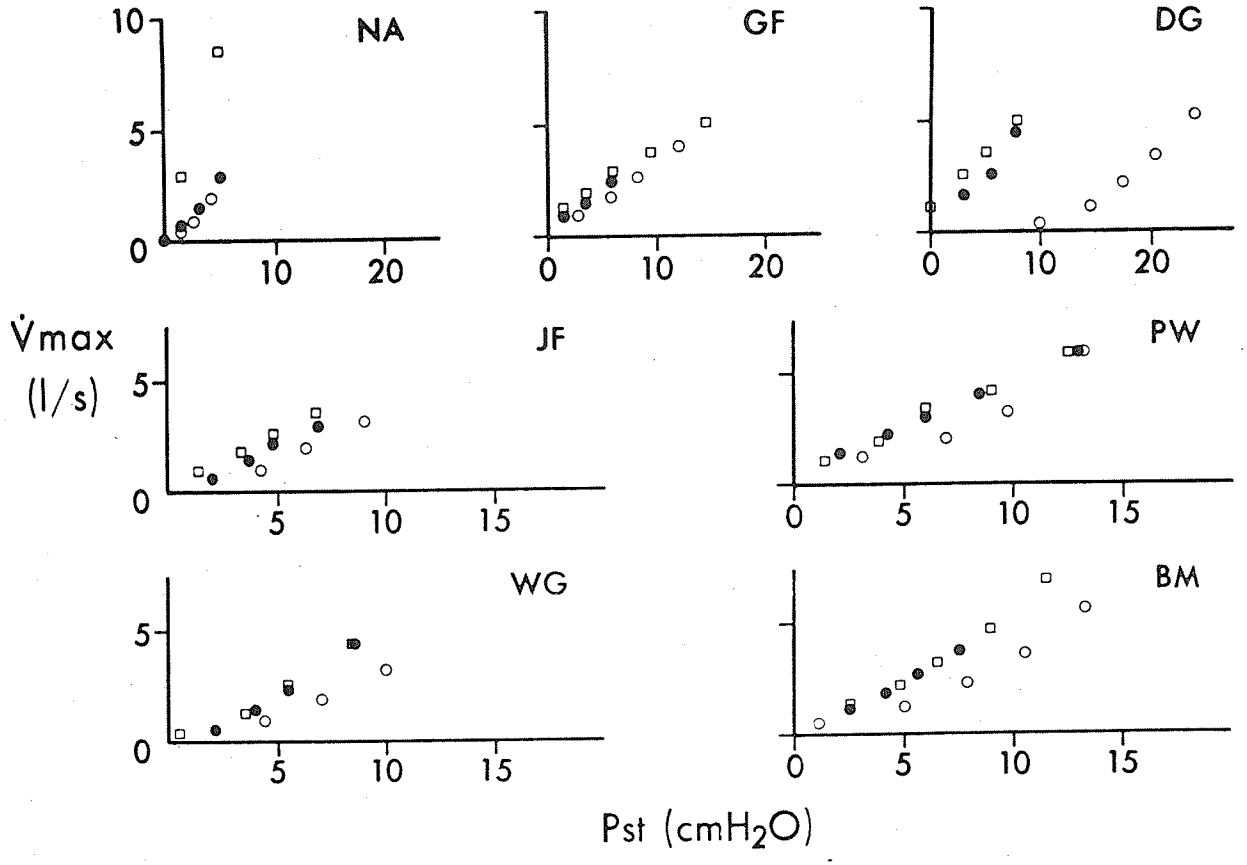




Figure 12: Maximum expiratory flow plotted against static recoil pressure during deflation in seven subjects. Solid dots represent a partial maneuver, open dots during rib cage restriction, and open squares during abdominal restriction.

TABLE 5: Effects of rib cage restriction and abdominal restriction on Rus.

| | Rus at 40% TLC (cm $H_2O$/l/s) | | |
|---|---|---|---|
| Subject | P | RC | ABD |
| J.F. | 2.53 | 3.25 | 1.94 |
| P.W. | 1.93 | 3.10 | 2.04 |
| W.G. | 2.62 | 3.50 | 2.15 |
| B.M. | 2.20 | 3.03 | 2.14 |
| N.A. | 1.91 | 1.18 | 0.68 |
| G.F. | 3.07 | 3.00 | 2.39 |
| D.G. | 2.00 | 7.12 | 1.45 |
| Mean | 2.32 | 3.45 | 1.83 |
| S.D. | 0.44 | 1.79 | 0.58 |
| S.E.M. | 0.16 | 0.68 | 0.22 |

P = partial, RC = rib cage restriction,
ABD = abdominal restriction.

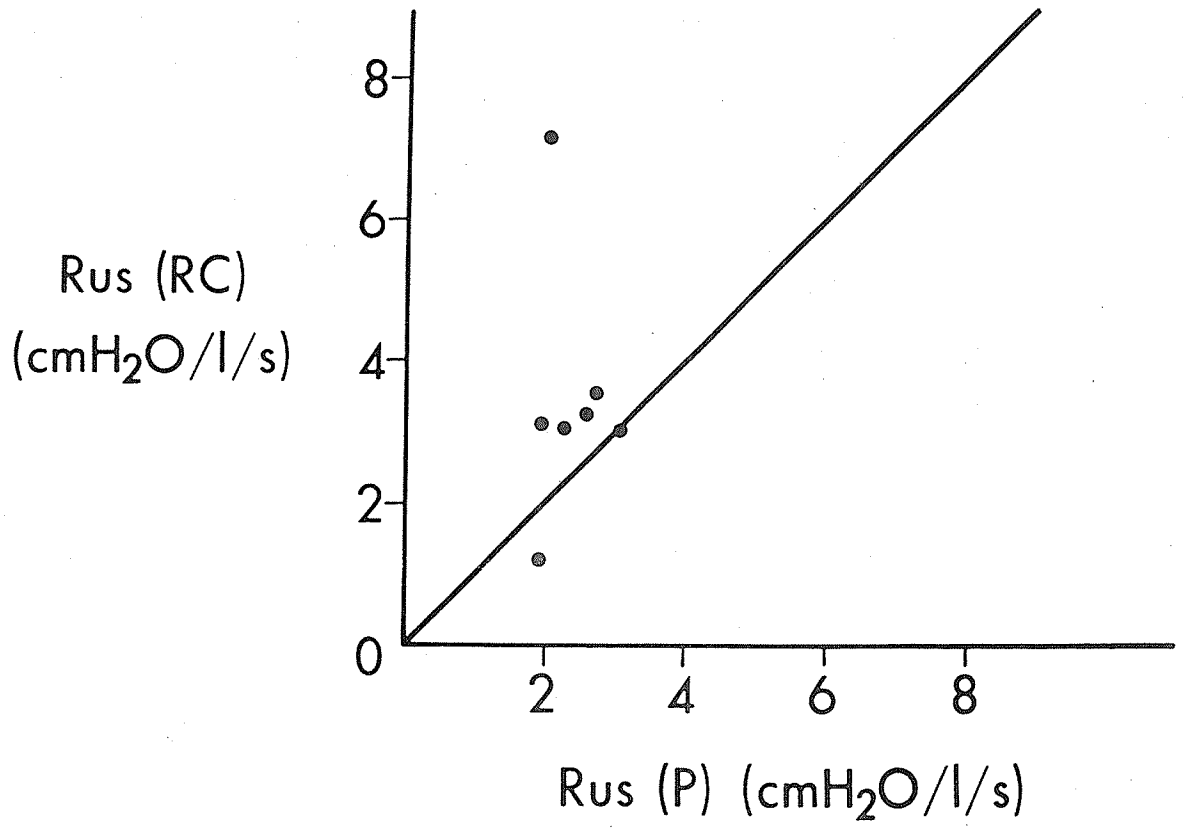


Figure 13: Resistance of the airways upstream from the equal pressure point (Rus) during a partial maneuver plotted against Rus during restriction of the rib cage for seven subjects. The solid line indicates the line of identity.

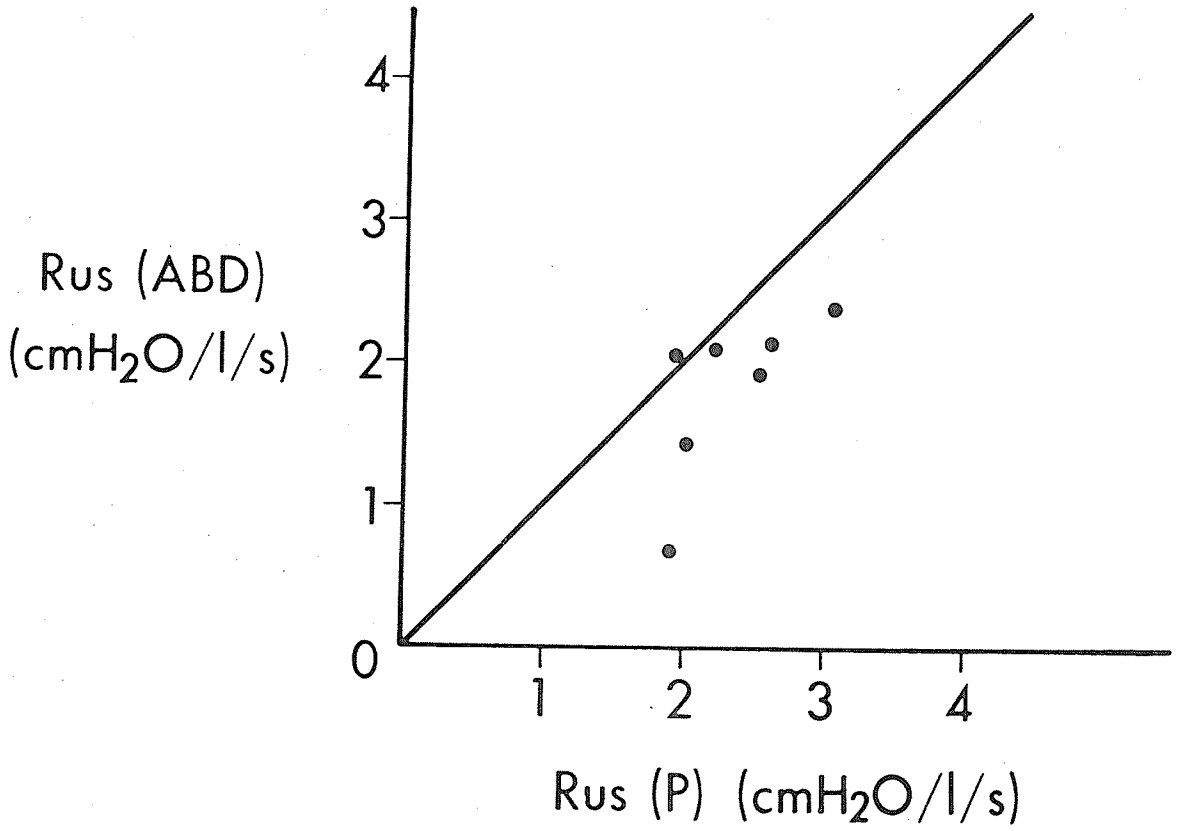


Figure 14: Resistance of the airways upstream from the equal pressure point (Rus) during a partial maneuver plotted against Rus during restriction of the abdomen for seven subjects. The solid line indicates the line of identity.

Mean $\dot{V}max$ at 40% control TLC was 2.7 L/s during both rib cage restriction and abdominal restriction. In both cases this value was significantly greater ($p < 0.05$) than the mean of 1.9 L/s measured during the partial maneuvers.

Maximum flow static recoil curves were constructed for seven subjects (Figure 12). For any given static recoil pressure, flow was less when the rib cage was restricted than it was when either the abdomen was restricted or when a partial maneuver was performed. Also in four of the seven subjects, at a given static recoil pressure, flow was greater during abdominal restriction than during a partial maneuver. Estimations of the resistance of the airway upstream from the equal pressure point (Rus) were taken as the ratio of static recoil pressure to maximum expiratory flow at 40% control TLC. These values are tabulated in Table 5. Although these values did not reach a level of significance, there was a tendency for Rus to increase during rib cage restriction, and decrease during abdominal restriction, when compared to data from partial meaneuvers. However mean Rus during rib cage restriction was significantly greater than during abdominal restriction ($p < 0.05$). Mean values of Rus rose from 2.31 cm $H_2O$/L/s during partial to 2.86 cm$H_2O$/L/s with rib cage restriction, and fell to 1.93 cm $H_2O$/L/s during abdominal restriction. A plot of the values of Rus during a partial maneuver against those during rib cage restriction (Figure 13) showed that all points except one lay above the line of identity, indicating that Rus was systematically greater during chest restriction in most subjects. A similar plot of partial versus abdominal restriction (Figure 14), indicated that Rus was systematically smaller during abdominal restriction compared to partial.

| Slope of Phase III ($\% N_2/1$) | | | |
|---|---|---|---|
| Subject | C | RC | ABD |
| J.F. | 1.05 | 2.30 | 1.20 |
| P.W. | 0.43 | 1.20 | 0.50 |
| W.G. | 0.50 | 1.73 | 0.85 |
| B.M. | 0.45 | 1.10 | 0.70 |
| N.A. | 0.30 | 1.40 | 0.49 |
| G.D. | 0.50 | 1.20 | 1.30 |
| G.F. | 1.25 | 3.40 | 2.00 |
| D.G. | 0.70 | 1.80 | 1.20 |
| Mean | 0.65 | 1.77 | 1.03 |
| S.D. | 0.33 | 0.77 | 0.51 |
| S.E.M. | 0.12 | 0.27 | 0.18 |

| Closing Capacity (L) | | | |
|---|---|---|---|
| Subject | C | RC | ABD |
| J.F. | 1.71 | 1.80 | 1.49 |
| P.W. | 1.44 | 1.58 | 1.63 |
| W.G. | 1.48 | 1.43 | 1.45 |
| B.M. | 1.42 | 1.42 | 1.48 |
| N.A. | 3.05 | 1.95 | 1.58 |
| G.D. | 1.53 | 1.25 | 1.33 |
| G.F. | 1.58 | 1.35 | 1.29 |
| D.G. | 2.22 | 1.65 | 1.55 |
| Mean | 1.80 | 1.55 | 1.48 |
| S.D. | 0.57 | 0.24 | 0.12 |
| S.E.M. | 0.15 | 0.12 | 0.04 |

TABLE 6: Measurements of the Slope of Phase III and closing capacity for Control (C), during rib cage restriction (RC) and during abdominal restriction (ABD).

Slope of phase III and the absolute volumes of closing capacity for all subjects are presented in Table 6. There was a significant increase ($p < 0.05$) in the slope of phase III during rib cage restriction compared to either control or abdominal restriction. During abdominal restriction the slope of phase III was slightly greater than control, but values did not reach the level of significance. Although the ratio of closing capacity to strapped TLC showed an increase during restrictive states, the absolute volumes of closing capacity were not significantly different from control.

# IV. DISCUSSION AND INTERPRETATION OF EXPERIMENTAL RESULTS

In this experiment rib cage restriction produced changes in the subdivisions of lung volume which were similar to those reported by previous studies (12,14,15,25). Like Caro, Butler and DuBois (12) we found that rib cage restriction produced both a significant increase in elastic recoil of the lung and a significant decrease in lung compliance. We found that the release of the chest strap did not cause a return of these parameters to normal, but that this occurred with the first deep breath to control TLC, again in agreement with previous workers (12,14, 15). We also found that rib cage restriction significantly increased maximum expiratory flow at a given lung volume. These results are similar to those reported by Sybrecht et al (15), but somewhat smaller than the increases found by Stubbs and Hyatt (14). At the low lung volumes considered, maximum expiratory flow was effort independent and related to static recoil of the lung, so these results also indicated that static lung recoil was increased. When a rib cage restricted subject was immersed fully to the neck in water, there was a further decrease in FRC (35% to 50%), but the VC was comparable to that of rib cage restriction alone (mean 2.70 L during rib cage restriction compared to 2.66 L during restriction while immersed. RV remained unchanged. Immersion of subjects with restriction produced no further change in the P-V curve of the lung (Figure 8), or in mean C(st) at 50% control TLC (Table 4). Similarly, no consistent changes were found when a comparison was made of the maximum expiratory flow volume curves in the two conditions (Figure II, Table 4). From this we conclude that the mechanical changes in the lung observed during restriction of the rib cage were not altered to any significant degree by immersion.

Prefaut, Lupi-H and Anthonisen (16) studied subjects immersed

in the unstrapped state. Immersion decreased FRC by an amount similar to chest strapping and they made measurements following a five minute period of shallow breathing at this reduced FRC. However, Prefaut et al (16) failed to show increased recoil or increased maximum expiratory flow at low lung volumes. In fact they found a decrease in both elastic recoil and maximum expiratory flow at low lung volumes indicating that not all forms of volume restriction increase the elastic recoil of the lung. The obvious differences between low lung volumes attained during immersion and chest-strapping are that the former is attended by shifts of blood into the chest (26) and is due almost entirely to diaphragmatic as opposed to rib cage displacement (27). In their original paper Caro, Butler and DuBois (12) reported a mean increase in elastic recoil associated with restriction of the abdomen. As previously noted these results are rather misleading, as of the three subjects studied, one showed a relatively large increase in elastic recoil while the remaining two showed no change from the unstrapped state (12). However on the assumptions that (a) abdominal restriction increases the elastic recoil of the lung and (b) that abdominal restriction in an analogue of immersion, Prefaut et al (16) suggested that their results might indicate that shifts of blood into the chest during immersion prevent an increase in recoil due to volume restriction. This study did not support this argument, as we found no discernible effect of immersion on the altered elastic recoil and maximum expiratory flow present during rib cage restriction. This led us to question the initial assumption made by Prefaut et al (16), that restriction of the abdomen increases elastic recoil. We extended the study of Caro, Butler and Dubois (12)

by examining the effect of restricting the abdomen in eight subjects, and further compared this to the changes observed during rib cage restriction in the same eight subjects.

Using pure abdominal restriction, we were unable to produce changes in lung volume which were comparable to those observed with rib cage restriction (Table 7). Thus, the fact that we found no change in elastic recoil with pure abdominal restriction might have been because the volume loss was not large enough. This was supported by the finding that a similar degree of rib cage restriction did not produce recoil changes (Figure 15, Table 8). We created lung volume changes comparable to severe rib cage restriction by combining maximal abdominal restriction with mild rib cage restriction. Because the latter alone caused no change in lung recoil or maximum expiratory flow, we believed that changes in these variables could be ascribed to either low lung volume breathing or to abdominal restriction. This form of abdominal restriction reduced lung volumes by an amount comparable to that seen with rib cage restriction. TLC was reduced by 43% with rib cage restriction, and by 40% with abdominal restriction. The reductions in VC were 52% and 48% respectively. However, the static deflation pressure-volume curve during abdominal restriction was not significantly different from that of a partial maneuver in the unstrapped state (Table 4, Figure 8). In spiteof this, the reduction in mean static compliance at 50% TLC during abdominal restriction was similar to that observed during rib cage restriction (Table 4). Also, measurements of $\dot{V}max$ made at lung volumes where flow was effort independent showed a similar increase during both forms of restriction.

That the increase in elastic recoil during rib cage restriction is real and not a reflection of esophageal artefacts is confirmed by the associated increase in $\dot{V}max$ during effort independent flow. Abdominal restriction also increases $\dot{V}max$ during effort independent flow, but we failed to observe an associated increase in elastic recoil at similar lung volumes. Were the two methods of restriction different, or did abdominal restriction produce some esophageal artefact which underestimates the true elastic recoil of the lung? If the latter were correct, restriction of the abdomen must have generated a falsely positive esophageal pressure. If this phenomenon did occur, and if immersion restricted lung volume in a similar fashion to abdominal restriction, then we would have expected an increase in esophageal pressure, and thus a decrease in elastic recoil, when a subject was immersed. However we found that elastic recoil did not change when a rib cage restricted subject was immersed. Furthermore, if abdominal restriction did apply a positive force to the esophageal balloon and thus mask the expected increase in recoil, removal of the abdominal binder should have been accompanied by a decrease in esophageal pressure and an increase of recoil. When we did this experiment, lung recoil did not change after removal of the binder which restricted the abdomen, until the subject inhaled to TLC (Figure 9b). This finding was similar to that noted by ourselves (Figure 9a) and others (12,14,15) in the case of rib cage restriction.

Therefore we concluded that there was no reason to believe our estimates of recoil were falsely low in restriction of the abdomen, that this state was not associated with an increase in recoil and, that there

was a real difference present between the two methods of restriction. This conclusion is supported by the fact that the different types of restriction had different influences on phase III slope (Table 6).

During rib cage restriction we found a distinct increase in the slope of phase III of the single breath $N_2$ test (17,22). Mean values of the slope were 0.65 $\%N_2/L$ during the control run and 1.77 $\%N_2/L$ during rib cage restriction. On the other hand during abdominal restriction the slope of phase III was not significantly different from that of control (1.03 $\%N_2/L$). Caro et al also used the single breath $N_2$ test and found an increase in the unevenness of ventilation during rib cage restriction (12).

In his original paper on uneven pulmonary ventilation, Fowler (17) noted that as inspired volume increased, the uniformity of expired alveolar gas increased. Thus if inspired volume is decreased, there would be an associated increase in the uneveness of distribution of inspired gas. The volume inspired during rib cage restriction was significantly less that that during a control inspiration in the unstrapped state. The increased uneveness of ventilation distribution noted by both us and Caro et al (12) could have been purely a volume related phenomenon. We performed partial closing volume maneuvers on three subjects. On a partial maneuver the mean slope of phase III was 1.84 $\%N_2/L$, and on the same three subjects the mean slope of phase III during rib cage restriction was 1.83 $\%N_2/L$. This tended to indicate that the slope of phase III during rib cage restriction was a consequence of volume reduction, and did not reflect a specific effect of restricting the rib cage.

If this were true however, it appeared that abdominal restriction decreased the uneveness of inspired gas. In other words our data could be interpreted to indicate that restriction of the abdomen led to improved distribution of ventilation, whereas during rib cage restriction it was unaltered. We are uncertain of what mechanisms caused this, but examination of the elastic recoil and maximum expiratory flow results may give some clues. During abdominal restriction we observed increased $\dot{V}max$ with no change in elastic recoil. Effort independent $\dot{V}max$ is governed by two factors: elastic recoil, which is the driving pressure, and airways resistance of the upstream segment (Rus). Since recoil was not significantly increased during abdominal restriction, Rus must have been decreased (Figure 14). If abdominal restriction decreased peripheral resistance, this might explain not only the observed increase in $\dot{V}max$, but also might contribute to the improved distribution of ventilation.

Maximum flow static recoil curves were constructed for seven subjects, and for any given static recoil pressure, maximum flow during rib cage restriction was less than that during partial maneuvers, or during abdominal restriction; thus Rus increased with rib cage restriction. In four of the seven subjects, at a given static recoil pressure, flow was greater during abdominal restriction than during a partial maneuver, indicating a trend for Rus to decrease with abdominal restriction. At 40% control TLC, Rus during partial maneuvers averaged 2.32 $cmH_2O/l/s$ increasing to 3.45 $cmH_2O/l/s$ with rib cage restriction and decreasing to 1.83 $cmH_2O/l/s$ with abdominal restriction. The difference in Rus between rib cage and abdominal restriction was significant ($p<0.05$).

In summary, our data indicated that restriction of the rib cage was different from abdominal restriction. Although both forms of restriction decreased the static compliance of the lung, and increased maximum expiratory flow, only rib cage restriction was associated with a substantial increase in recoil. Resistance of the airways upstream from the equal pressure point was significantly increased during rib cage restriction, and there was a significant difference between the two forms of restriction in terms of eveness of distribution of inspired gas.

Our finding of increased Rus during rib cage restriction agreed with that of Sybrecht et al (15). Stubbs and Hyatt (14) did not find an increase in Rus; in fact their maximum flow static recoil curves were similar to the ones we measured during abdominal restriction, in that they did not differ from those measured in the unstrapped state. They employed a "thoraco-abdominal restriction" during their study, and as the abdomen is a more deformable structure than the rib cage, it is reasonable to speculate that they restricted the abdomen more than the rib cage. A closer look at their study tends to support this hypothesis.

In our study the reductions in the vital capacity found during rib cage and abdominal restriction differed depending on the form of the restriction. We assessed this reduction by comparing the percentage reduction of the IC to that of the ERV. To allow comparison between our study and previous papers we quantified this numerically by dividing the percentage reduction of the IC by the percentage reduction of the ERV. Thus if both components of the VC were reduced by an equal proportion, this ratio would be close to unity. Alternatively a value less

than unity would indicate a greater reduction of the ERV than of the IC. During rib cage restriction in our study, both IC and ERV were reduced by similar amounts, the percent change in IC per percent change in ERV averaging 0.81. During abdominal restriction the reduction in ERV was relatively greater, and the ratio was 0.53. The studies of Caro, Butler and DuBois (12) and Sybrecht et al (15) both used a form of rib cage restriction, with no abdominal component. Computed values of our ratio in these studies were 1.20 and 0.85 respectively. In Stubbs and Hyatt's study (14) the ratio was 0.51, in close agreement with that noted with the abdominal restriction we employed.

Also, at 50% TLC Stubbs and Hyatt found a mean increase in static recoil pressure from 3.6 cm $H_2O$ during the control unstrapped situation to 5.9 cm $H_2O$ during restriction. This increase was similar to our mean increase from control to abdominal restriction, which at 50% control TLC was 4.7 cm $H_2O$ to 7.0 cm $H_2O$. However we found this increase could be explained by hysteresis of the lung, as mean static recoil at 50% control TLC during a partial maneuver was 6.2 cm $H_2O$, not significantly different from that during abdominal restriction. Stubbs and Hyatt stated that in preliminary experiments four subjects had performed partial pressure-volume curves, and at 50% TLC there was an average difference of 0.4 cm $H_2O$ between the partial and full pressure-volume curves (14). Our results disagree with theirs as we found an average increase of 1.53 cm $H_2O$ at 50% control TLC. Thus our data is consistent with the work of Stubbs and Hyatt (14) if it is assumed that their form of restriction was an analogue of our abdominal restriction, with small increases in recoil that might have been due to hysteresis, and MFSR curves that are indistinguishable from those measured during a full expiration.

Clinical disorders can reduce total lung capacity, and depending on the relative contributions of rib cage or diaphragm displacement, various alterations of lung function have been reported.

It has been shown that when subjects were anaesthetized in the supine position there was a significant reduction in FRC compared to the awake supine state (28,29,30) and as this effect was not abolished by paralysis, the reduction was not due to contraction of expiratory muscles (26,28). Westbrook et al (28) carefully measured pressure-volume curves in supine subjects in the awake, anaesthetized, and anaesthetized-paralyzed states. Elastic recoil showed a significant increase from the 2.8 cm $H_2O$ in the awake state to 12.2 cm $H_2O$ in the anaesthetized state, with no further change with paralysis. C(st) fell significantly with anaesthesia (0.204 to 0.143 L/cm$H_2O$) and again muscle paralysis produced no further change (28). They felt their data was not consistent with an increase in airway closure, as measurements of TGV by plethsmography and by a gas dilution technique did not differ. Also the changes they observed were not progressive, but occurred early and stabilized, and were not reversible by inflation to transthoracic pressures of 35-40 cm $H_2O$ (28). Their results are consistent with those found with chest restriction, a decrease in FRC together with an increase in $P_L$ and a decrease in C(st). Similarly, if our subjects inspired fully while still restricted, large transpulmonary pressures could be developed, without decreasing lung elastic recoil.

Westbrook and associates postulated that the initial effect of anaesthesia was to cause a change in the pressure-volume characteristics of the chest wall such that a reduction in FRC occurred. Subsequent

breathing at an abnormally low lung volume led to increased recoil and decreased compliance, either through a change in surface tension or atelectasis (28). Their results appear to contradict this, as their mean pressure-volume curve for the chest wall was not significantly different from that of control. This as well as the fact that in the sitting position there was no change in FRC or lung compliance following the onset of anaesthesia (31) makes the idea of a change in the chest-wall mechanical properties due to anesthesiology a little doubtful.

A possible difference between anaesthesia and rib cage restriction is apparent, in terms of the distribution of inspired gas. Both we, and Caro et al (12), found an increase in uneveness of distribution of inspired gas with rib cage restriction. During anaesthesia it has been shown that $N_2$ clearance improved (30) and the slope of phase III was decreased when compared to the awake supine state (20) suggesting a more uniform ventilation distribution during anesthesia. However in the anaesthesia studies mechanical ventilation was used both in the awake and the anaesthetized situation. Mechanical inflation of an awake unparalyzed person may not be totally comparable to voluntary inflation. All our studies were done with voluntary inflation and deflation, so perhaps the two situations are not comparable.

Another clinical disorder which alters lung function analogous to chest-strapping is kyphoscoliosis. Patients with kyphoscoliosis have reduced FRC and VC, but normal RV (9,10,32). Slight uneveness of the distribution of inspired gas has been shown in kyphoscoliotic children who demonstrated an increase in the slope of phase III (32).

Bergofsky in 1959 (11) showed increased lung recoil in kypho-

scoliotic adults, together with a marked reduction in C(st) (11,32). A similar reduction was found in dynamic compliance (11,32) and the latter was not frequency dependent (11). In the chest-restriction studies of both Caro, Butler and DuBois (12) and Stubbs and Hyatt (14) dynamic compliance was found reduced during strapping, but was not frequency dependent.

In a later study on adult kyphoscoliotic patients, Bergofsky demonstrated that a five minute period of intermittent positive pressure breathing at 25 cm $H_2O$ produced a 70% increase in dynamic compliance, which was sustained for 3 hours. Because FRC changed little, they believed that the opening of previously closed airways was an unlikely explanation for the increase in Cdyn (9). They speculated that the large changes in Cdyn could be due to reorientation of surface forces, produced by an alteration of surface active agents or by a change in geometric configuration of alveoli. This explanation had been used by Mead and Collier (33) who showed much greater changes in lung compliance than in FRC in normal animals when the lung was deflated and reinflated. Bergofsky (9) also examined MEFV curves in kyphoscoliosis patients and found that at low lung volumes they were convex to the volume axis suggesting a decreased conductance of more peripheral airways (5,7). After hyperinflation there was an increase of flow at low lung volumes. Because this improvement was associated with the post-hyperinflation increase in lung compliance (9), it was not attributable to increased lung recoil, strongly suggesting a decrease in Rus (9).

It is interesting to note that in 1961 Caro and DuBois studied a group of young kyphoscoliotic patients and found that their pressure-

volume curves were not dissimilar from normal (32). Measurements of the elastance of the thoracic cage showed it was either equal or just slightly less than normal (32). The rigidity of the chest cage appears to be a late complication of the disease and it appears that it is only when this distensibility has decreased that static recoil presssures of the lung are grossly affected (11). However, though chest wall mechanics were near normal in young kyphoscoliotics, lung compliance was reduced (32). This reduction in compliance was thought to reflect decreased lung volume as opposed to increased elastic recoil (32). When Bergofsky and associates combined both normal and kyphoscoliosis results they found a strong linear correlation between compliance and VC (11).

Rib cage restriction appears analogous to kyphoscoliosis, both reducing lung volumes, increasing elastic recoil of the lung, and decreasing static and dynamic compliance equally. Dynamic compliance is not frequency dependent in either situation, although an increase in Rus has been indicated in both. During both rib cage restriction and kyphscoliosis a slight increase in the uneveness in the distribution of inspired gas is found.

Clinical representations of abdominal restriction, would logically be situations in which there is a reduction in lung volume due to a cephalad displacement of the diaphragm, as in pregnancy and obesity.

During pregnancy there is a reduction in the FRC but no reduction in the vital capacity (34,35,36). Closing capacity (34,35) and compliance (36) are unchanged in pregnancy. Gee et al (36) postulated that pregnancy did not constitute a true restriction of diaphragm movement,

since the VC was unchanged during, and although they found a large increase in airway conductance comparable to that demonstrated by Caro, Butler and DuBois (12) they felt the analogy between pregnancy and abdominal restriction was invalid. They speculated that the decrease in airways resistance found (50% below normal) could be due to modified bronchomotor tone due to metabolic factors (increased progesterone, cortisone and related steroids, relaxin) (36). Airway conductance increased progressively and returned to normal by 2 months postpartum (36). If the increase were due to volume restriction, one would expect it to be restored to normal values immediately following postpartum. Changes during pregnancy appear to be due more to metabolic factors than due to volume restriction.

Extreme obesity is associated with reduced lung volumes, and unlike pregnancy is not accompanied by hormonal changes. Many workers have found FRC to be lower than normal during obesity, and as residual volume either rises or is unchanged, this is due to a reduction of ERV (37,38,39,40). There was an abnormally large decrease in FRC when an obese subject changed from a sitting to a supine position (39,41), and FRC was further reduced with anaesthesia (41). Douglas and Chong (38) found that VC was in the normal range, whereas Rochester and workers (37) found that the vital capacity was decreased with obesity, and others have supported this finding (41,42). The respiratory system demonstrated increased resistance (42), increased inertance and decreased compliance (37,41, 43) in obesity. Although lung compliance does decrease (37,41,42) in obesity, the chief factor responsible for the reduction in compliance of the

total respiratory system in obesity is the reduced compliance of the chest wall (41,42). Similarly, the depressed slope of the pressure-volume curve of the total respiratory system in the obese is due almost entirely to the decreased slope of the chest wall pressure-volume curve (41,42). Pressure-volume curves of the lung do not significantly differ from normal (38,41,42) and measurements of elastic recoil are within normal limits (38).

Thus it appears from a review of the relevant literature, that when reduced lung volumes are present with obesity, the static mechanical characteristics of the lung are analogous to those found during abdominal restriction.

What causes the changes in elastic properties of the lung secondary to rib cage restriction? Pulmonary vascular engorgement seems an unlikely explanation as immersion fails to alter measurements of either lung recoil or lung compliance in the same manner as rib cage restriction (16). Also Caro, Butler and DuBois (12) found an increase in venous pressure of only 1.2 cm $H_2O$ which was slight in contrast to the study of Bondurant (44,45) who increased central venous pressure by 25 cm $H_2O$ to obtain a 25% reduction in lung compliance.

The changes of the pressure-volume curve and MEFV curves we observed during rib cage restriction cannot be attributed to the hysteresis known to exist in the airways and the lung, as in all cases comparisons were made between partial maneuvers initiated at the same lung volumes. Caro, Butler and DuBois (12) suggested that rib cage restriction either produced airway closure and atelectasis, or an alteration of the elastic characteristics of the lung tissue or surface lining film.

They supported the airway closure theory as they found some evidence of trapped gas in their subjects following the release of the chest strap (12). In 1968 Burger and Macklem (13) studied subjects voluntarily breathing at low lung volumes. When the subjects breathed air at low lung volumes for some minutes there was a shift of the pressure-volume curve from the normal, and when the air was replaced by oxygen this shift was more exaggerated. This they felt added support to the theory that atelectasis was responsible for the shift of the pressure-volume curve while breathing $O_2$, but it failed to explain the discrepancy of the air breathing curves from normal. They found the shift of the oxygen breathing curves were present only when the subject was breathing with a tidal volume of RV + 750 ml. When the tidal volume was RV + 1000 ml the resulting pressure-volume curve was similar to control. From this they concluded that above RV + 750 ml essentially all airways are open (13). However, in our study our subjects were continually breathing at volumes above RV + 750 ml, and were also generating transpulmonary pressures greater than those estimated to open closed airways.

A subsequent study by Manco and Hyatt (25) failed to detect trapped air during chest restriction in eight out of thirteen subjects. In those exhibiting a small amount of gas trapping, no correlation was found between the amount of trapping and the severity of chest restriction, whereas there was a positive correlation between increased restriction and increased recoil (25).

If airway closure and atelectasis occurred during rib cage restriction, it would be reasonable to expect a decrease in the regional

ventilation of the dependent portions of the lung where airway closure is thought to occur. Sybrecht et al (15) found no changes in regional ventilation of the lung, and postulated that there was no increase in airway closure, but that the reduction in lung compliance seen with rib cage restriction was equally shared between all portions of the lung (15).

Animal studies have failed to correlate a reduction in lung compliance with atelectasis. Williams and co-workers (46) in a study of anaesthetized paralyzed rabbits and newborn lambs found large reductions in lung compliance (53% and 40% respectively) following 20 minutes of mechanical ventilation at a constant tidal volume. They found no evidence of trapped air, and morphometric studies showed patent airways. Young, Tierney and Clements (47) held freshly excised rat lungs at 3 cm $H_2O$ transpulmonary pressure and found a 42% reduction in lung compliance after 20 minutes. The lungs were then quick frozen with liquid propane and histological examination revealed no evidence of airway occlusion, atelectasis nor changes in the configuration of the alveoli and alveolar ducts (47). They repeated the experiment with a saline filled lung, thus eliminating surface forces, and found no change in lung compliance in 20 minutes. Thus the compliance decrease, they reasoned, was probably not due to changing tissue forces. Further animal studies showed that the decrease in compliance is present prior to any evidence of air trapping, atelectasis, or shunting (33,48,49).

Thus from the aforementioned studies it seems likely that airway closure and atelectasis are not the main mechanism operative during rib cage restriction. The evidence present in the literature seems more to

favour an alteration of surface forces within the lung. Indeed Tierney has shown with studies of lung extracts that compression of the surface is associated with a prompt increase in surface tension (50).

During both abdominal and rib cage restriction a reduction in lung compliance was found. Decreased lung compliance with low lung volume breathing has previously been found both in humans (51,52) and in animals (46) and has been attributed to an alteration of surface forces within the lung as previously discussed. Studies on both kyphoscoliosis patients and obese subjects have revealed that the reduction in compliance shows a strong correlation to the vital capacity (11,41, 42) and thus the reduction in compliance found during both rib cage and abdominal restriction can probably be attributed to the decrease in lung volume. The alveolus cannot maintain a low surface tension for long without occasional large inflations which replenish the surface with surface active material. Thus maintaining abnormally low lung volumes, as during rib cage and abdominal restriction, leads to an increase of surface tension, and as a direct consequence, a reduction of lung compliance. Recent in vivo work of Schurch, Goerke and Clements (53) does not support this theory however. They observed that the surface tension of rat lungs in situ did indeed reach very low values at FRC, but remained stable when held at this volume for a 30 minute period. They did not, however, do experiments at lung volumes lower than FRC.

Our study does not indicate the causes of the changes we observed during either rib cage or abdominal restriction. However it does indicate that, depending on the form of restriction utilized, the alterations

of lung function differ, and therefore cannot be ascribed to low lung volume breathing per se.

During rib cage restriction both elastic recoil of the lung and maximum expiratory flow at low lung volumes increased. However at a given elastic recoil pressure, $\dot{V}max$ was less than during rib cage restriction than during a partial maneuver, indicating an increase in Rus. Alternatively abdominal restriction produced the same change in lung volumes, and increased maximum expiratory flow at low lung volumes, but there was no corresponding increase in elastic recoil. This indicates a decrease in Rus during abdominal restriction. The difference between the two states is also attested to by the finding of less unevenness in the distribution of inspired gas during abdominal restriction compared to rib cage restriction.

Thus, lung volume restriction induced changes in lung function, but the nature of these changes depended on how the restriction was applied and not on the degree of volume restriction. We are unable to explain this finding, but suggest that it might relate to lung distortion. In the present context lung distortion can be defined as a change in lung shape resulting from forces which are unevenly applied. When an excised lung is inflated it is not distorted: both alveolar and "pleural" pressures are uniform. Since in this condition the lung is allowed to find its own shape, the energy (pressure) needed to inflate it is minimal. However, when the lung is distorted, that is it cannot freely change shape because some dimension is constrained by an unevenly applied force, some energy must be utilized to change the lung shape to fit the external constraint. Thus, inflation of a distorted lung will demand more pressure than similar inflation of an undistorted

lung. In the normal thorax lungs are apparently relatively undistorted since pressure-volume curves of lungs in situ agree well with excised lungs (54). It is very likely that rib cage restriction caused lung distortion. The rib cage was compressed in its antero-posterior diameter more than in its lateral diameter, and no compression was applied to the diaphragmatic surface. If the distortion were severe enough, PL would be increased at a given lung volume and because of this maximum flow increased. In addition, such distortion would not effect all lung units in the same way and because of this Rus might increase and the distribution of inspired gas become more uneven. Thus it is possible that severe lung distortion could account for the changes we observed during rib cage restriction.

During abdominal restriction, lung distortion probably was less than during rib cage restriction. Konno and Mead showed that when the abdomen was voluntarily compressed, the rib cage expanded in a uniform fashion, that is all changes in rib cage dimensions were proportional to the original dimensions (55). This is another way of saying that the shape of the rib cage changed little. Thus if changes in elastic recoil during rib cage restriction were due to distortion, one would expect much smaller changes with abdominal restriction. Further, one might expect any distortion due to abdominal restriction to be more evenly distributed, which might account for the relatively flat phase III and normal Rus observed in this condition.

The problem of course with the distortion argument is that changes in elastic recoil persisted after the restricting force was removed. Whether the lung "remembers" distortion or not is not known but it is difficult to see how it could be in the absence of surface tension

changes. However the hypothesis is testible. The influence of distortion on the elastic recoil of isolated lungs can be studied and more particularly, if distortion increases elastic recoil one can assess whether the increased recoil persists after the distorting force is removed.

V APPENDICES

APPENDIX I Preliminary Experiment

APPENDIX II Statistical Analysis

Appendix I.

Preliminary Experiments

Methods

Four healthy male volunteers (3 smokers) aged 22-44 years were studied under three conditions: unstrapped, abdomen-strapped and abdominal equivalent rib cage strap. The abdomen was strapped by placing an pneumatic cuff around the abdomen, held in position by an inextensible canvas corset, which was manually tightened while the subject breath-held at RV. The cuff was then inflated to not less than 60 mmHg, and was maintained at this pressure throughout the study. Abdominal-equivalent rib cage-strapping was obtained by manually tightening the canvas corset around the rib cage until the reduction in TLC was comparable to that seen during abdominal restriction.

In each subject lung volumes, deflation static pressure-volume (P-V) curves of the lung, MEFV curves and closing volume curves were measured while unstrapped (control) and during abdominal and abdominal equivalent rib cage strapping. During the unstrapped state, maneuvers generating deflation P-V curves and MEFV curves were initiated at 80% vital capacity (partial), as well as at total lung capacity (controls). Thus data acquired during abdominal strapping could be compared to maneuvers in the unstrapped state which began at the same lung volume. All the above parameters were measured in the same fashion as described in the previous method section.

Results

The effects of strapping on the subdivisions of lung volume can be seen in Table 7. Abdominal strapping produced an overall reduction in TLC of 20%, and in FRC of 27%. A similar decrease in TLC was seen

| | TLC, (liters) | | | | FRC, (liters) | | | | RV, (liters) | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Subject | C | PA | AS | $RC_A$ | C | PA | AS | $RC_A$ | C | PA | AS | $RC_A$ |
| N.A. | 8.0 | 6.0 | 6.2 | 5.7 | 4.2 | 4.2 | 2.5 | 3.2 | 1.9 | 1.9 | 1.6 | 1.8 |
| G.D. | 5.9 | 5.1 | 5.5 | 5.6 | 2.7 | 2.7 | 2.3 | 2.5 | 1.1 | 1.1 | 1.1 | 1.1 |
| G.F. | 6.3 | 4.9 | 4.8 | 5.1 | 2.9 | 2.9 | 1.7 | 2.1 | 1.2 | 1.2 | 1.2 | 1.2 |
| D.G. | 6.8 | 5.6 | 5.3 | 5.6 | 2.6 | 2.6 | 2.5 | 2.6 | 1.3 | 1.3 | 1.3 | 1.3 |
| Mean | 6.8 | 5.4 | 5.5 | 5.5 | 3.1 | 3.1 | 2.3 | 2.6 | 1.4 | 1.4 | 1.3 | 1.4 |
| S.D. | 0.92 | 0.49 | 0.58 | 0.27 | 0.74 | 0.74 | 0.38 | 0.45 | 0.36 | 0.36 | 0.22 | 0.31 |
| S.E.M. | 0.46 | 0.24 | 0.29 | 0.14 | 0.37 | 0.37 | 0.19 | 0.23 | 0.18 | 0.18 | 0.11 | 0.16 |

C = control, PA = partial, AS = abdominal strapping, $RC_A$ = abdominal equivalent rib cage strapping.

TABLE 7: Subdivisions of Total Lung Capacity

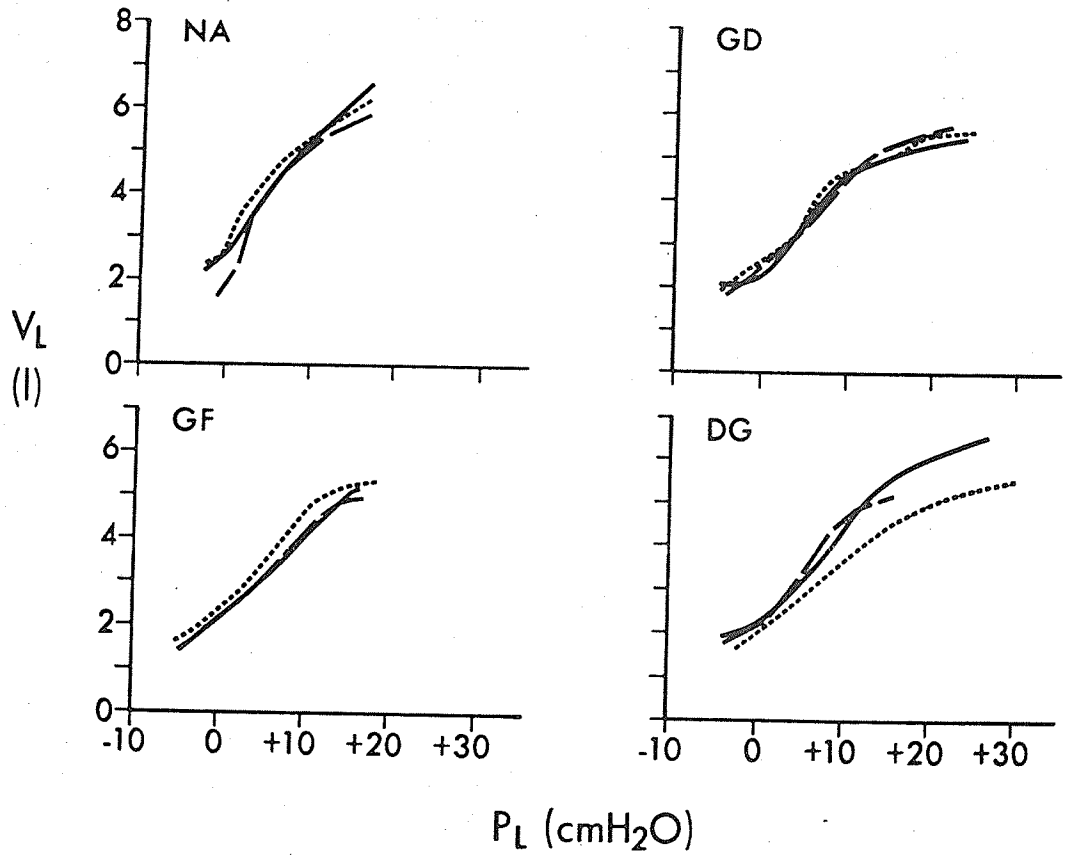


Figure 15: Static deflation pressure-volume curves in four subjects during a partial maneuver (———), during abdominal equivalent rib cage strapping (--------), and during abdominal strapping (— — —). Absolute lung volume is shown on the ordinate, transpulmonary pressure on the abscissa.

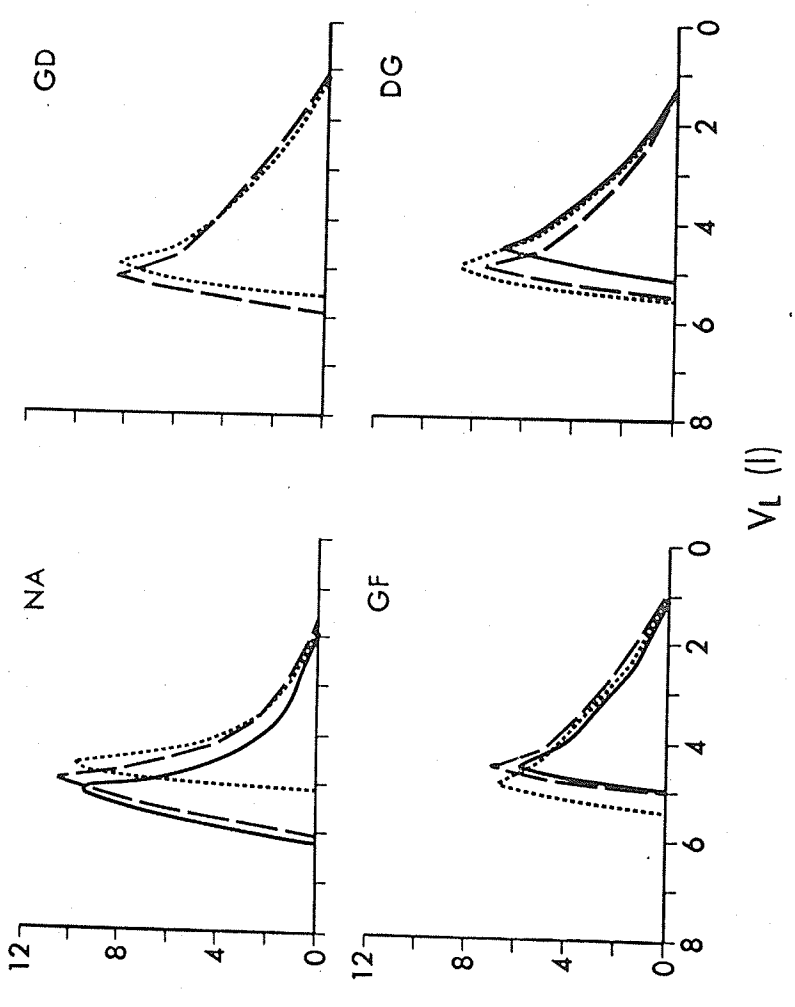


Figure 16: Maximum expiratory flow-volume curves in four subjects during a partial maneuver (———), during abdominal equivalent rib cage strapping (- - - - -), and during abdominal strapping (— — —). Maximum expiratory flow is shown on the ordinate, absolute lung volume on the abscissa.

| | $P_L$ at 50% TLC, ($cmH_2O$) | | | | MEF at 40% TLC, (1/s) | | | | Cst at 50% TLC, ($1/cmH_2O$) | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Subject | C | PA | AS | $RC_A$ | C | PA | AS | $RC_A$ | C | PA | AS | $RC_A$ |
| N.A. | 2.6 | 4.0 | 4.0 | 3.0 | 1.2 | 0.9 | 1.7 | 1.6 | 0.43 | 0.25 | 0.25 | 0.20 |
| G.D. | 4.8 | 3.2 | 3.0 | 2.7 | 1.8 | | 1.7 | 1.5 | 0.25 | 0.22 | 0.19 | 0.20 |
| G.F. | 5.4 | 5.4 | 5.7 | 3.7 | 1.6 | 1.3 | 1.8 | 1.6 | 0.25 | 0.20 | 0.20 | 0.20 |
| D.G. | 5.0 | 6.7 | 5.8 | 8.5 | 1.9 | 1.9 | 1.5 | 1.9 | 0.32 | 0.25 | 0.39 | 0.17 |
| Mean | 4.4 | 4.8 | 4.6 | 4.5 | 1.6 | 1.4 | 1.7 | 1.7 | 0.31 | 0.23 | 0.26 | 0.19 |
| S.D. | 1.25 | 1.55 | 1.36 | 2.72 | 0.31 | 0.50 | 0.13 | 0.17 | 0.085 | 0.024 | 0.092 | 0.015 |
| S.E.M. | 0.63 | 0.77 | 0.68 | 1.36 | 0.15 | 0.29 | 0.06 | 0.09 | 0.042 | 0.012 | 0.046 | 0.008 |

C = control, PA = partial, AS = abdominal strapping, $RC_A$= abdominal equivalent rib cage strapping

TABLE 8: Measurements of transpulmonary pressure, maximum expiratory flow and static compliance.

with the abdominal equivalent rib cage strapping, namely 19%, but FRC was decreased by slightly less (16%). Both restricted TLC's were significantly decreased from control ($p < 0.05$), whereas FRC and RV were not significantly different from control in either case. In all subjects neither form of strapping significantly displaced the static deflation P-V curve from that of the partial maneuver in the unstrapped state (Figure 15).

MEFV curves for all four subjects can be seen in Figure 16. Values of elastic recoil ($P_L$) taken at 50% control TLC, and static compliance (Cst) measured over the interval 50% control TLC + 0.5 liters are listed in Table 8. Mean recoil pressure in each condition did not differ significantly from each other, similarly there was no systematic change in measured static compliance. During both strapped conditions mean $\dot{V}max$ at 40% control TLC did not differ significantly from control or partial values.

Conclusion

Abdominal strapping failed to cause the increase in elastic recoil or maximum expiratory flow normally associated with chest strapping. However, strapping the rib cage to a comparable volume did not appear to alter the elastic recoil of the lung either. From this it was concluded that the level of restriction produced here was not sufficient to effect an alteration in recoil.

Thus in order to study the effects of abdominal strapping it seemed necessary to adopt a more severe form of restriction. This was achieved by applying an abdominal strap while simultaneously preventing expansion of the rib cage.

Appendix II.

Statistical Analysis

Definition and Explanation of Statistical Methods used.

Mean = $\bar{X} = \frac{\Sigma X}{N}$ where N = No. of observations

Standard deviation = S.D. = $\left[\Sigma (X - \bar{X})^2/(N - 1)\right]^{\frac{1}{2}}$

Variance = $S^2 = \Sigma (X - \bar{X})^2/(N - 1)$

Standard Error = S.E.M. = S.D./$\sqrt{N}$

To test for differences among the means of the two or more groups, an analysis of variance was performed. This analysis requires estimating the population variance in two ways, and then comparing these two estimates. The two estimations used are (a) a direct computation of the variance of the means multiplied by sample size and (b) a pooled estimate of the variance of separate groups. As estimation (a) is based on between group variance, and estimation (b) on within group variance, if the means of the groups are greatly different, then estimation (a) will be much larger than estimation (b).

Formulae used:-

Let the 1st subscript refer to the group number and the 2nd subscript refer to the observation. e.g. $X_{ij}$ is the jth observation of group i.

Let their be K groups, $n_i$ observations per group.

Let the total number of observations be $N = \sum_{i}^{k} n_i$.

Let $\bar{X}_{++}$ be the overall mean, and $\bar{X}_{i+}$ be the mean of group i.

$S_M^2$ = Mean square between group = $\frac{\text{Sums of squares between}}{\text{Degrees of freedom (D.F.)}}$

$$= \frac{\sum_{i=1}^{k} n_i (\bar{X}_{i+} - \bar{X}_{++})^2}{k - 1}$$

$$S_p^2 = \text{Mean square within groups} = \frac{\text{Sums of squares within groups}}{\text{Degrees of Freedom}}$$

$$= \frac{\sum\sum_{ij} (X_{ij} - \bar{X}_{i+})^2}{N - k}$$

If the groups are from populations having unequal means, the mean square between groups will usually be considerably larger than the estimate for the mean square within groups. We wish to reject the hypothesis of equal means if the observed means are significantly more disperse than we would expect when they are all obtained from the same population. The test statistic used is the F ratio, the ratio of the mean square between groups to the mean square within groups, and the hypothesis of equal means is rejected if the value obtained exceeds the critical value of the F table for k-1 and $\Sigma n_i - k$ degrees of freedom (56). For this test to be valid the assumption that the observations are randomly selected from normal populations with homogeneous variance must be satisfied.

Homogeneity of variance was tested as an approximation of Bartletts test (57,58) using $X^2$ as the test statistic. If variances were not homogeneous, the observations were transformed so that this assumption was satisfied.

If the hypothesis of equal means was rejected, one of two tests of multiple comparisons was employed to make pairwise comparisons.

(i) Tukey's test, which compares the range of the means to the mean square within groups. The test statistic used is $q = \frac{\bar{X}max - \bar{X}min}{Sp\sqrt{n}}$, where n = sample size per group. The hypothesis of equal means is rejected if the calculated q exceeds the critical value of q with K = No. of treatment means, and N-k degrees of freedom obtained from the distribution of the Studentized range statistic (59).

(ii) Dunnett's test for comparisons involving a control mean. The test statistic used is $t_{(Dunnett)} = \frac{\bar{X}_i - \bar{X}_{control}}{\sqrt{\frac{2(Sp)^2}{n}}}$

The hypothesis of equal means is rejected if the calculated $t_{(Dunnett)}$ exceeds the critical value of t with k treatment means excluding control (k-1), and N-k degrees of freedom obtained from the table of t for one-sided comparisons between (k-1) treatment means and a control (60).

Statistical Results

The critical value ($\alpha$) taken for all tests was at the 0.05 level. Thus when values have been stated to be statistically different, we are saying that this would occur by chance only 5% of the time. All statistical results are presented in the following manner:-

Section. Experimental Study.

List of treatment levels, or groups.

Number of groups (k)

Number of observation per group (n)

Total number of observations (N) = $\sum_i n_i$

Sub-section.

Name of variable being compared between treatment levels.

(i) Bartletts test for homogeneity of variances.

$X^2$ = $X^2(DF)\alpha = 0.05$ =

DF =

Transformation information if applicable.

(ii) Analysis of Variance.

F = $F(k-1, \Sigma n_i - k)\ \alpha = 0.05$ =

If the F ratio is significant, one of the following multiple

comparison tests will be presented. Only significant comparisons have been reported.

(iii) Tukey's test

Comparison performed $q = (k,\Sigma n_i - k)\alpha = 0.05 =$

Dunnett's test

Comparison performed $tD = (k-1,\Sigma n_i - k)\alpha = 0.05 =$

Section A. Preliminary Experiments

Group (1) Control, (C)

Group (2) Partial, abdomen ($P_A$)

Group (3) Abdominal equivalent chest-strap ($RC_A$)

Group (4) Abdominal strap (AS)

k = 4

n = 4

N = 16

A1 TLC

(i) $X^2 = 3.601$

DF = 3 $X^2(3,\alpha = 0.05) = 7.81$

(ii) F = 4.548 $F(3,12\alpha = 0.05) = 3.49$

(iii) Tukey's test

C: $P_A$ q = 4.400 $q(4,12\alpha = 0.05) = 4.20$

C: AS q = 4.269

C: $RC_A$ q = 4.106

A2 FRC

(i) $X^2 = 1.728$

DF = 3 $X^2(3,\alpha = 0.05) = 7.81$

(ii) F = 1.892 $F(3,12\alpha = 0.05) = 3.49$

Â3 RV

(i) $X^2 = 2.615$

DF = 3 $X^2(3, \alpha = 0.05) = 7.81$

(ii) F = 0.050 $F(3,12\ \alpha = 0.05) = 3.49$

A4 $P_L$

(i) $X^2 = 2.214$

DF = 3 $X^2(3, \alpha = 0.05) = 7.81$

(ii) F = 0.037 $F(3,12\ \alpha = 0.05) = 3.49$

A5 $\dot{V}max$

(i) $X^2 = 4.709$

DF = 3 $X^2(3, \alpha = 0.05) = 7.81$

(ii) F = 0.778 $F(3,11\ \alpha = 0.05) = 3.59$

A6 C(st)

(i) $X^2 = 9.472$

DF = 3 $X^2(3, \alpha = 0.05) = 7.81$

As the means of the groups vary proportionally with their standard deviations, a log transformation was performed on the data to endeavor to reduce scatter, to allow the assumption of homogeneity of variances to be satisfied.

Following log transformation.

(i) $X^2 = 5.950$

DF = 3 $X^2(3, \alpha = 0.05) = 7.81$

(ii) F = 2.943 $F(3,12, \alpha = 0.05) = 3.49$

Section B Immersion Experiments

Group (1) Control (C)

Group (2) Rib cage restriction (RC)

Group (3) Rib cage restriction while immersed (RCI)

k = 3

n = 4

N = 12

B1 TLC

(i) $X^2$ = 2.987

DF = 2 $X^2(2, \alpha = 0.05) = 5.99$

(ii) F = 26.151 $F(2,9, \alpha = 0.05) = 4.26$

(iii) Tukey's test

C: RC q = 9.027 $q(3,9, \alpha = 0.05) = 3.95$

C: RCI q = 8.677

B2 FRC

(i) $X^2$ = 4.357

DF = 2 $X^2(2, \alpha = 0.05) = 5.99$

(ii) F = 14.631 $F(2,9, \alpha = 0.05) = 4.26$

(iii) Tukey's test

C: RC q = 5.447 $q(3,9, \alpha = 0.05) = 3.95$

C: RCI q = 7.376

B3 RV

(i) $X^2$ = 1.013

DF = 2 $X^2(2, \alpha = 0.05) = 5.99$

(ii) F = 0.148 $F(2,9, \alpha = 0.05) = 4.26$

B4 $P_L$

(i) $X^2$ = 5.589

DF = 2 $X^2(2, \alpha = 0.05) = 5.99$

(ii) F = 4.519 $F(2,9, \alpha = 0.05) = 4.26$

(iii) Dunnett's test

C: RC tD = 2.588 $tD(3,9, \alpha = 0.05) = 2.18$

C: RCI tD = 2.619

Tukey's test

RC: RCI $q = 0.044$ $q(3,9, \alpha = 0.05) = 3.95$

B5 $\dot{V}max$

(i) $X^2 = 3.499$

DF = 2 $X^2(2, \alpha = 0.05) = 5.99$

(ii) $F = 2.949$ $F(2,9, \alpha = 0.05) = 4.26$

(iii) Tukey's test

RC: RCI $q = 1.878$ $q(3,9, \alpha = 0.05) = 3.95$

B6 C(st)

(i) $X^2 = 7.633$

DF = 2 $X^2(2, \alpha = 0.05) = 5.99$

As the means of the groups varied proportionally with their standard deviations, a log transformation was performed on the data.

Following log transformation.

(i) $X^2 = 5.394$

DF = 2 $X^2(2, \alpha = 0.05) = 5.99$

(ii) $F = 12.738$ $F(2,9, \alpha = 0.05) = 4.26$

(iii) Dunnett's test

C: RC $tD = 4.541$
C: RCI $tD = 4.179$ $tD(2,9, \alpha = 0.05) = 2.18$

Tukey's test

RC: RCI $q = 0.0512$ $q(3,9, \alpha = 0.05) = 3.95$

Section C Reported Study

Group (1) Control (C)

Group (2) Partial (P)

Group (3) Rib cage restriction (RC)

Group (4) Abdominal restriction (ABD)

k = 4

n = 8 (n was chosen as 8, by using results of previous experiments to determine an n such that at $\alpha = 0.05$, the power of the test would be greater than 80%, and thus minimize the risk of making a Type II error).

N = 32

C1 TLC

(i) $X^2 = 6.348$

DF = 3 $X^2(3, \alpha = 0.05) = 7.81$

(ii) F = 77.802 $F(3,28,\alpha = 0.05) = 2.95$

(iii) Tukey's test

C: P q = 17.481 $q(4,28, \alpha = 0.05) = 3.84$

C: RC q = 18.298

C: ABD q = 17.053

C2 FRC

(i) $X^2 = 9.653$

DF = 3 $X^2(3, \alpha = 0.05) = 7.81$

As the means of the groups varied proportionally with their standard deviations, a log transformation was performed on the data.

As Controls and Partial values were the same these were used as one group (k = 3, n = 8).

Following log transformation.

(i) $X^2 = 1.490$

DF = 2 $X^2(2, \alpha = 0.05) = 5.99$

(ii) F = 38.383 $F(2,21, \alpha = 0.05) = 3.47$

(iii) Tukey's test

C: RC q = 9.353 $q(3,21, \alpha = 0.05) = 3.58$

C: ABD q = 11.715

C3 RV

(i) $X^2 = 2.804$

DF = 3 $X^2(3, \alpha = 0.05) = 7.81$

(ii) F = 0.426 $F(3,28, \alpha = 0.05) = 2.95$

C4 $P_L$

(i) $X^2 = 10.382$

DF = 3 $X^2(3, \alpha = 0.05) = 7.81$

As the means of the groups varied proportionally with their standard deviation, a log transformation was performed on the data.

Following log transformation

(i) $X^2 = 1.487$

DF = 3 $X^2(3, \alpha = 0.05) = 7.81$

(ii) F = 5.286 $F(3,28, \alpha = 0.05) = 2.95$

(iii) Dunnett's test

C: RC tD = 3.917 $tD(3,28, \alpha = 0.05) = 2.15$

In this situation, it would be more technically correct to use the partial run as the control, so further comparisons were done, eliminating the control run, and comparing rib cage and abdominal restriction to the partial as a control.

Following log transformation

(i) $X^2 = 0.393$

DF = 2 $X^2(2, \alpha = 0.05) = 5.99$

(ii) F = 4.315 $F(2,21, \alpha = 0.05) = 3.47$

(iii) Dunnett's test

P: RC tD = 2.813 $tD(2,21\ \alpha = 0.05) = 2.03$

C5 MEF

(i) $X^2 = 5.014$

DF = 3 $X^2(3, \alpha = 0.05) = 7.81$

(ii) F = 4.548 $F(3,27, \alpha = 0.05) = 2.96$

(iii) Dunnett's test

C: RC tD = 2.704 $tD(3,27, \alpha = 0.05) = 2.15$

C: ABD tD = 2.817

Using the partial as control, and excluding control in the comparisons.

(i) $X^2 = 0.838$

DF = 2 $X^2(2, \alpha = 0.05) = 5.99$

(ii) F = 3.097 $F(2,20, \alpha = 0.05) = 3.49$

(iii) Dunnett's test

P: RC tD = 2.128 $tD(2,20, \alpha = 0.05) = 2.03$

P: ABD tD = 2.226

C6 C(st)

(i) $X^2 = 15.867$

DF = 3 $X^2(3, \alpha = 0.05) = 7.81$

As the means of the groups varied proportionally with their standard deviations, a log transformation was performed on the data.

Following log transformation.

(i) $X^2 = 3.065$

DF = 3 $X^2(3, \alpha = 0.05) = 7.81$

(ii) F = 13.958 $F(3,28, \alpha = 0.05) = 2.95$

(iii) Dunnett's test

C: RC tD = 6.089 $tD(3,28, \alpha = 0.05) = 2.15$

C: ABD tD = 4.776

Using the partial as control, and excluding control in the comparisons. No log transformation was necessary.

(i) $X^2 = 4.053$

DF = 2 $X^2(2, \alpha = 0.05) = 5.99$

(ii) F = 5.984 $F(2,21, \alpha = 0.05) = 3.47$

(iii) Dunnett's test

P: RC tD = 3.381 $tD(2,21, \alpha = 0.05) = 2.03$

P: ABD tD = 2.327

C7 Rus (using only groups 2,3,4)

(i) $X^2 = 12.530$

DF = 2 $X^2(2, \alpha = 0.05) = 5.99$

As the means of the groups varied proportionally with their standard deviations, a log transformation was performed on the data.

Following log transformation.

(i) $X^2 = 5.464$

DF = 2 $X^2(2, \alpha = 0.05) = 5.99$

(ii) F = 3.698 $F(2,18, \alpha = 0.05) = 3.55$

(iii) Tukey's test

RC: ABD q = 3.846 $q(3,18, \alpha = 0.05) = 3.61$

C8 Slope of Phase III (using only groups 1,3,4).

(i) $X^2 = 4.439$

DF = 2 $X^2(2, \alpha = 0.05) = 5.99$

(ii) F = 8.060 $F(2,21, \alpha = 0.05) = 3.47$

(iii) Tukey's test

C: RC q = 5.586 $q(3,21, \alpha = 0.05)=3,58$

RC: ABD q = 3.676

C9 Closing Capacities (using only groups 1,3,4).

(i) $X^2 = 14.158$

DF = 2 $\qquad\qquad$ $X^2(2, \alpha = 0.05) = 5.99$

Recent research has suggested that Tukey's test is not seriously affected by departures from normality and homogeneity of variances when there are equal sample sizes (61). So although we have violated the assumption of homogeneous variances, as we have equal sample size, we felt confident in doing a Tukey's test on the data.

(iii) Tukey's test

C: RC $q = 1.959$ $\qquad$ $q(3,21\ \alpha = 0.05) = 3.58$

C: ABD $q = 1.822$

RC: ABD $q = 0.617$

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