JCI The Journal of Clinical Investigation

RESPIRATORY MECHANICS DURING FORWARD ACCELERATION

John F. Watson, … , Neil S. Cherniack, Fred W. Zechman

J Clin Invest. 1960;39(11):1737-1743. https://doi.org/10.1172/JCI104196.

Research Article

RESPIRATORY MECHANICS DURING FORWARD ACCELERATION *

By JOHN F. WATSON, NEIL S. CHERNIACK AND FRED W. ZECHMAN †

(From the Acceleration Section, Biophysics Branch, Biomedical Laboratory, Aerospace Medical Division, Wright Air Development Division, Wright-Patterson Air Force Base, Ohio)

(Submitted for publication May 20, 1960; accepted July 28, 1960)

Tolerance to headward and footward accelerations is seriously limited by cardiovascular impairment. During forward acceleration, when the subject is so oriented that the inertial effect experienced is transverse to the long axis of the body and directed front to back, toleration is increased, but substernal pain, sensation of pressure on the chest and abdomen, dyspnea and difficulty in inspiring (1) become the limiting factors. Vital capacity and maximal breathing capacity decrease while respiratory frequency and minute volume increase (2). X-rays of the thorax reveal elevation of the diaphragm and shortening of the anteroposterior diameter resembling forced expiration (13). These changes in pulmonary physiology are similar to those that have been described during negative pressure breathing (4–7). Since the net inertial effect of forward acceleration resembles a compressive force applied to the body while the subject continues to breath ambient air, it seemed reasonable to assume that forward acceleration simulates negative pressure breathing in a manner analogous to breathing ambient air while submerged under water (5, 8). In order to test this hypothesis, measurements were made of the pressure-volume relationships and the changes in lung volumes occurring during forward acceleration.

METHODS

1. Subjects. Four healthy adult males between the ages of 23 and 30, experienced in riding the free-swinging cab of the human centrifuge at the Biomedical Laboratory, were used as subjects. Measurements were obtained while the subjects were lying on a nylon net seat facing the center of rotation with the trunk perpendicular to the inertial force, hips and knees flexed 90° and head and back tilted forward 12° from the horizontal (Figure 1).

2. Lung volumes. Vital capacity, expiratory reserve, inspiratory reserve and tidal air were measured at 1, 2, 3 and 4 G from a 9 L Benedict Roth recording spirometer with the CO_2 absorber in place. Residual volume at 1 and 4 G was determined by the method of Lundsgaard and Van Slyke (9) as modified by Rahn, Fenn and Otis (10). Residual volume at 4 G was measured by having the subject expire to his maximal end-expiratory position while at 4 G. The centrifuge was then rapidly stopped while the subject maintained this lung volume. The equilibration with 100 per cent oxygen required by the method was then carried out at 1 G. The concentration of N_2 in the expired gas sample was determined by a Waters Conley nitrogen analyzer.

The relative change in the residual air, but not its absolute volume, was measured in the following manner. At 1 G the subjects expired into a spirometer to their maximal end-expiratory position. While still in communication with the spirometer, they were then rapidly accelerated to 4 G. Any shift in the spirometer tracing then represented a change in residual volume.

3. Static relaxation pressure-volume curves. Lung volumes were measured as above and tabulated as percentage of vital capacity. To measure intrapulmonic pressure a polyethylene tube 0.5 cm in diameter and 40 cm in length was fitted into one nostril through a sponge rubber stopper. The tube was attached to a Statham strain gage pressure transducer and the results transcribed on a Sanborn multichannel recorder. Control values at rest, or 1 G, were obtained by the subject's inspiring from the maximal end-expiratory position to a given lung volume. Then while the subject held his breath at the desired lung volume, the mouthpiece was removed, and with mouth closed, glottis open and subject relaxed, intrapulmonic pressure was recorded. Each subject furnished approximately 130 pressure readings over the entire range of his vital capacity.

The above procedure was repeated in order to obtain the relaxation pressure-volume curves during forward acceleration. After a lung volume was obtained, the mouthpiece removed and the subject relaxed, the centrifuge was accelerated to 2, 3 and 4 G plateaus for 2 to 3 seconds each. Intrapulmonic pressure was then recorded for each lung volume at each G level.

In order to verify the pressure measurements obtained at each acceleration plateau, a few measurements of intrapulmonic pressure were obtained by first accelerating to the desired level and then obtaining a lung volume and recording a pressure. No essential difference was noted between the two methods. However, because of the

* Presented in part at the meeting of the American Physiological Society and Federation, Chicago, April 13, 1960.

† Department of Physiology, Miami University, Oxford, Ohio.

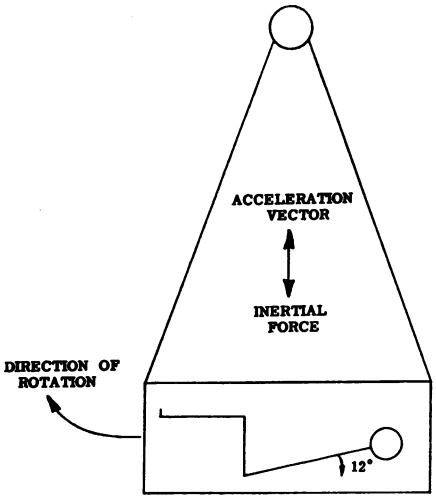

FIG. 1. ORIENTATION OF THE SUBJECT IN THE HUMAN CENTRIFUGE DURING FORWARD ACCELERATION WITH A 12° BACK ANGLE FROM THE HORIZONTAL.

difficulty in obtaining the curves in this manner, this method was not used throughout. Also, since the subjects could obtain larger lung volumes at 1 G than they could while being accelerated, the first method enabled us to extend the relaxation pressure-volume curves to 85 per cent of the 1 G vital capacity at all accelerations.

4. Dynamic pressure-volume loops. Estimation of the total pulmonary work of breathing at 1 and 4 G was obtained by measuring the areas of pressure-volume respiratory loops (11). Each subject was instructed to relax while being ventilated by a Seeler paramedic resuscitator (type XMA-4), whose line pressure and inflow resistance remained the same during both levels of acceleration. Mask pressure ranged between 0 and 30 mm of Hg at 1 and 4 G. Changes in volume were measured by electrical integration of the signal from a pneumotachygraph linear for flow rates between 0 and 107 L per minute. Pressure was measured from a tight-fitting face mask by polyethylene tubing connected to a Statham pressure transducer. The pressure-volume respiratory loops were recorded directly on an Electronics for Medicine recorder from the integrated flow signal and the mask pressure transducer signal. The recorder and the pneumotachygraph were calibrated dynamically with a constant-volume pump. The response was constant and independent of frequency. Pressure-volume respiratory loops were then recorded at both 1 and 4 G for all subjects.

The areas measured to determine the work of breathing at 1 and 4 G are shown in Figure 2. The total work was divided into its elastic and non-elastic components, and calculated by the usual method (5, 11, 12). For example, at 1 G elastic work is represented by the area ACD and the non-elastic work by the cross-hatched area. At 4 G, since the pressure-volume curve as shown in Figure 4 is displaced 15 mm of Hg to the right by the compressive force of acceleration, additional work must be done to overcome this force. Therefore, the origin of the pressure-volume loop at 4 G was placed at a positive 15 mm of Hg to illustrate this additional work of breathing. However, the recorded mask pressure at the beginning of inspiration regardless of the acceleration was actually zero. This is comparable with the method used by Rahn, Otis, Chadwick and Fenn (5) to calculate the work of respiration during positive pressure breathing. Elastic work is, therefore, calculated to be equal to the area EACD, while non-elastic work remains the cross-hatched area. Although, for simplicity, the work necessary to overcome this compressive force is included with the total

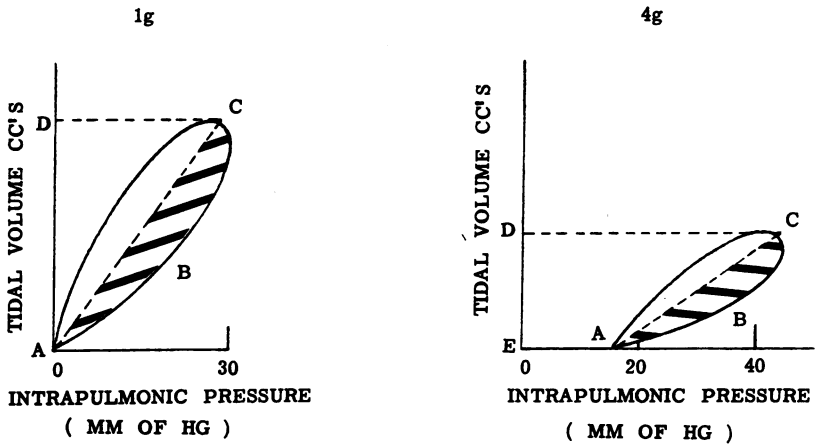

FIG. 2. SCHEMATIC PRESSURE-VOLUME RESPIRATORY DIAGRAMS TO ILLUSTRATE THE AREAS MEASURED FOR CALCULATION OF THE TOTAL WORK OF BREATHING (LUNG AND CHEST BELLOWS) DURING CONTROL (1 G) AND ACCELERATION (4 G). The loop at 4 G has been displaced 15 mm of Hg to the right to represent the shift of the pressure-volume curve by acceleration. Elastic work represents the area ACD at 1 G and EACD at 4 G, while the cross-hatched areas represent non-elastic work.

TABLE I

Changes in lung volumes during acceleration *

Acceleration	T.L.C.		V.C.		E.R.		I.R.		Tidal		R.V.	
G	*cc*	%†	*cc*	%	*cc*	%	*cc*	%	*cc*	%	*cc*	%
1	5,004		3,875 ± 343		761 ± 135		2,333 ± 191		781 ± 107		1,129 ± 143	
2			3,488 ± 437	90	502 ± 128	66	2,468 ± 183	106	518 ± 108	66		
3			3,219 ± 430	83	419 ± 123	55	2,282 ± 258	98	518 ± 108	66		
4	3,932	79	2,629 ± 460	68	360 ± 94	47	1,785 ± 310	77	484 ± 71	62	1,303 ± 165	116

* Mean of four subjects with SE. Abbreviations used: T.L.C. = total lung capacity; V.C. = vital capacity; E.R. = expiratory reserve; I.R. = inspiratory reserve; R.V. = residual volume.
† Per cent of 1 G value.

elastic work, it must be kept in mind that this work may not be elastic in its true meaning.

From these pressure-volume loops, total dynamic pulmonary compliance, i.e., the distensibility of the entire respiratory apparatus, including lungs and chest bellows, was measured from the slope of the line connecting points of zero air flow.

RESULTS

1. Lung volumes. The changes in lung volumes occurring during acceleration are shown in Table I and Figure 3 along with the standard errors and the percentages of the 1 G value. All the lung volumes decrease more or less proportionately (with the exception of residual volume which shows no significant change) by either of the two methods used.

2. Static relaxation pressure-volume relationships. The weighted mean static relaxation pressure-volume curves for 1, 2, 3 and 4 G are plotted in Figure 4, with the volumes expressed as percentage of the 1 G vital capacity. As acceleration increases, intrapulmonic pressure increases for a constant volume displacing the relaxation pressure-volume curve progressively downward and to the right. The difference between each curve by the paired *t* test is significant with a $p < 0.01$ (13).

The slopes of the relaxation pressure-volume curves for each G appear identical. However, if static compliance is measured from the end-expiratory position at each G and over a pressure change of 5 mm of Hg, the static total pulmonary compliance becomes progressively smaller as ac-

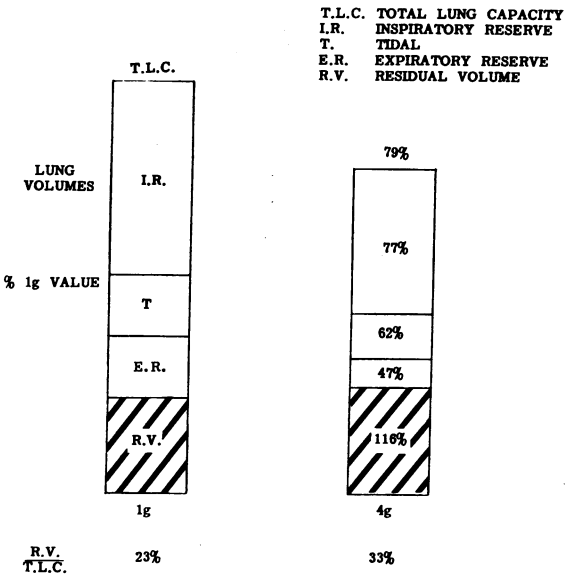

FIG. 3. SCHEMATIC REPRESENTATION OF THE CHANGES OCCURRING IN TOTAL LUNG CAPACITY AND ITS SUBDIVISIONS FROM CONTROL (1 G) TO 4 G FORWARD ACCELERATION.

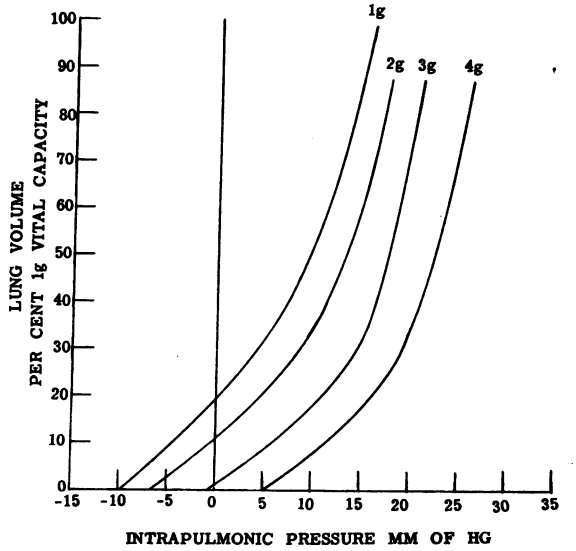

FIG. 4. THE STATIC RELAXATION PRESSURE-VOLUME CURVES DURING CONTROL (1 G), AND 2, 3 AND 4 G FORWARD ACCELERATION. All lung volumes were obtained at 1 G (see Methods).

TABLE II

*The work of breathing and total dynamic compliance * at 1 G*

Subject	Tidal	Pt	f	Ve	Work of breathing			Dynamic compliance
					Elastic	Non-elastic	Total	
	cc	*cm H_2O*		*L/min*		*kg/m/min*		*cc/cm H_2O*
J.F.	2,220	27	7	15.5	2.10	0.90	3.00	82
	2,220	27	7	15.5	2.10	0.83	2.93	82
	2,220	27	7	15.5	2.10	0.93	3.03	82
N.C.	2,070	30	10	20.7	3.07	0.62	3.69	70
	2,210	31	10	22.1	3.37	0.55	3.92	73
	2,070	30	10	20.7	3.12	0.59	3.71	70
J.W.	2,000	29	10	20.0	2.93	1.02	3.95	68
	1,840	30	13	24.0	3.55	1.31	4.86	62
	1,980	29	10	19.8	2.90	1.22	4.12	68
G.S.	1,880	28	13	24.0	3.38	1.01	4.39	68
	1,840	28	13	24.0	3.38	0.87	4.25	67
	1,900	28	13	24.7	3.47	1.50	4.97	68
Mean	2,090	29	10	20.5	3.04	0.95	3.99	72
SE				± 0.65	±0.16	±0.09	±0.20	± 2

* The distensibility of the entire respiratory apparatus, including lungs and chest bellows. Pt = intrapulmonic pressure at the end of the tidal volume; f = respiratory frequency; Ve = minute ventilation.

celeration is increased, being 80 cc per cm of H_2O (2.1 per cent of vital capacity per cm of H_2O) at 1 G and 52 cc per cm of H_2O (1.3 per cent vital capacity per cm of H_2O at 4 G). The static 1 G total pulmonary compliance agrees roughly with that reported by other investigators (5, 14, 15).

3. Total pulmonary work of breathing. Tidal volume, work of breathing and total dynamic pulmonary compliance are listed for each subject at both 1 and 4 G in Tables II and III, respectively.

Minute ventilation was constant at both accelerations. However, in order to maintain this constant and continue to be adequately ventilated during acceleration, it was found necessary to use large tidal volumes and hence minute ventilations. Since this resulted in abnormally high values for

TABLE III

*The work of breathing and total dynamic compliance * at 4 G*

Subject	Tidal	Pt	f	Ve	Work of breathing			Dynamic compliance
					Elastic	Non-elastic	Total	
	cc	*cm H_2O*		*L/min*		*kg/m/min*		*cc/cm H_2O*
J.F.	1,210	29	17	20.6	5.20	1.03	6.23	42
	1,280	29	17	21.8	5.55	0.84	6.39	44
	1,440	29	17	24.5	6.24	0.51	6.75	49
N.C.	840	30	24	20.2	8.23	0.51	8.74	28
	950	30	24	22.8	9.30	0.26	9.56	32
	950	31	24	22.8	9.30	0.39	9.69	31
J.W.	1,120	31	20	22.4	7.06	1.21	8.27	36
	1,120	31	20	22.4	7.06	1.13	8.19	36
	1,000	31	18	18.0	5.65	0.94	6.59	33
G.S.	1,020	30	16	16.3	3.87	0.77	4.64	34
	1,020	30	16	16.3	3.87	0.77	4.64	34
	1,170	30	16	18.7	4.47	0.77	5.24	39
Mean	1,093	30	19	20.6	6.32	0.76	7.08	37
				± 0.8	±0.56	±0.08	±0.52	± 2

* See footnote to Table II.

mechanical work (11), the increment of work between 1 and 4 G rather than the absolute value is meaningful.

The total work of breathing approximately doubled from 1 to 4 G, with the increase being entirely in the "elastic work" component. There was no change in non-elastic work.

The total dynamic pulmonary compliance decreased from 1 to 4 G. This change was highly significant by the method of significance of differences between means (13).

DISCUSSION

It has become evident that with the accelerations necessary for successful space flight, the subject must be oriented so that the inertial effect is transverse to the long axis of the body. In this position the anticipated rocket acceleration patterns for manned space vehicles are tolerable by humans. Indefinite tolerance to forward acceleration, however, is limited by respiratory embarrassment (1). It has been shown that the respiratory defect during transverse acceleration is primarily restrictive since the maximal breathing capacity and 0.5 second vital capacity decrease less rapidly than does the total vital capacity (2). It has also been demonstrated that oxygen consumption increases (16). Therefore, it appears that forward acceleration from a mechanical aspect applies an added load upon the muscles of respiration, decreasing lung volumes and increasing work without evidence of bronchial obstruction. The experiments reported in this paper offer further evidence for this concept.

As acceleration increases, intrapulmonic pressure increases for a constant lung volume. The increase is the same for each G regardless of the lung volume (Figure 4). This implies that while breathing ambient air the respiratory pump is working against an additional resistance which, in our studies, at 4 G is equivalent to approximately 15 mm of Hg. One might describe this accelerative force by saying that it effectively increases the weight of the thorax and lung which in turn compresses the volume of air within the alveoli. We have elected to term this force PG since it appears to be a constant for each level of acceleration. Rahn and co-workers (5) analyzed the relaxation pressure (Pr) as the algebraic sum of two fractions, the lung elasticity (Pl) and the chest elasticity (Pc) where the latter includes all of the elastic or gravitational factors other than those of the lung itself. During acceleration the relaxation pressure at each G is greater than the resting relaxation pressure by an amount equal to PG.

Non-elastic work between 1 and 4 G does not change. However, because of the additional resistance to inspiration offered by forward acceleration (PG), "elastic" and total work of breathing double. The change in compliance adds only a small fraction of this increase in "elastic" work. In other experiments, it has been observed that respiratory frequency increases while tidal volume decreases during increasing forward acceleration (16). This probably represents the optimal combination for minimizing the increase in the work of breathing (11, 17). The cause of the increased oxygen requirement during acceleration described by two of the authors (16) is in all probability partly the result of this increased work.

The static total pulmonary compliance for each G over the same volume range is the same, indicating that there is no definite change in the overall elastic properties of the thorax and lung. However, if the static compliance is measured from an end-expiratory position, it progressively diminishes with increasing acceleration. Since the relaxation pressure-volume curves (Figure 4) are parallel, this decrease is the result of the fall in functional residual capacity (FRC) and pulmonary mid-position (18–19). Dynamic compliance also decreases during acceleration and probably for the same reason. Christie (20) and others (21, 22) have shown that compliance decreases with increasing respiratory frequency when airway resistance is increased. However, we have no evidence of increased airway resistance during acceleration since there is no change in non-elastic work or timed vital capacity.

During 4 G forward acceleration the relaxation pressure (Figure 4) even at zero lung volume is positive. This was true regardless of the method used. The reasons for this are obscure. It is possible that blood pools into the pulmonary circuit upon relaxation after a forced expiration, or that during a forced expiration under acceleration there is air-trapping within alveoli, which is released upon relaxation. Regardless of the cause, it implies that inspiration during 4 G or greater for-

ward acceleration is always active, regardless of the end-expiratory position, and complete relaxation of the inspiratory muscles does not occur.

Respiratory frequency during forward acceleration increases linearly at least up to 12 G (16). Since forward acceleration resembles chest compression, the increase in frequency may in part be due to stretch or proprioceptive reflexes. Culver and Rahn (23) have demonstrated that chest compression by pneumatic cuff or Drinker respirator results in a decrease in tidal volume and an increase in respiratory frequency, both of which can be nullified by vagal blocking.

All of the lung volumes decrease proportionately during forward acceleration, with the exception of residual volume. The largest percentage of reduction is in the expiratory reserve volume. This implies a downward shift of the mid-pulmonary position, and a decrease in the FRC. Agostoni, Thimm and Fenn (24) considered the FRC as a buffer zone for maintenance of alveolar composition. They demonstrated that as the FRC decreased, respiratory frequency increased, maintaining within small limits changes in the composition of alveolar air. This relationship between respiratory frequency and FRC is evident during forward acceleration (16) and would theoretically minimize fluctuations in alveolar gas composition and rate of work.

Residual volume between 1 and 4 G does not significantly change. This is surprising, since it was expected that during forward acceleration residual volume would decrease as the result of thoracic compression and increase in pulmonary blood volume. Changes in residual volume secondary to shifts in pulmonary blood volume have been demonstrated by Fowler, Guillet and Rahn (7) using continuous positive or negative pressure breathing in a Drinker respirator. However, these changes were small, and the degree of negative pressure breathing that produced a significant change far exceeded that which would be equivalent to 4 G acceleration. During acceleration, residual volume becomes a larger percentage of total lung capacity. Whether or not this increased ratio is significant enough to be a factor in altering gas exchange remains to be evaluated. This pattern of lung volumes is the same as that seen in other situations of thoracic constriction

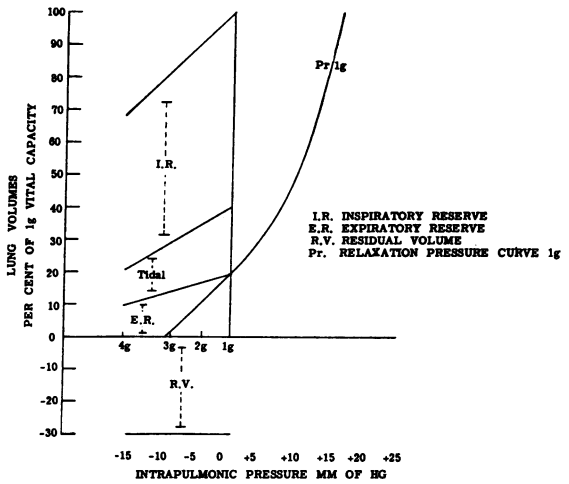

FIG. 5. PRESSURE-VOLUME DIAGRAM OF THE CHEST DURING INCREASING FORWARD ACCELERATION.

such as kyphoscoliosis and after encasing the thorax of a normal individual in a restricting corset (25, 26).

The result described here support the hypothesis that mechanically forward acceleration resembles negative pressure breathing. This may be clarified if all the existing information is combined in one diagram, using as a model the pressure-volume diagram of the chest described by Rahn and associates (5). They constructed the diagram from information obtained in relaxation pressure-volume curves, and lung volume changes as the result of negative and positive pressure breathing. We can construct a similar diagram (Figure 5) using the same information by assuming that PG at various accelerations is equivalent to a pressure applied to the body in a manner analogous to the pressure applied to the body by a Drinker respirator during negative pressure breathing. Therefore, we can place the 4 G lung volumes at a negative 15 mm of Hg, and the 1, 2 and 3 G lung volumes at their respective pressures.

It is not meant to imply that all the abnormalities occurring in respiratory physiology during forward acceleration are the result of negative pressure breathing, as inequalities of perfusion, diffusion and ventilation may be present. However, this study provides a basis for the concept that positive pressure breathing may reverse the abnormalities of respiratory mechanics and increase the tolerance to forward acceleration.

SUMMARY

The respiratory mechanics of forward acceleration were studied in four normal subjects. All of the lung volumes decreased proportionately with the exception of residual volume which became a larger fraction of the total lung capacity. The resting mid-pulmonary position decreased. As acceleration increased, intrapulmonic pressure increased for a constant volume, shifting the relaxation pressure-volume curve downward and to the right. Static and dynamic total pulmonary compliances decreased as a result of the decrease in functional residual capacity. Elastic work approximately doubled due to the work necessary to overcome acceleration resistance (PG). Nonelastic work did not change. Inspiration during 4 G acceleration was always active regardless of the end-expiratory position.

It is suggested that respiratory mechanics during forward acceleration and negative pressure breathing are similar.

REFERENCES

1. Clarke, N. P., Bondurant, S., and Leverett, S. D. Human tolerance to prolonged forward and backward acceleration. Aerospace Med. 1959, **30**, 1.
2. Cherniack, N. S., Hyde, A. S., and Zechman, F. W., Jr. Effect of transverse acceleration on pulmonary function. J. appl. Physiol. 1959, **14**, 914.
3. Hershgold, E. J. Roentgenographic study of human subjects during transverse accelerations. Aerospace Med. 1959, **31**, 213.
4. Holt, J. P. The effect of positive and negative intrathoracic pressure on cardiac output and venous pressure in the dog. Amer. J. Physiol. 1944, **142**, 594.
5. Rahn, H., Otis, A. B., Chadwick, L. E., and Fenn, W. O. The pressure-volume diagram of the thorax and lung. Amer. J. Physiol. 1946, **146**, 161.
6. Mills, J. N. The influence upon the vital capacity of procedures calculated to alter the volume of blood in the lung. J. Physiol. (Lond.) 1949, **110**, 207.
7. Fowler, R. C., Guillet, M., and Rahn, H. Lung volume changes with positive and negative pulmonary pressures *in* Studies in Respiratory Physiology, Air Force Technical Report no. 6528, August, 1951, p. 522.
8. Hamilton, W. F., and Mayo, J. P. Changes in the vital capacity when the body is immersed in water. Amer. J. Physiol. 1944, **141**, 51.
9. Lundsgaard, C., and Van Slyke, D. D. Studies of lung volume. I. Relation between thorax size and lung volume in normal adults. J. exp. Med. 1918, **27**, 65.
10. Rahn, H., Fenn, W. O., and Otis, A. B. Daily variations of vital capacity, residual air, and expiratory reserve including a study of the residual air method. J. appl. Physiol. 1949, **1**, 725.
11. Otis, A. B., Fenn, W. O., and Rahn, H. Mechanics of breathing in man. J. appl. Physiol. 1950, **2**, 592.
12. McIlroy, M. B., Marshall, R., and Christie, R. V. The work of breathing in normal subjects. Clin. Sci. 1954, **13**, 127.
13. Mainland, D. Elementary Medical Statistics. Philadelphia, W. B. Saunders, 1952, p. 149.
14. Nims, R. G., Conner, E. H., and Comroe, J. H., Jr. The compliance of the human thorax in anesthetized patients. J. clin. Invest. 1955, **34**, 744.
15. Butler, J., and Smith, B. H. Pressure-volume relationships of the chest in the completely relaxed anaesthetised patient. Clin. Sci. 1957, **16**, 125.
16. Zechman, F., Jr., Cherniack, N. S., and Hyde, A. S. Ventilatory response to forward acceleration. Fed. Proc. 1960, **19**, 376.
17. Cook, C. D., Sutherland, J. M., Segal, S., Cherry, R. B., Mead, J., McIlroy, M. B., and Smith, C. A. Studies of respiratory physiology in the newborn infant. III. Measurements of mechanics of respiration. J. clin. Invest. 1957, **36**, 440.
18. Nisell, O. I., and DuBois, A. B. Relationship between compliance and FRC of the lungs in cats, and measurements of resistance to breathing. Amer. J. Physiol. 1954, **178**, 206.
19. Cook, C. D., Helliesen, P. J., and Agathon, S. Relation between mechanics of respiration, lung size, and body size from birth to young adulthood. J. appl. Physiol. 1958, **13**, 349.
20. Christie, R. V. The elastic properties of the emphysematous lung and their clinical significance. J. clin. Invest. 1934, **13**, 295.
21. Otis, A. B., McKerrow, C. B., Bartlett, R. A., Mead, J., McIlroy, M. B., Selverstone, N. J., and Radford, E. P. Mechanical factors in distribution of pulmonary ventilation. J. appl. Physiol. 1955, **8**, 427.
22. Mead, J., Lindgren, I., and Gaensler, E. A. The mechanical properties of the lung in emphysema. J. clin. Invest. 1955, **34**, 1005.
23. Culver, G. A., and Rahn, H. Reflex respiratory stimulation to chest compression in the dog *in* Studies in Respiratory Physiology, Air Force Technical Report no. 6528, August, 1951, p. 78.
24. Agostoni, E., Thimm, F. F., and Fenn, W. O. Comparative features of mechanics of breathing. J. appl. Physiol. 1959, **14**, 679.
25. Bergofsky, E. H., Turino, G. M., and Fishman, A. P. Cardiorespiratory failure in kyphoscoliosis. Medicine (Baltimore) 1959, **38**, 263.
26. Caro, C. G., Butler, J., and DuBois, A. B. Some effects of restriction of chest cage expansion on pulmonary function in man: An experimental study. J. clin. Invest. 1960, **39**, 573.