

**AN INVESTIGATION OF THE PULMONARY FUNCTION OF
PARAPLEGICS**

Allan Hemingway, ... , Ernest Bors, Richard P. Hobby

J Clin Invest. 1958;**37**(5):773-782. <https://doi.org/10.1172/JCI103663>.

Research Article

Find the latest version:

<https://jci.me/103663/pdf>



AN INVESTIGATION OF THE PULMONARY FUNCTION OF PARAPLEGICS¹

By ALLAN HEMINGWAY, ERNEST BORS, AND RICHARD P. HOBBY

(From the Long Beach Veterans Administration Hospital, Long Beach, and the University of California Medical School, Los Angeles, Calif.)

(Submitted for publication October 21, 1957; accepted January 30, 1958)

In evaluation of pulmonary insufficiency in patients with paralytic respiratory disease, the contribution of various muscles and muscle groups is of considerable interest. The older observers, notably Keith (1), based their conclusions of the contribution of the various muscles to respiration on two types of study. One was the anatomical position of the muscle in relation to the direction of the fibers and the origin and insertion of the muscle. The other was the study of muscle function by observation or palpation of the contracting muscle. So influential has been the classical treatise of Keith that modern textbooks (2) base their teaching of respiratory muscle function on this work. The observations of recent years have consisted mainly of studies of electromyography—Campbell (3), Campbell and Green (4), Lewis (5), Dickinson (6) and Floyd and Silver (7). While these investigators have recognized the limitations of electromyography, such as the uncertainties when using surface electrodes in recognizing electrical activity of a deep muscle below a superficial muscle and the uncertainty of determining the exact location of the active tip of the less popular needle electrodes, much useful information has been obtained. In particular, electromyographic studies have revealed the association of electromyographic activity with muscle function during quiet and deep breathing, in hyperventilation, maximum inspiratory and expiratory effort against pressure, coughing, straining and in maintenance of posture. One muscle whose participation in respiration cannot be easily evaluated by electromyography is the diaphragm. As will be shown later, it is this muscle whose participation in respiration can be particularly evaluated by the present study of paraplegics.

The work reported here consists of a study of a group of patients who have varying degrees of

muscle paralysis caused by a traumatic lesion of the spinal cord. The characteristic feature of this group is that the lesion is complete and well defined, with absence of ventilatory function below the lesion and normal function above. By selection of patients with lesions at various spinal segments and testing with standard pulmonary function tests, it has been possible to evaluate the effect on breathing caused by functional elimination of the respiratory muscles below any spinal level. For patients with a "low" lesion, that is, in the lower lumbar region, the only loss of function of body muscles is that of the pelvic floor. With "high" lesions in the lower cervical region the only effective muscle remaining for respiration is the diaphragm. This method of evaluating the role of various groups of muscles as contributors to respiration differs from the usual methods of studying pulmonary muscle function mentioned above and provides supplementary information not obtainable by other methods.

METHODS

The patients studied were selected from approximately 400 patients of the Paraplegia Service of the Long Beach Veterans Administration Hospital. This particular service has special facilities for care, treatment, research, social and economic management of this type of patient. From this group 64 patients were selected for study. The selection was determined by completeness of the spinal cord lesion, freedom from complicating factors such as muscle spasm, willingness of the patient as a cooperative subject and absence of other respiratory disorders than that caused by the lesion. The patients were mostly in the 20 to 40 year age group. They were all men and were the victims of the accidents listed in Table I, the majority of these being automobile accidents. Before injury these men were in excellent health, were muscular and most were engaged in outdoor work or outdoor activities. Between the time of the injury and the time of measurement, weight loss occurred of approximately 5 to 20 per cent. The tests were made at a time after injury varying from 4 months to 10 years. The time interval between injury and testing, together with the ages of the subjects, is shown in Figure 1.

¹ Aided by a research grant (B-371) from the United States Public Health Service.

TABLE I
Number of patients tested and cause of accident

Cause of accident	No. patients	Per cent
Automobile	34	54.0
Gunshot	8	12.8
Diving	7	11.4
Falls	5	8.0
Logging	2	3.1
Airplane	2	3.1
Train	2	3.1
Mining	1	1.5
Ship explosion	1	1.5
Rupt. intervertebral disc	1	1.5
Total	63	100.0

The patients were divided into three groups according to the level of the spinal cord lesion. Group I included 21 patients with lumbar lesions; Group II, 14 patients with thoracic lesions; and Group III, 29 patients with cervical lesions. The extent of muscle paralysis of the three groups is shown in Figure 2. The members of Group I with lumbar lesions had paralysis of the pelvic floor and the lower limbs. If the muscles of the pelvic floor are excluded as contributing to respiration, then these men had respiratory functioning musculature which was normal. The members of Group II had lesions in the thoracic region. These extended from the first to the tenth thoracic level. Patients of this group were more difficult to obtain because injuries to this region of the back were fewer due to mechanical protection of the rib cage, and well defined spinal transections were less likely to occur. Group III includes the patients with lesions from the fourth to the eighth cervical segment. A few of those with the higher lesions had limited use of the arms and were unable to perform the exercise test described later. Patients in this group, as shown in Figure 2, had paralysis of all the muscles of respiration except the accessory muscles and the diaphragm.

A group of 17 normal men served as controls. These men were staff physicians, residents, interns, aids and technicians. Their age distribution is shown in Figure 1. These men were tested in the same manner as the patients except that the arterial blood studies were omitted.

There were four groups of tests made including *a*) spirometry, *b*) residual volume and nitrogen clearance during oxygen breathing, *c*) blood gas analysis and *d*) an exercise tolerance test. *a*) The spirometry tests consisted of a measure of inspiratory vital capacity, expiratory reserve volume, and maximum breathing capacity. The inspiratory vital capacity as defined here is the increase in lung volume caused by a maximal forced inspiration following a maximal expiration. The Collins 13 liter spirometer was used. Four determinations of inspiratory vital capacity were made and the largest value chosen. Nine measurements of expiratory reserve volume were made and the mean value used. Two measurements of the maximum breathing capacity were made

and the highest values used. *b*) Residual volume was measured by the open-circuit method of Darling, Courmand, and Richards (8). *c*) Arterial blood was drawn and analyzed for oxygen, carbon dioxide content and oxygen capacity using the Van Slyke manometric apparatus. Arterial pH was determined with the Cambridge glass electrode. *d*) An exercise test was devised which consisted of raising from the supine position two 5 pound hand weights, one in each hand, from the side to above the head. This weight lifting was performed once every two seconds for five minutes. This type of test was used because an arm movement exercise was the only type of exercise capable of being performed by these patients. Oxygen consumption rate, carbon dioxide production rate, respiratory quotient, ventilation rate, tidal volume, ventilation coefficients, and alveolar (end-tidal) carbon dioxide tensions were measured in a five minute resting period before the exercise, during the five minutes of exercise, and in a five minute postexercise period. Respiratory dead space was computed using the Bohr formula (9) and the values of CO₂ in expired and alveolar air. A mouthpiece with a Douglas valve was used with

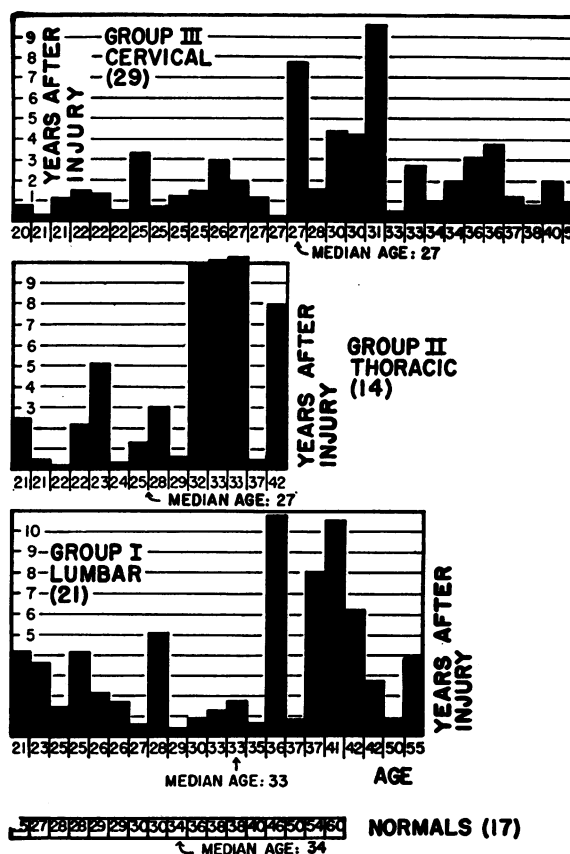


FIG. 1. AGE DISTRIBUTION, TIME INTERVENING BETWEEN INJURY AND TESTING AND NUMBER OF INDIVIDUALS (IN PARENTHESES) IN EACH GROUP

The age distribution of the 17 normal men of the control group is given at the bottom of the figure.

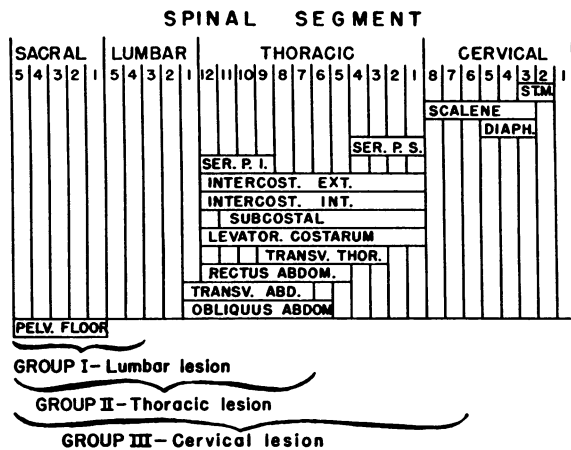


FIG. 2. ANATOMICAL EXTENT OF MUSCLE PARALYSIS IN EACH OF THE THREE GROUPS TESTED

a 120 liter spirometer for collecting expired air. Gas analyses were made with the Scholander (10) apparatus. End-tidal (alveolar) carbon dioxide was measured in two ways: 1) by collecting small aliquots of end-tidal air from approximately 10 exhalations beyond the expiratory value of the Douglas mouthpiece, or 2) passing the expired air through the seven-eighths inch tube of the Model 16 Liston-Becker carbon dioxide analyzer. The gain control and the zero control of the infra-red carbon dioxide analyzer were set immediately before the test with two carbon dioxide air mixtures of known composition. A calibration curve was made for the instrument in which instrument reading was plotted against alveolar air CO₂ determinations using the gas collection technique with chemical gas analysis. In the majority of determinations (approximately 95 per cent) both infra-red CO₂ analysis and chemical analysis were made. The chemical gas analysis values were considered more reliable and were chosen in preference to those of the CO₂ analyzer. The two (chemical and infra-red) differed only slightly with approximately 80 per cent of the two determinations differing by only plus or minus 1 mm. in the computed P_ACO₂.

RESULTS

Vital capacity

The inspiratory vital capacity of the control group was determined and from the data a linear regression formula relating vital capacity to age was derived. The formula is:

$$\text{Inspiratory vital capacity} = 2.857 - 0.101 \times \text{age} \pm 0.335 \text{ (liters, } 37^\circ \text{ C., ambient pressure saturated with water vapor).}$$

Another prediction formula was the "height formula" given by Baldwin, Cournand, and Richards

(11). Using these two prediction formulas, the predicted vital capacity of each patient was determined and from their measured vital capacity, the percentage of the predicted value was obtained. These percentages with standard deviations are given in Table II. For each group and for each prediction formula the probability of a significant difference from normal was determined using the Fisher test. In the table, if a group is significantly different from normal, it is designated S; if not significantly different, NS. It was considered significantly different if the probability of being different exceeded 99 per cent, or the probability, p, of nonsignificance was less than 0.01.

The patients of Group I (lumbar lesion) had vital capacities which did not differ significantly from normal. Group II patients (thoracic lesion) had vital capacities significantly smaller (82.9 per cent) than our control group but not significantly less than predicted by the Baldwin height formula. Group III patients had vital capacities significantly less than normal (approximately two-thirds of normal). Their vital capacities were surprisingly high considering the extensive paralysis and the availability for breathing of only one important respiratory muscle, the diaphragm.

TABLE II

Vital capacity of the three groups of paraplegics compared with the normal standard and a group of 17 normal individuals tested with the same procedure as the paraplegics

	Percentage values of the predicted (mean and S. D.)	p*
	%	
Group I (lumbar lesion)		
West formula†—22	99.4 ± 18.8	
Height formula‡—22	100.5 ± 18.3	NS
Normal group	94.0 ± 15.9	NS
Group II (thoracic lesion)		
West formula—16	90.9 ± 13.8	
Height formula—16	92.4 ± 19.6	NS
Normal group—17	82.9 ± 17.0	S
Group III (cervical lesion)		
West formula	70.3 ± 12.7	
Height formula	71.8 ± 13.1	S
Normal group	64.9 ± 10.5	S

* The letters in this column are used to indicate a significant difference (S) or no significance (NS) for a probability of being significantly different greater than 99 per cent; i.e., p less than 0.01.

† West formula, VC/M.² = 2.50 liters.

‡ Height formula, VC = (27.63 - 0.112 × age) (Ht. in cm.) (ml.).

TABLE III
Variation of expiratory reserve volume/vital capacity percentage with level of spinal injury

VC = EC + IC*		p†
EC/VC ratio	%	
Group I—22	19.0 ± 7.1	NS
Normal group—17	17.6 ± 5.2	
Group II—16	19.7 ± 4.0	NS
Normal group—17	19.5 ± 5.2	
Group III—24	8.9 ± 5.3	S
Normal group—17	18.9 ± 5.9	

* Vital capacity (VC) equals expiratory capacity (EC) plus inspiratory capacity (IC).

† NS, no significance; S, significant difference.

Expiratory reserve volume/inspiratory vital capacity ratio

It is to be expected that, with lesions of the spinal cord in the thoracic or cervical segments, the expiratory effort would be more likely to be impaired than the inspiratory effort presuming that the abdominal muscles are the powerful expiratory muscles. Considering the two components of the vital capacity as the inspiratory capacity and the expiratory reserve volume, and defining the expiratory reserve volume as the decrease in lung volume caused by a forced expiration starting from the resting expiratory level, one would expect that with abdominal muscle paralysis the expiratory reserve volume would be decreased more than inspiratory capacity. The ratio of expiratory reserve volume to vital capacity expressed as percentage is given in Table III with the probability of there being a significant difference between paraplegic and control groups. For the paraplegics with lumbar and thoracic spinal cord lesions there was no significant difference between normals and paraplegics. With cervical lesions, however, the expiratory reserve volume percentage was reduced significantly to approximately one-half.

Maximum breathing capacity

In Table IV are given the values of maximum breathing capacity expressed as percentages of the standard predicted values. These standards are those of Motley (12) and Baldwin, Cournand, and Richards (11). The maximum breathing capacity

of the control group of normal individuals was determined and a regression formula derived as a function of age. This formula is:

Maximum breathing capacity/square meter of body surface = $92.9 - 0.654 \times \text{age} \pm 13.6$ (liters, 37° C., ambient pressure saturated with water vapor).

The percentage of the maximum breathing capacity predicted by this formula is given in Table IV. The probability of a significant difference from the predicted, with $p < 0.01$, is indicated by S (see vital capacity). For Group I (lumbar lesion) the maximum breathing capacity did not differ significantly from the normal predicted values of the Baldwin formula or our control group. However, compared with the Motley formula (which has higher values than the Baldwin or our control group) the Group I paraplegics were significantly lower. For Groups II and III the maximum breathing capacity values are all significantly lower than predicted.

TABLE IV
Maximum breathing capacity as percentage of standard predicted values and predicted values from control group

		p*
Group I—21 Lumbar lesion		
Motley standard†	84.4 ± 25.3	S
Baldwin standard‡	94.5 ± 28.6	NS
Control group§	96.7 ± 28.6	NS
Group II—16 Thoracic lesion		
Motley standard	61.0 ± 15.6	S
Baldwin standard	70.4 ± 17.8	S
Control group	71.1 ± 15.6	S
Group III—24 Cervical lesion		
Motley standard	48.6 ± 15.4	S
Baldwin standard	56.3 ± 17.4	S
Control group	56.9 ± 17.4	S

* S, significant difference; NS, no significance.

† Motley standard: Maximum breathing capacity per M.² equals 97 minus age/2 (12).

‡ Baldwin standard: Maximum breathing capacity per M.² equals 86.5 minus 0.522 times age (liters B.T.P.S. per minute (11)).

§ Control group: Maximum breathing capacity per M.² equals 90.8 minus 0.68 times age.

Residual volume

The residual volume in liters B.T.P.S. per square meter of body surface for the paraplegics compared with normal controls is shown in Figure 3. A linear regression formula for the normal controls was computed and found to be:

$$\text{Residual volume} = 0.64 + 0.0073 \times \text{age} \pm 0.16$$

(liters per square meter body surface, 37° C., ambient pressure saturated with water vapor).

The regression line corresponding to this formula is shown as the center line of Figure 3 with the limits of standard error of estimate forming the two outer lines. On the same figure individual values of residual volumes for the three groups of paraplegics are indicated by points. The majority of the paraplegics have residual volumes greater than normal as is obvious from the location of the points on the graph. That they are significantly greater is shown by computation of the mean of each group and the mean of a normal group of the same age. The means are shown in the small inset table of Figure 3.

The residual volume/total capacity ratio for patients and normal controls of the same age is

TABLE V
Percentage ratio of residual volume/total capacity (RV/TC) for the three groups of paraplegics

	%	p*
Group I (lumbar lesion)—22	32.7 ± 8.0	S
Normal controls—17	26.3 ± 3.5	
Group II (thoracic lesion)—16	36.0 ± 9.2	S
Normal controls—17	25.0 ± 3.5	
Group III (cervical lesion)—24	41.3 ± 6.1	S
Normal controls—17	25.4 ± 3.5	

* Significant difference.

given in Table V. This ratio for the control group of 17 normal individuals was found to be:

$$\text{RV/TV} \times 100 = 19.50 + 0.201 \times \text{age} \pm 3.50$$

(per cent).

For each of the three groups of patients the mean ratio for a group of normals for the same age was obtained using this formula. This mean ratio for each normal group is given in Table V, the mean values for each normal group being slightly different because of differences in the mean age of each group. The residual volume/total ca-

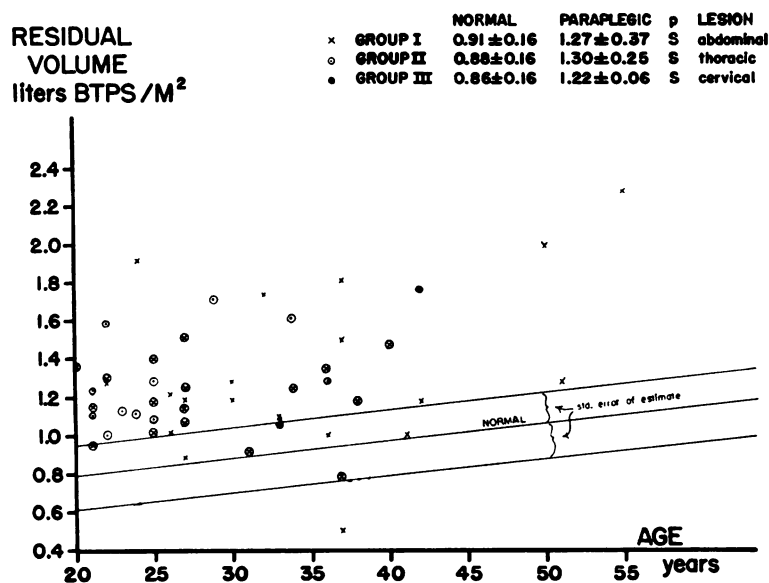


FIG. 3. VARIATION OF RESIDUAL VOLUME * PER SQUARE METER OF BODY SURFACE WITH AGE FOR NORMAL CONTROLS AND THREE GROUPS OF PARAPLEGICS

The center line is the linear regression line of the controls and the two outer lines are the limits of the standard error of estimate. Residual volume is expressed as liters at 37° C., ambient pressure, saturated water vapor.

TABLE VI
Arterial blood gases and pH for the three groups
of paraplegics *

	I Lumbar	II Thoracic	III Cervical
O ₂ content \bar{x}	17.6	17.2	17.5
σ	1.5	1.3	1.2
O ₂ capacity \bar{x}	18.8	18.3	18.6
σ	1.7	1.7	0.9
Per cent saturation of hemoglobin \bar{x}	94.4	95.4	95.8
σ	3.0	2.4	1.1
CO ₂ content \bar{x}	48.3	49.3	48.3
σ	3.6	4.5	2.5
pH \bar{x}	7.40	7.42	7.38
σ	0.07	0.09	0.08

* Oxygen content, oxygen capacity and carbon dioxide content are given as ml. of each gas STPD (0° C., 760 mm. pressure, dry) in 100 ml. of whole blood. \bar{x} is the mean value and σ the standard deviation.

capacity ratio is significantly greater than normal controls of the same age as is shown in Table V. The higher the lesion the greater is this ratio. The total capacity is the sum of vital capacity and residual volume.

Blood gases

The arterial blood oxygen and carbon dioxide, as well as arterial pH, are shown in Table VI.

Values given are the means and standard deviations for each group. These values are normal.

Exercise test

In Table VII are given the data for the five minute weight lifting exercise test. The data in rows opposite "rest" are those collected in a five minute preexercise resting period. The post-exercise period immediately following the exercise period lasted five minutes. The values in the table are the means (\bar{x}) and the standard deviations (σ) for the individuals of each group. With this "mild" exercise the oxygen consumption rate (\dot{V}_{O_2}) in ml., 0° C., 760 mm. Hg pressure, dry, per square meter of body surface per minute and the ventilation rate (\dot{V}_E) in liters, 37° C., ambient pressure, saturated with water vapor per square meter of body surface per minute, approximately doubled. As is characteristic of exercise, there was a slight rise in respiratory quotient and in ventilation coefficient (\dot{V}_{O_2}/\dot{V}_E).

Respiratory dead space

The respiratory physiological dead space values as determined by the Bohr formula (9) and using

TABLE VII
Ventilation data of normal controls and three groups of paraplegics for a five minute weight lifting exercise test *

Number		Normal		Lumbar		Thoracic		Cervical	
		\bar{x}	σ	\bar{x}	σ	\bar{x}	σ	\bar{x}	σ
O ₂ consumption rate (OCR) ml. STPD/min. M ²	Rest	124	18	121	14	124	14	132	22
	Ex	251	40	259	32	274	27	211	36
	Post Ex	141	19	153	26	155	13	145	32
CO ₂ production rate (CPR) ml. STPD/min. M ²	Rest	95	11	92	13	98	11	108	20
	Ex	210	37	200	25	227	21	180	31
	Post Ex	120	16	131	25	130	16	146	30
Respiratory quotient	Rest	77	5	76	9	79	7	82	5
	Ex	84	5	77	8	83	6	85	9
	Post Ex	85	5	85	11	83	10	83	6
Ventilation rate (\dot{V}_E) BTPS/min. M ²	Rest	3.12	0.42	3.67	0.17	3.50	0.64	3.81	0.80
	Ex	6.17	0.80	6.35	0.73	6.94	0.32	5.99	0.98
	Post Ex	3.85	0.35	4.88	0.96	4.46	0.55	4.30	0.75
OCR/ \dot{V}_E	Rest	39.8	3.5	33.4	3.4	35.3	5.9	34.7	4.2
	Ex	40.9	5.8	41.0	4.5	40.1	4.5	36.7	6.7
	Post Ex	37.0	4.9	31.5	4.4	34.3	3.4	33.8	5.0
O ₂ debt		214	13	198	20	148	64	108	60

* Each test was divided into three successive periods including a five minute preexercise (resting) period, the five minute exercise period, and a five minute postexercise period.

TABLE VIII

*Respiratory dead space (V_D) (in ml. per square meter of body surface) and the dead space/tidal volume ratio (V_D/V_T) expressed as percentage **

		Before exercise		During exercise		After exercise	
		V_D (ml.)	V_D/V_T (%)	V_D (ml.)	V_D/V_T (%)	V_D (ml.)	V_D/V_T (%)
Normal group (N = 14)	\bar{x}	87.9	31.6	110.0	23.3	87.7	24.8
	σ	9.2	3.1	25.8	7.5	17.3	5.9
Group I (N = 15)	\bar{x}	93.9	31.8	121.8	24.1	96.1	26.2
	σ	17.2	5.5	26.9	5.9	25.3	5.9
Group II	\bar{x}	98.8	29.5	94.1	17.2	97.1	27.2
	σ	20.7	5.2	20.3	4.8	16.2	4.8
Group III	\bar{x}	100.1	28.4	86.4	21.1	94.8	25.9
	σ	21.5	5.9	16.8	6.5	26.0	7.4

* \bar{x} is the average for the group and σ is the standard deviation. N is the number of individuals in the group.

carbon dioxide analyses of end-tidal (alveolar) air and expired air are given in Table VIII with values of the dead space/tidal volume ratio. For the normal control group and the patients with lumbar spinal transections the dead space volume increased with exercise and the dead space percentage of the tidal volume decreased, a finding in agreement with previous investigators (9). However, for the patients with thoracic lesions (Group II) the dead space decreased slightly (but insignificantly) with exercise, while for patients with cervical lesions the dead space fell more. If a statistical comparison is made of the dead space change with exercise and Group I, or the normal control group, compared with the Group III (cervical lesion), a statistically significant difference between the two groups ($p < 0.001$) exists.

DISCUSSION

In the management of paraplegics, respiratory dysfunction is not as serious a clinical problem as bowel and bladder regulation, decubitus ulcers and urinary infection. In tetraplegics, respiratory dysfunction occurs early in the post-traumatic phase when a tracheostomy may be needed and later if an intercurrent upper respiratory infection develops. With an upper respiratory infection, difficulty is experienced by the patient due to failure of active expectoration. Removal of tracheobronchial secretions can be aided if necessary by postural drainage, splinting of the abdomen and liquefaction of bronchial secretions with expectorants. Usually,

however, for routine activity of the tetraplegic the existing, but impaired, cough mechanism with tracheobronchial ciliary function is adequate.

It is evident from the data on vital capacity, maximum breathing capacity, exercise ventilation and blood gases that the ventilation of the paraplegics with lumbar lesions is essentially normal. This is readily explainable since the muscular paralysis of these patients is limited to that of the lower extremities and pelvic floor while the respiratory musculature is functioning normally. The patients of Group III with lower cervical lesions have paralysis of abdominal and intercostal muscles, and the sole remaining effective respiratory muscle is the diaphragm. In spite of this extensive respiratory muscle paralysis, the entire ventilation as performed by this one remaining functional muscle is surprisingly effective. Approximately, for this group, the vital capacity is two-thirds of normal and the maximum breathing capacity one-half of normal.

The expiratory reserve volume is the decrease in volume of the lung caused by a forced expiration commencing from the resting expiratory level. For this forced expiration the expiratory muscles are active participants. Since with thoracic spinal cord lesions part of the expiratory muscles are paralyzed, and with cervical lesions all are paralyzed, it is to be expected that the expiratory reserve volume would be decreased. It was found that with the normal control group and Group I (lumbar lesion) and II (thoracic lesion) the ex-

piratory reserve volume percentage of the vital capacity was approximately the same, *i.e.*, approximately 17 to 19 per cent. With Group III (cervical lesions) the expiratory reserve volume was approximately 9 per cent of the vital capacity, a reduction from the normal value of approximately one-half. The surprising finding is that these patients with no functional expiratory muscles can exhale forcibly this much air from the resting expiratory level. The reason for this forcible exhalation is not readily apparent. Possibly the shoulder girdle muscles participate in exhalation more than is generally believed. Another explanation is that the residual volume of these patients is increased approximately 500 ml., as is shown in Figure 3, which would indicate that they are breathing from a more inspiratory chest position than normal. The term resting expiratory position of the chest, although it implies minimal or no diaphragmatic muscle tonus, does not necessarily mean that such tonus is absent. The work of Culver and Rahn (13) and the earlier work on pressure breathing (14) have provided evidence that at the resting expiratory level there may be partial contraction of the respiratory musculature, and it is possible that in the abnormal condition of respiratory muscle paralysis where the diaphragm is the only muscle of respiration, an increased diaphragmatic tone can occur at the resting expiratory level. This would permit an expiratory volume of air to be exhaled from the resting expiratory level simply by relaxation of diaphragmatic tonus. Although this might explain the unanticipated high value of the expiratory reserve volume, it does not explain why the residual volume is elevated. The residual volume was found to be elevated in all of the three groups of paraplegics and did not seem to be dependent on the extent of respiratory muscle paralysis. The only paralysis common to the three groups was paralysis of the lower limbs and pelvic floor muscles. In addition all the patients led a sedentary or supine type of living while the controls were actively working. These differences do not afford a simple explanation for the increased residual volume. One explanation which is proposed reservedly is that the pelvic floor muscles support the abdominal viscera. Paralysis of these muscles withdraws some support from the abdominal vis-

cera causing the diaphragm to descend to increase residual volume. If this occurs, it imparts to the pelvic floor more of a supporting role for the viscera than is generally believed to occur.

The values observed for blood gases and ventilation studies during exercise were essentially the same for the patients as the controls. These observations indicate that the extensive paralysis of respiratory muscles causes no serious interference with blood gas exchange in the lung and that a mild exercise (equal to approximately two and a half times the resting oxygen consumption rate) can be performed without ventilatory difficulty. The fact that oxygen consumption rate of the paraplegics being essentially the same as the controls suggests that widespread denervation of the skeletal muscle does not reduce oxygen demand of the tissues. This is an observation of theoretical interest in the control of metabolism and the needs for homothermy.

In the response to mild exercise a normal individual increases his ventilation more by deep breathing than by increased respiratory rate. Associated with this increased tidal volume the respiratory physiological dead space as measured by the CO₂ method and using the Bohr formula is increased while the dead space/tidal volume fraction is reduced (9). This response with values which were similar to those in the literature were found for the individuals of the normal control group and those of Group I (lumbar lesions). With Group II (thoracic lesions) the mean respiratory dead space for the group fell slightly, the decrease being of questionable significance. With Group III, however, the respiratory dead space was reduced considerably more with exercise, the mean value decreasing from 100.1 to 86.4 (ml., 37° C., ambient pressure, saturated with water vapor per square meter of body surface). If a statistical comparison of the changes in dead space before and during exercise is made for Group I (or the normal control group) and Group III (cervical lesions) a significant difference ($p < 0.001$) for the dead space change of the two groups is found. The reason for this reduction in dead space with exercise for the Group III paraplegics is not clear. Some individuals of this group breathed irregularly and erratically during exercise. This group, more than the others, during

exercise increased their respiratory rate and their tidal volume increase was less. This irregular breathing increased the difficulty of sampling end-tidal air. However, large errors in this sampling probably did not occur since the following end-tidal mean values of CO₂ (dry, volumes per cent) before (B) and during (D) exercise were obtained: controls, (B) 5.39, (D) 5.43; Group III, (B) 4.75, (D) 4.75. The problem of measuring respiratory dead space by the CO₂ method is one on which some of the most heated controversies of exercise physiology have been waged and many of the problems are still unsettled (9). It is concluded from the observations here reported that with diaphragmatic breathing of cervical paraplegics, physiological dead space does not increase but may decrease with exercise. This might be due to the fact that diaphragmatic breathing moves the lower parts of the lung adjacent to the diaphragm while the upper part of the lung containing the large airways, and hence the dead space, remains relatively immobile.

SUMMARY

Pulmonary function studies have been made on a group of 63 paraplegic patients with complete spinal transections and a group of 17 normal controls. The patients were divided into three groups: 1) those with lumbar spinal lesions who had paralysis of the lower limbs and pelvic floor but no paralysis of the muscles of breathing; 2) those with thoracic spinal lesions with paralysis of the lumbar muscles and lower intercostals; and 3) those with lower cervical spinal lesions with paralysis of all muscles of breathing except the diaphragm and the accessory muscles of respiration. Measurements were made of vital capacity and components, residual volume, maximum breathing capacity, arterial blood gases and pH, and ventilation during exercise. It was found that the vital capacity and maximum breathing capacity of patients with lumbar lesions were normal, while the group with lower cervical lesions had a vital capacity approximately two-thirds of normal and a maximum breathing capacity of one-half of normal. Paraplegics with thoracic lesions had vital capacities and maximum breathing capacities between the values for the

cervical and lumbar lesions groups. The resting expiratory reserve volume expressed as a percentage of the vital capacity was below the normal percentage only for the group of paraplegics with cervical lesions. For this group the percentage value was one-half normal, which is higher than was anticipated for patients with only inspiratory muscle function. Residual volumes of all groups were significantly higher than the normal group. Arterial blood gases of all groups were normal and the ventilatory response to exercise for all groups of paraplegics did not differ from normal.

ACKNOWLEDGMENT

The authors gratefully acknowledge the cooperation and assistance of Dr. Ben Klaumann, formerly Chief of the Pulmonary Function Laboratory, and Dr. Donald Leik, Chief of Medicine, of the Veterans Hospital. Technical assistance was ably rendered by Dr. Wayne Hull, Lucille Baumgartner, Beverly Lim, Ray Power and Wilson Henderson.

REFERENCES

1. Keith, A. The mechanism of respiration in man *in* Further Advances in Physiology, Leonard Hill, Ed. London, Arnold, 1909, p. 182.
2. Schmidt, C. F. Medical Physiology, Philip Bard, Ed. St. Louis, Mosby, 1957, page 287.
3. Campbell, E. J. M. An electromyographic study of the role of the abdominal muscles in breathing. *J. Physiol.* 1952, 117, 222.
4. Campbell, E. J. M., and Green, J. H. Variations in intra-abdominal pressure and the activity of abdominal muscles during breathing. *J. Physiol.* 1953, 122, 282.
5. Lewis, L. The role of the abdominal musculature in respiration. Conference on Respiratory Physiology in Poliomyelitis, National Foundation for Infantile Paralysis, Los Angeles, 1955.
6. Dickinson, D. G. An electromyographic study of some of the muscles used in respiration. Conference on Respiratory Physiology in Poliomyelitis, National Foundation for Infantile Paralysis, Los Angeles, 1955.
7. Floyd, W. F., and Silver, P. H. S. Electromyographic study of patterns of activity of the anterior abdominal wall muscles in man. *J. Anat. (Lond.)* 1950, 84, 132.
8. Darling, R. C., Cournand, A., and Richards, D. W., Jr. Studies on the intrapulmonary mixture of gases. III. An open circuit method for measuring residual air. *J. clin. Invest.* 1940, 19, 609.

9. Rossier, P. H., and Buhlmann, A. The respiratory dead space. *Physiol. Rev.* 1955, **35**, 860.
10. Scholander, P. F. Analyzer for accurate estimation of respiratory gases in one-half cubic centimeter samples. *J. biol. Chem.* 1947, **167**, 235.
11. Baldwin, E. D., Cournand, A., and Richards, D. W., Jr. Pulmonary insufficiency: I. Physiological classification, clinical methods of analysis, standard values in normal subjects. *Medicine* 1948, **27**, 243.
12. Motley, H. L. The use of pulmonary function tests for disability appraisal: Including evaluation standards in chronic pulmonary disease. *Dis. Chest* 1953, **24**, 378.
13. Culver, G. A., and Rahn, H. Reflex respiratory stimulation by chest compression in the dog. *Amer. J. Physiol.* 1952, **168**, 686.
14. 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.