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Relationship Between Respiratory Muscle Strength and Vital Capacity during Partial Curarization in Awake Subjects

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To determine the relationship between respiratory muscle strength and changes in normal vital capacity (VC), graded levels of muscle weakness were produced in six healthy supine male subjects by four successive doses of *d*-tubocurarine (0.05 mg/kg each). The maximal effect of *d*-tubocurarine abolished hand-grip strength and ability to sustain head lift for 5 s, but VC was decreased to only 66 ± 3 per cent of control. At each level of weakness decreases in VC were significantly less than were decreases in respiratory muscle strength (RMS) monitored by maximum static inspiratory and expiratory pressures. The first dose decreased RMS to 86 ± 3 per cent of control, but VC was unchanged. Following the second dose, VC (97 ± 1 per cent of control) was minimally affected compared with RMS (71 ± 4 per cent). The VC after the third dose was still 85 ± 3 per cent of control, while RMS had decreased to 58 ± 2 per cent. Following the final dose of *d*-tubocurarine (cumulative total 0.20 mg/kg), RMS was 39 ± 2 per cent of control, compared with VC, 66 ± 3 per cent of control. The relationship between VC and RMS was curvilinear and conformed to predictions based on the static mechanical characteristics of the normal respiratory system. These findings demonstrate that while VC is relatively spared during partial curarization, this sparing of VC does not indicate a similar extent of preserved RMS. Rather, it reflects the relative ease with which weakened respiratory muscles are able to drive the normal respiratory system in the supine subject. The same weakened muscles may be unable to generate sufficient force to handle mechanical challenges such as coughing and vomiting. (Key words: Lung: compliance; function; vital capacity. Measurement techniques: neuromuscular blockade. Monitoring: ventilation. Neuromuscular relaxants: *d*-tubocurarine.)

WEAKNESS OF THE RESPIRATORY MUSCLES is commonly seen in patients who have neuromuscular disease and during recovery from general anesthesia in which muscle relaxants have been used. This decreased respiratory muscle strength is usually associated with a reduced vital capacity (VC).[1,2] The loss of VC has traditionally been used to monitor the extent of weakness and to predict impending respiratory difficulties.[3] Since the extremes of inspiration and expiration required for the VC maneuver are achieved with maximal respiratory muscle effort, one might assume that decrements in VC would relate directly to the extent of respiratory muscle weakness. However, the shape of the pressure–volume curve of the normal relaxed respiratory system is curvilinear near the extremes of vital capacity, such that large pressure changes are associated with small changes in volume.[4] This predicts that marked decreases in respiratory muscle strength (*i.e.*, the ability of respiratory muscles to generate pressure) should occur before VC decreases significantly. In patients who have chronic neuromuscular disease the decreases in VC fail to conform to this prediction because of coexistent pulmonary disease[5] and skeletal deformities.[6] However, in normal seated subjects acute respiratory muscle weakness resulting from infusion of *d*-tubocurarine (*d*Tc) also produced decreases in VC that exceeded predictions at various levels of weakness.[7] Additional data obtained in supine subjects are inconclusive because during curarization weakness progressed abruptly from very slight to severe.[8]

The decrepancies between theoretical and observed changes in VC with acute muscle weakness may result from several factors. Among these are decreased compliance of the lung, altered length–tension behavior in curarized muscles, and erroneous assumptions about normal respiratory system recoil. In the present study, by producing progressive partial paralysis with *d*Tc, we sought to test these hypotheses and to further examine the relationship between decreased respiratory muscle strength and vital capacity in the absence of pulmonary disease.

Methods

Six healthy non-smoking men (ages 23–32 years) served as subjects. All had normal baseline pulmonary function and were familiar with maximal respiratory maneuvers. The protocol was approved by the Human Studies Committee of the University of Virginia Medical Center, and each subject gave informed consent. The experiment was performed with the subject in the supine position. A large rubber mouthpiece together with a nose clip maintained an airtight seal during all maneuvers. Vital capacity (VC) and its subdivisions, inspiratory capacity (IC) and expiratory reserve volume (ERV), were measured with a waterless rolling-seal spirometer (model 840, Ohio Medical Products). The spirometer was calibrated with a 3-liter super-

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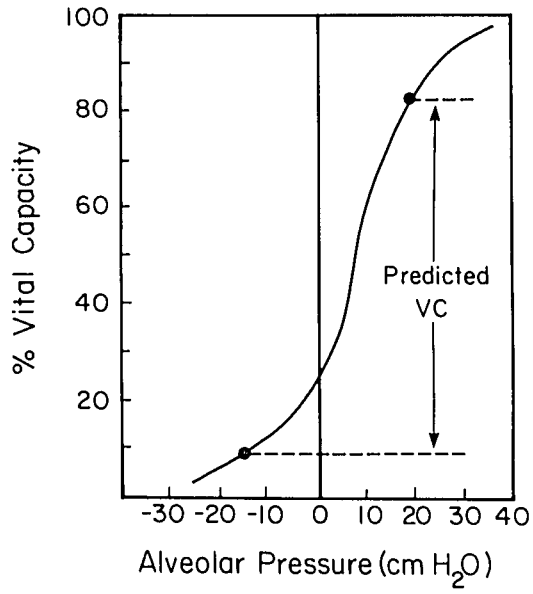

FIG. 1. Respiratory system recoil curve for Subject 5 illustrates the method of predicting changes in vital capacity (VC). The static pressure–volume relationship of the total respiratory system was obtained by measuring mouth (alveolar) pressure as the subject relaxed at various lung volumes between the extremes of VC. At functional residual capacity this pressure is zero. In this subject, when respiratory muscle strength was decreased to 60 per cent of control, the predicted VC was 82 per cent of control. The two points along the pressure–volume curve represent the alveolar pressure 60 per cent of maximum in each direction and define the maximum inspiratory and expiratory lung volumes that should be achieved. The vertical distance between the two points defines the expected VC.

syringe and provided a volume accuracy of ±30 ml. All volumes were converted to body temperature and pressure saturated with water vapor (BTPS).

We assessed respiratory muscle strength by measuring mouth pressure during maximal inspiratory and expiratory efforts against an occluded mouthpiece (a small 2-mm orifice in the mouthpiece produced a small leak to minimize artifacts produced by the facial muscles). The pressures measured were maintained for at least 1.0 s. Maximum static inspiratory ($P_{I_{max}}$) and maximum static expiratory pressure ($P_{E_{max}}$) were measured with a differential pressure transducer (Validyne MP-45, range ± 250 cm H_2O) and recorded with lung volume on the strip-chart recorder (Gould 220). Maximal static inspiratory and expiratory efforts were made at total lung capacity (TLC), residual volume (RV), functional residual capacity (FRC), FRC + 1 liter, and FRC + 2 liters. Values for $P_{I_{max}}$ and $P_{E_{max}max}$ were corrected for respiratory system recoil pressure (P_{rs}) to derive the actual pressure developed by the respiratory muscles (P_{mus}). At FRC, P_{rs} is zero, and $P_{I_{max}}$ and $P_{E_{max}}$ are equal to P_{mus}.

Respiratory system recoil pressure (P_{rs}) was estimated from measurements of mouth pressure during stepwise exhalation from total lung capacity (TLC) to residual volume (RV). Subjects inspired fully to TLC and relaxed as completely as possible against an occluded mouthpiece for 2–3 s. Then, as they exhaled slowly, the mouthpiece was occluded at intervals of about 500 ml and the relaxation maneuver again performed. Mouth pressure sensed by a differential pressure transducer (Validyne MP-45, range ± 50 cm H_2O) and lung volume (VC) were recorded simultaneously on a strip-chart recorder (Gould 220). Following each deflation maneuver, subjects breathed in and out as far as possible to establish the extremes of VC and to relate them to the volumes at which mouth pressures were recorded. At least five deflation maneuvers were performed by each subject. A line of best fit was drawn by eye through the plots of pressure and volume between TLC and RV after small volume corrections for compression or expansion of gas in the lung.[2]

Since the performance of the VC maneuver requires maximum effort on the part of both inspiratory and expiratory muscles, we utilized an index that incorporates the effects of both muscle groups. Respiratory muscle strength (RMS) during partial curarization was calculated as the average of $P_{I_{max}}$ and $P_{E_{max}}$, expressed as percentage of control values for each.[9] Measurements of $P_{I_{max}}$ and $P_{E_{max}}$ at FRC were used for the calculations. The FRC was assumed to remain constant throughout the study.

Values for VC were predicted from P_{rs} curves obtained from supine subjects.[4] The method for constructing these predicted curves is illustrated on the P_{rs} curve for Subject 5 (fig. 1), whose RMS was reduced to 60 per cent of control. When this reduced RMS of the actively driven respiratory system is translated to the relaxed or passively stretched respiratory system, an alveolar pressure that is 60 per cent of the maximum in either direction will define the maximum inspiratory and expiratory lung volumes. These two points are indicated on the respiratory system recoil curve. The vertical distance between them defines the VC that should result with this degree of weakness provided respiratory system recoil is not altered by curarization.

The prediction of VC using RMS assumes the $P_{I_{max}}$ and $P_{E_{max}}$ are decreased equally, which is often not the case. However, we tested a model in which the decline in RMS was due solely to either $P_{I_{max}}$ or $P_{E_{max}}$ and one in which $P_{E_{max}}$ was reduced more than $P_{I_{max}}$ (as in this study). In either case the predicted VC did not differ from that obtained using RMS expressed as the average of $P_{I_{max}}$ and $P_{E_{max}}$.

To estimate lung recoil, esophageal pressure was measured by use of a balloon–catheter system. The 10-cm-long balloon, containing 0.5 ml air, was passed

into the mid-esophagus and connected to a differential pressure transducer (Validyne MP-45, range ± 50 cm H_2O) by polyethylene (PE 200) tubing 100 cm long. The opposite transducer port was connected to a pressure tap in the mouthpiece. Transpulmonary pressure ($P_{st}L$) was expressed as mouth pressure minus esophageal pressure. Compliance of the lung was estimated by expiratory quasi-static pressure–volume curves. Lung volume was plotted against pressure on a direct-writing X-Y recorder (Hewlett-Packard 7041A) as subjects inhaled to TLC and then slowly exhaled (about 0.2 l/sec) to RV. A line of best fit was drawn through at least two closely agreeing curves with the greatest inspiratory volume and transpulmonary pressure. Lung recoil pressure ($P_{st}L$) was plotted at 10 per cent intervals of control VC, and compliance of the lung estimated as the slope of the curve 0.5 l above FRC (about 25–35 per cent of VC). The pressure–volume curves were measured in the control state and following the final dose of *d*Tc.

The ability to sustain head lift for 5 s and hand-grip strength (Lafayette Dynamometer) were measured to estimate non-respiratory-muscle strength. Blood pressure was checked intermittently and the electrocardiogram monitored continuously throughout the study.

Following measurements of P_{rs} and several trial runs to obtain constant baseline measurements for VC and maximum static respiratory pressures, subjects received 0.6 mg atropine intravenously to reduce salivary secretions. Fifteen minutes after the atropine administration, control estimates of VC, $P_{I_{max}}$, and $P_{E_{max}}$ at the various lung volumes were obtained, and quasi-static pressure–volume curves constructed to measure lung recoil. All control values for VC, $P_{I_{max}}$ and $P_{E_{max}}$ are the means of three determinations. During the period of *d*Tc administration all values for $P_{I_{max}}$ and $P_{E_{max}}$ were the results of single determinations. The VC and its subdivisions each represent the averages of two determinations, one prior to and one immediately following pressure measurements.

Progressive acute muscle weakness was produced by a total dose of 0.2 mg/kg *d*Tc, given intravenously during a 28-min period as four doses of 0.05 mg/kg at intervals 7 min apart. The RMS and VC were measured during a period of peak drug effect 4–6 min following each dose.[10,11] This quasi-static steady state of drug effect at each level of weakness was verified by VC measurements at the beginning and end of each period. (Values for VC varied less than 5 per cent). Following measurements after the last dose of *d*Tc, subjects were allowed to recover partially for 10–15 min, then residual weakness was reversed by intravenous administration of atropine (1.0 mg) and neostigmine (2.5 mg).

Results were analyzed using Student's *t* test for paired data. $P < 0.05$ was considered significant. Standard least-squares linear regressions and logarithmic regressions were used to compute correlations.

TABLE 1. Changes in Respiratory Muscle Strength (RMS), Vital Capacity (VC), and Non-respiratory-muscle Strength during Progressive Partial Curarization (Means ± SEM Expressed as Percentages of Control)

	Grip	Head Lift†	RMS	VC
Dose 1	89 ± 1*	+	86 ± 3*	100 ± 1
Dose 2	67 ± 3*	+	71 ± 4*	97 ± 1
Dose 3	15 ± 5*	+	58 ± 2*	85 ± 3*
Dose 4	3 ± 2*	−	39 ± 2*	66 ± 3*

* Significant difference from control, $P < 0.01$, by *t* test for paired data.
† Head lift = ability to sustain head lift for 5 s.

Results

Baseline vital capacity in the six subjects was 6.1 ± 0.4 l (mean ± SEM), while $P_{I_{max}}$ and $P_{E_{max}}$ measured at FRC were 135 ± 12 and 124 ± 12 cm H_2O, respectively. Total doses of *d*Tc administered to individual subjects averaged 16.7 mg. The intial dose (0.05 mg/kg) produced slight diplopia. Grip strength and RMS decreased slightly, while VC was unchanged (table 1). Following the final dose of *d*Tc (cumulative total 0.20 mg/kg), subjects experienced difficulty swallowing and were unable to raise their heads. Hand-grip strength was too slight to measure (<1.0 kg) in three of the six subjects. At this maximal level of peripheral muscle weakness, RMS was 39 ± 2 per cent of control, while VC was 66 ± 3 per cent of control.

At all levels of partial curarization, RMS was decreased significantly more than VC ($P < 0.01$) when expressed as percentage of control. The curvilinear nature of the relationship between RMS and VC is evident in figure 2. A line fitted to the individual data points was best described by the logarithmic curve $y = 36.5\ (1n\ x) - 64.6$ ($r = 0.85$; $P < 0.0001$). The mean values for VC were slightly but significantly higher than those predicted from P_{rs} curves in supine subjects ($P < 0.05$) after the first two doses of *d*Tc. Although this predicted curve (*B*) was constructed from the data of Agostoni[4] for P_{rs}, predictions based on P_{rs} data for the subjects in this study did not differ significantly.

At all levels of weakness, expiratory muscle strength ($P_{E_{max}}$) was significantly more depressed (table 2) than was inspiratory muscle strength ($P_{I_{max}}$). Therefore, we examined the relationships between $P_{I_{max}}$ and IC (fig. 3), and between $P_{E_{max}}$ and ERV (fig. 4). As can be seen, neither departed significantly from the predicted relationship.

Muscle forces (P_{mus}) developed at lung volumes

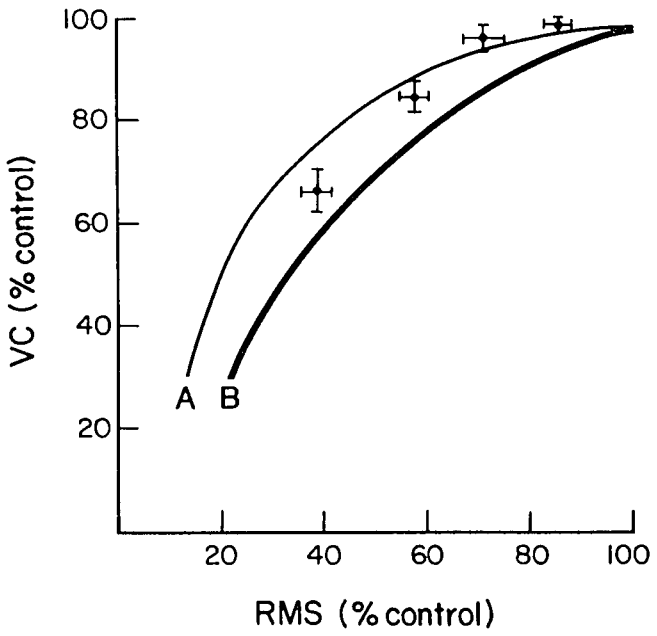

FIG. 2. The effects of decreased respiratory muscle strength (RMS) on vital capacity (VC). All values are means ± SEM expressed as percentages of control. Curve *A* indicates the theoretical effect of respiratory muscle weakness on VC, predicted by the respiratory system recoil from data of Agostoni[4] in seated subjects, while curve *B* describes the relationship in supine subjects.

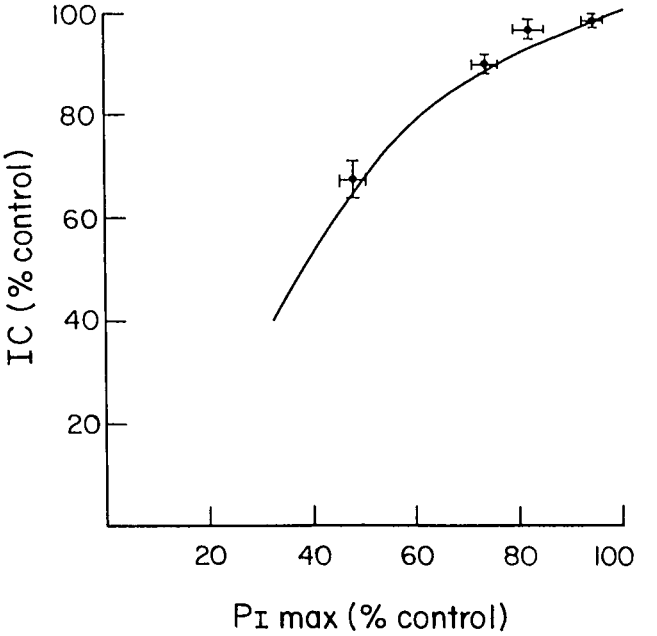

FIG. 3. The effects of decreased inspiratory muscle strength ($P_{I_{max}}$) on inspiratory capacity (IC). Values are plotted as means ± SEM expressed as percentages of control. The solid line indicates the theoretical relationship predicted from the data of Agostoni[4] using the inspiratory portion of the respiratory system recoil curve (between functional residual capacity and total lung capacity).

more than and less than FRC following the last two doses of *d*Tc are shown in figure 5. Decreases in expiratory and inspiratory pressures did not differ significantly from predictions, which assumed decrements in P_{mus} at all lung volumes similar to those measured at FRC.

At the time of maximal muscle weakness, mean $P_{st}L$ was 14 cm H_2O at full lung inflation (66 per cent of control VC). This value was 45 per cent of $P_{st}L$ at control TLC (fig. 6). Mean $P_{st}L$ at a comparable control lung volume was 11 cm H_2O, a value that did not differ significantly from the value for curarized subjects ($P > 0.05$). Values of $P_{st}L$ at FRC averaged 3.5 cm H_2O in the control state and did not change significantly following partial paralysis. Similarly, control lung com-

TABLE 2. Respiratory Muscle Strength during Progressive Partial Curarization (Means ± SEM Expressed as Percentages of Control, Measured at Functional Residual Capacity)

	$P_{I_{max}}$	$P_{E_{max}}$
Dose 1	94 ± 2	75 ± 6*
Dose 2	82 ± 3	63 ± 7*
Dose 3	75 ± 2	40 ± 4*
Dose 4	48 ± 3	29 ± 2*

* Significant difference ($P < 0.01$) compared with $P_{I_{max}}$ (Student's *t* test for paired data).

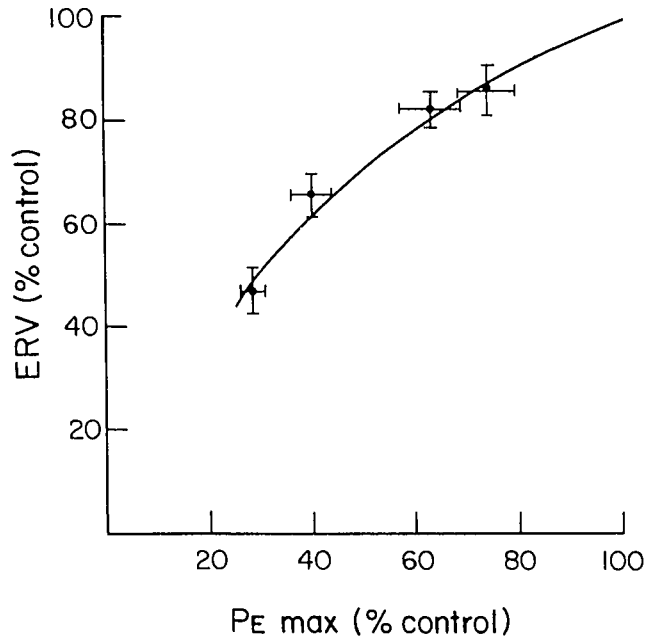

FIG. 4. The effects of decreased expiratory muscle strength ($P_{E_{max}}$) on expiratory reserve volume (ERV). Values are plotted as means ± SEM expressed as percentages of control. The solid line indicates the theoretical relationship predicted from the data of Agostoni[4] from the expiratory portion of the respiratory system recoil curve (between functional residual capacity and residual volume).

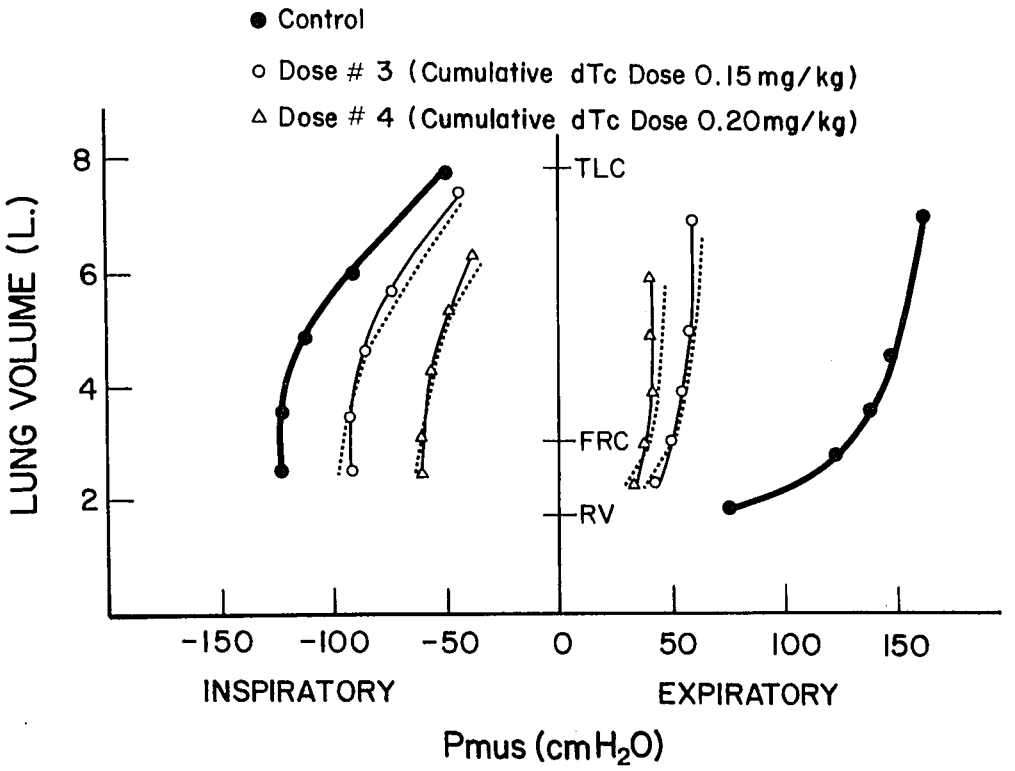

FIG. 5. Maximum static inspiratory and expiratory muscle pressures (P_{mus}) plotted at lung volumes between residual volume (RV) and total lung capacity (TLC). Mean control values are compared with those after the third and fourth doses of *d*-tubocurarine. Dotted lines are theoretical vlaues constructed by assuming decrements of P_{mus} at all lung volumes proportional to those at functional residual capacity (FRC).

pliance (0.35 ± 0.02 l/cm H_2O) was unchanged by partial curarization (0.33 ± 0.33 l/cm H_2O).

Discussion

The scheme of *d*Tc administration in this study produced graded levels of weakness to an extent that virtually abolished hand-grip strength and ability to sustain head lift. The VC at this point was still about two-thirds of its control value. Of itself this suggests considerable sparing of respiratory muscle function. On the other hand, the more direct assessments of respiratory muscle strength with $P_{I_{max}}$ (48 ± 3 per cent of control) and $P_{E_{max}}$ (29 ± 2 per cent) reflected far greater degrees of weakness. These findings are significant for two reasons. First, they indicate that measurements of $P_{I_{max}}$ and $P_{E_{max}}$ are not only specific but also more sensitive than VC in monitoring respiratory muscle function, particularly at slight levels of weakness. Second, they help to further clarify the concept of "respiratory sparing" suggested when VC is used to monitor partial curarization. The respiratory muscles are not as weak as are peripheral muscle groups, but they are also not spared to the extent suggested by loss of VC. Rather, the apparent preservation of VC reflects the relative ease with which weakened respiratory muscles are able to drive the normal respiratory system in the supine position.

The results of this study differ from observations in seated curarized subjects, in whom loss of lung volumes reflected respiratory muscle weakness more directly.[7] Several possibilities exist to explain this difference. First, the normal P_{rs} curve obtained with the subject in the supine position is slightly steeper than that obtained while the subject is sitting.[4] Thus, the curve describing the normal relationship of muscle pressure to

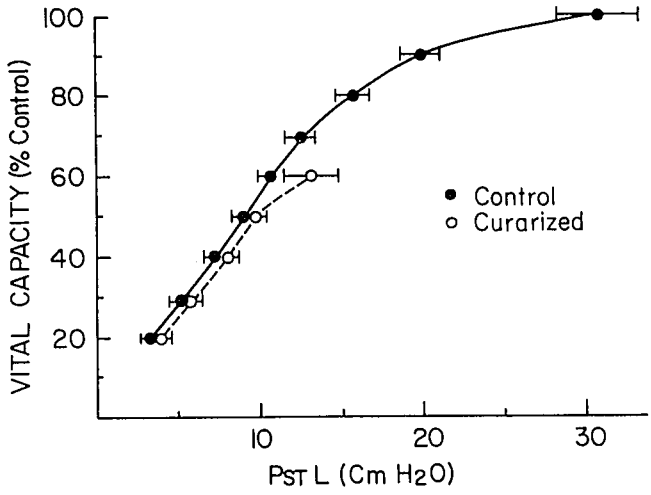

FIG. 6. Quasi-static deflation (expiratory) pressure–volume curves before (control) and after the final (fourth) dose of *d*-tubocurarine. Transpulmonary pressure ($P_{st}L$) is plotted as mean ± SEM for the six subjects at 10 per cent intervals of control vital capacity.

volume is not as curvilinear (fig. 2, line *B*) compared with the curve for the seated position (fig. 2, line *A*). The FRC is also less in supine subjects, such that inspiratory capacity comprises a greater portion of the VC. Not only is the diaphragm more efficient in the supine position because of increased abdominal compliance,[12] but the inspiratory muscles in turn are less affected by curarization (table 1). Furthermore, in the seated curarized subjects reduced inspiratory volume was reflected primarily by reduced rib-cage expansion. Normal maximal inspirations while seated are accomplished predominantly by this increase in rib-cage dimensions, but the latter is far less important in the supine position.[13] Finally, the theoretical curve relating RMS and VC assumes that respiratory system recoil is unaltered by muscle weakness. Data recently obtained in seated subjects given pancuronium intravenously suggest that respiratory system recoil was altered by decreased outward pull of the chest wall.[14] The result was a rightward shift of the P_{rs} curve not unlike that seen in the supine position. Since VC in our supine subjects conformed to predictions, it appears reasonable to assume that the normal supine P_{rs} curve was not altered significantly by curarization.

Another factor that must be considered concerns the use of pressure measurements made at FRC to estimate muscle strength. Normally, maximum static expiratory force is achieved at TLC, where the expiratory muscles are optimally stretched prior to contraction. Similarly, at RV the inspiratory muscles are most stretched and maximum inspiratory force is developed. It is reasonable to wonder whether *d*Tc affected the respiratory muscles to different extents at lung volumes other than FRC, which are associated with other muscle lengths. Our data (fig. 5) indicate that the percentage reductions in force for curarized muscles were approximately the same at all lung volumes. This suggests that curarization reduced tension to the same extent at all muscle lengths.

A final factor that must be addressed is a possible change in FRC. In this study we assumed that FRC remained constant during the period of partial curarization. DeTroyer *et al.*[14] demonstrated a 15 per cent decrease in FRC in seated subjects during slight neuromuscular blockade with pancuronium.

They attributed the reduction in FRC to alterations in the pressure–volume curve of the chest wall.‡ In the lower portion of VC the tendency of the chest wall to recoil outward was decreased, whereas at greater lung volumes its inward pull was increased. This same phenomenon occurs as a normal consequence of gravity as one changes from upright to supine postures, except that total respiratory system compliance also increases. Further reductions in lung volume, however, are mediated principally through the diaphragmatic component of the chest wall. Complete diaphragmatic paralysis is associated with a further cephalad displacement of the diaphragm and reduced supine FRC.[15] However, since such extreme weakness was not present in our subjects, significant changes in FRC are unlikely. Indeed, values obtained by measurements of FRC with multiple-breath N_2 washout in six similar supine subjects curarized to a point of abolition of head lift and hand grip did not differ significantly from the mean value of 2.80 ± 0.17 l in the control state. The largest change found was a 210-ml increase in FRC.§ In the present study $P_{st}L$ at FRC did not change significantly with curarization, supporting the likelihood that FRC was unchanged. Small changes in FRC may have caused slight variations in estimates of IC and ERV, but would not affect the relationship between RMS and VC. Changes in maximum static pressure–volume relationships would likewise be negligible.

Patients who have chronic neuromuscular disease involving the respiratory muscles have marked decreases in compliance of the lung and increases in lung recoil pressure.[1] Similar changes have been seen in normal subjects whose lung volumes were reduced acutely by chest strapping.[16] In this study the reduced lung volumes produced by partial curarization did not increase lung recoil pressure ($P_{st}L$), nor was compliance of the lung altered in the range of normal tidal ventilation just above FRC. However, the decreased ability to inflate the lungs completely was associated with decreased $P_{st}L$ on full inflation, *i.e.*, loss of maximal distending pressure. Because of normal pressure–volume behavior, hysteresis tends to reduce expiratory compliance measured immediately below this point, and hence flatten out the pressure–volume curve.

These observations of the respiratory system during partial curarization provide insight concerning the effects of acute muscle weakness on respiratory function. Unfortunately, the data do not allow us to reach firm conclusions regarding the level of neuromuscular weakness associated with adequacy of ventilation. The dysphagia seen in the subjects after the final dose of *d*Tc suggests an inadequate ability to overcome upper airway obstruction and remove secretions, despite an ability to maintain a major portion of normal VC. The mean levels of VC (48 ml/kg) and $P_{I_{max}}$ (65 cm H_2O) at maximum levels of weakness were far in excess of

‡ Chest wall refers not only to the rib cage but to the diaphragm, abdominal contents, and abdominal wall as well.[4]

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minimal values for VC (15–20 ml/kg) and $P_{I_{max}}$ (20–25 cm H_2O) needed for "adequate" respiratory function.[17] Although these clinical measurements in patients reflect muscle weakness, they are influenced to a great extent by decreased cooperation because of pain, sedation, and residual anesthesia. As such, they would not be expected to approximate the values achieved by maximal effort in highly motivated volunteers.

In conclusion, we have shown that normal supine subjects during acute muscle weakness produced by administration of *d*Tc undergo reductions of VC that are significantly less than decreases in RMS. This relative sparing of VC is in agreement with predictions based on normal pressure–volume relationships, and reflects the ease with which weakened muscles are able to drive the normal respiratory system. The agreement between observed and predicted values also reflects the lack of significant change in lung recoil, and suggests that total respiratory system recoil was similarly unaffected by partial curarization.

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References

1. Gibson GJ, Pride NB, Newson-Davis J, et al: Pulmonary mechanics in patients with respiratory muscle weakness. Am Rev Respir Dis 115:389–395, 1977
2. Black LF, Hyatt RE: Maximal static respiratory pressure in generalized neuromuscular disease. Am Rev Respir Dis 103: 641–650, 1971
3. Ferris BG, Warren A, Beals CA: The vital capacity as a measure of spontaneous breathing ability in poliomyelitis. N Engl J Med 252:618–621, 1955
4. Agostoni E: Statics, The Respiratory Muscles. Edited by EJM Campbell, E Agostoni, J Newson-Davis. Philadelphia, W. B. Saunders, 1970, pp 63–69
5. Harrison BDQ, Collins JV, Brown KGE, et al: Respiratory failure in neuromuscular disease. Thorax 26:579–584, 1971
6. Caro CG, DuBois AB: Pulmonary function in kyphoscoliosis. Thorax 16:282–290, 1961
7. Saunders NA, Rigg JRA, Pengelly LD, et al: Effect of curare on maximum static PV relationships of the respiratory system. J Appl Physiol 44:589–595, 1978
8. Gal TJ, Smith TC: Partial paralysis with *d*-tubocurarine and the ventilatory response to CO_2: an example of respiratory sparing? ANESTHESIOLOGY 45:22–28, 1976
9. Braun NMT, Rochester DF: Muscular weakness and respiratory failure. Am Rev Respir Dis 119 (suppl):123–125, 1979
10. Foldes FF, Monte AP, Brunn HM, et al: Studies with muscle relaxants in ananesthetized subjects. ANESTHESIOLOGY 22: 230–235, 1961
11. Unna KR, Pellikan EW, Macfarlane DW, et al: Evaluation of curarizing drugs in man. I. Potency, duration of action and effects on vital capacity of *d*-tubocurarine, dimethyl *d*-tubocurarine, and decamethylene. J Pharmacol Exp Ther 98: 318–324, 1950
12. Goldman MD, Grassino A, Mead J, et al: Mechanics of the human diaphragm during voluntary contraction. J Appl Physiol 44:840–848, 1978
13. Sharp JT, Goldberg NB, Druz WS, et al: Relative contributions of rib cage and abdomen to breathing in normal subjects. J Appl Physiol 39:608–618, 1975
14. DeTroyer A, Bastenier-Geens J: Effects of neuromuscular blockade on respiratory mechanics in conscious man. J Appl Physiol 47:1162–1168, 1979
15. Froese AB, Bryan AC: Effects of anesthesia and paralysis on diaphragmatic mechanics in man. ANESTHESIOLOGY 41: 242–255, 1974
16. Stubbs SE, Hyatt RE: Effect of increased lung recoil pressure on maximal expiratory flow in normal subjects. J Appl Physiol 32:325–331, 1972
17. Pontoppidan H, Geffin B, Lowenstein E: Acute respiratory failure in adults. Boston, Little, Brown, 1973, p 60