TITLE: RELATIONSHIP BETWEEN RESPIRATORY MUSCLE STRENGTH AND VITAL CAPACITY DURING CURARE-INDUCED WEAKNESS

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INTRODUCTION: Weakness of respiratory muscles reduces vital capacity (VC). The shape of the normal respiratory system recoil curve is such that respiratory muscle strength (RMS) should decrease markedly before VC falls. Studies in curarized volunteers indicate that the relation between RMS and VC fails to conform to predictions in seated subjects[1]. Data in supine subjects is inconclusive[2]. Possible explanations include undetected alterations in lung compliance and aberrant length-tension behavior in curarized muscles. The present study sought to test these hypotheses and to further clarify relationships between RMS and VC during progressive paralysis with d-Tubocurarine.

METHODS: Six healthy males (mean age 25) were studied supine. Informed consent and approval by the Human Studies Committee were obtained. Subdivisions of VC were measured spirometrically. Respiratory system recoil (Prs) was recorded from mouth pressures during stepwise deflation from total lung capacity (TLC) to residual volume (RV). Expiratory quasi-static P-V curves with an esophageal balloon estimated lung recoil. Respiratory muscle strength (RMS) was assessed by measuring mouth pressure during maximum efforts against an obstructed mouthpiece. Maximum static inspiratory pressure (P_I max) and maximum static expiratory pressure (P_E max) were recorded at several lung volumes between RV and TLC. These were corrected for Prs to yield pressure actually developed by the muscles (Pmus). The index RMS was taken as (P_I max + P_E max/2 measured at functional residual capacity (FRC). Head lift and hand grip ability (dynamometer) reflected peripheral muscle strength. Control measurements were made 15 minutes after subjects received atropine 1.0 mg intravenously. Then dTc 0.2 mg/kg was given intravenously as 4 doses of 0.05 mg/kg each 7 minutes apart. RMS and VC were measured during a quasi-steady state of maximal drug effect 4-6 minutes after each dose.

RESULTS: All levels of curarization decreased RMS significantly more than VC. Dose #3 reduced grip to 30% of control; RMS was 50% and VC 82% of control. Dose #4 abolished head lift and hand grip; RMS at 42% of control was significantly less ($P<0.01$) than VC (61% control). Relationships between VC and RMS conformed to predictions based on Prs (Figure 1). Inspiratory Capacity (IC) and Expiratory Reserve Volume (ERV) also were similar to values predicted by P_I max and P_E max respectively. Curarization reduced Pmus at all lung volumes to the same extent as at FRC. The acute reduction in lung volume during curarization did not change lung compliance.

DISCUSSION: Although partial curarization decidely reduced VC, decreases in RMS were significantly greater until severe levels of weakness were reached. The relationship between VC and RMS closely agreed with predictions from Prs curves in supine subjects. With curarization, the length-tension relation of weakened muscles had the same general shape as the normal curve except for reduction in contractile force. Therefore, estimation of RMS with measurement at FRC can be extrapolated to respiratory muscle strength at other lung volumes. We conclude that acute curarization in normal volunteers has no effect on lung compliance or respiratory system recoil other than that explainable by the muscle weakness alone.

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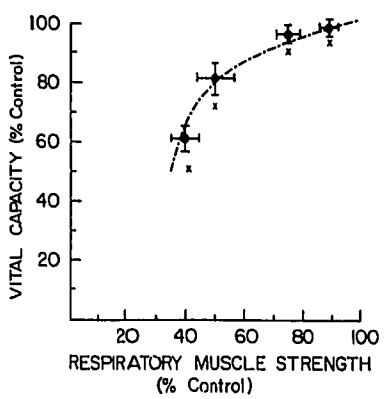

Figure 1: VC plotted as function of RMS in six subjects at four levels of paralysis with dTc. Values are percent of control (mean ± SEM). Dotted line is predicted relationship from data of Agostoni[3] for recoil of respiratory system (Prs). X's are mean VC's predicted from Prs curves of our subjects.