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TITLE: CHANGES IN PULMONARY FUNCTION TESTS (PFT) DURING HIGH-DOSE LUMBAR EPIDURAL ANESTHESIA (LEA)

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Cephalad local anesthetic spread may result in high-level abdominal (AB)/intercostal (IC) muscle paralysis. Inspiratory (insp) compromise (IC paralysis and less effective zone of apposition mechanics due to AB paralysis) and expiratory (exp) compromise (IC and AB paralysis) may occur.

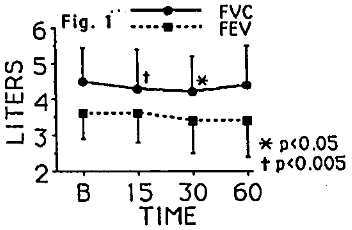
Previous investigations of LEA and spinal anesthetics have found small or no reductions in PFT in small patient groups who received heterogeneous anesthetics. We previously found more profound chest wall distortion when high-dose LEA was used compared to spinal anesthesia[1]. We therefore investigated high-dose LEA temporal effects on PFT.

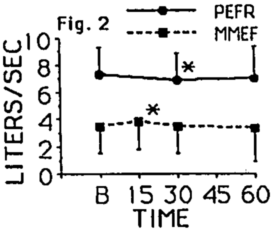
After institutional approval and written informed consent, we studied 22 consecutive patients (ASA I-II), age 17 - 37 having cruciate ligament repairs. No premedication or sedation were given. 25 - 30 mL 2% lidocaine with 5 μg/mL epinephrine was injected over 2 min. at L3-4 in the right lateral decubitus position. Exp and insp abilities were evaluated by 2 forced exhalations into a computerized spirometer (Collins) followed by an exp/insp flow-volume curve by Wedge spirometry with X-Y output to an oscilloscope. PFT data were collected at baseline and 15, 30, and 60 min following anesthetic injection. Statistical analysis was by paired T-test.

Mean anesthetic level at 30 min was T-5.6 to pinprick (T2 - T10). Large decreases in all parameters were seen in some patients. Maximum individual % decreases in forced vital capacity (FVC) = 17.7%, forced expiratory volume 1-sec (FEV1) = 30.9%, peak expiratory flow rate (PEFR) = 30.3%, maximum mid-expiratory flow rate 25-75% (MMEF) = 26.5%, and peak inspiratory flow rate (PIFR) =36.6%. As expected, for the group, there was no statistically significant decrease in PIFR since the main insp muscles (diaphragm and accessory neck muscles) are intact during LEA. There were significant decreases in the more effort-dependent exp parameters, FVC and PEFR (Fig 1 and 2), which depend on intact exp muscles. This effect regressed along with recession of sensory level by the 60 min measurement (Fig 1). Mean FVC decreased 176 mL ($p<0.02$) and PEFR by 0.34 L/sec ($p=0.05$). The more effort- independent parameters showed no significant change (FEV1) or a significant increase (MMEF = 0.26 L/sec) which we believe was due to systemic epinephrine effect, as it peaked at 15 min (Fig. 2) and coincided with increases in heart rate and blood pressure. This subtle but significant effect of epinephrine on exp flow occurred even in this healthy patient population and merits further investigation.

Despite previous demonstrations of large decreases in maximal AB and thoracic pressures with cough during spinal or LEA[2,3], actual changes in PFT are smaller. This is evidence that very little exp muscle function is necessary to reach effort-independence of exp flow limitation. Therefore mainly airway conduction properties govern PFT results even with high-dose or high level conduction anesthesia.

References:
1. Regional Anesthesia 15:1S:70, 1990
2. Anesthesiology 22:882, 1961.
3. Regional Anesthesia 13:1S:41, 1988





A1155

TITLE: BLOOD-GAS SOLUBILITY AND ACINAR ANATOMY JOINTLY INFLUENCE DEADSPACE

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INTRODUCTION A computer model based on a numerical solution of the classic single-path airway convection-diffusion equation for a normal lung (Weibel's Model A), including a source term allowing for the evolution of CO_2 from the blood to the alveolar air, produces CO_2 expirograms that closely approximate those from normal human subjects. Despite absence of changes in the anatomy of the conducting airways (i.e. no change in V_DANAT), expirograms produced by models that contain alveolated airways of reduced cross sectional area show increases in V_DBOHR in proportion to the degree of narrowing. To help elucidate the influence of airway narrowing on blood gas exchange, we investigated the retention and excretion of six inert gases with partition coefficients (pc) between 0.0076 and 333, produced by models with reduced airway area and with redistribution of acinar blood flow.

METHODS The control was based on Weibel's model A. A narrowed model (Area/3) was created by reducing the cross sectional areas of the seventeenth through the twenty-third generations by a factor of three while FRC was held constant by increasing the length of the narrowed airways. Two different blood flow patterns were investigated. In WMA, blood flow was distributed to the alveolated generations in proportion to the number of alveoli per generation reported by Weibel. In WMT, all blood flow was to the 23rd generation. For each of the four models expirograms were generated and the end-tidal, arterial, and mixed expired concentrations of CO_2 and the six inert gases calculated. In all cases V_T was 500 ml, RR 10, cardiac output 5 liters/min, FRC 2.5 l, and mixed venous CO_2 pressure 45 mm Hg.

RESULTS As the trumpet was narrowed the V_DBOHR for WMA increased from 156 ml to 242 ml and for WMT from 156 ml to 251 ml. Retention and excretion fractions for the six inert gases are plotted for WMA in Figure 1. Narrowing of the airways caused a small increase in the retention excretion differences for all of the test gases. The effect of narrowing was most apparent for gases with a partition coefficient between one and ten. Retention and excretion fractions for WMT are plotted in Figure 2. The retention excretion differences are considerably larger in this model than in WMA for all gases with a partition coefficient greater than one.

DISCUSSION Narrowing of the distal alveolated airways increases deadspace as surely as enlarging the volume of the conducting airways, but the increase may be offset by redistribution of acinar blood flow towards the mouth. The change in deadspace produced by redistribution of blood flow is more pronounced for soluble than for insoluble gases.