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12/8/102473

In vivo estimation of septal lung tissue volume and correlation with diffusing capacity in lung volume reduction surgery

To the Editor

We read with great interest the recent paper by J. C. Chen and associates[1] about the diffusing capacity limitations of the extent of lung volume reduction surgery (LVRS) in animal models of emphysema. The authors induced diffuse emphysema by aerosol elastase, a model similar to the homogenous type of human emphysema. However, patients with emphysema who are good candidates for LVRS tend to have heterogeneous targeted areas for resection,[2] as Cooper has mentioned.[1] In these patients, improvement in respiratory system compliance is prominent even after resection of a large volume of the lung. In contrast, diffusing capacity deteriorated when the resected volume exceeded a threshold. In the setting of major lung resection, diffusing capacity may predict the postoperative morbidity and mortality.[3] We believe that the importance of diffusing capacity in LVRS needs to be emphasized. The goal of LVRS should be a balance between improving mechanical function of the lung and diaphragm without excessive loss of diffusing capacity or of the pulmonary vascular bed. We congratulate Chen and associates for raising this important issue.

In Dallas, we[4,5] have performed extensive studies to determine the diffusion limitation after major lung resection at rest and during exercise. Here, we would like to introduce a method of assessing the diffusing capacity and septal lung tissue volume in vivo using combined radiologic and physiologic techniques. We believe this approach has potentially important applications in LVRS. With the use of an acetylene and a carbon monoxide rebreathing method, lung air volume, tissue volume, diffusing capacity, and cardiac output can be simultaneously and noninvasively measured.[4] In addition, tissue volume and air volume were also separately estimated by computed tomographic (CT) scan, from which topologic distribution of tissue and air volumes are obtained.[6] We compared tissue volume measured by these 2 techniques in immature dogs at different ages. Half the dogs had undergone resection of the right lung; the other half had undergone thoracotomy without lung resection. We[6] found significant correlations ($P < .01$) between tissue volume measured by CT and rebreathing and between tissue volume and diffusing capacity in both groups (Fig 1, *A* and *B*). These data suggest that tissue volume is an anatomic correlate of gas exchange capacity.

The article by Chen and associates reinforces the point that the key functional parameter of gas exchange is not total lung volume, but diffusing capacity and tissue volume. Measurement of diffusing capacity and tissue volume may aid the functional evaluation of patients with emphysema, although their predictive value in the setting of LVRS requires further investigation. For example, preoperative measurement of diffusing capacity and tissue volume by the rebreathing method could identify patients with insufficient gas exchange reserves who would not benefit from LVRS regardless of improvements in mechanical lung and respiratory muscle function. In addition, one could potentially use CT scan to map out the topologic distribution of tissue volume and to target regions with a low tissue volume (high air/tissue volume ratio) for resection.

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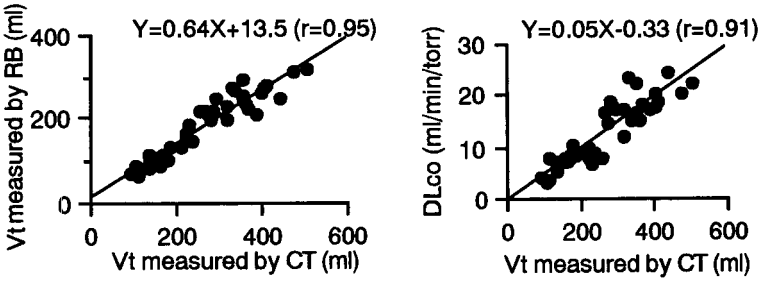

Fig 1. Measurement of tissue volume and diffusing capacity by computed tomography and rebreathing. *Vt*, Tissue volume; *RB*, rebreathing; *CT*, computed tomography; DL_{CO}, diffusing capacity of carbon monoxide.

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Pulmonary surfactant and cardiopulmonary bypass in infants

To the Editor:

We read with interest the recent article by Paul and colleagues[1] describing changes in pulmonary surfactant after cardiopulmonary bypass (CPB) in a group of infants having surgery for congenital heart disease.

In this article the authors report concentrations of phospholipid and, indirectly, protein in returned fluid from tracheal lavage. There is no apparent attempt to correct concentrations for variable recovery of epithelial lining fluid in these specimens. To define the concentration of surfactant components in sampled secretions, a marker of dilution should be used, allowing the result to be expressed as concentration in epithelial lining fluid.[2] Results expressed as concentrations in raw lavage fluid are impossible to interpret meaningfully.

Paul and colleagues do report the phospholipid/protein ratio of tracheal lavage specimens. This ratio does nothing to clarify the data and certainly cannot be interpreted as an attempt to correct for dilution, given their later statement that alveolar protein concentration is known to be increased after CPB. A useful marker of dilution of epithelial lining fluid must not be present in increased concentration in the damaged lung. For this reason protein (along with albumin and sphingomyelin) is not suitable in this population.[3]

Paul and colleagues report a significant fall in total phospholipid concentration immediately after CPB. In their discussion they state: "Our data support the findings of McGowan and colleagues, who demonstrated an alteration in surfactant composition in older infants and children after CPB." In fact, these findings are at odds with those of McGowan and colleagues,[4] who found no difference in total phosphatidylcholine recovered by bronchoalveolar lavage before and after CPB. In the other published study looking at phospholipid after CPB in children, LeVine and colleagues[5] showed no difference in phosphatidylcholine levels between a group of children who had undergone CPB and a control group. Both of these studies involved greater numbers of patients having CPB than in that of Paul and colleagues, and both are also subject to the same criticism of not appropriately correcting results for dilution.

There may well be significant abnormalities of pulmonary surfactant that contribute to postoperative lung dysfunction in this patient population. McGowan and colleagues[4] did find a change in the proportion of phospholipid in pulmonary surfactant subtypes after CPB (a measurement not influenced by dilution of specimens), which would have important functional implications. This subject warrants further investigation, but care must be taken to express findings in a way that will add to our understanding of the consequences of CPB on the composition and function of pulmonary surfactant.

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12/8/102476

Reply to the Editor:

We thank Millar and colleagues for their interest in our article, "The Role of Cardiopulmonary Bypass and Surfactant in Pulmonary Decompensation after Surgery for Congenital Heart Disease."[1] We concur that our data have certain limitations and must be interpreted with a degree of caution. Although we did not account for the variable recovery of epithelial lining fluid by using a marker of dilution, we made every effort to standardize the timing and technique of the lavage fluid with each procedure. Although a measurement of dilution may have been helpful, even this method has potential limitations[2] and, as mentioned in the letter, our method is similar to that used in other studies in children undergoing bypass for congenital heart disease.[3,4] Millar and colleagues also pointed out the potential limitations to using total protein as the denominator. However, as stated in our conclusions, we