ter clarify the role of video-assisted thoracoscopic surgery (VATS) in the treatment of thymic diseases and related disorders.

We all understand the enthusiastic optimism for this new therapeutic modality, but we do not completely agree with the authors. VATS is by its very nature limited, and it cannot achieve a comprehensive resection of thymic tissue as it is required in the treatment of myasthenia gravis in patients with or without known thymoma. The multicentric nature of thymomas or failure to remove all nonneoplastic thymic tissue has been claimed to be responsible for poor outcome of the operation in some myasthenic patients and for occurrence of recurrent or second primary tumor. Because of the wide cervical and intrathoracic extent of thymic tissue,1,2 we believe that VATS cannot adequately search for and remove potential ectopic thymic tissue, especially if it is performed only through a hemithorax as reported by Yim and colleagues (CHEST 1995; 108:1440-43).

In fact, VATS does not offer a wide exposure to the pleural cavities, mediastinum, and cervical spaces, which is particularly important in surgery of the thymus because the thymus requires an aggressive surgical approach. It has been demonstrated that as little as 2 to 3 g of residual thymic tissue can be severely symptomatic and its removal therapeutic.3 In conclusion, we believe that, at present, thoracoscopic does not represent an effective alternative to transcervical-transsternal maximal thymectomy.

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REFERENCES


To the Editor:

We appreciate the comments from Drs. D’Andrea and Biancardi on thoracoscopic thymectomy. We agree with them that complete thymectomy is important in the surgical treatment of myasthenia gravis. Prior to thoracoscopic, we resected the thymus through a median sternotomy. We have found that we are now able to perform an equivalent operation through the thoracoscopic approach by examination of the thymic bed and the resected specimen. Our initial experience was very encouraging, and the combined experience from a few centers using this approach was gathered for a presentation at the American Association for Thoracic Surgery meeting in the Spring of 1996 by Dr. Michael Mack.

Limited access is traditionally thought of as the equivalent to limited exposure. This has changed revolutionarily with the development of video assistance and selective one-lung ventilation. We are cautious in advocating this new approach as our experience is still limited and our data preliminary. However, we do not believe that a procedure should be rejected just because it utilizes minimal access. We are closely following up our patients to see if the long-term results are equivalent to that provided by the other approaches.

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Pitfall in the Pco2 Gradient

To the Editor:

 Liu and colleagues (CHEST 1995; 107:1218-24) present interesting data on the relationship of end-tidal to arterial Pco2 during exercise. In interpreting their findings, they emphasize the close inverse linear correlation of the difference between the end-tidal and the arterial carbon dioxide concentration (PET-aCO2) with the ratio of the dead space to the tidal volume (VD/VT). Unfortunately, two methodologic flaws mar their statistical analysis. Each subject contributed about ten data points to the regression analysis (from two different experimental conditions), violating the assumption of the method that individual data are independent of one another. The authors acknowledge this faux pas but downplay its importance. A more serious violation is the regression of two calculated variables (VD/VT, PET-aCO2), which share a measured variable (PaCO2) in common. This practice can result in an apparent correlation where none exists or can spuriously amplify a modest correlation,1 as we believe to be the case here (CHEST 1995; 107:1218-24) and elsewhere.2,3

To illustrate this pitfall, we computed the Pearson correlation coefficient (r) for PET-aCO2 on VD/VT using computer-generated values for the three measured variables, PetCO2, PaCO2, and PeCO2 (mixed expired Pco2). We simulated 84 data points (the number in Fig 1 by Liu et al) using pseudorandom numbers drawn from the following distributions: PetCO2=38±6 mm Hg, PaCO2 =35±4 mm Hg, and PeCO2=30±4 mm Hg. Using principal components analysis, the three sets of numbers were constrained to show no correlation (pairwise r=0). These random data yielded a significant correlation (r=0.35, p<0.001) between the CO2 gradient and "physiologic" dead space, which is solely due to the method of calculation, i.e., the use of PaCO2 to derive both the independent and dependent variables. In a real data set there would also be experimental measurement error, which could further increase the apparent "correlation" because of mathematical coupling of common error.4

To mimic the use of repeated measures from the same subject in the regression, we simulated the data by Liu et al using 12 values for each of 7 "subjects" (n=54). The intersubject correlations between the 3 variables (n=7) were again zero, but we constrained the intrasubject r for each variable (n=12) to approximately 0.50. Adding this degree of nonrandom intrasubject "error" improved the apparent r between CO2 gradient and dead space to -0.41 (p<0.001).

We do not doubt that the relatively high r values reported by Liu et al, Luft et al,2 and Lewis et al also included a component due to true correlation of the variables in question. Such a relationship is physiologically reasonable and would be predicted for incremental exercise (CHEST 1995; 107:1218-24) or for patients showing a wide range of VD/VT at rest;4 however, it is likely to be much weaker than reported in these studies!

Of the three mechanisms offered by Liu et al to explain values of PetCO2 which exceed PaCO2, the first is questionable, because one expects "slow" lung units to contribute proportionately less to expired gas as breathing frequency increases during exercise. Their second explanation could account for less negative, but not positive, values of PetCO2. Their third consideration approaches the mark: during exercise CO2 production, CO2 tension in mixed venous blood, and VT all increase, resulting in greater CO2 excretion per breath and larger tidal swings in PaCO2. This is the main