the role of TBNA in the mediastinal staging of NSCLC. Even more false-negative, lymphocyte-based TBNA aspirates can be observed in patients with sarcoid LNs, a condition in which the use of a 19-gauge histology needle makes it much easier to recover granulomas. By considering these data, we tend not to rely exclusively on an adequate negative TBNA cytology specimen—even in the presence of lymphocytes only—to rule out a more specific diagnosis mainly if the clinicoradiologic picture is evocative.

In conclusion, we think that EBUS can be a useful tool to guide TBNA in some specific settings, such as “difficult mediastinal LN areas” (mainly 2, 3, 4L) and small LN size (< 1 cm), although the technique is costly and requires a considerably long apprenticeship. For the time being, conventional TBNA has to be considered less costly, easier to learn, and offers similar yields in the LN stations more frequently involved by NSCLC, among those accessible to the technique.

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References


Measuring the Work of Exercise

To the Editor:

Drs. Irvin and Kaminsky (January 2004) have stated that “... often there is a discordance between work (watts) and [oxygen uptake] V̇O₂ in clinical testing, making the interpretation and final determination of exercise tolerance difficult.” The authors suggested that normal exercise should be predicted as the maximal number of watts rather than the maximal V̇O₂.

Our exercise laboratory uses the predicted maximal V̇O₂ based on ideal body weight, and we have had good agreement between the percent predicted values for these two measurements in our patient population. I think that discrepancies in these two measurements may be due to the usage of inappropriate predicted equations or weights, especially in obese patients. It makes sense that the measured work on a cycle ergometer (expressed in watts) is only part of the patient’s total work done. Additional work by the muscles of respiration and upper arm musculature cannot be measured at the pedals but can be measured as V̇O₂. If there are no technical errors, a discrepancy between maximal work done at the pedals (watts) and V̇O₂ may actually be important information indicating a significant component of nonleg work. Therefore, I suggest that readers not necessarily abandon the use of maximal V̇O₂ as a measure of total work done.

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Reference

1 Irvin CG, Kaminsky DA. Exercise for fun and profit. Chest 2004; 125:1–3

To the Editor:

We thank Dr. Kirsch for his comments regarding the use of work vs oxygen uptake (V̇O₂) to assess exercise tolerance. Most of the time, these two measures are in close agreement in terms of percent predicted and can be used interchangeably as objective measures of exercise tolerance. Dr. Kirsch correctly points out some of the reasons for the discrepancies between these two measures, such as the selection of appropriate predicted values, especially in obese individuals, and the performance of different types of exercise. Most studies relating exercise capacity to important outcomes such as survival or the ability to tolerate lung resection surgery use V̇O₂ as the measure of exercise capacity because it reflects the physiologic health of the individual in terms of their global ability to utilize oxygen. Our suggestion that work may be a more appropriate measure of exercise tolerance is based on our view of exercise expressed as power output (work per unit of time) rather than oxygen utilization. Indeed, many subjects achieve a percent predicted for work that is higher than their maximal percent predicted V̇O₂, likely indicating a motivational ability to sustain exercise beyond the anaerobic threshold. In real-world terms of the ability to perform various tasks and activities, the amount of work someone is able to do seems more relevant than the amount of oxygen they can consume. In this regard, defining exercise tolerance in terms of work is also more appropriate when prescribing exercise or explaining the results of exercise testing to patients. As we stated in the editorial, we invite more discussion in this area of the definition of exercise tolerance.

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25-Year Study of Lung Fibrosis Following Carmustine Therapy for Brain Tumor in Childhood

To the Editor:

Our group in Manchester, UK, previously reported the existence of active and progressive lung fibrosis in patients up to 20 years after undergoing chemotherapy with high-dose carmustine therapy for brain tumors in childhood.1,2 We now present follow-up data for these patients up to 25 years from the date of carmustine treatment (mean time, 23 years; range, 18 to 25 years).

Nine of 17 brain tumor survivors (53%) have died of pulmonary fibrosis. Two survivors (12%) died within the first 3 years after undergoing chemotherapy, four more died between 6 and 13 years after treatment, and another three have died since then (ie, 13 to 25 years after treatment). Patients who were treated at an earlier age seemed to be at greater risk of developing pulmonary fibrosis. Of the eight patients still alive, we have follow-up data for seven, and they all have radiologic and physiologic (ie, lung function) evidence of upper zone pulmonary fibrosis. These patients have been followed up with serial lung function data at the North West Lung Centre at Wythenshawe Hospital, as presented in Figure 1 (which includes the lung function data of two other patients who received follow-up treatment and survived the first 13 years following treatment but have died since then). Three of the patients have had stable lung function for >2 decades, but the rest have slowly progressive restrictive defects.

Patients to whom carmustine was given in childhood developed an unusual progressive upper zone fibrosis. This model continues to give insight into progressive pulmonary fibrosis in which an early transient insult results in progressive pulmonary fibrosis. The rate of progression depends on the severity and timing of the insult, and probably on genetic profibrotic factors.

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REFERENCES

Figure 1. Serial FVC measurements of the study patients.