The letter by Drs. Tessler and Kuper (see page 1408 in this issue) and the editorial by Banner et al. (see page 1141 in this issue) raise some salient points that we would like to address. First, we wholeheartedly agree with Tessler and Kuper in their assertion that “an individualized treatment plan and close follow-up will result in safe and rapid weaning for a majority of patients.” We also agree that work of breathing (WOB) measurements should be reserved for those patients whose clinical courses have been particularly complicated and/or those patients who have repeatedly failed weaning attempts. The literature has long demonstrated that simple weaning parameters can predict successful extubation in better than 90% of patients. The difficult population to evaluate involves those patients with chronic lung disease in whom the negative predictive value of these simple weaning parameters is less than 60%. It is this population that may remain intubated and ventilated longer than physiologically necessary and who may benefit from more sophisticated bedside monitoring of pulmonary mechanics. This statement is, of course, based on the assumption that a positive impact on outcomes can be demonstrated in controlled trials, which is not yet the case. Meanwhile, it falls to the bedside clinician to be fiscally responsible in the application of all such expensive, high-tech monitoring devices.

We have some concerns about the issues and questions raised by Banner et al (see page 1141). The purpose of our study was to evaluate the value of bedside measurements of WOB as a weaning parameter using a commercially available monitor. We did not incorporate the Campbell diagram into our measurements as this is not an available option on these bedside monitors. It is misleading for Banner et al to imply that data from esophageal pressure, tidal volume, and chest wall compliance may be processed using a commercially available monitor that “calculates total WOB using the Campbell diagram.” In fact, these monitors (Biore; Allied Health-care Products; Riverside, Calif) are not currently available with Campbell diagram calculations. Measurement of imposed WOB is also not available at the bedside. Therefore, our calculations much more accurately reflect what is available to the bedside critical care clinician. These monitors are in common clinical use, and the information obtained from these monitors is frequently utilized in the weaning process. As clinical investigators, our interest lies in producing information that will assist clinicians in the optimal care of critically ill patients. Therefore, we choose to evaluate the technology in the manner in which it is available to clinicians. To that end, we believe that we have demonstrated that calculations of WOB alone from these bedside monitors may be unreliable for important extubation decisions in mechanically ventilated patients.

We also find it interesting that Banner et al choose to describe 5 to 8 cm of precostobuction pressure support ventilation as “low level” and refer to their own study, which demonstrated that 13-cm PSV is required to decrease imposed WOB to zero. While this may eventually be accepted, their data has not yet been reproduced, and most clinicians view 5- to 10-cm PSV as adequate to overcome the resistance of the endotracheal tube.

We applaud the attempts of Banner et al as well as the work of Kirton et al in evaluating the value of bedside measurements of WOB. We also agree that the bedside measurement of WOB is more complex than initially believed and that measurements of imposed WOB may eventually provide invaluable information during the weaning process. For now, the data remains scarce and inconclusive, and we believe it is best to remind clinicians to exercise caution when utilizing data from newer technologies for which there are little positive outcome data.

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**Lung Health in Sawmill Workers Exposed to Pine and Spruce**

**To the Editor:**

I read with interest the article “Lung Health in Sawmill Workers Exposed to Pine and Spruce” by Hessel and colleagues (CHEST 1995; 108:642-46) where sawmill workers were shown to have an obstructive lung function impairment and also reported shortness of breath and wheeze with chest tightness. The symptoms were more likely to occur when the employment time was more than 3 years. The authors state that it is somewhat surprising that there is lack of data on the health effects of workers exposed to spruce and pine because of the extensive use of these woods throughout the world. I would therefore like to supplement the findings by Hessel et al with some of our findings published during the last years.

To determine whether wood trimmers in a modern sawmill exhibited a lung function impairment and/or alveolar inflammatory reaction, and if so, this was related to the presence of precipitating antibodies against mold spores; lung function studies and bronchial provocation tests were performed in 28 wood trimmers (11 with precipitins) before and after a week of work. Occupational exposure to wood dust, mold spores, and bacteria, measured with personal-equipped equipment, was low. The workers, who had a mean employment time of 13 years, showed slight obstructive lung function impairment at the beginning of the week, and the seropositive workers showed a further decrease in FEV1 at the end of the week (p<0.05). The workers reported an increased prevalence of cough and breathlessness. Bronchoalveolar lavage was performed in 19 workers with (n=9) or without precipitins. The alveolar cell concentrations of various cells did not differ from those in a nonexposed reference group. However, the alveolar concentrations of albumin, fibronectin, and hyaluronan were significantly increased (p<0.001 for all), indicating a low-intensity alveolar inflammation. Seropositive and seronegative workers did not differ regarding alveolar findings.

Thus, even low-grade exposure to sawdust and its components

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