Oximeter Performance and Diagnostic Accuracy of Sleep Studies

To the Editor:

I am writing to comment on the article by Davila et al1 (November 2002), which highlighted the importance of understanding instruments, mainly oximeters in his example, when acquiring data to be used in medical diagnosis. Davila and colleagues1 showed that, depending on the acquisition parameters set on the instrument and the mechanism of storage of the data, the results of variables such as oxygen desaturation index could vary tremendously, with the result that the settings on the instrument can result in patients being misdiagnosed. There are many oximeters on the market, and there are many data acquisition systems that have oximeters built in, and many people using these systems are not aware of the problems that Davila and colleagues1 have pointed out in their article.

Although response time of the oximeter and mechanism of storage are very important, we showed that oximeter and sensor type and location may also dramatically affect the oximetry signal. Using the same instrument, we demonstrated that an ear sensor is preferable to a finger sensor, although it is a bit more inconvenient to use; but because of the faster response, we are able to assess variables such as heart-to-ear circulation time, which is a useful measure, for example in patients with cardiac failure.2 In those few patients in whom obtaining a signal from the ear is problematic because of perfusion problems, application of a tiny amount of topical vasodilator improves the signal. We applaud Davila et al1 for reminding us all once again how important it is to understand the instruments we are using to diagnose our patients. The fine print in the instruction manual really is important.

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To the Editor:

Our study focused on downstream issues, in terms of differential averaging, storage, and display of data once it had reached the oximeter. As pointed out by Kryger, it is important to realize that upstream factors, relating to differences in the sensor types and locations, can be significant sources of variability in the data as well. We agree that it is important for the clinician to appreciate the vulnerabilities in all the links of the chain of detection, transmission, computation, storage, and display of oximetry data.

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Is Mushroom Workers’ Chronic Cough the Same as Byssinosis and What Should the Occupational Exposure Limit Be for Endotoxin?

To the Editor:

Tanaka and colleagues (September 2002)1 reported that most of the mushroom workers in their study group had experienced chronic cough, which they further characterized as organic dust toxic syndrome, hypersensitivity pneumonitis, runny nose (ie, postnasal drip syndrome), cough-variant asthma, and eosinophilic bronchitis.

These diseases (except for two cases of hypersensitivity pneumonitis) were not defined on an etiologic basis. Some cases of asthma or rhinitis could be due to an immunologic mechanism, but the authors did not measure IgE against Hypsizigus marmoreus (commonly called, Jade Gill mushrooms) spores. The remaining cases could be an irritant effect from airborne fungal spores, mycotoxin, organic dust, or endotoxin.

We suggest that the mushroom workers’ chronic cough is the same (or a variant) disease as that reported in cotton textile workers, byssinosis.2 The following points form the basis for thinking that endotoxin is the agent of chronic cough in mushroom workers.

First, symptoms reported by those with chronic cough were observed “to improve or disappear after weekend holidays.” This is similar to the reported Monday-morning effect experienced by those in the cotton textile industry, which has identified endotoxin as the causative agent.

Second, the rapid occurrence of chronic cough in this population of workers is similar to that in cotton textile workers, among whom Wang et al3 reported a rapid decline in FEV1 for newly hired, nonsmoking female cotton dust workers after 3 months of exposure.

Third, since 71.4% of workers reporting chronic cough developed symptoms within 3 months after starting employment, sensitization to fungal spores is an unlikely responsible factor. Endotoxin (lipopolysaccharide [LPS]) air concentrations in the harvesting room (three samples) and packing room (three samples) were reported (Table 1) to be well above the suggested no-observed-effect level for airway inflammation (ie, 10 ng/m3),4 although a range from 9 to 170 ng/m3 has been reported.5 In the cultivating room, the LPS concentration was below the no-observed-effect level but was above the level observed to cause an acute decrease in airway function (ie, 53 endotoxin units [EU]/m3, or approximately 5 ng/m3),6 to reduce lung function (ie, 4 ng/m3)7 and to elicit a physiologic response (ie, 2 ng/m3).8,9

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Since airborne endotoxins are highly variable, the relatively low levels of LPS reported by Tanaka et al require confirmation before these findings can be fully accepted. LPS concentrations should be expressed as EU per cubic meter and not as nanograms per cubic meter. Reporting endotoxin values as nanograms per cubic meter does not allow an accurate comparison with other measurements.

Additional data might be useful in the establishment of an occupational exposure limit (OEL) for endotoxin that minimizes the incidence of occupational disease. It is proposed here that the OEL (time weighted average, 8 h) be established between the concentration reported for reducing lung function and the physiologic response (ie, 20 to 50 EU/m³). This will provide some margin of safety in protecting the majority of people. This proposed value is similar to the suggested OEL (50 EU/m³/8 h, or about 4.5 ng/m³) initially proposed by the National Health Council in the Netherlands, although a higher value was finally adopted (200 EU/m³).

The study by Tanaka et al illustrates the diversity of industries in which workers are exposed to levels of endotoxin that are sufficient to cause respiratory diseases. Endotoxin may even be one of the major causes of health effects from outdoor pollutant particulates. It should be mentioned that beneficial effects of LPS resulting from occupational exposure have been suggested (notably, reduced lung cancer rates). Thus, occupational exposure may be a two-edged sword. It has been suggested that the beneficial effects of occupational exposure also be reported in studies rather than dismissing these results as bias or as some form of the healthy-worker effect.

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3 Wang X-R, Pan LD, Zhang H-X, et al. Follow-up study of respiratory health of newly-hired female cotton textile workers reporting chronic cough that developed within 3 months in one patient and 3 months in the other (unpublished data). Based on these cases, 3 months might be enough time to be sensitized by mushroom antigens, in which case all 71.4% of workers reporting chronic cough that developed within 3 months after the start of employment might not be the result of endotoxin inhalation alone. We speculate that a high concentration of airborne mushroom spores may shorten the sensitization period in the contemporary mushroom farm worker. Thirty percent of workers were sensitized to the spore in the first year and 93% in the second year. We are now developing a system for measuring serum IgG and IgE levels in response to the spore antigens in our laboratory.

Our results on the effects of mushroom antigens on peripheral blood mononuclear cells in mushroom workers have now been published. Levels of CD14+ -positive monocytes, acting with innate immunity and contributing to a primary defense mechanism in mucosal tissue, increased in mushroom workers. CD14 is a ligand of lipopolysaccharide, and its signal acts through the

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To the Editor:

We thank Lange et al for their comments. They have raised an important issue about airborne endotoxin exposure in occupational chronic cough. We reported that airborne endotoxin, as well as antigens from mushroom spores, was one of the important causes of cough in mushroom workers. As Lange et al have suggested, the phenomenon occurring in the mushroom farm workers is similar to that in cotton textile workers (byssinosis). Actually, some mushroom workers noticed cough in the first 2 weeks. This seems too short a time in which to be sensitized by mushroom spore antigens, therefore, we think that the cough resulted from exposure to chemicals, for example, endotoxin. As stated in our report, the latent time period from the start of working to the onset of symptoms of organic dust toxic syndrome (average, 1.8 months) was the shortest, the second was eosinophilic bronchitis (average, 3.5 months) and the third was cough-variant asthma (average, 4.1 months). However, we recently have seen two cases of hypersensitivity pneumonitis due to another kind of mushroom spore in which the latent period was only 2 months in one patient and 3 months in the other (unpublished data). Based on these cases, 3 months might be enough time to be sensitized by mushroom antigens, in which case all 71.4% of workers reporting chronic cough that developed within 3 months after the start of employment might not be the result of endotoxin inhalation alone. We speculate that a high concentration of airborne mushroom spores may shorten the sensitization period in the contemporary mushroom farm worker. Thirty percent of workers were sensitized to the spore in the first year and 93% in the second year. We are now developing a system for measuring serum IgG and IgE levels in response to the spore antigens in our laboratory.

Our results on the effects of mushroom antigens on peripheral blood mononuclear cells in mushroom workers have now been published. Levels of CD14+ -positive monocytes, acting with innate immunity and contributing to a primary defense mechanism in mucosal tissue, increased in mushroom workers. CD14 is a ligand of lipopolysaccharide, and its signal acts through the
toll-like receptor 4. Therefore, sensitivity to lipopolysaccharide is speculated to be enhanced in mushroom workers. We agree that airborne endotoxin plays an important role in the mechanism of chronic cough in mushroom workers, but the contribution seems to be only a part of the cause.

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Microdrainage Via Open Technique in Severe Subcutaneous Emphysema

To the Editor:

The very interesting and original reports in CHEST by Beck et al (February 2002)1 and Leo et al (October 2002)2 gave us the occasion to review our experience with subcutaneous emphysema and its treatment in patients who have undergone lung parenchyma resections. In the period between January 1990 and September 2002, we performed 1,561 lung resections. No substantial changes in the surgical technique regarding the parenchyma occurred during this period (essentially, mechanical stapling either at the paren-

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Communications to the Editor

Risk of Bleeding Associated With Transbronchial Lung Biopsy

To the Editor:

We read with interest the prospective report of Herth et al (October 2002).1 which examined the role of aspirin as a risk factor for bleeding associated with transbronchial lung biopsy (TBBX). Two hundred eighty-five of the 1,217 study subjects had consumed aspirin within 24 h of undergoing the procedure. No bleeding was defined as traces of blood after finishing the biopsies without the need for continued suctioning. Mild bleeding was defined as the need for continued suctioning of blood from the airways after the procedure, moderate bleeding was defined as a requirement for intubation of the biopsied segment with the flexible bronchoscope into the wedge position, and severe bleeding was defined as the need for an additional intervention, such as placement of a temporary bronchus blocker, the application of a fibrin sealant, admission to a critical care unit, or the need for blood products.

There are several unanswered questions regarding the methodology used in this article. TBBX may be performed with or without wedging the bronchoscope.2,3 Was the bronchoscope wedged to obtain the TBBX? If it was, then when was the wedge position removed? With the wedge technique, the bronchoscope is reinserted or rewedged if there is evidence of bleeding postbiopsy and suctioning is discouraged. If the TBBX was performed without wedging the bronchoscope, then at what stage was suctioning stopped and the bleeding segment wedged with the bronchoscope? Furthermore, this appears to be a collaborative work from two centers. Was the same technique used to obtain TBBX in both centers? What criteria were used to ensure that the need for clinical intervention to judge the amount of bleeding associated with TBBX was uniform in both the centers?

Some bronchoscopists may not wedge the bronchoscope at all and may continue suctioning with the “back-and-forth technique” until bleeding stops spontaneously.3 Were further TBBXs abandoned because of the severity of bleeding?

As none of the patients in the study required intubation, admission to critical care areas, or blood transfusion, the article highlights the fact that TBBX may be undertaken with controllable bleeding complications in patients who have received aspirin within 24 h of the procedure. Bleeding during TBBX is usually recorded as the volume of mixed blood and lavage fluid that is collected through the suction system of the bronchoscope at the end of the procedure.2–5 However, this study does not clearly assess the role of aspirin in the risk of bleeding associated with TBBX, as the quantity of bleeding was not recorded, irrespective of whether it was controlled with bronchoscopic methods and despite the prospective nature of the study. The identification of the severity of bleeding following TBBX in patients who have taken aspirin within 24 h of the procedure and its management in this large series remains elusive.

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