dynamic response to exercise are the determination of the early normalized oxygen deficit during square wave exercise testing. Due to the inappropriate (suboptimal) increase of the cardiac output at the early onset of exercise, the organism has to rely on anaerobic energy sources with an excess lactic acid accumulation. Therefore, at the onset of exercise, VO₂ is subtracted from the steady-state value, all breaths are cumulated, and the total oxygen deficit is expressed as a percentage of the total oxygen cost of the exercise level, above the resting value. Previous studies on this topic, in patients with chronic heart failure and in patients with congenital heart disease, have shown that the normalized oxygen deficit is increased in these patients groups, which suggest an inadequate oxygen delivery to the exercising tissue or a metabolic inertia of the muscular tissue, which can be due to deconditioning.

Finally, the measurement of VO₂ kinetics at low intensity (subanaerobic threshold) exercise may provide useful information about the efficiency of the oxygen delivery to the exercising tissue and may give some insight about the mechanisms of exercise limitation in patients with cardiovascular disease.

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Polysomnography in the Diagnosis of the Obstructive Sleep Apnea-Hypopnea Syndrome

Where Do We Draw the Line?

In this issue of CHEST (see page 353), Le Bon et al report the largest series to date of consecutive polysomnograms (PSGs) in patients suspected of having the obstructive sleep apnea-hypopnea syndrome (OSAHS). The authors conclude that a single negative PSG does not exclude a diagnosis of OSAHS. The evidence provided plus a review of the literature strongly supports this conclusion for any apnea-hypopnea index (AHI) that is considered to be clinically relevant below an AHI of 20.

According to a recent recommendation, an AHI of 5 to 15 is consistent with mild OSAHS. Furthermore, it is important to recognize such patients, since they may benefit from therapy such as continuous positive airway pressure (CPAP) with improvement in subjective daytime sleepiness and quality of life.

In determining the diagnostic accuracy of the PSG to measure AHI, its sensitivity should be assessed. For the purposes of this analysis, I will accept the authors’ view that the highest AHI is the one to be used for diagnostic purposes. That is, a positive test on either night 1 or night 2 is 100% specific. This
approach is designed, in part, to include symptomatic patients who may benefit from intervention. I will also confine most of my analysis to AHI thresholds of 5 and 10, since both have been recommended for clinical purposes.1,3

For the most part, I have analyzed the authors’ data to include only the 142 patients with 2 nights of data in which the first night had an AHI < 20. I have done this for several reasons. First, this is the only group in which consecutive unselected nights were performed. Second, it is the variability of AHI in patients with borderline or low AHIIs that presents diagnostic problems.1,4–10 In this group, the sensitivity of the first night for an AHI ≥ 5 was 75% (85 of 113 patients); for an AHI ≥ 10, it was 53.6% (37 of 69 patients). Since performing a second night involves more expense and inconvenience, it is important to examine the diagnostic yield of 2 nights in the face of a negative first night. For an AHI of < 5, the diagnostic yield of a second night was 49.1% (28 of 57 patients); for an AHI of < 10 it was 30.4% (32 of 105 patients). The yield is greater and the number of repeat PSGs is less when using an AHI of ≥ 5. This concept is further supported since the AHI on night 1 did not predict the AHI on night 2. For example, the 25 patients with an AHI ≤ 20 on night 1 and ≥ 20 on night 2 had AHIs on night 1 that ranged from near 0 to near 20.

This analysis of the current study plus the potential for clinical intervention supports an AHI ≥ 5, in an appropriate clinical setting, to be consistent with a diagnosis of OSAHS. A recommendation has been made to standardize such a clinical setting.1 Typically, such patients are sleepy during the day and snore at night.

The current study also provides perspective for the relative number of patients that are underdiagnosed with 1 night of polysomnography using AHIs of 5 and 10 as thresholds. The percentage of total patients presenting (in the current study) with an AHI < 5 and who had an increase to > 5 was 11.5% (28 of 243 patients); for an increase from < 10 to ≥ 10, it was 13.2% (32 of 243 patients).

The current study can be compared to limited data in the literature for consecutive night studies. The diagnostic yield of combining the studies of both nights, combining all patients,2,7,11 for an AHI of ≥ 5 was 48.3% (14 of 29 patients); for an AHI ≥ 10, it was 32.6% (14 of 43 patients).5,7,9 The percentage who were underdiagnosed using an AHI of 5 is 14% (31 of 218 patients)4,6–9, for an AHI of 10, it was 7.7% (12 of 155 patients).4,7,9 In general, the results of the current study by Le Bon et al are consistent with data in the literature.

The reason for the variability between the 2 nights with a greater yield on the second night is not defined. The current study controlled the sleep environment, standardized the method of scoring the record, and accounted for the patients’ sleeping position. One issue that may have impacted the results is the routine of waking patients at 7:00 AM, regardless of their usual waking time. However, the authors recognized this and made efforts to analyze this effect that did not appear to affect the results. The authors attempts to define a predictive relationship, including variables of sleep staging, between subject characteristics and a higher AHI on the second night were unrewarding.

One possible explanation for the relatively greater increase in AHI in this study in patients with lower AHI may be regression to the mean of the AHI combined with the fact that the lower values can increase more than decrease. This may occur since there is no “ceiling” on the increase but there is a “floor,” namely an AHI of 0, on the decrease.

I have confined my comments to the use of AHI to define respiratory disturbance during sleep. In addition, recent information indicates that more subtle respiratory disturbances may lead to arousals, symptoms, and response to therapy such as CPAP consistent with OSAHS.3,12 These respiratory effort-related arousals4 are increasingly being incorporated in scoring of the PSG. How adding these events to the total will affect the threshold for a study to be consistent with OSAHS is currently undefined.

Future research in this area should focus on correlating respiratory events with symptoms and response to therapy. This will ideally produce an approach to combining pretest probability with post-test AHI and predicted response to therapy to estimate the likelihood that a patient has clinically relevant OSAHS. Only by putting the recommended OSAHS syndrome definition to the test of clinical science will validation or refinement of the recommended threshold of five respiratory events per hour of sleep1 be possible.

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Editorials

Hemoptysis Season

The clinical presentation of hemoptysis often leads to difficult questions: is this merely due to bronchitis, as in most cases? Or is this a symptom of a more ominous problem, such as cancer, tuberculosis, or one of the many other serious conditions that can cause hemoptysis? Case series reports have identified a few clinical and historical clues that help to guide decisions about the need for pursuing an extended workup for the cause of the bleeding, including chest radiograph abnormalities, patient age, and smoking history. In this issue of CHEST (see page 440), Boulay et al suggest another, possibly important, consideration—the season.

A few clinical investigators have noted that the incidence of hemoptysis appears to increase during the colder months of the year, but none of the studies have had sufficient power to examine this issue. To investigate the role of seasonality in hospitalizations for hemoptysis, Boulay and colleagues examined the monthly incidence of hemoptysis in 29 teaching hospitals in France over a 3-year period. A total of 6,349 patients with hemoptysis were identified using discharge diagnosis codes; 3,672 of these patients (58%) had a second diagnosis that could explain the cause of the bleeding, and the remainder were labeled as cryptogenic. This study elegantly demonstrates that the incidence of hemoptysis does, indeed, vary quite significantly throughout the year, with a peak incidence during the late winter and a nadir in late summer. The variation closely correlates with the seasonal variation in respiratory tract infections, which are also known to affect the incidence of asthma exacerbation.

It is interesting that in this study there was no difference in the seasonal pattern between the cryptogenic and noncryptogenic groups. This is surprising because the hemoptysis-associated diseases in the noncryptogenic group, including lung cancer and cardiovascular diseases, are dissimilar. This suggests that the etiologic mechanisms for the seasonal variation in both groups were similar. Seasonal variation in the incidence of respiratory infection appears to be the most likely explanation for the observed variation in hemoptysis incidence; however, it is also possible that cold air, which is also dry air, may have a direct irritating effect on respiratory mucosa. In either case, it is possible that some of the cases of hemoptysis among patients with chronic lung conditions such as bronchiectasis and chronic bronchitis could be prevented. Further investigations into the mechanisms of hemoptysis among patients with chronic lung disease and the potential for preventive interventions are needed.

Differences in the seasonal pattern among specific causes of hemoptysis could have important implications on the decision to pursue the possibility of malignancy or other serious causes. For example, if the incidence of hemoptysis due to cancer is relatively stable throughout the year, while the incidence of hemoptysis due to infection varies widely, then the proportion of patients with hemoptysis caused by malignancy will also fluctuate throughout the year. Thus, a patient presenting with hemoptysis during the warmer months of the year could merit a more aggressive workup to rule out cancer than one presenting in late winter during the peak of the cold and flu season. There was some difference in the amplitude of the seasonal variation by age, which raises the possibility of confounding effects among age, chronic disease, and acute infections. Hopefully, in the near future, Dr. Boulay’s group or other researchers with access to large patient databases will be able to describe the seasonal variation of the various conditions associated with hemoptysis and to identify clinical factors that help to predict the risk of underlying malignancy.

Dr. Boulay’s study is an excellent example of how large electronic clinical data files can be used to address pertinent clinical questions and to identify

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