When an intermediate type I patient needs invasive ventilation, we maintain mechanical ventilation for 3 weeks. Then, after the tube is removed, the patient continues with nasal nocturnal ventilation and intermittent positive-pressure ventilation for 20 min three times a day. If, despite this management, the patient requires invasive ventilation more than three times during the same year or requires nasal ventilation not only during sleeping, but also while awake, a tracheostomy is performed.

Treatment has changed over the years. We agree with Dr. Bach that patients with respiratory paralysis benefit from preventive noninvasive treatment with hyperinsufflation and noninvasive nocturnal ventilation, which is associated with help in coughing. At the present time, we also use percussion therapy with respiratory physiotherapy every day at home, and these treatments delay the acute respiratory worsening.

In our experience, patients with tracheostomy tubes are no longer hospitalized until spine surgery and are less dependent on their environment. Severely affected children cannot attend a boarding school while using noninvasive ventilation. So, even now we perform a tracheostomy in ventilator-dependent children who require day-long therapy. Dr. Bach said that “many of their patients needed to be intubated on 10 or more occasions before age 5” and that “patients … use high span BPPV [bilevel positive pressure ventilation] at least when sleeping and as many as 60 use it up to 24 hours a day.” In our opinion, for a patient who needs continuous nasal ventilation, or requires invasive ventilation more than three times during the same year, we prefer the use of tracheostomy, which allows a better quality of life. Mechanical ventilation with good thoracic expansion prevents pectus excavatum.

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Surprisingly, they were not able to find a difference between the COPD patients and the control subjects. Their conclusion was that neutrophil apoptosis in COPD patients occurred at a rate similar to that found in healthy individuals and smokers with normal lung function.

We agree with the methodology used; however, we believe that this conclusion is misleading. An increase in systemic cytokines (eg, interleukin-6), inhibiting the apoptosis of circulating neutrophils, is present in stable-state COPD patients but is not pronounced as in those with acute exacerbations. In addition, the spontaneous apoptosis rates of circulating neutrophils show a high degree of interindividual variation. Therefore, a larger number of subjects than that investigated by Noguerà et al might be needed to find a statistically significant difference between healthy volunteers and COPD patients in the stable state.

According to the guidelines of the Global Initiative for Chronic Obstructive Lung Disease, exacerbations are the main factor in the progression of COPD. Therefore, we performed a very similar study but enrolled only hospitalized patients who had experienced an acute exacerbation of COPD. Neutrophil apoptosis was measured at hospital admission, after 3 to 5 days, and at hospital discharge. In addition, we assessed spontaneous apoptosis rates in healthy volunteers. We were able to show that neutrophil apoptosis is significantly suppressed at the beginning of an acute exacerbation (p < 0.0001 [paired t test, and the parametric distribution of the data was confirmed by the Shapiro-Wilk test]), and that it increases progressively after treatment and clinical remission, reaching the values of healthy subjects on the day of hospital discharge. Therefore, we believe that delayed neutrophil apoptosis is an important key feature of COPD pathogenesis and deserves further investigation to avoid missing any potential new treatment options for COPD, which is a disease with a high burden.

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### References


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### Apoptosis of Circulating Neutrophils in COPD Patients

**To the Editor:**

We read with great interest the study by Noguerà et al (May 2004) on the apoptosis of circulating neutrophils in COPD patients. Neutrophils exhibit a short half-life and are primarily removed by apoptosis. In contrast to apoptosis, neutrophil necrosis results in the release of aggressive enzymes, leading to tissue destruction and increased inflammation.

Noguerà et al compared ex vivo the spontaneous apoptosis rates of circulating neutrophils from COPD patients in stable condition with those from smokers with normal lung function and healthy nonsmokers. Since it is known that spontaneous neutrophil apoptosis is decreased in various inflammatory conditions and that neutrophils are considered to play a crucial role in COPD pathogenesis, they expected to find a decreased or delayed apoptosis. They incubated the cells and measured spontaneous apoptosis rates after 2, 8, and 24 h by flow cytometry.
course, in vitro conditions may differ very substantially from in vitro ones. Thus, the comments made by Drs. Pletz and Lode with respect to our work are well taken. We would like, however, to highlight another potential source of confusion. In the article by Pletz et al., patients were studied during exacerbations. As such, they were receiving intense steroid therapy, among other types of therapy. As is known by the authors, steroids can interfere both with systemic inflammation and neutrophil apoptosis. We therefore fully agree with Drs. Pletz and Lode that the role of neutrophil apoptosis in COPD deserves further study.

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REFERENCES

SNAP Technology and Sleep Apnea

To the Editor:

As a Fellow of the American College of Chest Physicians, a medical adviser to SNAP Laboratories, and a SNAP Laboratories test user of many years, I read with surprise the study by Liesching et al (March 2004), as well as the accompanying editorial by Collop. Using data derived from a poorly designed study, Liesching et al unjustifiably concluded that the agreement between the apnea-hypopnea index determined by SNAP testing and polysomnography can only be characterized as “fair.” The authors then offered the opinion that SNAP studies may not accurately assess the severity of obstructive sleep apnea. In an additional reply to this article, Collop correctly pointed out that “the study is not without faults.”

As noted, the study by Liesching et al was a retrospective comparison of nonsimultaneous studies performed in the laboratory with those performed at home. In actuality, the mean time frame between tests that were compared was 5 months. The decision to compare data collected from different and nonsequential nights is a major flaw. Despite the rebuttal of one study by Liesching et al, there is a substantial body of research that documents the first-night and night-to-night variability of sleep apnea. This variability is compounded by the week-to-week or month-to-month variability inherent in their study. Therefore, their data were clearly impacted by variables such as changes in body mass index, medications, and site of testing. In addition, of the original 39 subjects, only 31 had complete data for analysis. Considering the limitations of the study, it is remarkable that it was considered to be an empirical foundation for any conclusions. A more appropriate conclusion, if any were to be drawn, would be that SNAP home testing variability is no greater than that documented between nights in a sleep center.

In addition to pointing out this study’s shortcomings, Collop has noted previously that “given the outdated sleep staging rules, inconsistent equipment between labs, variable scoring parameters...it is a wonder that there can be any consistent scoring of PSGs [between trained sleep professionals].” In another publication Collop concluded that “clinicians should be aware that there is tremendous variability among polysomnography technologists regarding the scoring of polysomnography.” Given the disagreement levels reported by Collop in this study (eg, the record of the same patient yielded apnea-hypopnea index scores of 5 and 74 by two differently trained technicians), the SNAP results criticized by Liesching et al are in fact impressive. Thus, even if the SNAP test had been in perfect agreement with the scoring done by the Brown Sleep Laboratory physician used in this study, scoring by personnel at another laboratory would in all likelihood differ. This would lead Liesching et al to the same conclusions that they reached in their study.

SNAP testing is being held to a different standard from that of the sleep industry sets for itself. The sleep medicine community has been aware for years of the high level of disagreement between two trained professionals scoring the same record on the same night. Why then should they be surprised when SNAP results vary between a review by one technician and that of another technician performed months later in an entirely different environment? Perhaps one should question why we should subject patients to laboratory-based polysomnography at significantly greater expense and inconvenience when same-test scoring agreement is no more accurate than that reported by a SNAP home test.

The SNAP test is not a “black box,” as suggested in the editorial by Collop, but instead requires a full night of continuous recording of at least four data channels. These raw data are analyzed by a trained technician who analyzes the full night of digitally recorded raw data, just as is done in the sleep laboratory. All data with marked events are available to the referring physician on request. The SNAP test technology and scoring procedure have been validated on five separate occasions by accredited sleep centers. All of these studies used a protocol of recording SNAP data and conventional polysomnography data on the same night in sleep laboratories, with quite impressive results. Previous attempts to publish these side-by-side blind studies have been met with strong resistance by journals with review committees dominated by sleep specialists. In other cases, after the data were collected and analyzed, the sleep laboratory involved in the study simply refused to submit the results for publication.

Despite these obstacles, a recent study performed at the University of Chicago was published that simultaneously compared SNAP to PSG, as was suggested by Liesching et al. The authors concluded that “the results of this validation study demonstrate that there is good correlation between SNAP and PSG in quantifying RDI with reliable sensitivity, specificity, positive and negative predictive values in a laboratory setting.” Thus “SNAP is an excellent tool for the diagnosis of OSAS.”

As a medical adviser I have been assured by SNAP Laboratories of their willingness to participate in any well-designed, objective study that will yield a fair and accurate assessment of SNAP sleep testing.

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