Obstructive Sleep Apnea Syndrome

To the Editor:

On the basis of the figures depicting raw waveforms reproduced in the paper by Coppola and Lawee, "Management of Obstructive Sleep Apnea Syndrome in the Home," published in the July issue of Chest,1 I take exception to their statement regarding the accuracy of four-channel sleep apnea recordings in the diagnosis of moderate to severe obstructive sleep apnea syndrome (OSAS). The authors have not documented obstructive hypopnea in Figure 1A. Airflow as measured with nasal-oral thermistors reflects changes of air velocity and cannot be equated to volume because cross-sectional area is undefined. Even the relative flat portion of the airflow waveform labeled apnea shows oscillations that might be a result of high grade hypopnea. The tracing labeled hypopnea shows slowed respiratory frequency. Possibly the authors made the diagnosis of hypopnea because of the concomitant fall of SaO2, but relying on levels of SaO2 alone is dangerous without confirming their validity with an associated normal pulse oximetry waveform. In fact, there are a number of spikes on the SaO2 display suggesting movement artifacts. In Figure 1B, I am unable to distinguish respiratory related movements on the chest wall impedance waveform from electrical noise. Therefore, I would conclude from their study that wide fluctuations in SaO2 from home monitoring with pulse oximetry in patients with suspected moderate to OSAS are consistent with such a diagnosis and that nasal continuous positive airway pressure abolishes these fluctuations. But for greater certainty of diagnosis, substitution of the pulse oximetry, pulse waveform for their respiratory-related signals in this four-channel recorder would lead to more accurate assessment of SaO2 levels. The value of SaO2 home monitoring has been previously pointed out by Williams et al.2 I do not advocate such an approach because diagnosis is best made by accurately estimating the number and types of apneas rather than values of SaO2 with pulse oximetry, which may be affected adversely by motion artifacts that often occur in the arousal period after the event. After all, the condition is designated the obstructive sleep apnea/hypopnea syndrome.

Marvin A. Sackner, M.D., F.C.C.P.,
Mt. Sinai Medical Center,
Miami Beach, Florida

REFERENCES
1 Coppola MP, Lawee M. Management of obstructive sleep apnea syndrome in the home: the role of portable sleep apnea recording Chest 1993; 104:19-25

To the Editor:

Dr. Sackner makes a number of comments that question the validity of the four-channel sleep apnea recording used in our report (Chest 1993; 104:19-25). We did not design the paper to be a validation of this device, but a description of how that device might be used in a clinical setting. Two previous studies cited in our paper found this device to be accurate when compared with standard polysomnography.1,2 Sensitivity and specificity for moderate to severe obstructive sleep apnea syndrome (OSAS) were 95 percent or better in these two reports.

Dr. Sackner asserts that he cannot detect whether a hypopnea was present in the ample tracing provided. We have strict scoring criteria for the determination of a hypopnea that parallels the definition used in standard polysomnography laboratories. An oral-nasal thermistor is used to reflect airflow through the upper airway. Airflow thermismy is an accepted noninvasive method of detecting airflow.3 We feel that thermistors are well suited for the home environment, as they are very stable in the unattended setting. We have recorded over 1,000 of these studies and have never had a thermistor fail. Dr. Sackner is concerned that these thermistors measure velocity and not flow. Actually, they measure temperature change that accurately parallels changes in airflow. More invasive methods may produce more accurate physiologic data, but measurement of endtidal CO2, esophageal pressure measurements, and other methods may not be appropriate for the home.

The ability to detect an event labeled as apnea was also questioned by Dr. Sackner, as he was not able to detect whether airflow or "electrical noise" was present in the tracing. We perform a breath-hold maneuver at the commencement of each recording to assist the scorer in differentiating cardiac pulsations and other "noise" from airflow. We have been able to detect sleep disordered breathing accurately with this recording device and regularly recognize both obstructive apneas, hypopneas, central apneas, and Cheyne-Stokes respirations. The tracing labeled as a hypopnea (Fig 1A) clearly showed respiratory effort with significant reduction in airflow with desaturation, consistent with a hypopnea. Complete cessation of measurable airflow consistent with an apnea is also seen in this example. These physiologic findings are characteristic of the OSAS. Motion artifact in the oximetry channel probably represents arousals, as they are seen near the termination of events. We think that these motion artifacts actually contribute to the diagnosis of OSAS. We agree that the distinction between apnea and hypopnea may not be clinically important. The tracing shown is irrefutably positive for OSAS. "If it quacks like a duck, it must be a duck."

Dr. Sackner mentions oximetry as being preferable to four-channel studies for diagnosing the syndrome. The literature does not support this statement. Published studies on oximetry alone have failed to produce the greater than 95 percent
accuracy seen with the Edentrace device used in our and other studies. We believe oximetry alone may not be able to distinguish between the different types of sleep disorders breathing as well as the four-channel sleep device described in our report.

Dr. Sackner has expressed his concerns about the physiologic appropriateness of the particular measurement tools used in the portable sleep apnea recording devices. Certainly more complex data acquisition may give purer physiologic data, but we are not convinced this would significantly affect the outcome. It is doubtful that any more invasive technique would have arrived at a different diagnosis in our example patient whose apnea-hypopnea index of 64 with oxygen desaturations to 50 percent confirmed the diagnosis of OSAS. We believe that the literature published to date shows that these sleep apnea recorders can be quite accurate. Unless data to the contrary become available, Dr. Sackner's concerns appear to be moot. The OSAS is a common disorder that is being underdiagnosed. Simplified accurate testing may help to address this problem.

A duck is a duck.

Michael P. Coppola, M.D., F.C.C.P.,
Springfield, Massachusetts

Reprint requests: Dr. Coppola, 2150 Main St., Springfield, MA 01104

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Subcutaneous Emphysema:
Spontaneous or Iatrogenic?

To the Editor:

The article of Herlan et al, "Massive Spontaneous Subcutaneous Emphysema," is interesting; the treatment suggested by the authors is attractive. However, the use of the term "spontaneous" in relation to the four cases of subcutaneous emphysema is most surprising. The New Webster's Dictionary and Thesaurus" defines "spontaneous" as "happening without external cause or control." Is this really what happened to the four patients described in the article?

Patient 1 underwent an "uncomplicated" transhiatal esophagectomy. According to the description, he had a severe paroxysm of cough that was followed by massive subcutaneous emphysema. Was the operation really uncomplicated and the subcutaneous emphysema spontaneous? It seems obvious that the emphysema was a complication of the operation. While barium contrast study of the cervical esophagus ruled out a leak of barium under normal condition of pressure, it did not rule out a leak of air during the paroxysm of cough, when the pressure was markedly increased. Thus, patient 1 suffered a complication of his operation.

Patient 2 underwent thoracotomy with laser ablation of giant pulmonary bullae. The massive subcutaneous emphysema occurred short after tracheal extubation. There was nothing spontaneous here.

Patient 3 was on mechanical ventilatory support for 3 days with peak airway pressure exceeding 65 mm Hg. Subcutaneous emphysema is a well-known complication of ventilatory support. Another case of spontaneity?

Patient 4 underwent coronary artery bypass grafting with division of pleural adhesions. There is certainly a possibility of injuring lung tissue during division of adhesions and causing an air leak. Indeed, massive subcutaneous emphysema developed immediately after removal of the mediastinal drainage tubes.

We can speculate on the mechanism that led to the development of subcutaneous emphysema in every case, but one thing is certain: the occurrence of subcutaneous emphysema in each patient was a complication of treatment (three operations, one ventilatory support), not spontaneous.

Of course, this does not detract from the value of the treatment suggested by the authors. It is attractive, and in view of the good results, probably worth trying.

Don Weissberg, M.D., F.C.C.P.,
Department of Thoracic Surgery,
Tel Aviv University Sackler School of Medicine,
Holon, Israel

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Role of Closed-Needle Biopsy in the Diagnosis of Malignant Mesothelioma of the Pleura

To the Editor:

The article by Beauchamp et al,1 which appeared in the October 1992 issue of Chest, purports to test the hypothesis that closed-needle biopsy may be sufficient in many cases to make a diagnosis of malignant mesothelioma of the pleura. In a study of 20 consecutive patients with a histopathologic diagnosis of mesothelioma, the diagnosis was made by closed-needle biopsy alone in 12 cases. Therefore, the authors argue, closed-needle biopsy is sufficient to make a diagnosis of mesothelioma. The circular nature of this reasoning is obvious, and in and of itself is sufficient to invalidate the conclusions of the study.

However, there are additional problems with the study design. The authors suggest that Alcin blue or colloidal iron staining of tumor supports a diagnosis of mesothelioma over metastatic adenocarcinoma. These histochemical stains react equally well with hyaluronic acid produced by mesothelioma and other acid mucopolysaccharides produced by adenocarcinoma.4 6 Although the authors note that hyaluronidase pretreatment increases the specificity of this staining procedure, there is no evidence in the text that hyaluronidase was used in their cases. Periodic acid-Schiff stain (PAS) with diastase, which provides useful information in selected cases,4 was used by the authors in only one open biopsy specimen. (The entry [8C] specimens opposite "PAS with diastase" in Table 1 in their article actually refers to antibody to carcinoembryonic antigen [CEA] according to the text.)

Immunohistochemical studies performed by the authors are similarly suboptimal. Keratin and vimentin immunostaining do not discriminate between malignant mesothelioma and metastatic adenocarcinoma involving the pleura.4 6 In my own experience, CEA is the most useful immunostain available to make this distinction,6 an observation supported by others.3 6 However, staining procedures that employ monoclonal antibodies B72.3 and Leu-M1 are also quite useful,6 and examples of metastatic adenocarcinoma that

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