communications to the editor

Communications for this section will be published as space and priorities permit. The comments should not exceed 350 words in length, with a maximum of five references; one figure or table can be printed. Exceptions may occur under particular circumstances. Contributions may include comments on articles published in this periodical, or they may be reports of unique educational character. Specific permission to publish should be cited in a covering letter or appended as a postscript.

"Saw-tooth" Pattern in the Flow-volume Loop

To the Editor:

We read with interest Doctor Schiffman's description of a saw-tooth pattern of the flow-volume loop in a patient with Parkinson's disease (A "saw tooth" pattern in Parkinson's disease. Chest 1985; 87:124-26). His report confirms the results of our recently published study concerning pulmonary function in patients with Parkinson's disease and other extrapyramidal disorders. In that study, we emphasized the peculiar aspect of the flow-volume loop, consisting of flow oscillations similar to the saw-tooothing described in the obstructive sleep apnea syndrome. We demonstrated that the flow oscillations had a frequency of 4 to 8 Hz, similar to the patient's extremity tremor. They were not due to tremor of the respiratory pump muscles but were caused by involuntary movements of the intrinsic laryngeal muscles, as evidenced by rhythmic vocal cord abduction-adduction visualized through fiberoptic endoscopy procedures. Furthermore, this dysfunction of the upper airway musculature resulted in clinical or physiological airflow limitation. We agree with Doctor Schiffman that saw-tooothing of the flow-volume loop indeed is probably not specific for the obstructive sleep apnea syndrome, particularly since it has been described also in burn victims with thermal injury to the upper airways. In this respect, we would like to mention that in a retrospective review of 2,800 flow-volume loops performed at our pulmonary function laboratories during 1982 and 1983, we observed 31 instances of flow oscillations not due to the obstructive sleep apnea syndrome. Sixteen of these 31 patients either had organic upper airway obstruction or neurological disease involving the bulbar musculature (including neuromuscular and extrapyramidal disorders). In our opinion, therefore, flow oscillations on the flow-volume loop reflect nonspecific upper airway instability resulting from disorders involving the upper airway, either directly or indirectly, via a disturbed neuromuscular control of the upper airway musculature. Flow oscillations detected on the flow-volume loop should lead to further investigation of the upper airway.

W. Vincken, M.D., Intensive Care Unit, Academic Hospital, University of Brussels, Brussels, Belgium; and M. G. Cosio, M.D., Respiratory Division, Department of Medicine, Royal Victoria Hospital, McGill University, Montreal, Canada;

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1 Vincken WG, Gautier SG, Dolffus BE, Hanson BE, Daraway CM, Cosio MG. Involvement of upper-airway muscles in extra-


To the Editor:

I am in agreement with Drs. Cosio and Vincken that "saw-toothing" is not specific for sleep apnea syndrome or any other disease. However, its occurrence in the patient I reported on and in several other patients with Parkinson's disease subsequently seen in our laboratory: in 18 of 27 patients reported by Vincken et al; and in the many other patients with sleep apnea syndrome suggests that such a pattern may be associated with these conditions (ie, Parkinson's disease and sleep apnea syndrome), a fact worth knowing by those who interpret flow-volume loops.

I am puzzled as to why Vincken and Cosio are so certain that the etiology of these oscillations is not tremor (note that the case report speculated that the oscillations were due to tremor but never speculated as to its location: diaphragm, chest wall or upper airway muscles). Granted, all evidence points to anatomic abnormalities of the upper airway as the cause of saw-toothing in sleep apnea syndrome and upper airway burns. However, some circumstantial evidence may suggest an alternate etiology in extrapyramidal disease. Just as the oscillation frequency in our patient was in the range reported for tremor frequency in Parkinson's disease patients, Vincken et al mentioned "the frequency of flow oscillations to be similar to that of the tremor in the extremities—4 to 8 Hz..." in their patients with "Type A" patterns on flow volume loops. Moreover, Estenne and colleagues reported repetitive bursts of EMG activity in the scalene and parasternal muscles in the range of 4 to 8 Hz in two patients with Parkinson's disease. Lastly, does Vincken and Cosio's description of involuntary movements at a rate of 4 to 8 Hz constitute the observation of a tremor? Thus, I believe that at this time, the etiology of the saw tooth pattern in these patients is still speculative.

Philip L. Schiffman, M.D., F.C.C.P.
Associate Professor of Medicine
University of Medicine and Dentistry of New Jersey, Rutgers Medical School
New Brunswick

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2 Sanders MH, Martin RJ, Pennock BE, Rogers RM. The detection of sleep apnea in the awake patient: the "saw tooth" sign. JAMA 1981; 241:14-18
Preventing PPC

To the Editor:

In the editorial entitled "Prevention and treatment of postoperative atelectasis" (Chest 1985; 87:1-2), Dr. O'Donohue depicts a very negative picture of the state-of-the-art in the prevention and treatment of postoperative pulmonary complications (PPC). He correctly points out that the problem should be addressed differently when dealing with patients undergoing cardiopulmonary surgery than in those having nonthoracic procedures. It is in this last and most numerous group where significant progress has been made.

The following factors help identify those patients at high risk for the development of PPC who are benefited by preventive treatment:1, 2 patients undergoing upper abdominal operations, those with lung disease and abnormal pulmonary functions, the elderly, the obese, and patients who have prolonged surgery. In such a high risk group, Stein et al.3 used a multifaceted approach consisting of antibiotics, weight loss, bronchodilators, postural drainage and smoking cessation, and demonstrated a significant decrease in the incidence of PPC in the treated group as compared with untreated control subjects. Thoren et al.4 studied 392 patients who underwent cholecystectomy and evaluated the use of routine chest physical therapy in preventing PPC. He documented the importance of beginning therapy before surgery when he showed that, in this subgroup, the incidence of PPC was the lowest (12 percent). This result was significantly different from those patients whose therapy was begun after surgery (27 percent) and from untreated controls (42 percent). These two studies provide an answer to one of Dr. O'Donahue's questions, "When to treat?" The evidence suggests that it is better to begin before the surgical procedure.

The second question posed by Dr. Donahue is, "How to treat the patients at risk?" We have reported the results of a study which meets the requirements recommended in his editorial. It was controlled, randomized, preventive in nature and looked at length of stay as a way to evaluate the benefit of the routine application of a costly form of therapy.5 Our results showed that supervised maneuvers that encourage lung expansion, whether with mechanical aids such as incentive spirometry and intermittent positive pressure breathing or without mechanical aids, such as deep breathing exercises, were equally effective in decreasing the incidence of PPC. We also found that in the high risk group of patients undergoing upper abdominal surgery, incentive spirometry and, to a lesser extent, deep breathing exercises not only decreased the incidence of PPC, but they were associated with a decrease in the post-operative length of stay.

We believe that significant advances have been made in the understanding and prevention of PPC, and that there is some wisdom to the pattern of usage of different therapeutic modalities in the hospital survey reported in the same issue of Chest.6

Bartolome R. Celli, M.D.; Gordon L. Snider, M.D., F.C.C.P.
Boston University School of Medicine.
Pulmonary Center
Boston, MA

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To the Editor:

I very much appreciate the comments of Drs. Celli and Snider and certainly agree that it is most important to identify patients who are at risk for postoperative complications. There is no question that the multiple therapies which are applied, both preoperatively and postoperatively, are beneficial in preventing respiratory complications. In relation to the modalities used specifically for lung expansion, there is clearly reason for a "negative picture of the state-of-the-art" because, in fact, there is no clearly defined state-of-the-art at a time when these modalities are being used extensively in virtually every hospital in the United States, as documented by the national survey.

As cited in their letter, the recent study by Celli, Rodriguez and Snider demonstrates very clearly some of the difficulties encountered in current investigative techniques. In this study three modalities of lung expansion (intermittent positive pressure breathing, IPPB, incentive spirometry, IS, and deep breathing exercises, DBE) were compared with each other and with a control group of patients for the prevention of postoperative pulmonary complications. All lung expansion techniques were used four times daily (presumably every six hours). The use of any modality of lung expansion so infrequently in the immediate postoperative period is not likely to make any significant difference in the development of atelectasis or other respiratory complications (as demonstrated by the findings in this study). The protocol for IPPB indicated that it was administered with a preset pressure of 15 cm H2O for 15 min. This is certainly not a technique for effective lung expansion and probably serves only as an expensive aerosol generator. Failure to measure the lung volumes achieved with IPPB virtually invalidates any comparison of deep breathing techniques. The attempt to make valid conclusions about which technique is the most or least preferred treatment modality is a prime example of the problems which this editorial attempts to address.

My comments are not intended to be unkind to the authors of this well-done study, but only to point out that this very recent report by respected investigators serves as an excellent example of the absence of a state-of-art for this type of therapy, in terms of when it should be used, how frequently, and what are the most effective techniques for usage. Very importantly, this study in no way addresses which of these modalities for lung expansion may be most effective in treating postoperative atelectasis once it has occurred. This was not the intention of the authors but it does represent an entirely different side of the coin, in that some modalities which may be impractical or too expensive to be justified for prophylactic use may be very effective and cost saving when used more frequently for treatment of clinically significant atelectasis once it occurs—we really don't know!

Walter J. O'Donohue, Jr., M.D., F.C.C.P.
Professor and Associate Chairman
Department of Internal Medicine
Chairman, Pulmonary Medicine Division
Creighton University
Omaha, Nebraska