Tracheoesophageal Fistula Following Blunt Chest Trauma

R. S. Rampaul, V. Naraynsingh and V. S. Dean

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Systemic Distribution of Talc

To the Editor:

The recent article by Werebe and colleagues (January 1999) concerning the systemic distribution of talc was a fascinating and timely study. This group has a very large experience with talc pleurodesis, and they are to be commended in pursuing not only the efficacy of the procedure, but also the safety. As they are aware, a large (n = 500) multicenter trial is underway in the United States exploring the efficacy of talc pleurodesis—administered either through a chest tube as a slurry or via video-assisted thoracoscopy as an insufflated mist—in patients with a malignant pleural effusion. As co-principal investigator of this trial, I have been very sensitive to the potential safety issues relative to talc insufflation. For this reason, I believe we need further studies exploring the results of disseminated talc.

Somewhat surprisingly, all talcs are not equal. The crystal structure and size of the talc particle may vary depending on the location of the talc mine! Perhaps, it is this variability in the size or the structure of the talc that determines the permeability or distribution, and thus potential toxicity of intrapleural talc.

Approximately 250,000 people each year in the United States have a malignant pleural effusion. Talc is the most effective sclerosing agent identified to date. Thus, it is important to confirm its safety. I was very pleased to read this study by Werebe and colleagues and hope that further discussion and research is engendered.

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REFERENCE

Zafirlukast and Churg-Strauss Syndrome

To the Editor:

The case report by Knoell et al (July 1998) of a patient with Churg-Strauss syndrome associated with zafirlukast is very similar to the case we reported in JAMA in February 1998 of eight patients who developed a similar syndrome. In all of the cases, the patients developed at least four of the six criteria required for patients to be considered as having the Churg-Strauss syndrome in association with zafirlukast. In their case report, within 1 month of taking zafirlukast for worsening asthma, the patient developed a biopsy-proven eosinophilic vasculitic rash, pulmonary infiltrates, and extravascular eosinophils on lung biopsy. Unlike the cases reported in our series, Knoell et al found no evidence of cardiac involvement in their patients.

The authors contrast their case report with our series by stating that all of our cases occurred in the setting of corticosteroid withdrawal. However, their patient had “experienced multiple asthma exacerbations that required treatment with prednisone” and subsequently had been maintained on inhaled corticosteroids when the syndrome occurred. It is our contention that this patient’s severe asthma that required frequent courses of corticosteroids was the heralding event of incipient Churg-Strauss syndrome. We believe that the corticosteroids that were given to treat severe asthma likely masked the development of other systemic eosinophilic manifestations that occurred subsequently. Even the inhaled steroids that the patient was receiving could have masked the syndrome. While there was an association with zafirlukast use, there is still no convincing evidence that the medication can be causally linked because the patient’s course remains consistent with the natural course of the Churg-Strauss syndrome (ie, indolent allergic disease that evolves into asthma with multiple exacerbations that may progress to eosinophilia and then finally progress to multiorgan systemic eosinophilic vasculitis). We agree that health care providers must remain wary of the syndrome, especially in subjects with steroid-dependent asthma, but we also feel strongly that all new cases should be reported to the drug’s manufacturer and to the US Food and Drug Administration so that proper assessment of disease pathogenesis can occur.

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Dr. Drazen has served (within the past 5 years) as a consultant to the following pharmaceutical companies: Abbott, Bayer, Biogen, Eli Lilly, Forest Laboratories, Genetics Institute, Genome Therapeutics, Glaxo, Marion-Merrill-Dow, Merck, Pfizer, Roche Bioscience, Schering, Spacorac, and Zeneca. Dr. Drazen’s laboratory has received support for asthma trials from Abbott, Astra, Genetics Institute, Immunologies, Merck, Millenum, Schering, Wyeth-Ayerst, and Zeneca. Dr. Drazen holds no equity position in any of these entities. He serves on the scientific advisory board for the following companies: Astra, Genetics Institute, Immunologies, Merck, Millenium, Schering, Wyeth-Ayerst, and Zeneca.
of HiLife Health Systems, Inspire Pharmaceuticals, and Aradigm Medical Products. As compensation for service on these boards, Dr. Drazen holds equity positions in each of these entities.

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References

Tracheoesophageal Fistula Following Blunt Chest Trauma

Diagnosis in the ICU—the “Breathing Bag” Sign

To the Editor:

In the Intensive Care Unit (ICU), when patients are intubated and/or sedated, the diagnosis of many conditions often depends solely on the recognition of physical signs. Complications, such as an acquired tracheoesophageal fistula (TOF) in the critically ill, is one such condition, which may be difficult to diagnose and carries a high mortality.1,2 We wish to describe a new clinical sign that may be of value in the early diagnosis of this condition as it presents in patients in the ICU receiving positive-pressure ventilation: the “breathing bag” sign.

A 43-year-old male victim of a road traffic accident was admitted with a flail chest and severe bilateral pulmonary contusions. Due to respiratory embarrassment and deteriorating blood gases, he was intubated and put on intermittent positive-pressure ventilation. Four days following injury and admission, abdominal distension was noted. Later that day, his nasogastric bag was observed to fully distend and deflate cyclically in phase with the ventilator. These movements reminded us of an anesthesiologist’s breathing bag during spontaneous respiration. Tracheal tube placement was reconfirmed by auscultation and capnography. A TOF was diagnosed on the basis of the above observations and clinical findings. Inspection with a fiberoptic bronchoscope confirmed this at 2 cm above the carina.

TOF complicating blunt chest trauma is rare, and although it is difficult in this case to directly ascribe its etiology, either from blunt chest trauma, overinflation of the endotracheal cuff leading to tracheal necrosis, or both, early diagnosis and surgical intervention carry a good prognosis.1,3 We wish to describe a new clinical sign that may be of value in the early diagnosis of this condition as it presents in patients in the ICU receiving positive-pressure ventilation: the “breathing bag” sign.

IV Immunoglobulin for Asthma?

To the Editor:

Landwehr et al (November 1998) conclude that IV immunoglobulin (IV Ig) “provides a potentially important adjunctive therapy in severe asthma, reducing oral steroid requirements and steroid side effects without deterioration of lung function.” They make this statement definitively and without apparent equivocation based on an open-label study done without controls for a disease known to be highly variable in its clinical course. They identified the greatest benefit to be in adolescents, a population known to have a high rate of noncompliance with effective inhaled maintenance medications, such as inhaled corticosteroids, without considering the potential influence of closer monitoring during a study on their adherence to other aspects of the medical regimen. No credible mechanism is proposed to explain the alleged benefit that IV Ig has in patients with asthma other than the argument by analogy of its benefit for patients with other diseases known to be highly variable in its clinical course. They report for these 11 cases are sufficient to argue for a controlled clinical trial, it appears premature and imprudent to state, as the authors do, that “this study extends and confirms the response of patients with severe, steroid-dependent asthma to IV Ig therapy.” Since one of the authors of this manuscript also coauthored the previous report showed that IV Ig on asthma. Although the results reported for these 11 cases are sufficient to argue for a controlled clinical trial, it appears premature and imprudent to state, as the authors do, that “this study extends and confirms the response of patients with severe, steroid-dependent asthma to IV Ig therapy.” Since one of the authors of this manuscript also coauthored the previous report showed that IV Ig on asthma. Although the results reported for these 11 cases are sufficient to argue for a controlled clinical trial, it appears premature and imprudent to state, as the authors do, that “this study extends and confirms the response of patients with severe, steroid-dependent asthma to IV Ig therapy.” Since one of the authors of this manuscript also coauthored the previous report showed that IV Ig on asthma. 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To the Editor:

Dr. Weinberger has raised several important issues regarding our recent publication on the benefits of high-dose IV immunoglobulin (IVIg) in patients with severe steroid-dependent asthma (November 1998). ¹ He questions our use of definitive language, our conclusions based on a population with a high failure rate of compliance, our failure to propose a credible mechanism of action, and the absence of a randomized placebo-controlled clinical trial.

We believe that throughout the paper, we discuss in a non-definitive manner the potential for IVIg therapy and the need for further studies that define the mechanism of action and the optimal treatment regimen. The issues raised about compliance are certainly valid. In the design of the study, we made vigorous attempts during the 2-month run-in to optimize therapy during regular visits. In addition, because these patients were regularly followed at the National Jewish Medical and Research Center, they were under close monitoring prior to the initiation of the study. During the study, the patients were only seen monthly. As to the mechanism of action, most investigators believe that IVIg exhibits potent anti-inflammatory effects, and one target is the inhibition of cytokine production. This has been documented in several publications including our own.² ³ ⁴ A novel mechanism of action proposing that IVIg may affect steroid sensitivity has been reported.⁵ The final issue questions why a randomized placebo-controlled trial has not been carried out. There is no doubt in any of our minds that such a trial is absolutely required. Some trials have been carried out in a double-blind fashion but at lower doses. Nonetheless, their findings support our conclusions.⁶ However, in light of the current limitations of IVIg availability and the reluctance of pharmaceutical companies to support such a trial at the recommended doses during the current crisis period, we have continued to examine all issues surrounding IVIg use in this small group of asthmatics by using a cross-over design. In this way, we believe that progress has been made and that patients have benefited. We have been cautious in our conclusions, and we do not consider any of our statements as “premature or imprudent.”

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Assessing Respiratory Resistance Without Spirometry

To the Editor:

The paper by Ducharme and Davis (June 1998)¹ highlights the usefulness of forced oscillation methods for assessing respiratory resistance noninvasively in young or acutely unwell subjects who are unable to perform spirometry. The authors stated that there is only one previous report estimating the intraclass correlation coefficient for repeated measures using the technique. In fact, this is not the case. Using the Siregnost FD5 10-Hz device (Siemens; Erlangen, Germany), I found correlation coefficients of 0.77 in the respiratory resistance averaged during quiet breathing in 26 healthy subjects across three separate visits.² Gating to end-inspiration, these intraclass correlation results improved slightly to 0.80, then at end-expiration to 0.78. In asthmatics and in patients with COPD studied via a tracheostoma, gating to the respiratory phase also had a marked influence on the responsiveness of the respiratory resistance, with the greatest responses seen at end-expiration at 10 Hz after bronchodilatation.³

Ducharme and Davis highlight the particular usefulness of measurements of resistance, especially when made between 5 to 10 Hz, in terms of their excellent correlation to the FEV₁ and other markers of clinical response to bronchodilatation. Another important need for this technique is to standardize the test frequencies and sampling modes used in making these measurements. This should help to further improve the cross-comparisons that can be made among these important studies.

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Successful Management of Adenoid Cystic Carcinoma of the Trachea by Laser and Irradiation

To the Editor:

Segmental resection and reconstruction performed in a single stage and adjuvant pre- and post-operative irradiation have long been used to offer the best chance for cure for adenoid cystic carcinoma (ACC) of the trachea. For cases in which the ACC is nonresectable, palliative methods that yield good success rates are available. We report a case of ACC that was successfully managed with Nd-YAG laser photoresection and external beam irradiation and high-dose-rate (HDR) brachytherapy.

A 44-year-old white man with recurrent shortness of breath and multiple episodes of pneumonia for 18 months was referred to us for the management of an exophytic mass involving the trachea detected on flexible bronchoscopy. A CT scan of the chest showed a tracheal mass with no hilar or mediastinal lymphadenopathy. Repeat bronchoscopy at our institution showed a large tracheal submucosal and exophytic mass starting 3 to 4 cm below the vocal cords, which extended 1 cm above the carina with a vertical length of 6 cm and caused an 80% obstruction of the mid and lower trachea (Fig 1). Bronchial fine needle aspiration revealed atypical cells derived from an epithelial neoplasm, which was consistent with ACC.

The patient’s respiratory sounds were stridorous, and he was in moderate respiratory distress. Arterial blood gas analysis revealed a pH of 7.39, a PCO₂ of 40 mm Hg, a PO₂ of 110 mm Hg, an HCO₃⁻ of 24 mEq/L, and an arterial oxygen saturation of 98% on 4 L of O₂. As a precaution, helium-oxygen mixture was kept at the patient’s bedside until bronchoscopic resection was performed. The patient underwent Nd-YAG laser photoresection with a flexible bronchoscope under general anesthesia using a laryngeal airway mask at two sittings, and 90% patency of the trachea was established. There was significant relief of symptoms after the endobronchial photoresection.

The patient was started on external beam radiation, and he received a dose of 50 Gy in 25 fractions. He also received endobronchial radiation therapy in three fractions using a 102Ir device with a total dose of 21 Gy. Repeat flexible bronchoscopy a month after the radiation therapy revealed a complete patency of the trachea (Fig 2). The results of the cytology tests on tracheal washings were negative for malignant cells. We plan to follow the patient with flexible bronchoscopy every 4 months for any recurrence of the ACC.

ACC is a slowly growing, late metastasizing, and locally recurrent tumor with a prolonged natural history. As reported in our case above, debulking and irradiation can provide excellent palliation. This can be achieved with good precision using the laser. The role of adjuvant therapy is difficult to evaluate with certainty. Endobronchial brachytherapy is now widely used to increase the total dose of irradiation and to improve local tumor control. Follow-up bronchoscopies and frequent monitoring for spread of the tumor are required after initial debulking and irradiation.

Our case report supports the literature in that a combination therapy of endoscopic laser photoresection, external beam radiation, and endobronchial radiation therapy may provide better palliation and survival in patients with a nonresectable ACC. Also, in a patient presenting with critical airway obstruction, endoscopic laser photoresection allows for better ventilation of the airway. Because radiation therapy has the potential to further compromise the already obstructed airway, we prefer to perform endoscopic laser photoresection prior to radiation therapy.

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Figure 1. Bronchoscopic image of the ACC of the trachea on initial presentation.

Figure 2. Patency of the trachea reestablished after Nd-YAG laser photoresection of the ACC.
Questioning Flutter Therapy

To the Editor:

The recent investigation by Homnick et al (October 1998) was designed to compare two modes of chest physiotherapy in hospitalized patients with cystic fibrosis (CF). The authors alluded that physiotherapy using a "flutter device" (which generates rhythmic variations in positive expiratory pressure) appeared to be as effective as "standard chest physiotherapy" (a nebulous term) in the clinical management of this patient group.

These authors failed to objectively describe the treatment procedures used in their investigation. They reported the use of guidelines from the Cystic Fibrosis Foundation or from the manufacturers of the flutter device; however, without citing references, this precludes the proper interpretation of this investigation. Williams reports that the poor definition of physiotherapy techniques in most CF research is "the most confounding factor when reviewing the literature."

Baseline demographics were reported as being similar between the two groups studied; however, trends in the data indicated that there were younger patients and patients with lower FVC and FEV1 in the standard chest physiotherapy group, even though the differences were not statistically significant. These differences may have impacted the treatment outcome and thus limited the study conclusions.

Homnick et al only partially identified the limitations of their investigation. Most CF physiotherapy research includes inadequate sample sizes that often lead authors to incorrectly conclude that there are no differences between the two therapy groups in baseline characteristics or clinical outcome, because there were no statistically significant differences (type II error). The authors reported that a sample size of 219 would be required to achieve 80% power; however, the details of the method used for this power calculation are not provided. Homnick et al failed to report important trends in the data that indicated greater improvements in clinical outcome ("clinical score" that included sputum volume, respiratory rate, pulmonary function, body temperature, and weight gain) in the standard chest physiotherapy group, even though the results were not statistically significant.

The increased staffing costs required for the provision of standard chest physiotherapy led the authors to argue for the use of flutter device therapy, considering the equivalence in clinical outcome. The misleading references made by Homnick et al to Reisman et al that imply that standard chest physiotherapy is potentially traumatic are of concern. Reisman et al demonstrated the benefits of a chest physiotherapy regimen that included postural drainage, chest wall percussion, and forced expiratory technique, which reduced the deterioration in pulmonary function and general health when compared to a less intensive form of physiotherapy (forced expiratory technique alone), thereby contradicting the statement by Homnick et al. Although Homnick et al may advocate the use of more cost-effective physiotherapy, there is inadequate evidence to support flutter device therapy. Pryor et al report significantly reduced clearance of airway secretions with the use of a flutter device, indicating that a simplistic approach to promoting a "generic" therapy could be detrimental to patient outcome. The impact of modifying physiotherapy treatment on long-term patient outcome in this patient group must be rigorously investigated before modifications to treatment are suggested. Randomized controlled multicenter investigations, with adequate patient numbers and descriptions of the treatments and measurement tools are recommended before we change patient care. As pointed out by Williams, disease severity, treatment compliance, airway lability, and adjunctive medications are highly variable, and "it may be more appropriate to consider which physiotherapy regimens are more effective for individual patients rather than any one technique being the most effective for all patients with cystic fibrosis."

Chest physiotherapy that includes postural drainage, forced expiratory technique, and chest wall percussion, has been demonstrated to significantly enhance the clearance of airway secretions. The role of these treatment regimens in the prevention of pulmonary dysfunction and morbidity must be further investigated, as recommended by Reisman et al. CF patients have successfully been instructed in the use of standard chest physiotherapy regimens independently of physiotherapists and respiratory therapists, which may reduce the potential cost issues previously cited. Patient compliance with physiotherapy is of relevance to the use of the flutter device. The authors have not provided any evidence that patients would be any more compliant with the flutter device. A recent investigation by Scherer et al that high frequency oral and chest wall oscillation was as effective as "conventional chest physical therapy" in augmenting sputum clearance in 14 stable CF outpatients and that these modalities could be a useful way to reduce health costs. These authors, however, defined "conventional chest physical therapy" as including postural drainage, mechanical chest wall percussion/vibration and coughing, yet another variant of "standard" chest physiotherapy! Aside from other methodological flaws with this research, the "conventional chest physical therapy" used in this study is definitely not the most optimal form of physiotherapy for secretion clearance. The aims of these researchers is to justify the use of "novel" approaches to therapy to reduce health care costs, may "we" at least compare the most optimal form of physiotherapy treatment, rather than using substandard or poorly described treatment regimens. We must design better clinical research if we are to discern the most effective treatment approaches.

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To the Editor:

In regard to Dr. Ntoumenopoulos’s response to our article (October 1998),1 we agree that most studies of airway clearance devices and techniques in cystic fibrosis consist of insufficient numbers to provide adequate statistical power to draw absolute conclusions about their comparative efficacy. However, there are also few studies such as ours that attempt to define the sample size necessary to answer this question. Of course, every study must define its variables, including methods used, and we clearly stated that our interventions were based on our hospital protocols, which are available to anyone or any institution requesting them. Regarding the comments about trends in our data, we believe that trends are trends and that they are only of interest as topics for future investigation. Extensive comment on data that is not statistically significant is not wise in our opinion. Concerning issues surrounding the potential trauma of chest physiotherapy, our use of the word trauma was not meant to imply a potentially poorer outcome; rather, it simply referred to physical trauma. Many of us who have treated children with cystic fibrosis for many years have noted the potential for chest wall trauma when manual chest physiotherapy is applied too vigorously (muscle soreness, ecchymoses, and even rib fracture in unusual cases, etc). Our attempts to introduce and study alternative airway clearance therapies is based on our evolving needs to provide for patient independence and the desire for something with potentially greater efficacy while maintaining low costs and simplicity of use.

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