was detected. However, those studies would presumably be clinically indicated. It is the goal of all of us to reduce the cost of testing that does not further the clinical outcome of the patient.

Larger studies as well as investigations of different patient populations will be needed to answer all the questions surrounding the topic of routine chest radiographs in patients with respiratory failure who are treated with mechanical ventilation. Such studies should be performed in a randomized, controlled protocol, and meaningful clinical outcomes should be sought.

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Metabolic Alkalosis and Cystic Fibrosis

To the Editor:

I read with interest the study by Holland et al (August 2003) describing the metabolic acid-base status in patients with acute exacerbations of adult cystic fibrosis (CF) and COPD. I congratulate them on an interesting descriptive study of acid-base status and electrolytes in COPD and CF. However, I disagree with their conclusion that "metabolic alkalosis contributes to hypercapnic respiratory failure with acute exacerbations of CF." No arterial blood gas or electrolyte data is provided to establish a premorbid baseline in either the COPD or CF groups to support the inference that one or both groups are indeed in acute hypercapnic respiratory failure. The arterial blood gas and electrolyte data on presentation in both groups could be interpreted as chronic hypercapnic respiratory failure with appropriate compensatory metabolic alkalosis. In fact, the mean inorganic strong ion difference (Na\(^+\) + K\(^-\) – Cl\(^-\)) in both the CF and COPD groups is approximately equal (∼45.7 mmol/L and 45.1 mmol/L or milliequivalents per liter, respectively) and consistent with appropriate renal compensation for chronic hypercapnea. An equal mean inorganic strong ion difference between the CF and COPD groups reveals that the magnitude of the metabolic alkalosis attributed to electrolyte differences is equal in both groups and does not account for the minor difference in mean pH. The CF group manifested a more severe hypoalbuminemia (less plasma weak-acid content) relative to the COPD group, and fully accounts for the alkaline difference in mean pH between the groups as assessed by physicochemical analysis. The inference of the author is of clinical concern since overzealous correction of a compensatory metabolic alkalosis may unmask a severe respiratory acidosis. As well, aggressive ventilation for chronic hypercapnic respiratory failure may unmask a severe metabolic alkalosis. Insufficient data are present to support the conclusion that a metabolic alkalosis in adult CF in this setting contributes to hypercapnic respiratory failure.

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To the Editor:

We thank Dr. Omron for his interest in our article examining acid-base status in patients with cystic fibrosis (CF) and COPD. His comments focus on two main points: whether the differences in acid-base balance do in fact represent a primary metabolic alkalosis in addition to respiratory acidosis in the CF group, and what the mechanism of the observed changes may be.

In the setting of an acute exacerbation of lung disease accompanied by hypercapnia, we observed a significantly more alkaline pH in the CF group than in the group of patients with COPD. Although the mean difference in pH between the groups was modest in absolute terms, we noted that 13 of 14 patients with CF (93%) had a pH > 7.4, which would be an unusual feature of a partially compensated respiratory acidosis. In addition, in 13 of 14 of the patients with CF (93%), the observed renal response to elevated Pa\(_{CO_2}\) was greater than would be expected to compensate for a chronic respiratory alkalosis. We therefore feel confident that the observed differences indicate both primary metabolic alkalosis and primary respiratory acidosis in the patients with CF.

This was a cross-sectional study in which we described acid-base status in hypercapnic patients with CF and COPD presenting with an acute exacerbation of lung disease. As such, our article did not aim to determine the cause of the observed metabolic alkalosis in CF. We concur with Dr. Omron that hypoalbuminemia may play an important role in acid-base disturbance, and the data we presented support this hypothesis. However, in the light of previous work showing an association between chloride depletion and metabolic alkalosis in children with CF, along with observed changes in cerebrospinal fluid chloride levels with changes in plasma chloride, the contribution of electrolyte status requires evaluation. We are currently conducting further work in this area.

We agree with Dr. Omron that the clinical implications of this study are as yet unclear. It is possible that correction of metabolic alkalosis in this patient group may not be advantageous; however, this has not been examined. In addition, the effects of noninvasive ventilation on respiratory drive in patients with metabolic alkalosis are unknown. These issues are yet to be examined in clinical trials. It is clear, however, that acid-base analysis in patients with multisystem diseases such as CF needs to encompass more than a cursory glance at Pa\(_{CO_2}\).

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References


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Plication for Diaphragmatic Eventration

A Simple Technique, Not a Simple Problem

To the Editor:

The case recently reported in CHEST by Hwang et al (July 2003)1 left three important questions open to readers. Even if technically simple, are we justified to propose surgical correction of a diaphragmatic eventration only on the basis of progressive dyspnea on exertion and chest radiograph findings? We do not think so.

At least two steps in the evaluation of this patient are missing.

1. Clinical history: if the patient has no previous chest radiograph, in our opinion the case has to be classified as a recently diagnosed eventration (ie, one known for < 2 years). In this case, a long period of observation (at least 1 year) is needed to exclude the possibility of diaphragmatic function recovery. Moreover, in the assessment of diaphragmatic eventration, a detailed clinical history is crucial to determine the most likely cause. The presented case concerns a patient with a mediastinal deviation, which can cause troubles with cardiac rhythm. Has this aspect been investigated? In cases in which patients are aware of their eventration, dyspnea can be very difficult to quantify because they can easily translate their anxiety into a wide variety of respiratory symptoms. If this is the case, more qualifying signs, such as orthopnea, have to be searched for.

2. Radiologic evaluation: a CT scan is essential to rule out thoracic or abdominal disease, to assess the anatomic area of the phrenic nerve, and to evaluate lung parenchyma. On the basis of the presented chest radiograph, how can authors exclude the presence of a left main bronchus lung cancer with lymph node metastases of the aortopulmonary window?

The proposed technique is not that different from the videoassisted technique that we presented in 19962 and performed in 12 patients in the last 10 years. Do the proposed modifications improve the technique? This is questionable for several reasons. The only theoretical advantage to the modifications is the avoidance of a 5-cm minithoracotomy by the insertion of two 5-mm ports in the eighth and ninth intercostal spaces (with possible injury to two different intercostal nerves). In such a situation, the surgeon starts the suture in the simplest position possible injury to two different intercostal nerves). In such a situation, the surgeon starts the suture in the simplest position but ends up in the most difficult one, with the largest part of the eventration from the midline of the posterolateral diaphragmatic dome to the level of the phrenic nerve. A good correction shows a diaphragm progressively ascending from the attachments just below the posterior minithoracotomy, assuming a horizontal portion at the level of the cardiophrenic angle. If the diaphragm overcomes this angle, it has been undercorrected. If the diaphragm becomes a linear, flat, oblique, and hard plane between its costodiaphragmatic portion and the cardiophrenic angle, it has been overcorrected (Fig 1).

Finally, a single running suture cannot be enough for a structure such as the diaphragm, which continuously changes its profile.

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REFERENCES

To the Editor:

We appreciate the interest in our case report (July 2003)1 and the comments made by Leo and colleagues. They pointed out several problems, including evaluation of the patient, minithoracotomy, and direction of the repair.

The most important step in the evaluation of the patient is the search for the cause of disease. In our case, we had been observing the patient for about 6 years when the operation was performed. A chest CT scan obtained with the patient in the posteroanterior position 6 years before the operation showed...