Peripheral lesion in a particular segment, but no lesion is seen during the initial bronchoscopic inspection.

The technique of saline distension involves rapidly instilling 10 to 20 mL of 0.9% saline solution via the suction channel of a flexible bronchoscope that is wedged in a segmental or subsegmental bronchus. This saline distension often allows a more-distal view of the bronchus and may indicate the nature of any narrowing, if present.

Saline distension is a technique that can reveal hidden peripheral tumors, and it can provide the operator with an immediate indication, at the time of flexible bronchoscopy, as to whether an area of narrowing in a segmental or subsegmental bronchus may be true or apparent. This, in turn, may help guide the physician in arranging the priority and urgency of future investigations. It may also help the bronchoscopist in deciding the type and amount of specimens to be taken from that area.

In a recent audit of a total of 319 flexible bronchoscopy procedures, carried out in our unit to investigate possible lung cancers during a 12-month period, the technique of saline distension was used in 47 cases. It was not associated with any significant complications. The technique of saline distension revealed hidden tumors in approximately 1% of all patients undergoing flexible bronchoscopy procedures, although the yield was much higher if used in a selected group of patients where a plain chest radiograph suggested a possible tumor in a medium-sized airway. The operator had made use of the technique in nine cases in which, on initial inspection, no lesion had been seen, despite a plain chest radiograph suggesting a likely tumor in a segmental or subsegmental bronchus. This technique revealed the presence of hidden tumors (not visible at routine bronchoscopy because of peripheral location; Fig 1) in three of these patients (one submucosal and two endobronchial tumors). All had positive brush cytology findings.

If the bronchoscope is wedged in a small bronchus, the saline solution will remain static, giving a clear view for a minute or more. This can allow “underwater” photographs and brush and biopsy samples to be taken. The technique can also provide a clear view in bronchi that are partially occluded by blood or mucus that cannot be aspirated due to the small size of the airways.

During inspection with a flexible bronchoscope, the operator may discover apparent narrowing of a segmental bronchus. Use of the technique of saline solution distension in this circumstance can help the bronchoscopist in the assessment of the underlying nature of the narrowing, although sensitivity will be low. If the bronchus distends easily when saline solution is instilled, this indicates that fixed narrowing due to peribronchial tumor is less likely. There were 21 cases in our study in which a narrowed bronchus had easily distended with saline solution, revealing a macroscopically normal distal airway. All subjects had negative cytology findings, although 3 of 13 subjects did have CT-scan findings suggestive of peribronchial tumor. If the narrowed bronchus is nondistendable, the chances of an underlying tumor in the airway are much greater. In our study, 15 of 17 patients (88.2%) with a fixed narrow bronchus had evidence of a peribronchial tumor by cytology and/or histology results, or CT-scan findings. Only one patient had an apparently fixed narrow bronchus at flexible bronchoscopy but no evidence of tumor on a CT scan or from cytology/histology.

There is a strong correlation between an abnormal result during saline distension (ie, failure of bronchus to distend or distension revealing an endobronchial tumor) and the chances of either positive cytology/histology or abnormal CT-scan findings. The information is immediately available to the physician. The physician must remain aware that the technique should be used to give an indication, but it is not diagnostic of malignancy; other causes of airway rigidity, such as posttuberculous scarring, are possible in addition to malignant stenoses. This technique is both quick and easy to perform. It does not require any additional equipment by the bronchoscopy unit. In our own experience, it is not associated with any significant complications. We conclude that the technique of saline distension is a useful tool in the bronchoscopist’s armory.

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Transient Lactic Acidosis as a Side Effect of Inhaled Salbutamol

To the Editor:

Transient increase of lactate levels (lactatemia) with or without metabolic acidosis has been seldom reported as a complication of 2-adrenergic agents administered during an asthma attack or for preterm labor therapy. The mechanism of this complication is poorly understood. In previous reports,1–3 lactatemia or lactic acidosis were associated with IV administration of β2-agonists or aminophylline, or a combination of inhaled β2-agonists and IV aminophylline.

During the last 6 months, transient lactatemia and/or lactic acidosis were observed in five patients admitted in our department for an asthma attack (Table 1). All of these patients were initially treated with 5 mg of inhaled salbutamol before blood gas (and lactic acid) analysis was performed. No IV bronchodilators were administered, and methylprednisolone, 40 mg/d, was ad-

Table 1—Functional and Metabolic Characteristics of Asthmatic Patients With Transient Lactatemia Attributed to the Administration of Inhaled Salbutamol

<table>
<thead>
<tr>
<th>No.</th>
<th>Patient</th>
<th>FEV1, L</th>
<th>Po2, mm Hg</th>
<th>PCO2, mm Hg</th>
<th>Arterial pH</th>
<th>Lactate, mmol/L</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td></td>
<td></td>
<td>T1  T2</td>
<td>T1  T2</td>
<td>T1  T2</td>
<td>T1  T2</td>
</tr>
<tr>
<td>2</td>
<td></td>
<td></td>
<td>2.1 4.49</td>
<td>82 85</td>
<td>34 44</td>
<td>7.36 7.38</td>
</tr>
<tr>
<td>3</td>
<td></td>
<td></td>
<td>0.8 1.9</td>
<td>83 85</td>
<td>42 44</td>
<td>7.34 7.41</td>
</tr>
<tr>
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<td>85 90</td>
<td>38 43</td>
<td>7.38 7.42</td>
</tr>
<tr>
<td>5</td>
<td></td>
<td></td>
<td>1.7 2.9</td>
<td>65 86</td>
<td>30 40</td>
<td>7.43 7.41</td>
</tr>
</tbody>
</table>

*T1 = measurement after first hour of treatment; T2 = measurement 24 h later.
ministered only after the first blood analysis. No clinical or laboratory indications of any alternative cause of lactatemia (eg, hypoxemia, hypoperfusion, sepsis) were identified, in accordance with previous reports. Serum lactate levels were not related with the severity of the airway obstruction or the degree of dyspnea. However, in all five cases, resolution of asthma symptoms was accomplished by intensive treatment with inhaled salbutamol. After 24 h, serum lactate levels had returned to normal without any specific treatment.

To the best of our knowledge, this is the first report of transient lactatemia associated with the sole administration of inhaled salbutamol. The reason why only a small proportion of asthmatic patients treated with β-agonists develop lactic acidosis remains to be elucidated. Lactatemia, besides its metabolic consequences, may increase the sensation of dyspnea and compensatory hyperventilation. This situation could be easily misinterpreted as a sign of treatment failure and lead to inappropriate intensification of treatment.

Physicians who treat patients for severe bronchospasm should be aware of this side effect of bronchodilators, which might prove of clinical significance in the more severe cases where other causes of metabolic acidosis coexist.

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To the Editor:

We read with interest the article by López-Peláez et al (June 2001). This reminded us of a 28-year-old man of Chilean origin who was under detention due to an immigration offense. He was admitted to our emergency department with a painfully swollen face and neck. The physical examination revealed subcutaneous emphysema in the face, neck, and supraclavicular region with normal cardiorespiratory function. Chest radiography and CT of the mediastinum showed a pneumomediastinum with a small bilateral pneumothorax. An esophageal radiogram with contrast did not detect any leakage.

The etiology of the condition could not be explained by these diagnostic procedures, and the symptoms disappeared without active treatment within an observation period of 3 days in the patient ward of the detention facility. However, during the following weeks, the subcutaneous emphysema reappeared two times. Again, no active treatment was considered necessary. In the absence of any other possible cause, a self-induced injury was suspected. Further interviews with the patient indeed resulted in the admission that after self-induced punctures in the oral cavity with a sharp object, he had repeatedly performed a Valsalva maneuver. In addition, he explained that this procedure was common knowledge in South American prisoners, and was used frequently to at least achieve better living conditions by bringing about a transfer from a prison to a medical facility and possibly a better opportunity of escaping from detention.

That this technique is not only national South American “know how” but is common also in Europe is documented by several similar reports in the German-language literature that have been published at least as early as 1969. Sluga and Grünberger reported such self-induced injuries by prisoners in general.

In summary, differential diagnosis of recurring swelling of face and neck including subcutaneous emphysema and pneumomediastinum should also include self-induced oral injury followed by Valsalva maneuver, especially in patients under detention.

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REFERENCES


Self-Induced Subcutaneous Emphysema and Pneumomediastinum

Peroxisome Proliferator-Activated Receptor-γ Expression in Lung

To the Editor:

We are pleased to accept the hypothesis by Momoi et al, by adding our experimental results. Momoi et al have demonstrated that thiazolidinedione (TZD) inhibits monocyte chemoattractant protein (MCP)-1 protein and messenger RNA expression in cytokine-treated human lung epithelial cells (type II-like epithelial cells). Showing the gene expression of peroxisome proliferator-activated receptor (PPAR)γ in the lung epithelial cells, they raised the possibility that the efficacy of TZD on the lung epithelial cells may be mediated by the activation of the nuclear receptor.

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