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.4 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 achieved 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 and lead to inappropriately intensified treatment. 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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Self-Induced Subcutaneous Emphysema and Pneumomediastinum

To the Editor:

We read with interest the article by López-Peláez et al (June 2001).1 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 1969.2–4 Sluga and Grünberger5 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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Peroxisome Proliferator-Activated Receptor-γ Expression in Lung

To the Editor:

We are pleased to accept the hypothesis by Momoi et al,1 by adding our experimental results. Momoi et al have demonstrated that thiazolidinedione (TZD) inhibits monococyte 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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Communications to the Editor
We recently examined the expression of PPAR-\(\gamma\) in normal lung tissues by reverse transcriptase-polymerase chain reaction (RT-PCR), Western blot analysis, and immunohistochemistry.\(^2\) By using RT-PCR, we detected the expression of PPAR-\(\gamma\) messenger RNA in two of three normal lung tissues. By Western blot analysis, we detected the expression of PPAR-\(\gamma\) protein in three of five normal lung tissues. Immunohistochemistry demonstrated that PPAR-\(\gamma\) expression was detected in four of six normal lung tissues. In addition, we graded the intensity and extent of expression of immunoreactive PPAR-\(\gamma\) for each tissue specimen on a scale of 0 to 4 according to the grading methodology previously described.\(^3\) As a result, immunoreactive PPAR-\(\gamma\) was weakly expressed in bronchial epithelial cells, type I pneumocytes, and vascular endothelial cells (mean \(\pm\) SD immunohistologic scores were 1.0 \(\pm\) 0.3, 1.2 \(\pm\) 0.4, 0.8 \(\pm\) 0.3, respectively). However, we found the moderate expression of immunoreactive PPAR-\(\gamma\) in type II pneumocytes (mean immunohistologic score was 1.8 \(\pm\) 0.6), suggesting that type II pneumocytes may be target cells in which PPAR-\(\gamma\) is activated by TZD.

In conclusion, we would like to support the hypothesis that TZD inhibits MCP-1 secretion in interleukin-1\(\beta\)-treated human lung epithelial cells through activation of PPAR-\(\gamma\).

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Erratum
In the April 2002 issue, the article, “Two-Year Retrospective Economic Evaluation of Three Dual-Controller Therapies Used in the Treatment of Asthma” (CHEST 2002; 121: 1028–1035) by O’Connor et al, contained a typographical error. In the abstract, the number of subjects in the ICS-plus-LTM cohort should be 360, not 30.