Too Much Testing?

To the Editor:

In their recent article in CHEST (January 2014) on the treatment of acute asthma with albuterol, Lewis et al show that high levels of lactate during such treatment are highly prevalent; in addition, high plasma albuterol concentration is significantly correlated with hyperlactatemia. In a previous case involving a woman with asthma, we described lactic acidosis (pH 7.29) and a high level of arterial lactate (10.47 mmol/L) after treatment with bronchodilator aerosols (terbutaline). This level represents the highest level of lactate, without any grave consequences, that we were able to find in the literature. We concur with Lewis et al that hyperlactatemia, with or without lactic acidosis, is not necessarily a sign of immediate gravity and has no clinical consequences in these patients.

We also question the utility of measuring lactates in acute asthma. Advanced tests have dramatically changed how diseases are diagnosed and treated. A great evolution in medicine! However, we have to keep in mind that clinical examination is more important than the results of these tests, even if hyperlactatemia is present. Laboratory tests should be targeted, and physicians should not order tests if they do not think they will aid in patient management. The study by Lewis et al helps demonstrate this concept.

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Response

To the Editor:

We appreciate the letter from Dr Claret and colleagues questioning the need for serum lactate measurements in patients being treated for acute exacerbation of asthma. Our study, part of a clinical trial protocol, required repeated measures of a number of laboratory values, including plasma albuterol, serum electrolytes, and serum lactate concentrations.1 We agree with Dr Claret and colleagues that this study and others have demonstrated that most patients treated for acute exacerbation of asthma who subsequently develop elevated serum lactate concentrations have no discernable worsening of outcomes and require no alteration in management.2,3 Thus, the routine use of serum lactate measurements in these patients is not warranted.

However, we and others have described occasional subjects with worsening dyspnea and stable pulmonary function who were found to have hyperlactatemia and who clinically improved when beta-agonists were withheld.4,5 It is not unreasonable to consider testing of serum lactate concentrations (particularly rapid point-of-care testing) in patients with worsening subjective dyspnea, in the face of stable pulmonary function based on clinical or spirometric features, to determine if hyperlactatemia and acidosis may be the cause.

Finally, the lactate concentration Dr Claret and colleagues report of 10.47 mmol/L is higher than the maximum lactate concentration measured in any of our subjects. However Koul et al6 reported a lactate concentration of 13 mmol/L in a case report of a 17-year-old who recovered uneventfully.

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