base disturbances in critically ill patients with cirrhosis. *Liver Int* 2007;27(7):901-909.

4. Nguyen BV, Vincent JL, Hamm JB, Ab-alain JH, Carre JL, Nowak E, Ahmed MO, et al. The reproducibility of Stewart parameters for acid-base diagnosis using two central laboratory analyzers. *Anesth Analg* 2009;109(5):1517-1523.
5. Guérin C, Bayle F, Poggi B, Germain-Pas-tene M, Nesme P, Bourdin G, Richard JC. Quantitative analysis of acid-base disorders in patients with acute on chronic respiratory failure and acute respiratory distress syndrome: a prospective, one center study. *Open Clin Chem J* 2008;1:27-37. DOI: 10.2174/1874241600801010027.
6. Crapo RO, Jensen RL, Hegewald M, Tash-kin DP. Arterial blood gas reference values for sea level and an altitude of 1,400 meters. *Am J Respir Crit Care Med* 1999;160(5 Pt 1):1525-1531.
7. Fencel V, Jabor A, Kazda A, Figge J. Diagnosis of metabolic acid-base disturbances in critically ill patients. *Am J Respir Crit Care Med* 2000;162(6):2246-2251.
8. Adrogue HJ, Madias NE. Secondary responses to altered acid-base status: the rules of engagement. *J Am Soc Nephrol* 2010; 21(6):920-923.
9. Martinu T, Menzies D, Dial S. Re-evaluation of acid-base prediction rules in patients with chronic respiratory acidosis. *Can Respir J* 2003;10(6):311-315.

The author responds:

I was very interested in the comments from Drs Bruno, Berend, and Engels about our recent report.¹ Dr Bruno pointed out that in our report the fact that the significantly higher mean bicarbonate value in group 3 (unstable patients) than in group 1 (stable patients) suggests the presence of a concomitant metabolic alkalosis in many of those patients, whereas the normal bicarbonate in groups 2 and 4 does not exclude the presence of a metabolic disorder. We should say that higher bicarbonate value in group 3 than in group 1 might also reflect that chronic hypercapnia was greater and

ongoing for a longer period than in patients in group 3.

Furthermore, Dr Bruno proposed using a slightly modified traditional approach to detect mixed acid-base disorders. It is true that we did not use the approach suggested by Dr Bruno. The traditional approach we used in groups 1 and 3 (Table 1) was clearly unable to detect any single disturbance. We agree that this is too simplistic and a more complex approach would have been of value. Therefore, the comparison between the Stewart approach and the approach proposed by Dr Bruno should be done, since apparently it has not been done. It is likely, however, that the stepwise approach that relies on different measurements, namely biological and clinical, has first to be assessed in terms of diagnostic performance, with pre-test odds, positive and negative likelihood ratios, and post-test odds measurements. Dr Bruno also asked for additional data from our patients, which, unfortunately, were not collected in our case report form. Dr Bruno's conclusion that "I think their conclusion of better performance with the Stewart method is not justified. The traditional approach, with only minor adjustments, can provide the same practical information." is, however, not supported by data, as outlined above.

Drs Berend and Engels pointed out 5 issues in our report.¹ I do agree with their first comment, that there is no accepted standard to compare when assessing acid-base abnormalities. Second, to try to circumvent the lack of agreement between laboratories regarding the computation of the contributing variables in the Stewart approach, as suggested by Berend and Engels, we determined normal ranges locally.

I don't yet understand the third issue Berend and Engels raised. Setting the pH threshold below 7.36 to define acute respiratory failure is all but conservative. In a randomized controlled trial in COPD patients admitted for acute respiratory failure in the pneumology ward, Plant et al² enrolled COPD patients in acute respiratory

failure with pH range 7.25–7.35. The definition we used was composite and centered around dyspnea. Furthermore, we enrolled not only COPD patients but also any patient with chronic respiratory failure, defined with a priori criteria.

Fourth, Fencel et al³ separated respiratory and non-respiratory acid-base disorders. We enrolled a prospective cohort of patients who were thought by definition to have a respiratory disorder.

I also agree with Berend and Engels' fifth comment, on the hypothesis by Martinu et al,⁴ a study we quoted in our report.¹ There is obviously the need for further pathophysiological investigations to better understand the complex acid-base derangements in the setting of chronic respiratory failure.

Claude Guérin MD PhD

Service de Réanimation Médicale et
d'Assistance Respiratoire
Hôpital de la Croix Rousse
Lyon, France

The author has disclosed no conflicts of interest.

REFERENCES

1. Guérin C, Nesme P, Leray V, Wallet F, Bourdin G, Bayle F, Germain M, Richard JC. Quantitative analysis of acid-base disorders in patients with chronic respiratory failure in stable or unstable respiratory condition. *Respir Care* 2010;55(11):1453-1463.
2. Plant PK, Owen JL, Elliott MW. Early use of non-invasive ventilation for acute exacerbations of chronic obstructive pulmonary disease on general respiratory wards: a multicentre randomised controlled trial. *Lancet* 2000;355(9219):1931-1935.
3. Fencel V, Jabor A, Kazda A, Figge J. Diagnosis of metabolic acid-base disturbances in critically ill patients. *Am J Respir Crit Care Med* 2000;162(6):2246-2251.
4. Martinu T, Menzies D, Dial S. Re-evaluation of acid-base prediction rules in patients with chronic respiratory acidosis. *Can Respir J* 2003;10(6):311-315.