The recent study by Burri and colleagues examined the usefulness of arterial blood gases (ABG) in the diagnosis and prognosis of 530 dyspneic patients with ABG drawn upon presentation to the emergency department [1]. The study was a retrospective analysis of prospectively collected data performed at a single center. They concluded that ABG cannot be used to distinguish between pulmonary and other causes of dyspnea. On the other hand, arterial pH was highly predictive of ICU admission and outcome. Until large clinical studies show equivalence between peripheral venous and ABG, we will continue to advocate the use of ABG in the evaluation of acute dyspnea.

Prior to the introduction of automated blood gas analyzers, blood PO2 and PCO2 could be obtained only by laborious and often inaccurate laboratory methods, such as gas tonometry and Van Slyke manometric extraction of plasma total carbon dioxide. Improvements in electrode technology came with the development of the Astrup pH electrode [2], the Stow/Severinghaus-type PCO2 electrode [3,4] and the polarographic oxygen Clark electrode [5]. Although pulse oximetry has largely superseded the use of arterial PO2 (PaO2), the ease by which ABG can now be measured has made this test de rigueur in the workup of dyspneic patients.

We agree with Burri and colleagues in that PaO2 values serve mainly to triage and to guide treatment, rather than to differentiate among the causes of dyspnea. Except for anxiety hyperventilation, it is difficult to rely solely on ABG to identify a specific clinical syndrome. This is not new information. The poor predictive value of PaO2 in diagnosing patients with pulmonary embolism is well known. In the PIOPED I study of patients with angiographically proven pulmonary embolism and no prior cardiopulmonary disease, 26% had PaO2 > 80 mmHg [6]. This figure was 38% in the PIOPED II study (n = 42) [7,8]. The probability of diagnosing an acute pulmonary embolism based on changes in PaO2 did not achieve statistical significance. Further, no combination of PO2 and PCO2 values could reliably exclude pulmonary embolism.

Burri and colleagues also report that arterial pH was a significant predictor of short-term and long-term outcome. Multiple physiological buffers act to preserve the concentration of hydrogen ions in blood within a relatively small range. Decreases in arterial pH are likely to reflect severe impairments or even exhaustion of systemic compensatory mechanisms in patients with acute dyspnea. The predictive value of the arterial pH in dyspneic patients noted by Burri and colleagues supports this hypothesis.

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Burri and colleagues propose the use of venous blood pH in the initial evaluation of acute dyspnea, based on data from several single-center studies reporting relatively close limits of agreement (−0.11 to +0.04) between arterial pH and venous blood pH [9,10]. One must keep in mind, however, that muscular activity or regional microcirculatory alterations could result in misleadingly low regional pH values in septic or agitated patients.

Substituting venous blood pH for arterial pH in the evaluation of dyspneic patients is an appealing notion, but one that requires testing in multicenter, prospective clinical studies. Until these studies corroborate the equivalence between arterial and peripheral venous blood gases, or until non-invasive techniques to monitor arterial PCO2 and pH become available, we shall continue to support the use of ABG in the initial evaluation and treatment of patients with acute dyspnea.
Abbreviations
ABG, arterial blood gases; ICU, intensive care unit; PaCO₂, arterial partial pressure of carbon dioxide; PCO₂, partial pressure of carbon dioxide; PO₂, partial pressure of oxygen.

Competing interests
The authors declare that they have no competing interests.

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