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François Jardin
Antoine Vieillard-Baron

Weaning failure from cardiovascular origin

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In a recent editorial titled "Weaning failure from cardiovascular origin," C. Richard and J.L. Teboul focused on the paramount importance of early identification of patients in whom the transfer from mechanical ventilation to spontaneous breathing can result in acute cardiac dysfunction [1]. In these patients, as emphasized by F. Lemaire 15 years ago, a pre-weaning approach using diuretics and/or inotropic agents may facilitate successful weaning [2]. One can but agree with this concept.

However, the method advocated to identify these patients—invasive monitoring of pulmonary artery occluded pressure (PAOP)—is inaccurate. Acute left ventricular dysfunction actually increases left ventricular end-diastolic pressure (LVEDP). Evaluation of this increase by right heart catheterization was largely successful in the 1980s, because it was a last resort for intensivists. However, in a dyspneic patient exhibiting large swings in pleural pressure, an accurat measurement of PAOP is particularly difficult, and weakly reflects LVEDP [3].

Conversely, left ventricular dysfunction induced by acute weaning can be detected by bedside echocardiography. First, left ventricular dysfunction, which may appear as localized dyskinesia or as a global hypokinesia, is perfectly revealed by two-dimensional real-time imaging. Second, a careful analysis of acute changes in Doppler profile of mitral flow permits reliable evaluation of acute changes in left ventricular diastolic function [4]. Furthermore, availability of tissue imaging has now improved noninvasive measurement of LVEDP [5].

Over the last 20 years, increasing availability of sophisticated ultrasound techniques has led to advances in hemodynamic evaluation of the critically patient. With these advances, there is no longer any need for invasive monitoring by pulmonary artery catheterization.

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F. Jardin (✉) · A. Vieillard-Baron
Hôpital Ambroise Paré, Réanimation Médicale,
Boulogne, France
e-mail: francois.jardin@apr.aphp.fr
Fax: +33-1-49095892