The following is the abstract of the article discussed in the subsequent letter:

**Bendjelid, Karim, Peter M. Suter, and Jacques A. Romand.**

The respiratory change in pre-ejection period: a new method to predict fluid responsiveness. *J Appl Physiol* 96: 337–342, 2004; 10.1152/japplphysiol.00435.2003.—The accuracy and clinical utility of preload indexes as bedside indicators of fluid responsiveness in patients after cardiac surgery is controversial. This study evaluates whether respiratory changes (Δ) in the pre-ejection period (PEP; ΔPEP) predict fluid responsiveness in mechanically ventilated patients. Sixteen post-coronary artery bypass surgery patients, deeply sedated under mechanical ventilation, were enrolled. PEP was defined as the time interval between the beginning of the Q wave on the electrocardiogram and the upstroke of the radial arterial pressure. ΔPEP (%) was defined as the difference between expiratory and inspiratory PEP measured over one respiratory cycle. We also measured cardiac output, stroke volume index, right atrial pressure, pulmonary arterial occlusion pressure, respiratory change in pulse pressure, systolic pressure variation, and the Δdown component of SPV. Data were measured without positive end-expiratory pressure (PEEP) and after application of a PEEP of 10 cmH2O (PEEP10). When PEEP10 induced a decrease of >15% in mean arterial pressure value, then measurements were re-performed before and after volume expansion. Volume loading was done in eight patients. Right atrial pressure and pulmonary arterial occlusion pressure before volume expansion did not correlate with the change in stroke volume index after the fluid challenge. Systolic pressure variation, ΔPEP, Δdown, and change in pulse pressure before volume expansion correlated with stroke volume index change after fluid challenge (r2 = 0.52, 0.57, 0.68, and 0.83, respectively). In deeply sedated, mechanically ventilated patients after cardiac surgery, ΔPEP, a new method, can be used to predict fluid responsiveness and hemodynamic response to PEEP10.

**Prediction of fluid responsiveness: searching for the Holy Grail**

*To the Editor:* We have read with great interest the study by Bendjelid et al. (2) proposing a new tool to answer one of the most common clinical questions in patients with shock: Can we improve stroke volume, cardiac output, and hence hemodynamics by giving fluid? Indeed, Bendjelid et al. (2) demonstrated that the respiratory variation in pre-ejection period (the time interval between the Q wave on the ECG and the upstroke of the radial arterial pressure) is directly proportional to the stroke volume increase induced by a fluid challenge.

Fluid responsiveness has become an increasingly popular concept over the past few years (1, 8, 10). To date, 10 clinical studies (2–7, 12–15) have demonstrated the potential value of dynamic parameters in detecting patients who may benefit from volume loading. The arterial systolic pressure variation (SPV) and its expiratory component (Δdown) and the arterial pulse pressure variation (ΔPP), the Doppler aortic blood velocity variation, the pulse contour stroke volume variation, and the pre-ejection period variation (ΔPEP) have successively been shown to be useful tools to predict fluid responsiveness. In contrast, static indicators of cardiac preload (e.g., cardiac filling pressures or dimensions) have been shown to be of minimal value (1, 8). Therefore, the superiority of dynamic over static parameters is now well documented.

However, what is the best dynamic parameter remains to be determined. Indeed, only very few studies have compared the value of dynamic parameters (2, 6, 15). Interestingly, Bendjelid et al. (2) compared the value of SPV, Δdown, ΔPP, and ΔPEP by looking at the r2 correlation coefficients (respectively, 0.52, 0.68, 0.83, and 0.57) between these parameters and the percent increase in stroke volume in response to volume loading. Using the same method, we provide in Fig. 1 a comparison of all dynamic parameters that have been shown to be related to the hemodynamic effects of a fluid challenge. When all studies are taken into account, the mean r2 correlation coefficient is 0.63, suggesting that the dynamic parameters, although much more valuable than static parameters, could still be improved.

In this regard, some concerns have recently been raised about the influence of tidal volume and lung or chest wall compliance on dynamic parameters. There is no doubt that the magnitude of changes in pleural pressure (which depends on both tidal volume and respiratory mechanics) influences the value of dynamic parameters (11). However, whether the magnitude of changes in pleural pressure may also influence fluid responsiveness remains to be determined (9). Interestingly, in patients with static compliances of the respiratory system ranging from 26 to 69 ml/cmH2O and ventilated with tidal volumes ranging from 5 to 10 ml/kg, Bendjelid et al. (2) have reported a very tight relationship (r2 = 0.83) between ΔPP and the percent increase in stroke volume. This finding supports the notion that the predictive value of dynamic parameters is relatively independent of tidal volume and respiratory mechanics. However, further studies are definitely required to clarify this issue.

**REFERENCES**


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**REPLY**

To the Editor: We thank Drs. Michard and Schmidt for their interest in our article (2) and for their additional comments. We take the opportunity to respond to their letter, which puts forward the potential value of dynamic parameters in detecting patients who may benefit from volume loading, in regard to our recent study (2). The comments provided were appreciated and in some measure correct.

Indeed, they have reanalyzed data from 14 studies in an attempt to demonstrate that dynamic parameters have been shown to be related to hemodynamic effects of a fluid challenge (their Fig. 1). However, Michard and Schmidt have omitted to add the study of Wiesenack et al. (4), which suggests, in contrast to other recent studies (1, 3), that stroke volume variation derived from pulse contour analysis could not serve as an indicator of fluid responsiveness in cardiac surgical patients. This work reminds us that critical care pathophysiology is fascinating but not simple.

**REFERENCES**


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