

Karim Bendjelid
Jacques-A. Romand

**Reply to comment
on “Fluid responsiveness
in mechanically ventilated
patients: a review of indices
used in intensive care”**

Received: 26 March 2003
Accepted: 30 March 2003
Published online: 22 May 2003
© Springer-Verlag 2003

Sir: We read with interest the comments by Michard and Reuter regarding our review article entitled “Fluid responsiveness in mechanically ventilated patients: a review of indices used in intensive care” [1] and would like to make the following remarks about their comments.

First, we have never stated that cardiac preload and fluid responsiveness (cardiac responsiveness to volume loading) are synonyms. Moreover, we disagree with these authors that improvement in technological measurements of selected static cardiac preload indices should not increase the predictive value of fluid responsiveness. As they highlight in their letter, we propose three-dimensional echocardiography to meet this challenge. We chose this technique as it allows the measurement of both ventricular volumes and contractility and the estimation of pericardial restraint. Therefore for physiological reasons we can accurately predict fluid responsiveness simply by assessing baseline cardiac preload when the technique used allows the discrimination of normal from failing ventricle (systolic and/or diastolic failure). Moreover, it seems to us that in particular cases such as cardiac tamponade (mechanical diastolic dysfunction) echocardiography could be a better index of fluid responsive-

ness than dynamic parameters. Faehrich et al. [2] have recently demonstrated effectively in an animal study that the respiratory change in mitral flow under positive pressure ventilation becomes nonexistent when cardiac tamponade is present. In this particular case the hypotended patient could be considered a nonresponder to a fluid challenge by stroke volume variation indices, when giving him more fluids should be useful to increase cardiac output and/or mean arterial pressure.

Second, Starling’s law has shown that preinfusion cardiac preload, ventricular contractility, and afterload all influence the stroke volume response to volume load. Nevertheless, in the particular case of a patient with a left ventricular systolic dysfunction, clinicians should keep in mind that positive pressure ventilation acts as a circulatory pump [3] that relatively improves contractility and decreases afterload.

Third, although we agree that the question “does our fluid challenge effectively increase patient cardiac preload?” is essential to explain and validate the physiological background of a cardiac preload index [4], the answer to this question has no consequence for clinical practice if the index has been validated to be an accurate parameter for predicting the response of cardiac output and/or mean arterial pressure after volume infusion in all pathological situations. Practically, clinicians want to determine not the variation in the end-diastolic volume but rather whether the mean arterial pressure will increase (related to increase in cardiac output) after volume infusion.

Finally, cardiac responsiveness to volume loading depends on different Starling curves (each reflecting a different inotropic state of the ventricles) and on the ventricular interdependence [5]. Therefore the optimal index to predict the fluid responsiveness, in all pathological situations, must assess these two estimations. As stated in our review [1], there are presently only few works clearly relevant to the specific fluid responsiveness, and the most useful parameters for predicting cardiac output response to fluid challenge in mechanically ventilated patients are the dynamics parameters. However, because the limits of these indices have not yet been demonstrated in par-

ticular cases, and the echocardiography should be revitalized by three-dimensional technique, it seem to us that the challenge continues.

References

1. Bendjelid K, Romand JA (2003) Fluid responsiveness in mechanically ventilated patients: a review of indices used in intensive care. *Intensive Care Med* 29:352–360
2. Faehrich JA, Noone RB Jr, White WD, Leone BJ, Hilton AK, Sreeram GM, Mark JB (2003) Effects of positive-pressure ventilation, pericardial effusion, and cardiac tamponade on respiratory variation in transmitral flow velocities. *J Cardiothorac Vasc Anesth* 17:45–50
3. Beattie C, Guerci AD, Hall T, Borkon AM, Baumgartner W, Stuart RS, Peters J, Halperin H, Robotham JL (1991) Mechanisms of blood flow during pneumatic vest cardiopulmonary resuscitation. *J Appl Physiol* 70:454–465
4. Tavernier B, Makhotine O, Lebuffe G, Dupont J, Scherpereel P (1998) Systolic pressure variation as a guide to fluid therapy in patients with sepsis-induced hypotension. *Anesthesiology* 89:1313–1321
5. Jardin F (2003) Ventricular interdependence: how does it impact on hemodynamic evaluation in clinical practice? *Intensive Care Med* 29:361–363

This reply refers to the comment available at <http://dx.doi.org/10.1007/s00134-003-1846-4>

K. Bendjelid (✉) · J.-A. Romand
Surgical Intensive Care Division,
Geneva University Hospital,
1211 Geneva 14, Switzerland
e-mail: Karim.Bendjelid@hcuge.ch
Tel.: +41-22-3827452
Fax: +41-223827455