Commentary

Can passive leg raising be used to guide fluid administration?

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See related research by Lafanechère *et al*., http://ccforum.com/content/10/4/R132

Abstract

Predicting fluid responsiveness has become a topic of major interest. Measurements of intravascular pressures and volumes often fail to predict the response to fluids, even though very low values are usually associated with a positive response to fluids. Dynamic indices reflecting respiratory-induced variations in stroke volume have been developed; however, these cannot be used in patients with arrhythmia or with spontaneous respiratory movements. The passive leg raising (PLR) test has been suggested to predict fluid responsiveness. PLR induces an abrupt increase in preload due to autotransfusion of blood contained in capacitance veins of the legs, which leads to an increase in cardiac output in preload-dependent patients. This commentary discusses some of the technical issues related to this test.

In many instances, hemodynamic optimization requires the use of fluids. However, the response to fluids may be quite variable and cannot be adequately predicted from the measurements of intravascular pressures (central venous pressure or pulmonary artery pressure) [1] or volumes. Indeed, the relationship between stroke volume and preload varies considerably between the patients. Accordingly, extreme values only can predict fluid responsiveness. Dynamic indices reflecting respiratory-induced variations in stroke volume have been developed [2], but these cannot be used in patients with cardiac arrhythmias or in patients with spontaneous respiratory movements [3] or ventilated with a low tidal volume [4]. Recently, the so-called passive leg raising (PLR) test has been proposed. This test is based on the principle that PLR induces an abrupt increase in preload due to autotransfusion of blood contained in capacitance veins of the legs. This abrupt increase in preload leads to an increase in cardiac output in preload-dependent patients but not in other patients. However, the test requires the determination of cardiac output with a fast-response device, because the hemodynamic changes may be transient. In a previous issue of *Critical Care*, Lafanechère and colleagues [1] used esophageal Doppler to monitor cardiac output and reported that a PLR-induced increase in cardiac output higher than 8% can predict fluid responsiveness in critically ill patients. The predictive value of the PLR test was similar to that of respiratory-induced variations in pulse pressure. Although this study basically confirms the results of Monnet and colleagues [5], it brings some new pieces of information to the field, but also raises important questions.

Indeed, the 22 patients investigated by Lafanechère and colleagues [1] were all in acute circulatory failure and treated with high doses of epinephrine or norepinephrine. However, the use of vasopressor agents may be of paramount importance in determining the response to dynamic tests. In an experimental study, Nouira and colleagues [6] reported that norepinephrine decreased respiratory-induced variations in pulse pressure in dogs subjected to severe hemorrhage. In their study, Lafanechère and colleagues [1] observed that variations in pulse pressure predicted fluid responsiveness in these patients treated with vasopressor agents, and the cutoff level was similar to that found in other series [2,7]. Vasopressor agents may also affect the response to PLR. Under physiologic conditions, the blood volume contained in capacitance veins in the legs and recruited during PLR is estimated to be close to 300 ml [8]. Although norepinephrine and epinephrine may decrease the amount of recruited blood, because vasopressor agents also induce venous vasoconstriction, the impact of these agents on PLR was negligible in this study [1] because PLR predicted fluid responsiveness in patients treated with high doses of vasopressor agents. In addition, the changes in cardiac output induced by PLR were correlated with changes in cardiac output obtained after the administration of 500 ml of saline, with a slope of the regression line close to 1. These results suggest that dynamic tests are useful in patients treated with high doses of vasoactive agents.

PLR = passive leg raising.

However, the exact cutoff value for changes in cardiac output measured with esophageal Doppler that should be used to separate responders from non-responders remains to be determined. Indeed, the characterization of responders and non-responders is a key issue. A 15% increase in cardiac output is usually considered to be significant and is used to characterize responders. This value takes into account error in measurements. With thermodilution, this error is considered to be close to 7% (it depends on the number of boluses averaged; this value is accepted for three boluses, it may be lower when at least five boluses are averaged), hence a 15% (7% + 7%, rounded to 15%) difference between two measurements is required to ensure that the difference is real and cannot be ascribed to random errors in measurements. With esophageal Doppler determination of cardiac output, this value may differ. The intraobserver variability needs to be defined, because without this information it is difficult to distinguish responders from non-responders. In their study, Lafanechère and colleagues [1] arbitrarily used a 15% cutoff. Because the respiratory variation in pulse pressure separating responders and non-responders was similar to values reported in the literature [2,5,9], it is likely that this 15% cutoff value was adequate. However, it is quite evident that the cutoff for PLR-induced changes in cardiac output cannot be lower than 15%, because this represents the cumulative errors in measurements. Accordingly, the 8% cutoff value for PLR-induced changes in cardiac output proposed by Lafanechère and colleagues [1] is probably too small. With esophageal Doppler, cutoff values for fluid responsiveness prediction ranging between 10% and 18% have been reported for PLR-induced changes in cardiac output PLR [5] and for respiratory variations in aortic blood flow [7]. Further studies should be performed to define the exact cutoff value that should be used; these studies should include an evaluation of the magnitude of random errors in cardiac output measurements with esophageal Doppler.

Conclusion

This study confirms that PLR and respiratory-induced variations in pulse pressure can be useful to predict fluid responsiveness in patients treated with high doses of vasoactive agents. However, further studies should be performed to determine more precisely the cutoff value for PLR-induced changes in cardiac output that should be used to discriminate between responders and non-responders with esophageal Doppler.

Competing interests

The author declares that they have no competing interests.

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