Commentary

Predicting volume responsiveness in spontaneously breathing patients: still a challenging problem

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See related commentary by Heenen *et al*., http://ccforum.com/content/10/4/R102

Abstract

The prediction of which patients respond to fluid infusion and which patients do not is an important issue in the intensive care setting. Assessment of this response by monitoring changes in some hemodynamic characteristics in relation to spontaneous breathing efforts would be very helpful for the management of the critically ill. This unfortunately remains a difficult clinical problem, as discussed in the previous issue of the journal. Technical factors and physiological factors limit the usefulness of current techniques.

It is becoming increasingly apparent that excess fluid is detrimental to patient outcome [1], and that there is a limit to volume responsiveness of the heart [2]. It would therefore be potentially useful to be able to predict when volume infusions will be futile for the management of critically ill patients.

Perel and colleagues [3], followed by other workers [4], introduced the use of respiratory variations in systolic pressure for the prediction of fluid responsiveness; methods of detecting these variations have even been automated with monitoring devices. Based on the presumed physiological mechanism [5], however, the prediction was that that these techniques probably would not work in subjects with spontaneous efforts; and this was indeed supported in an earlier study by Rooke and colleagues [6]. In the previous issue of *Critical Care*, Heenen and colleagues [7] confirmed that pulse pressure variations are a poor predictor of cardiac output responsiveness in spontaneously breathing subjects. Similar to other studies [8,9], they also found that approximately one-half of the subjects did not respond to fluids.

Importantly, Heenen and colleagues also found that a test we previously described based on respiratory variations in right atrial pressure (P_{ra}) also failed to predict fluid responsiveness [7]. The authors presented a number of possible reasons for the difference from our previous studies [8,9].

First, their study included patients who did not have adequate inspiratory efforts, whereas we only included patients with adequate efforts. I can understand how one would include these patients when examining overall usefulness of a test for the whole population, but this test is based on the response of P_{ra} when pleural pressure falls, and if it does not fall then the test cannot be used. In their discussion Heenen and colleagues argued that a difference in the findings may also be because we had disconnected patients from the ventilator. We only did this on a few occasions, however, to ensure that we did not miss an inspiratory effort – and thus this should also have had a minimal effect on our results.

The most significant difference was that almost 30% of our subjects had no inspiratory fall in P_{ra}, whereas only three patients or 14% had no inspiratory fall in P_{ra} in Heenen and colleagues' study. This suggests differences in the assessment of respiratory variation in P_{ra}. Heenen and colleagues indicate that they took the difference between end-inspiration and end-expiration, but we are not told what happened when there was a forced expiration. This can raise P_{ra} at end-expiration and make it appear that there is an inspiratory fall when in fact there is only a fall in the expiratory P_{ra} back to baseline. We excluded such measurements from our studies because that change should not predict fluid responsiveness. My actual practice at the bedside is to try to identify a cycle with no forced expiration at any phase during the respiratory cycle; if I cannot identify such a cycle, then I do not use the test. Another potential problem is the interpretation of an increased 'x' or 'y' descent as an inspiratory fall in P_{ra}, rather than using the base of the 'a' wave.

P_{ra} = right atrial pressure.

I am not surprised that respiratory variations in P_{ra} were not sensitive for predicting fluid responsiveness. We previously argued [8] that patients who are close to the plateau of the cardiac function curve would have a significant fall in P_{ra} but a minimal response to volume. This leads to perhaps a major experimental issue in this field. I argue that the real question should not be the potential to predict an increase in cardiac output, but rather the potential for there *not to be* a response to volume. The clinician therefore needs to know when not to give fluid rather than when to give it. Based on this reasoning, the low specificity for our test observed by Heenen and colleagues is of greater concern. Their estimate was based on only three patients, however; hardly a large enough sample size to give much confidence to this estimate.

Another point of note is that the use of colloids also confounds the assessment of volume responsiveness, because there is a suggestion that colloids sometimes give a hemodynamic benefit that is not related to a Starling mechanism [10] and would thus not be predicted by dynamic measures. What is often seen is a rise in cardiac output without a rise in P_{ra}, a change in heart rate or a change in afterload, which means that there had to be a change in cardiac contractility [10]. Possible mechanisms could be a reduction of edema in the heart or electrolyte shifts that effect cardiac function.

Finally, one last technical issue. Pressures are measured relative to an arbitrary reference level. Although not stated, Heenen and colleagues probably used the mid-axillary line, which gives values ~3 mmHg higher than our values referenced to 5 cm below the sternal angle.

In summary, Heenen and colleagues have shown that respiratory variations in pulse pressure do not predict the volume responsiveness of cardiac output in spontaneously breathing subjects. Their data also raise potential concerns regarding the predictive value of respiratory variations in P_{ra}. Before this approach is rejected, however, it is important to be certain that the technical details are correct, and that the population size is adequate.

Competing interests

The authors declare that they have no competing interests

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