“Let’s Give Some Fluid and See What Happens” versus the “Mini-fluid Challenge”

EVALUATING the response to fluids in critically ill patients remains a clinical challenge. Regardless of its cause, a sustained alteration in organ perfusion can lead to multiple organ failure. Fluid administration is thus not just a matter of correcting hypovolemia but, by increasing ventricular preload, is one method by which cardiac output can be increased.1 The risk associated with increasing the blood volume (in the absence of concurrent losses of identical magnitude) is an increase in hydrostatic pressures in the vascular system, resulting in edema formation with resultant impaired organ function. Hence, fluid administration must be considered in terms of its benefit (increase in cardiac output): risk (increase in hydrostatic pressures) ratio. Unless there are simultaneous fluid losses, giving fluids to a patient without a resulting increase in cardiac output can only be harmful. In this issue of Anesthesiology, Muller et al.2 propose using a “mini-fluid challenge” to avoid giving too much fluid to patients who may not benefit.

There is a consensus that static evaluation of a patient’s fluid or hemodynamic status, even by the most astute clinician, provides little information about the likely fluid responsiveness of that patient. Variables such as blood pressure, cardiac filling pressures, heart rate, cardiac output, mixed venous oxygen saturation, capillary refill time, urine output, or blood lactate concentration alone or in combination are neither sensitive nor specific for fluid responsiveness. Hence, the “reference” method, when the likely response of a patient to fluid is uncertain, is to dynamically assess a patient’s response to a fluid infusion. This so-called “fluid challenge” should be performed according to some strict rules.3 Optimally, there are four predefined elements to a fluid challenge, which can be encapsulated in the TROL mnemonic: Type of fluid (T), rate of fluid administration (R), objective (O), and limits (L). The fluid challenge is, of course, immediately discontinued if the limits (an excessive increase in cardiac filling pressure) are reached before the objective (usually an increase in arterial pressure or a decrease in heart rate beyond a predefined level). Under optimal clinical circumstances, a fluid challenge should be positive in approximately half the patients in whom it is applied.

Some experts have argued that even when the rules are respected, a fluid challenge may result in excessive fluid administration in patients who do not respond, especially when such challenges are repeated several times a day. There have, therefore, been many attempts to predict fluid responsiveness using other techniques, including evaluation of pulse pressure variation,4 stroke volume variation,5 or changes in aortic blood flow6 during the respiratory cycle in mechanically ventilated patients, or evaluation of changes in stroke volume during passive leg raising.7 However, each of these approaches has limitations, and none is very reliable in patients who are not sedated or anesthetized. Clinical confounders, such as intraabdominal hypertension, also decrease the accuracy of this evaluation.8 Thus, the fluid challenge remains widely used.

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“The general concept is … that the response to fluid challenge can be evaluated rapidly after a very limited amount of fluid . . .”
The mini-fluid challenge technique proposed by Muller et al. involved an infusion of only 100 ml of colloid over 1 min and was found to predict the fluid responsiveness of a full fluid challenge with an additional 400 ml given over the next 14 min. The general concept is not entirely new but has a sound scientific basis, and reemphasizes that the response to fluid challenge can be evaluated rapidly after a very limited amount of fluid is given. The most original aspect of this study was the use of transthoracic echocardiography to evaluate the changes in subaortic velocity time index during the test. In 39 patients with acute circulatory failure, the authors found that variations in velocity time index had excellent sensitivity and specificity for predicting fluid responsiveness. Incidentally, while on this subject, it is interesting to consider whether we should put more emphasis on good sensitivity (so as to identify all responders) or good specificity (so as to avoid failure). This is an important question that is rarely addressed. The authors propose using a cutoff velocity time index value of 10%, which had a sensitivity of 95% and a specificity of 78%. From their figure 2, one can deduce that a cutoff value of approximately 20% would have excluded all nonresponders, whereas a value of approximately 0% (i.e., no change) would have included all responders. Perhaps different cutoff values should be considered depending on the clinical context. For example, in the presence of severe respiratory failure, we would prefer a high specificity to limit excess fluid administration but a lower sensitivity may be acceptable in such patients.

Several important questions need to be asked when evaluating this study. First, is the transthoracic echocardiography approach that the authors suggest too complex? Well, perhaps still for some of us, but echocardiography should now be considered a standard tool for every anesthesiologist involved in intensive care and we should all be receiving proper training in these techniques. Second, is the method used applicable to all acutely ill patients? No: The patients were deeply sedated, as in most studies evaluating fluid responsiveness (to obtain good data!), so these results may not be achievable in non-sedated patients. Moreover, arrhythmias present a key obstacle to this approach; in this study, one fourth of patients were excluded for this reason. In addition, it is not always possible to obtain reliable images with transthoracic echocardiography, especially in mechanically ventilated patients. Third, is echocardiography the only technique available for this purpose? No, thermodilution, transesophageal Doppler, or other techniques could also be considered. The main advantages of the technique used here are the lack of invasiveness and the rapid changes in velocity time index during fluid administration. Is it even necessary to measure the cardiac response directly? I’m not entirely sure: If the patient has tachycardia because of a fluid deficit, the heart rate will decrease immediately when fluid is administered; if cardiac output increases in the hypotensive patient after fluid administration, so will arterial pressure. In these situations, monitoring the cardiac response is perhaps superfluous. Admittedly, in the patient with oliguria, some form of cardiac output monitoring may provide useful information before there is any obvious increase in urine output.

So, let’s go back to the basics. All too often, if it is believed that a patient may benefit from fluid infusion, a vague order is given, “let’s give some fluid and see what happens.” But what does the “see what happens” really mean? Without clear endpoints and defined targets, this approach often results in the infusion of large amounts of fluid (fluid loading) without any real understanding of whether this fluid is actually needed or an indication of whether it is beneficial (for example, in situations such as this, we may hear, “the blood pressure seemed to increase but we had also stopped the sedatives” or “the patient improved, but the nurse also suctioned the trachea”). “Fluid challenge” and “fluid loading” are, after all, different terms based on different concepts. Perhaps the fundamental take-home message underlying this interesting study by Muller et al. is that there is a need to develop a simple and effective technique (such as changes in velocity time index) by which the response to a fluid challenge can be rapidly and objectively assessed so that fluid loading is reserved only for those who will benefit.

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References

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