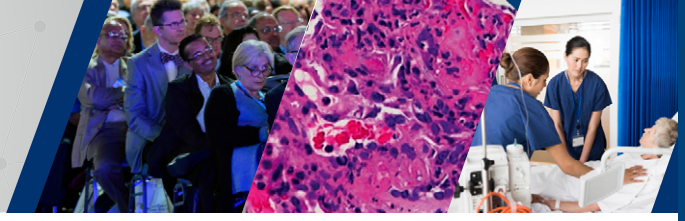

Intravascular Volume Assessment in the Critically Ill Patient

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Introduction

An 80-year-old patient is admitted with generalized peritonitis and a systolic BP of 75 mm Hg. I give 1 L of 0.9% NaCl rapidly and add NE to correct hypotension (salvage phase). I do an echocardiogram and insert a central venous catheter to be able to give vasopressors safely and measure central venous pressure (CVP), changes in which help to interpret tolerance to fluid (optimization). After a couple of hours, the patient's hemodynamic status stabilizes on a small dose of NE (stabilization), but edema is clearly present. I reduce fluids, discontinue the NE infusion, and consider a diuretic (de-escalation).

Hypervolemia and hypovolemia can both have harmful effects on organ function: hypervolemia by inducing edema and hypovolemia by limiting oxygen delivery to the organs (1). As an encapsulated organ, the kidney is particularly sensitive to the effects of edema, but decreased perfusion associated with hypovolemia may increase the risk of kidney failure (1). Intravascular volume assessment and appropriate fluid management are, therefore, crucial to avoid these two extremes of volume status. In terms of fluid administration, the time factor, as developed in the salvage, optimization, stabilization, and de-escalation paradigm, must be taken into account (2). In the initial phases, a patient may require large amounts of fluids to help achieve a minimum perfusion pressure but may as a result become hypervolemic, and the patient may require interventions to achieve a negative fluid balance during subsequent phases. Underestimating the importance of these phases by, for example, using diuretic therapy in hypovolemia or continuing fluid administration when there is no need can result in substantial harm.

How to Assess Hypervolemia

Hypervolemia has significant clinical implications, and therefore, identification is important. If a patient is hypervolemic, he/she usually requires minimal fluid administration and may need diuretic therapy and ultrafiltration if kidney function is altered; these strategies can be harmful in the absence of hypervolemia. Inotropic support may also be added if there is altered cardiac function. Hypervolemia means that blood volume is increased and, therefore, there is an inherent risk of edema; it is typically associated with elevated cardiac filling pressures, although the opposite is not always true. Edema can be due to elevated hydrostatic pressures of any cause (including heart failure), alterations in capillary permeability, or hypoalbuminemia alone or in combination. Diagnosing hypervolemia only on the basis of the presence of edema is unacceptable, because edema may coexist with normovolemia and even hypovolemia. Accordingly, I try to avoid the term "fluid overload" and simply refer to the presence of edema (3). Basing a diagnosis of hypervolemia on the presence of a positive fluid balance may also be misleading, because a patient with a positive fluid balance may still require more fluid.

How to Assess Hypovolemia

Identifying hypovolemia is also important, but I think that the more relevant question is whether this patient could benefit from fluid administration. Giving more intravenous fluid should only be attempted if there are signs of tissue hypoperfusion, such as arterial hypotension, oliguria, altered skin perfusion, or altered mentation.

When assessing whether a patient may be fluid responsive, I use a dichotomous approach depending on the presence or absence of spontaneous respiratory movements (Figure 1). A deeply sedated or anesthetized patient requires mechanical ventilation and will not spontaneously trigger the ventilator. In these very specific conditions, typically encountered during surgery under general anesthesia, hypovolemia is shown by fluctuations in venous return and, therefore, cardiac output, when intrathoracic pressures increase during gas insufflation in the lungs. The transient changes in stroke volume are translated into changes in arterial waveforms and quantified by calculation of the pulse pressure variation. This concept is quite easily recognized when an arterial catheter is in place but can also be observed with new noninvasive, plethysmography-based monitoring systems, and it may even be used within a closed loop system (4). Following the same principles, some cardiac output monitors also display the stroke volume variation. Use of fluctuations

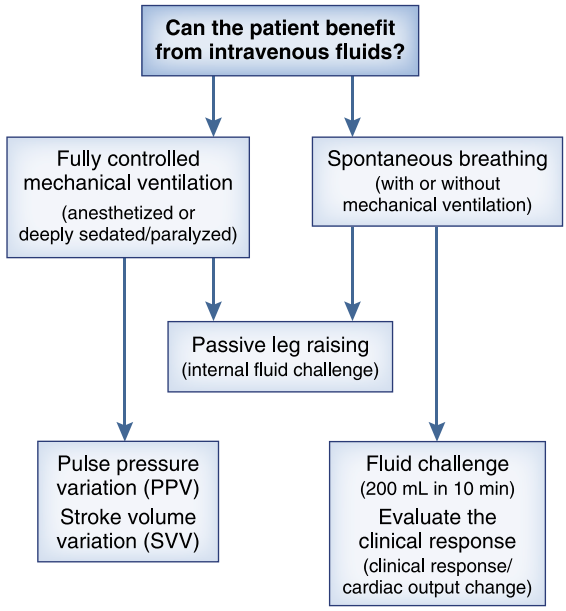

Figure 1. | **How I assess fluid requirements in the acutely ill patient.**

in the size of the vena cava during mechanical ventilation to predict fluid responsiveness has resulted in variable results (5), and I do not trust this approach. Even estimation of respiratory changes in superior vena cava diameter using transesophageal echocardiography is not very precise (6).

Today, profound sedation is rarely used; therefore, even mechanically ventilated patients have some spontaneous breathing, and the associated changes in intrathoracic pressure mean that respiratory variations are no longer reliable for assessment of fluid responsiveness. Evaluation of static variables, such as arterial pressure, heart rate, urine output, and even CVP, does not predict the response to fluids well, and a dynamic test is usually preferred. The fluid challenge technique combines evaluation of benefit (increase in cardiac output) and risk (increase in venous pressures). I use the classic test with rapid administration of a fluid bolus of 200 ml crystalloid or 100 ml colloid over (5 to) 10 minutes (7). If blood flow increases (estimated by cardiac output measurements or even clinical evaluation of organ perfusion) and the increase in CVP is minimal, fluid administration can be continued, and the test can be repeated. If, in contrast, the increase in blood flow is negligible and there is a marked increase in CVP, additional fluid administration is likely to be harmful, and fluids should be stopped. This strategy is recommended even in children and neonates with sepsis (8). Some physicians might use larger amounts of fluid over 20–30 minutes, but I do not support this approach, because patient status can change considerably during such a long period. Others may use "mini-fluid challenges" over 1 minute to be completely sure that conditions are unlikely to change; this is particularly applicable in the operating room. In all cases, care needs to be taken to avoid patient stimulation and changes in therapy during the procedure, which may make the results uninterpretable. Because the goal is to increase cardiac output by the Frank–Starling relationship, ideally cardiac output should be measured. When vascular tone is preserved, such as in hypovolemic shock, a positive response to fluid will result in an increase in arterial pressure, but this may not be the case when vascular tone is decreased, such as in sepsis. In patients with oliguria, urine output cannot be used to assess the effects of fluids, because the response is too slow, but a positive urine output response to a fluid challenge supports the need to give more fluid.

Passive leg raising is a clever procedure, which effectively amounts to an "internal fluid challenge" with a transient fluid shift from the lower part of the body into the thoracic compartment induced by lifting the legs (9). However, although passive leg raising sounds easy, it is not. First, changes are short lived and relatively subtle: changes in BP are not reliable, and changes in stroke volume need to be detected. Second and more importantly, the rapid change in inclination of the bed is stressful for the patient, and the associated increase in arterial pressure and heart rate may be inaccurately interpreted as a positive response to fluids. Evaluation of volume assessment is essential to ensure that patients are neither hypo- nor hypervolemic, because these extremes of volume status both have harmful effects on organ function. However, time should not be wasted on assessing volume status or fluid responsiveness if it is clear that fluids are needed (*e.g.*, in a patient with acute bleeding) (1). All measures of fluid responsiveness have limitations, and I prefer to base ongoing fluid decisions on the results of repeated fluid challenges. However the fluid volume is assessed, ongoing fluid management must be directed according to the salvage, optimization, stabilization, and de-escalation phase as shown in our patient history: in the initial phase of resuscitation, when tissue perfusion is altered, there may be a place for fluids, and there is definitely no place for fluid restriction or diuretics. When the patient is "stabilized" (10), attempts should then begin to limit edema.

Disclosures

Dr. Vincent has nothing to disclose.

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