A Responsividade a Fluidos Não é o Mesmo que Benefício de Fluidos

Fluid Responsiveness is Not the Same as Fluid Benefit

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Resumo:

A responsividade a fluidos tem sido um tema quente há algum tempo. Embora tenha uma definição conceptual fácil (a resposta à expansão de volume com aumento do débito cardíaco), a sua avaliação na prática tem sido assunto de investigação, debate e alguma controvérsia nos últimos 15 ou 20 anos. O problema é que a responsividade a fluidos não é sinónimo de benefício da administração de fluidos. E temos andado a gastar tempo a investigar formas de prever a responsividade a fluidos. E eu realmente não quero saber se o doente é responsivo a fluidos ou não (não o somos todos?), mas antes se aquele doente específico beneficia ou não da administração de fluidos naquele momento específico. Nós avaliamos se os doentes em choque são ou não responsivos a fluidos. Se, seja qual for o método utilizado, verificamos que o são, administramos fluidos. E só paramos essa administração de fluidos se uma de duas coisas acontece: se o doente já não está em choque, ou se o doente deixa de ser responsivo a fluidos. Nunca usaríamos um fármaco com efeitos deletérios comprovados, em especial se o seu benefício não estivesse comprovado. No entanto continuamos a usar fluidos em cenários em que o seu prejuízo está bem demonstrado, mas o seu benefício não. Precisamos de uma mudança de paradigma. Temos que deixar de procurar formas de prever a responsividade a fluidos. Precisamos de encontrar formas de identificar que doentes beneficiam da expansão de volume, depleção de volume ou de uma estratégia de balanço neutro. Os novos ensaios deverão, de forma prospectiva, comparar estratégias bem definidas de gestão de fluidos (expansão, depleção ou neutra) a serem aplicadas de acordo com critérios pré-determinados. Até lá, continuaremos com o mesmo problema: será que este doente, neste momento, beneficia de uma estratégia de expansão, depleção ou de balanço neutro?

Palavras-chave: Equilíbrio Hidroeletrolítico; Hidratação

Abstract:

Fluid responsiveness has been a hot topic for some time. Although with an easy conceptual definition (responding to volume expansion by increasing cardiac output), its practical assessment has been the subject of research, debate and some controversy, for the past 15 to 20 years. The problem is that fluid responsiveness is not the same as fluid benefit. And we have been wasting time researching in ways to predict fluid responsiveness. I really do not want to know if the patient is fluid responsive or not (are not we all?), but rather if fluid expansion is beneficial or detrimental to that specific patient, on that specific moment. We test patients in shock for fluid responsiveness. If, whatever the method we use, we find them to be responsive, we do intravenous fluids. We only stop fluid loading/fluid expansion if one of two things happen: if the patient is no longer in shock, or if the patient is no longer fluid responsive. We would never use a drug with proven harm, especially if its benefit was insufficiently proven. Nevertheless we continue to use fluids in scenarios in which their harm is proven, but their benefit is not. We need a paradigm shift. We need to stop looking for ways to predict fluid responsiveness. We must search for ways to identify which patients benefit from fluid expansion, fluid depletion or a neutral fluid strategy. New trials should prospectively compare well defined fluid strategies (expansion, depletion or neutral) to be applied depending on a set of predetermined tests. Until then, we will end up with the same question: will this specific patient, at this specific moment, benefit from fluid expansion, fluid depletion or a neutral fluid strategy?

Keywords: *Fluid Therapy; Water-Electrolyte Balance*

Fluid responsiveness has been a hot topic for some time. Although with an easy conceptual definition (responding to volume expansion by increasing cardiac output),[1] its practical assessment has been the subject of research, debate and some controversy, for the past 15 to 20 years.[2] I have been working in intensive care for most of those years. Yet, 20 years later, using everything from the oldie central venous pressure to the many "dynamic" methods for assessing fluid responsiveness, I often find myself exactly with the same question: will this patient benefit from fluid expansion, fluid depletion or a neutral fluid strategy?

What have we been missing?

The problem is that fluid responsiveness is not the same as fluid benefit. And we have been wasting time researching in

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ways to predict fluid responsiveness. And I do not know if that has any relevance at all. I really do not want to know if the patient is fluid responsive or not (are not we all?), but rather if fluid expansion is beneficial or detrimental to that specific patient, on that specific moment. And being fluid responsive gives me no answer to that question.

What do we usually do? We test patients in shock for fluid responsiveness. If, whatever the method we use, we find them to be responsive, we do intravenous fluids (fluid loading, fluid expansion, fluid infusion, whatever you want to call it). What fluids, what rate, for how long, that depends on who we are and where we work.[3-6]

And then we analyze the consequences of what we did. Blood pressure, vasopressor need, lactate kinetics, urine output. Then we assess fluid responsiveness, again. And do it all over again. We only stop fluid loading/fluid expansion if one of two things happen: if the patient is no longer in shock, or if the patient is no longer fluid responsive. We do it because it is so hard to stop doing fluids when the patient is still in shock and still fluid responsive. Even when we all know that this strategy often leads to fluid overload and all its harmful consequences (e.g. longer mechanical ventilation and increased mortality).[7-9]

More recently, some authors are ready to abandon fluid responsiveness and embrace "signs of tissue hypoperfusion" as the trigger that would make us decide to do fluids.[10] Nonetheless we find ourselves in the same predicament. Like with fluid responsiveness before it, are "signs of tissue hypoperfusion" a marker of fluid benefit?

We would never use a drug with proven harm, especially if its benefit was insufficiently proven. Nevertheless we continue to use fluids in scenarios in which their harm is proven, but their benefit is not (the Surviving Sepsis Campaign guidelines mandate the administration of IV fluids at a dose of 30 mL/kg -2400 mL for an 80 kg patient- given within the first 3 hours, as a possible "life--saving procedure", although there is no randomized controlled trial to support this statement).[10,11] And our fluid management strategies are driven mainly by expert opinion[9] (but are not we all experts?), systematic reviews,[12] meta-analysis,[13] retrospective and observational[14] studies. If this was a drug…

We need a paradigm shift. We need to stop looking for ways to predict fluid responsiveness or "signs of tissue hypoperfusion".

We must search for ways to identify which patients benefit from fluid expansion, fluid depletion or a neutral fluid strategy.

New trials should prospectively compare well defined fluid strategies (expansion, depletion or neutral) to be applied depending on a set of predetermined tests (namely "hypovolemia tests", which can be many of the so called fluid responsiveness tests, and "hypervolemia tests", e.g. an E/E'>8 on echocardiography, B lines on lung ultrasound, extravascular lung water with transpulmonary thermodilution, or pulmonary wedge pressure with a pulmonary catheter).

Outcomes should not be surrogate end-points (fluid overload, fluid responsiveness, blood pressure, vasopressor need, lactate kinetics, urine output), but rather hard outcomes (like mortality, mechanical ventilation days, renal failure).

Until then, we will continue with the same discussions, expert opinions and algorithms. And end up with the same question: will this specific patient, at this specific moment, benefit from fluid expansion, fluid depletion or a neutral fluid strategy? ■

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