LETTERS TO THE EDITOR

Ultrafiltration for Heart Failure: Assessment of Intravascular Volume Status through ECG Modifications

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To the Editor

We read with great interest the article of Madias and Guglin recently published in your journal.¹ This study evaluated the effect of ultrafiltration on the amplitude of QRS complexes in patients with heart failure (HF), and recommended its use as an index of effectiveness of therapy. Since extracorporeal ultrafiltration was originally used for kidney diseases, nephrologists are frequently consulted to assist in management of refractory HF using conventional hemodialysis machines.² Although excited about the interesting results of this study, we think a few points merit consideration for further evaluation of the authors' conclusions.

First, while the authors have extensively discussed the potential correlation between intravascular volume status and the amplitude of QRS complexes, the concept of "plasma refill rate" (PRR) has not been considered. During extracorporeal ultrafiltration, removal of fluid from intravascular sector leads to a shift of fluid from the extravascular sector into the intravascular compartment in order to replace the loss and maintain hemodynamic stability. A mismatch between plasma refill and ultrafiltration rates thus is a major cause of intravascular volume depletion and hypotension.³ The rate of this shift (i.e., PRR) depends on a number of factors including serum albumin level. It is known that many patients with HF, similar to other chronic disease states and wasting syndromes, suffer from malnutrition-inflammation complex syndrome.⁴ Hence, it is conceivable that these patients would manifest an impaired PRR due to low serum albumin, which will result in

a very slow shift of fluid into intravascular sector during ultrafiltration. In other words, the increase in the amplitude of QRS complexes observed after the ultrafiltration session in this study can be the reflection of a relatively low PRR (and temporary intravascular contraction) rather than the effectiveness of the treatment. The borderline P values for some volume variables observed in this study can therefore be explained by a significant variation in PRR among patients rather than just a type-2 error as suggested by the authors. Measurement of serum albumin could help clarify this point. Moreover, it would have been of interest to have obtained a second ECG a few hours after completion of therapy to assess whether the modification in QRS complexes persisted at a time when compartmental fluid shifts may have equilibrated.

Second, the authors suggest that modification of QRS complexes be used as a marker for effectiveness of ultrafiltration therapy. This is similar to the concept of using hemoconcentration (increase in hematocrit) as a noninvasive method for assessment of intravascular volume status, which is a known management strategy in the field of dialysis. Indeed, many newer dialysis machines have this tool incorporated in their design. It is noteworthy that even such a well-recognized method can prove to be harmful when incorporated into simplistic algorithms, and thus not modified in complex clinical cases using the clinical judgment and insights of a fully trained staff.⁵ Therefore, even if the authors could justify the use of ECG for evaluation of effectiveness of ultrafiltration therapy, its potential

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impact on the safety of the patients still remains to be evaluated.

Third, as correctly pointed out by the authors, clinicians need to be cognizant of changes in the ECG due to axis shifts induced by volume removal: a phenomenon we previously described in hemodialysis patients undergoing ultrafiltration.⁶

Lastly, it is admirable that this study was performed with the background experiences from the UNLOAD trial. As we have discussed previously,⁷ it would be especially helpful for this type of investigation to also consider and report the various costs and potential financial benefits of their suggested interventions.

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