

LETTER TO THE EDITOR RESPONSE

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When one cannot see the forest for the trees: femoral dP/dt_{\max} , LVEF and pulse pressure in critically ill patients

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Dear Editor,

Whether the peak rate of pressure rise (dP/dt_{\max}) in peripheral arteries is influenced by left ventricular (LV) contractility or by loading conditions remains controversial. We, therefore, appreciate the letter by Monge-García et al. [1] and have read it with interest.

Our colleagues challenge the use of LV ejection fraction (LVEF) as a marker of LV systolic function and its comparison with femoral dP/dt_{\max} to assess the reliability of the femoral dP/dt_{\max} to track changes in LV contractility [1]. We entirely agree that LVEF is not a pure marker of LV contractility, as widely documented by others [2], and as clearly acknowledged in the discussion section of our article [3]. Furthermore, we agree that the invasive measure, LV end-systolic elastance (Ees), is the gold standard methodology to estimate LV contractility, but we were unable to measure it for ethical and technical reasons. In our paper [3], we presented LVEF data, as is commonly done in similar studies, but in contrast to the inference of our colleagues' letter [1], our conclusion was not based upon LVEF results.

In our study [3], we measured femoral dP/dt_{\max} by pulse contour analysis before and after varying, LV systolic function (dobutamine infusion), preload (volume expansion and passive leg-raising) and afterload (norepinephrine) in 19 critically ill patients with cardiovascular failure [3]. Femoral dP/dt_{\max} changed, not only in response to dobutamine infusion, but also following changes in cardiac loading, particularly following changes in afterload induced by variations in

norepinephrine dose. In addition, changes in femoral dP/dt_{\max} were strongly related to changes in arterial pulse pressure (PP) in each intervention and when all interventions were pooled (each $R > 0.90$). Interestingly, Sharman et al. have also documented strong associations between radial dP/dt_{\max} and brachial PP in various populations of non-critically ill patients [4, 5]. Since PP is mainly influenced by arterial stiffness and stroke volume, and given the fact that PP explained $> 80\%$ of the variance of femoral dP/dt_{\max} , we concluded that femoral dP/dt_{\max} was mainly influenced by pulsatile arterial load [3]. These findings suggest that femoral dP/dt_{\max} is highly sensitive to changes in loading conditions, especially afterload, and thus may not be directly interchangeable with LV contractility.

In previous work [6], our colleagues have documented a linear relationship between Ees and femoral dP/dt_{\max} in 10 anesthetized pigs during different loading and contractile conditions. However, while the authors present good concordance results, the strength of this relationship was rather moderate ($R^2 = 0.33$) [6]. To our knowledge, the only other experimental study documenting the link between contractility (Ees) and femoral dP/dt_{\max} is Morimont et al. [7] who studied 6 anesthetized and mechanically ventilated pigs after endotoxin-induced shock and norepinephrine infusion. In their work, authors reported that Ees and femoral dP/dt_{\max} were significantly, albeit weakly correlated ($R = 0.51$, i.e., $R^2 = 0.26$). Therefore, two independent experimental studies have documented that factors other than LV contractility (Ees) explain the vast majority (67% to 74%) of the variance of femoral dP/dt_{\max} in response to LV contractility and ventricular loading changes [6, 7].

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In conclusion, our results showed that changes in femoral dP/dt_{max} are essentially analogous to changes in femoral PP and therefore subject to the same dependence to variations in cardiac loading conditions, especially pulsatile arterial load. Our hypothesis is consistent with the previous results obtained at the upper arm level in non-critically ill patients [4, 5] and with results from two independent experimental studies, having failed to demonstrate strong correlation between LV contractility (Ees) and femoral dP/dt_{max} in various experimental settings [6, 7].

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Authors' contributions

SV, DCH and XM contributed equally to the writing of this letter. All authors read and approved the final manuscript.

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The original study was supported by the clinical institutions where it was developed. The research team performed this study as part of their regular clinical work.

Availability of data and materials

The datasets used and/or analyzed during the original study are available from the corresponding author upon request.

Ethics approval and consent to participate

The original study was approved by local ethics committees of both participating institutions (Comitè Ètic d'Investigació Clínica de la Corporació Sanitària Parc Taulí CEIC2013616 and Comité pour la protection des personnes Ile-de-France VII). All patients or next of kin gave their consent to participate to the study.

Consent for publication

Not applicable

Competing interests

Prof. XM is a member of the medical advisory board of Pulsion Medical Systems. The remaining authors declare that they have no competing interests.

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