

Is the Carotid Artery a Window to the Left Ventricle?

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Sir,

I read with interest recent research by Chowhan et al.¹ They ask whether the common carotid artery velocity time integral (cVTI) can be used to infer how the left ventricle responds to a passive leg raise (PLR) maneuver in critically ill patients categorized as either non-septic controls, septic or septic shock.

They observed that % change (% Δ) in cVTI during PLR detected a 15% change in stroke volume (SV) in the non-septic, control group with an acceptable area under the receiver operator curve (AUROC) value of 0.74. In the septic group, the % Δ cVTI had an excellent AUROC (i.e., 0.90) for detecting a 15% change in SV, but in the septic shock group, the AUROC was only 0.69. The optimal % Δ cVTI thresholds for these three groups were approximately 11%, 6%, and 15%, respectively.¹

Yet, the authors also reported poor correlation between the cVTI and SV calculated by transthoracic echocardiography. This is slightly surprising given a recent study showing good correlation between % Δ cVTI and % Δ LVOT VTI in critically ill patients receiving controlled, mechanical ventilation.² Admittedly, the study of Pace et al. evaluated % cVTI and % aortic VTI variation during the respiratory cycle and not in response to the PLR.² Nevertheless, in a human model of central hypovolemia and resuscitation comparing approximately 50,000 cardiac cycles, we observed a strong, linear correlation between % Δ SV measured by noninvasive pulse contour analysis and % Δ cVTI measured by a wearable Doppler ultrasound.³ Why might the findings of Chowhan et al. be disparate?

The correlation that Chowhan et al. revealed in their Figure 2 is the change in (i.e., “delta”) SV and cVTI. It appears that they regressed % cVTI against % SV, as they did for their AUROC analyses. However, there are zero data points above “delta SV 15” in the control group (their Fig. 2A) and only 1 data point above “delta SV 15” in the septic shock group (their Fig. 2C). Given that they report that 35% (i.e., 7/20) in the control group and 35% (i.e., 7/20) in the septic shock group were fluid responders—defined as an increase in SV of at least 15%—should not there be 7 data points in each of these groups above “delta SV 15”?

If, instead, Chowhan et al. regressed absolute change in SV (in milliliters) against absolute change in VTI (in centimeters), this relationship may not be a fair comparison and could explain discordance. Finally, the authors are absolutely correct to assert that time delay between measurements made at the LVOT and common carotid artery,⁴ inadequate beat sample size,⁵ human factors,⁴ and cerebral autoregulation all might, additionally, mediate discrepancy. Accordingly, cVTI may remain a useful, albeit

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transient, window to the left ventricle; this is most likely observed under ideal measurement conditions and when the unit of interest is relative change.

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