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Passive leg raising, peripheral perfusion, and cardiac output: insights into hemodynamic coherence

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We read with great interest the study by Morin et al. assessing passive leg raising (PLR)–induced changes in skin blood flow (SBF) to predict microvascular fluid responsiveness in septic patients [1]. Their findings closely parallel our previous work published in *Critical Care* in 2019, in which PLR-induced changes in capillary refill time (CRT) accurately predicted peripheral perfusion response to volume expansion, despite the absence of correlation with cardiac output (CO) [2].

At the time, we hypothesized that this dissociation might partly reflect methodological limitations in the assessment of both CRT and CO. The present study, using high-precision laser Doppler monitoring of SBF, demonstrates that this dissociation persists even with refined microcirculatory measurement, thereby reinforcing the concept of a loss of hemodynamic coherence during PLR and subsequent fluid loading.

While dissociation between macrohemodynamics and the microcirculation is now well established at baseline in critically ill patients [3], it is more difficult to reconcile in the specific context of a quasi-experimental preload challenge explicitly designed to modify CO via the Frank–Starling mechanism. In this respect, the convergence of results from two independent studies using distinct techniques (CRT and SBF), yet observing a similar dissociation during PLR, raises an important and unresolved physiological question.

Although the authors acknowledge similarities with CRT-based approaches, PLR-induced CRT changes were not reported. Given the strong association between SBF and CRT observed in the present study, comparable findings might reasonably be expected. It should be noted that the characterization of CRT assessment as too time-consuming in our study primarily reflected methodological choices aimed at ensuring blinding and reproducibility rather than intrinsic feasibility limitations [2]. Moreover, the Ait-Oufella group has previously demonstrated excellent reproducibility and sensitivity to small CRT changes without such time-intensive procedures [4].

At present, no definitive mechanism can fully explain why preload-induced variations in CO fail to correlate with changes in skin perfusion. Conceptually, four physiological scenarios can be identified:

1. **No increase in CO and no increase in skin perfusion**,
2. **Increase in CO accompanied by increased skin perfusion**.

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These two situations reflect preserved hemodynamic coherence during PLR and fluid loading and would be expected to result in a strong correlation between CO and SBF (or CRT).

To account for the observed lack of correlation, the following two situations must therefore be frequent:

3. **Increase in CO without improvement in skin perfusion**

This scenario may represent an extension of baseline hemodynamic incoherence, whereby the skin microcirculation remains vasoconstricted and the additional flow is preferentially redistributed toward other vascular beds (e.g., splanchnic or renal). While physiologically plausible, this interpretation challenges our understanding of the strong prognostic value of CRT [5] and its relevance as a resuscitation target if skin perfusion were entirely dissociated from perfusion of other critical organs.

4. **Improvement in skin perfusion without a measurable increase in CO**

This situation is the most challenging from a physiological standpoint. The following interpretations should therefore be viewed as hypothesis-generating. PLR and fluid loading may trigger baroreflex-mediated reductions in sympathetic tone due to changes in atrial and central venous pressures. A trade-off between preload recruitment and reduced sympathetic drive could theoretically result in stable CO despite improved peripheral vasodilation. In addition, a reduction in sympathetic tone may increase unstressed volume, limiting effective preload augmentation despite venous blood mobilization capillary bed recruitment.

Furthermore, preferential redistribution of blood flow toward highly compliant vascular beds such as the skin may occur even in the absence of a substantial increase in global flow. Finally, microvascular perfusion is exquisitely sensitive to changes in perfusion pressure and critical closing pressure, such that modest hemodynamic shifts may result in marked capillary recruitment.

Taken together, the consistency of findings across studies suggests that PLR- and fluid loading–induced changes in CRT or SBF may reflect complex microvascular reactivity and baroreflex-mediated modulation of sympathetic tone rather than perfusion changes driven solely by CO increases associated with preload responsiveness. Further dedicated physiological studies are clearly needed to elucidate the mechanisms underlying this unexpected dissociation in a context classically viewed as CO-driven.

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