



Rebuttal to: “As simple as possible, but not simpler: estimating the effective arterial elastance at bedside”

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We would like to thank to Chemla et al. for their valuable comments about our recent experimental study. These authors suggested that effective arterial elastance (Ea) using systolic arterial pressure (SAP) incorporates the oscillatory component of arterial load, whereas Ea using mean arterial pressure (MAP) does not. They implied the role of arterial stiffness in the discrepancy between MAP and Pes. The same group has recently published a clinical study using 90% of SAP for estimating Ea, and they suggested to use femoral SAP as surrogate of left ventricular end-systolic pressure (Pes) [1].

Effective arterial elastance attempts to simplify the complexity of arterial input impedance into one single variable. This simplification, however, is not without its drawbacks. As Ea is derived from a simple lumped model of arterial system as the three-element Windkessel [2], it does not include the impact of arterial wave reflections. This is a well-known and inherent limitation of Ea [2, 3]. Moreover, mechanical properties of the arterial system affect to a different extent to Ea: the sensitivity of Ea to a change in arterial resistance is three times higher than to a similar change in arterial compliance [4, 5]. Just as reading the synopsis of a book gives us a general idea of the story but does not tell us about its

protagonists, Ea lumps the steady and pulsatile components of arterial load into one single value, but it does not inform about their relative contribution [5]. Therefore, Ea cannot replace arterial impedance and it should be used only as an integrative measure of arterial load [3]. Particularly, Ea is a valuable index if we aim to assess the interaction between the heart and the arterial circulation, since both the ventricular and the arterial elastances can be related in a single ratio [7].

Ignoring stroke volume, any peripheral estimation of Ea will be valid as long as the arterial surrogate is close to Pes. In this regard, MAP, dicrotic notch pressure and 90% of aortic systolic pressure offer similar performance. But *aortic*, not femoral or radial pressure. As the first author of this comment already pointed out in a previous paper: “The 0.9SAP approximation applies strictly to central pressure recordings and not to brachial artery pressure, given the physiological increases in systolic pressure observed from the aorta to periphery (pulse wave amplification phenomenon)” [4]. Moreover, in their recent study, the authors acknowledged this limitation: “The amount of [systolic pressure amplification] varies depending upon the peripheral arterial site. Thus, peripheral estimates of Ea are expected to differ from central estimate of Ea, depending on the amount of systolic pressure amplification at the peripheral arterial site where [systolic arterial pressure] is measured” [1]. Moreover, these authors mentioned that if arterial stiffness increases, as in hypertensive patients, there could be a discrepancy between MAP and Pes. However, if so, this discrepancy will be always greater for SAP! If we look carefully to the Fig. 1 in their comment, even if MAP overestimate Pes, this difference will be always less than for the 90% of SAP. Moreover, if arterial stiffness increases, the arterial pulse wave velocity will increase boosting the impact of arterial wave reflections on peripheral pressure measurements. If this is the case, peripheral SAP will significantly overestimate Pes. On the other hand, during septic shock peripheral pressure could

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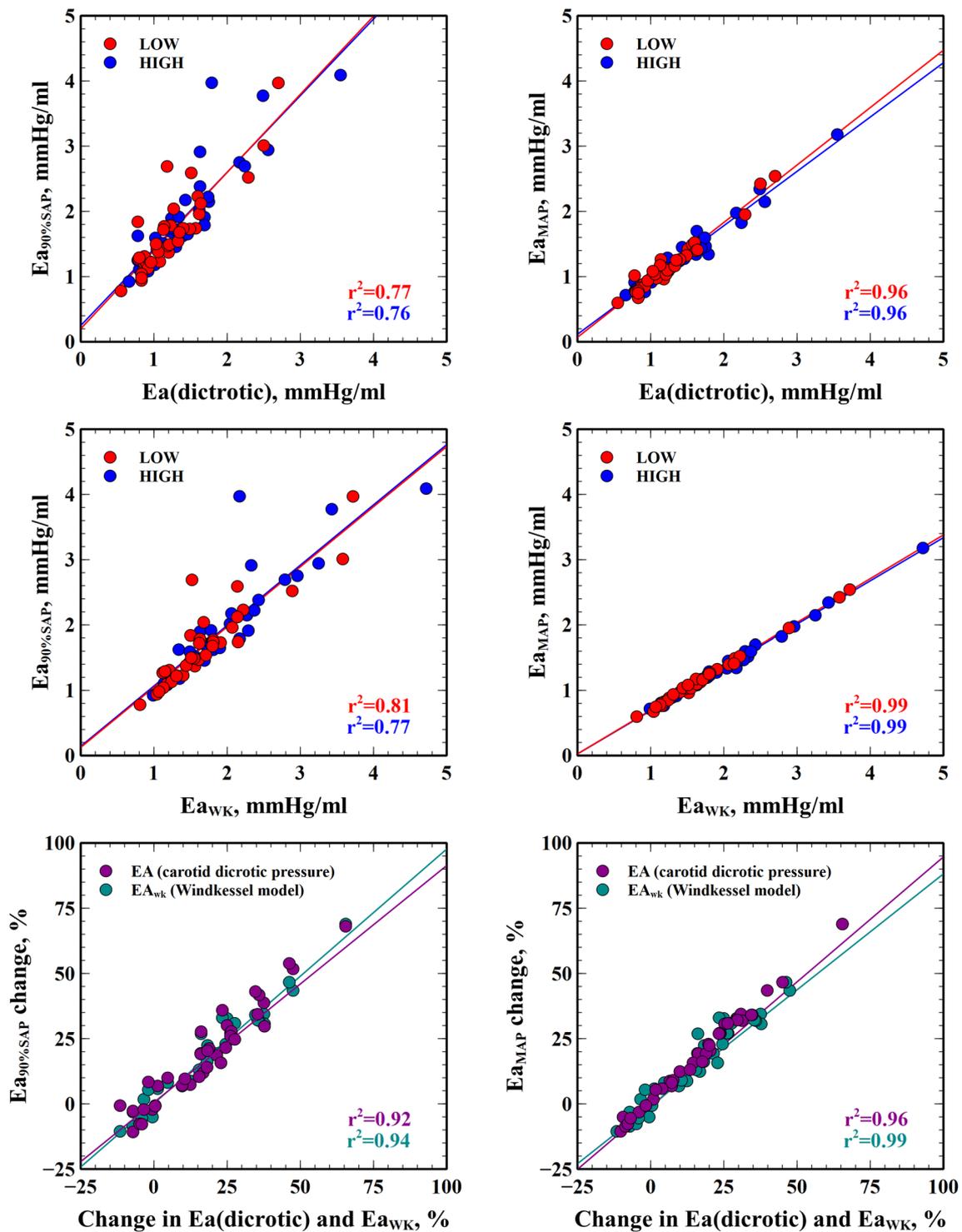


Fig. 1 Relationship between peripheral estimates of Ea using 90% of systolic arterial pressure (Ea_{90%SAP}) and mean arterial pressure (Ea_{MAP}), and central Ea using carotid dicrotic pressure [Ea(dicrotic)] and three-element Windkessel model derived Ea (Ea_{WK}). LOW and

HIGH refer to the lowest and highest dose of norepinephrine. At the bottom, relationship between changes peripheral estimates and both central Ea estimates [Ea(dicrotic) and Ea_{WK}] during norepinephrine dose changes

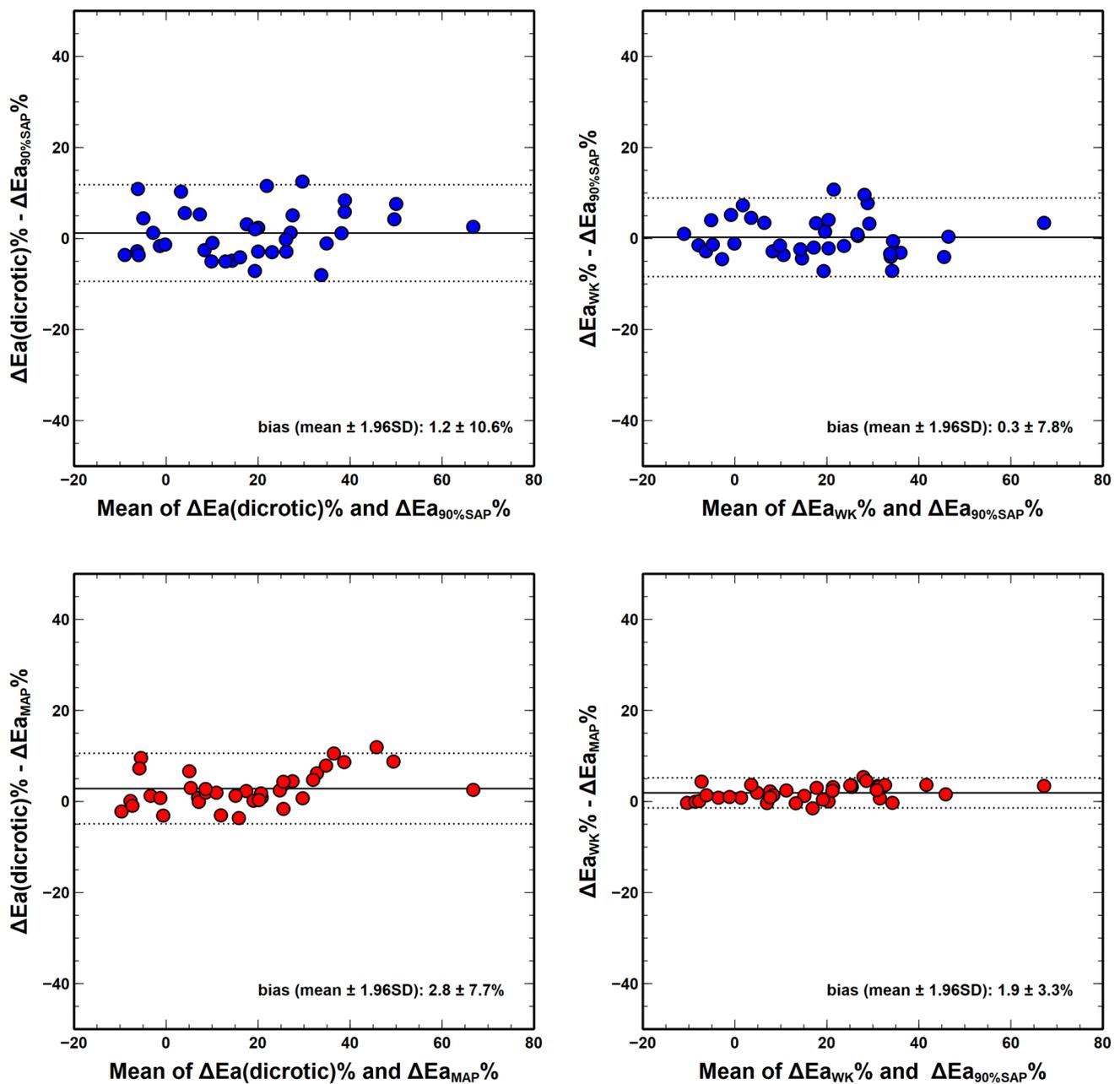


Fig. 2 Bland-Altman analysis for percentage changes for different arterial estimates of Ea ($Ea_{90\%SAP}$ and Ea_{MAP}) and both central Ea (dicrotic and Ea_{WK})

underestimated Pes, as a central-to-peripheral vascular tone could be decoupled [6]. In summary, the impact of changes in arterial stiffness will be always more evident in systolic arterial pressure, and in all cases, MAP will be superior as a surrogate for Pes.

Finally, the authors have recently published the results of a clinical study using $0.9SAP$ as surrogate for Pes. They have studied 50 stable and spontaneously breathing patients. In 30 of those patients a 500 ml fluid challenge was performed. The authors concluded that “effective arterial elastance may be

reliably estimated at bedside ($0.9 \times$ systolic femoral pressure/stroke volume)”. Interestingly, we have recently published a similar research in 37 septic shock patients with a very similar methodology [7]. In this study, we have assessed arterial variables during an incremental change in norepinephrine dose (from 0.11 ± 0.13 to $0.20 \pm 0.16 \mu\text{g}^{-1} \text{kg}^{-1} \text{min}^{-1}$). We used applanation tonometry-derived carotid pressure for estimating the central pressure and calculated Ea using dicrotic pressure from the carotid pressure and stroke volume from esophageal Doppler. We also calculated Ea (Ea_{WK}) using a

three-element Windkessel model including both resistive and pulsatile components of arterial load [3]:

$$Ea = R_T / [t_s + \tau * (1 - e^{-td/\tau})]$$

R_t is total vascular resistance (carotid MAP/esophageal Doppler cardiac output), t_s and t_d are systolic and diastolic periods, respectively, and τ the diastolic time constant ($R_T * C_{art}$) [3]. Arterial compliance (C_{art}) was estimated by the Liu method [8]: $C_{art} = \text{esophageal} - \text{Doppler stroke volume} / [K * (P_1 - P_2)]$, where K represents the ratio of the total area under the central pressure and the diastolic area, and P_1 and P_2 are the maximum central pressure after dicrotic and the diastolic pressure, respectively. Radial and carotid MAP were calculated as the time-averaged pressures during the cardiac cycle. Carotid pressure was calibrated using the values of the invasive diastolic and radial MAP. We have estimated peripheral Ea from the invasive radial pressure using both MAP (Ea_{MAP}) and 90% of systolic arterial pressure ($Ea_{90\%SAP}$) as surrogates for Pes .

According to our analysis, the relationship of MAP estimates of Pes was better than for 90%SAP estimates for both Ea and Ea_{wk} , and the limits of agreements narrower (Figs. 1, 2). Moreover, the conclusion drawn from our analysis is stronger than those reported by the authors of this comment, as we have altered the arterial system modifying the vasopressor dose, whereas in their study Ea was assessed during stable conditions or during a fluid challenge, which it does not represent a significant challenge to the arterial system. Therefore, even using their own methodology, the results are in agreement with our previous experimental study, which it is grounded in a more robust and validated methodology.

Finally, we would like to subscribe the Albert Einstein's words mentioned by the authors of this comment: "*everything should be made as simple as possible, but not simpler*". However, the use of systolic arterial pressure as surrogate for Pes neglected the complex nature of arterial system, since it ignores the heterogenous and finite design of the

arterial circulation. This approach overlooks the effects of arterial wave reflections and the impact on arterial pressure measurements. Therefore, the use of femoral or radial systolic pressure simplifies the arterial system to a mere conduit with a constant pulse pressure. This simplification, indeed, is less helpful than using MAP, as our results have already demonstrated.

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