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LETTER TO THE EDITOR

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

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We have read with interest the recent experimental study performed by Monge Garcia et al. in twelve anesthetized and mechanically ventilated adult pigs [1]. In their conclusion, the authors recommend the clinical use of the mean arterial pressure over stroke volume ratio (MAP/SV) as a robust and preferable estimate of the effective arterial elastance (Ea = ESP/SV, where ESP is the left ventricular end-systolic pressure). Besides the originality of the study results obtained under various experimental conditions, the current article [1] may also be viewed as supporting the use of the MAP/SV ratio as an estimate of Ea in patients with septic shock [2]. Improving the estimation of Ea at the bedside may be an important issue. Indeed, Ea incorporates the main components of arterial load, and the ratio of Ea to left ventricular end-systolic elastance allows quantifying the mechanical coupling between the left ventricle and its load [3–5]. However, we feel that the oversimplification of the estimation of Ea may hamper the precise evaluation of the left ventricular-arterial coupling, in the saying attributed to Albert Einstein that: *"everything should be made as simple as possible, but not simpler"*. For sake of clarity for the reader of the *Journal of Clinical Monitoring and Computing*, we are concerned by the fact the authors [1] do not discuss the role of arterial stiffness in the estimation of Ea.

It is indicated that the effects of common pathological conditions on arterial load may be reliably estimated at bedside by using MAP/SV "*in a more comprehensive way than the gross calculation of arterial resistance*" [1], but this statement is hard to comprehend. In most hemodynamic studies including the present one [1], arterial resistance is estimated as MAP/cardiac output (total peripheral resistance). Thus the MAP/SV ratio is simply the product of total peripheral resistance multiplied by heart rate [4], and its added value over resistance in an attempt to characterize arterial load in a more comprehensive way is thus questionable.

The difference between Ea and the MAP/SV ratio lies in the discrepancy between ESP and MAP. The current article [1] reactivates the initial proposal made by Sunagawa et al. 34 years ago, namely that MAP is a reasonably good estimate of ESP, and that MAP/SV matches Ea [4]. However, two subsequent studies [6, 7] have clearly demonstrated that there may be a considerable difference between MAP and ESP, especially in subjects with stiff vasculature (Fig. 1). On average, Ea exceeded MAP/SV by 22% in the 10 subjects studied by Kelly *et al*. at baseline (4 young normotensives and 6 older hypertensives) [6]. In the study by our group [7] Ea exceeded MAP/SV by 19 ± 5% (mean ± SD) in 20 controls and by 23 ± 5% in 46 hypertensive patients, and the difference linearly increased with total arterial stiffness. These two studies [6, 7] reported that ESP matches 90% systolic aortic pressure, and this approximation is the one most commonly used nowadays [5].

We do not intend to challenge the previously documented observation that MAP/SV only slightly underestimates Ea in the setting of low to normal left ventricular loading conditions [6, 8]. It must be noted however that in the Kelly et al. study [6], MAP/SV still underestimated Ea by 14%

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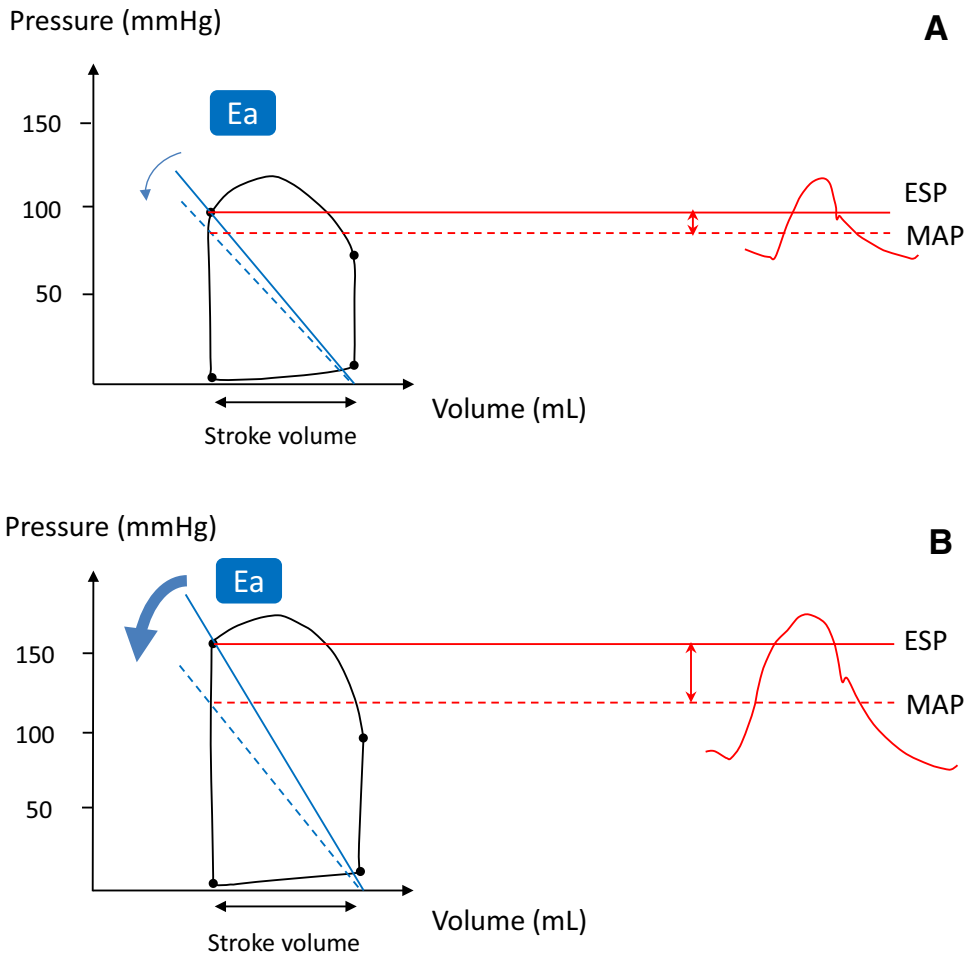

Fig. 1 Graphs show disparity of estimating the effective arterial elastance (Ea) using left ventricular end-systolic pressure ESP (solid lines) or mean arterial pressure MAP (dotted lines). Two pressure–volume loops are displayed in a normotensive (**a**) and hypertensive (**b**) subject. Average data have been extracted from Refs. [6, 7] to draw these theoretical, typical examples. For subject **a**, as in normal animals, there is little disparity between ESP and MAP. As a result, the MAP/SV ratio is slightly lower the ESP/SV ratio (SV = stroke volume). However, in subject **b**, there is a considerable difference between the two points due to the larger influence of the pulsatile component of arterial load. As a result, MAP/SV is markedly lower than ESP/SV. The important point is that in humans, particularly those with stiff or hypertensive vasculatures, ESP and MAP can deviate substantially

following decreases in left ventricular loading conditions. Based on the Tables 1–3 from Ref. [1], the ESP was < 88 mmHg on average in 11 of the 12 study subgroups, namely the 6 baselines and all the interventions performed except phenylephrine (fluid infusion, dobutamine, sodium nitroprusside, bleeding and esmolol). It is thus expected that the corresponding systolic aortic pressure was < 98 mmHg on average (i.e., 88 mmHg/0.9). No need to say that this is a rather low prevailing pressure level, which most often corresponds to normal MAP rather than normal systolic pressure in humans. Consistently, in the representative pressure–volume loop presented [1], systolic aortic pressure is circa 95 mmHg and aortic pulse pressure is narrow. Besides the fact that animals were anesthetized, this may be due to the fact that young adult, healthy pigs were studied. Importantly, it is also expected that their ESP minus MAP difference is minimal given their highly compliant, healthy arteries [6, 7]. Thus, in our opinion, the authors' conclusion [1] strictly pertains to conditions associated with low prevailing pressure and highly compliant arteries (low stiffness). Conversely, in cardiovascular population studies as well as in the standard ICU population including patients with shock states, there is a high prevalence of elderly patients with comorbidities including hypertension, both conditions being associated with stiffened arteries.

We agree that unlike systolic arterial pressure, MAP does not suffer from amplification from aorta to peripheral large arteries and is thus interchangeable when measured in any arterial site [1]. However, replacing ESP by MAP in the Ea formula implies neglecting the contribution of the pulsatile component of arterial load. We have recently documented that Ea is more reliably estimated at the bedside by using femoral rather than radial *systolic* arterial pressure in 50 hemodynamically stable, critically ill patients [9]. In particular, the 0.9 × femoral systolic pressure/SV ratio only slightly overestimated central (carotid) Ea by 8 ± 8%. This may be explained by the fact that the amount of the natural systolic pressure amplification is moderate at the level of the largest proximal arteries, especially in middle-aged and elderly subjects [10, 11], while a significant amount of pressure amplification occurs more distally (e.g., brachial-to-radial pressure amplification) [10].

Finally, our esteemed colleagues are opinion leaders in the hemodynamic field and it is surprising to note that

contrary to their previous works [12, 13], they now clearly favor a description of arterial load based on the rigid tube model which considers steady load only and neglects the pulsatile component of load [1]. Their viewpoint is not shared as it is at variance with the increasing number of studies demonstrating the importance of arterial stiffness in health and diseases, both at baseline and following therapeutic interventions [14]. Finally, their recommendation also contradicts the very background of the study [1], namely that the interest of Ea is that it lumps the steady and pulsatile components of arterial load.

In conclusion, in our opinion, the MAP/SV approximation of Ea [1, 2, 4] must be avoided in a general population, especially when elderly or hypertensive subjects are included. If one still decides to use the MAP/SV ratio to approximate Ea at bedside, it must be restricted to patients with low to normal prevailing pressure, bearing in mind that MAP/SV it is nothing else but the product of total peripheral resistance multiplied by heart rate. By doing so, only the steady component of arterial load will be documented, and the potential differences in arterial stiffness between subjects will be neglected, as well as the potential beneficial effects of treatment on arterial stiffness.

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