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LETTER TO THE EDITOR

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Diastolic shock index (DSI) works… and it could be a quite useful tool

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Dear Editor

We thank Dr. Dalmau for his interest in our recent manuscript [1]. Vasodilatory shock is fundamentally characterized by a failure of peripheral vascular smooth muscle cells to constrict, resulting in altered tissue perfusion and organ dysfunction. Although severe inflammation is perhaps the most prominent factor triggering vasodilatory shock, other non-inflammatory-related mechanisms could also be implied. Arterial hypotension is the natural consequence of decreasing arterial tone and represents one of the cardinal signs of vasodilatory shock along with a progressively impaired response by the vascular smooth muscle to endogenous circulating and exogenous vasoconstrictors. Nevertheless, hypotension observed during septic shock results from a complex interaction between relative and absolute hypovolemia, myocardial dysfunction, vasodilation, and altered blood flow distribution. In addition, alteration in the balance among sympathetic, cholinergic and anticholinergic inflows can directly affect inflammatory and immunologic response beyond their direct effects on the heart and vessel walls.

Clinicians facing septic shock clearly recognize low diastolic arterial pressure (DAP) as a classical sign of vasodilation, which is thought to be explained, at least in part, by lowering vascular tone. In normal conditions, vascular tone of the resistance arteries and arterioles determines peripheral vascular resistance, contributing in turn to the regulation of blood pressure and blood flow to, and within the tissues. Vascular tone, defined as the activation degree of vascular smooth muscle cells, provides a measure of vascular regulation in response to external stimuli as hormonal, myogenic, neural and endothelial-derived factors. Nevertheless, direct in vivo assessment of vascular tone during dynamic clinical conditions is not quite evident and it has been typically based on indirect measurements of vascular resistance: blood flow; arterial pressure/volume, pressure/diameter, and compliance/distensibility–pressure relationships [2, 3].

In general, an index is the ratio of one dimension of a thing (such as a physiological parameter) to another dimension. As such, diastolic shock index (DSI) represent a simple ratio between DAP and heart rate (HR), as a magnitude possibly reflecting how severe cardiovascular dysfunction is. DSI is based on three simple sequential thoughts: (a) under isovolemic conditions and constant arterial compliance, shortening of diastolic time is associated with higher DAP while a prolonged diastole leads to an opposite effect; (b) acute reductions in arterial pressure are normally compensated by increased sympathetic activity, which usually leads to tachycardia; (c) consequently, simultaneous and opposite variations in DAP and HR could reflect more severe cardiovascular alteration, with progressively high HR unable to compensate DAP drops as consequence of a gradual increased vasodilation. Interestingly, even though Dr. Dalmau suggests that DSI should not represent vascular tone because [times^{-1}].[mmHg] units do not correspond to any mechanical property of the vascular walls (a situation with which we fully agree), he supports the presence of a direct link, or dependence, between DAP and arterial vasomotor tone.

Operational definitions of shock have classically included the reduction of mean (MAP) and/or systolic

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arterial pressure (SAP), assuming the pivotal role of both MAP and SAP on organ perfusion, in addition to the clinical prognostic value of sustained low MAP values. Nevertheless, although DAP is not widely mentioned and it is not considered to categorize the severity of shock, evaluation of DAP could have significant clinical implications especially when the underlying mechanism of shock is vasodilation. Although admittedly, hypotension observed during septic shock results from a complex interaction among altered pump, "pipes", and volume factors, as previously discussed.

Peripheral resistance, arterial compliance, pulse wave velocity, and the timing of pulse wave reflections affect both steady (i.e., the mean arterial pressure) and dynamic components (i.e., the systolic and diastolic pressures) of arterial pressure. In turn, arterial compliance is affected by the blood pressure changes per se, and vascular biomechanics dependent on the composition of vessel walls. Admittedly, alterations in vascular tone should also influence SAP as correctly suggested by Dr. Dalmau. Nevertheless, changes of arterial pressure after fluid loading in septic "fluid-responders" usually translates into SAP increases with minimal or no effect on DAP. Certainly, one classical characteristic of resuscitated vasodilatory shock is the increased pulse pressure, which obeys to the stroke volume rise with no immediate variations in peripheral resistance. Indeed, classical observations demonstrated that variations of pulse pressure are greater when compliance changes at constant resistance than when resistance changes at constant compliance. Importantly, when cardiac output increases, the arterial compliance determines the rate at which the mean arterial pressure will attain its new, elevated value but will not determine the magnitude of the new pressure.

The arterial system has a dual function: first, as a simple conduit to adequately supply blood to the tissues; and second, as a converter of pulsatile flow generated by the heart beating into a continuous flow of blood at the periphery (i.e., the Windkessel phenomenon). In his letter, Dr Dalmau correctly signals, according to the two-element Windkessel model, that as the DAP is mainly determined by the diastolic time constant RC (i.e., arterial resistance times compliance), one could interpret it as being representative of the arterial tone, while the dependence of systolic arterial pressure on the arterial RC is more directly confounded by other parameters—namely, heart function parameters—than the DAP is. Nevertheless, the Windkessel model does not determine diastolic pressure itself (although it clearly influences it).

Recent experimental and observational data suggest that a very early start of vasopressor support could be beneficial [4, 5]. Nevertheless, there are no clear signals indicating when vasopressor support should be started. In this way, very early signals of severe vasodilation should alert on its possible immediate requirement and as such, DSI could be a quite useful tool on this purpose.

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References

1. Ospina-Tascón GA, Teboul JL, Hernandez G, Alvarez I, Sánchez-Ortiz AI, Calderón-Tapia LE, et al. Diastolic shock index and clinical outcomes in patients with septic shock. Ann Intensive Care. 2020;10(1):41.
2. Guerrisi M, Vannucci I, Toschi N. Differential response of peripheral arterial compliance-related indices to a vasoconstrictive stimulus. Physiol Meas. 2009;30(1):81–100.
3. Zheng D, Allen J, Murray A. Non-invasive in vivo assessment of changes in peripheral arterial properties with estimation of arterial volume compliance. Physiol Meas. 2007;28(10):1317–27.
4. Byrne L, Obonyo NG, Diab SD, Dunster KR, Passmore MR, Boon AC, et al. Unintended consequences; fluid resuscitation worsens shock in an ovine model of endotoxemia. Am J Respir Crit Care Med. 2018;198(8):1043–54.
5. Ospina-Tascón GA, Hernandez G, Alvarez I, Calderón-Tapia LE, Manzano-Nunez R, Sánchez-Ortiz AI, et al. Effects of very early start of norepinephrine in patients with septic shock: a propensity score-based analysis. Crit Care. 2020;24(1):52.

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LETTER TO THE EDITOR

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The diastolic shock index works… but, what is it?

Rafael Dalmau*

Editor

The recent study by Ospina-Tascón et al. [1] presents a novel hemodynamic index—the "diastolic shock index" (DSI), defined as the ratio of heart rate (HR) and diastolic arterial pressure (DAP)—distinguishing itself from the "classical" and "modified" shock indices [2], which are based on the systolic and the mean arterial pressure, respectively.

On the basis that "DAP depends [directly] on vascular tone and [inversely] on the duration of the cardiac cycle [i.e., the reciprocal of HR]" [1], the authors targeted the DAP, with the following goals: to assess the state of the vascular tone; to predict the severity and clinical outcomes of patients undergoing septic and other vasodilatory shock states; and to guide early vasopressor therapy.

With this rationale in mind, there are three matters that, given the intuitive and empirical nature of this index, might have been overlooked in its formulation. The first two address the meaning of the ratio between HR and arterial pressure (AP), and the third one is exclusively devoted to the DSI.

The word "index" has more than one meaning, but, as a general rule, an index is supposed to relate quantities independent of each other, in order to obtain additional and different information from that given separately by the individual quantities [1]. More precisely, mean arterial pressure − mean central venous pressure = CO × TPR.

In the case of the shock indices, the variables involved—HR and PA—are already interrelated by a preexisting function, so taking their ratio is redundant. With PA = CO × total peripheral resistance (TPR)[2] and CO = HR × stroke volume (SV), we can rewrite PA = HR × SV × TPR; in other words, the ratio HR/PA is already implied in the functional relationship between them.

Secondly, there is the principle of dimensional consistency, by which two entities with different dimensions cannot represent the same thing. If the ratio of HR and AP is supposed to represent something else (here, certain "state" of the cardiovascular system in shock), the shock index should have dimensions (and the corresponding units) of that quantity.

If the DSI, in particular, is to represent the vascular tone [1], then the units of the quantity representing it should be of [Time^{-1}] × [Pressure^{-1}], which, evidently do not correspond to any mechanical property of the vascular wall related to its "tone" (whether it is wall tension, arterial resistance, or arterial compliance), which leads to the third and final point.

The direct link, or dependence, between DAP and arterial vasomotor tone is well known in the literature, and is

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[1] For instance, the "cardiac index" relates CO to body surface area, yielding a *new* quantity relating heart performance to the size of the individual, and which corresponding units are consistent with the new information which is not found in the definition of either of the two separate magnitudes. Likewise, if the ratio of HR/DAP is to represent the vascular tone, the units should be compatible.

[2] More precisely, mean arterial pressure − mean central venous pressure = CO × TPR.

the basis for the present DSI. However, there seems to be no apparent reason as to why the DAP should *preferentially* reflect the vascular tone, or more than the systolic pressure does. In other words, both the systolic *and* diastolic pressures are determined by vascular factors.

This matter is not miscellaneous, as with no basis for linking DAP specifically to arterial tone there would be no justification for distinguishing the DSI from the classical or the modified shock indices. A plausible answer to this question may be found in simple "Windkessel" theory [3]:

$$P(t) = P(t_d)e^{\frac{-(t-t_d)}{(RC)}},$$

where the exponential pressure decay from the start of diastole (t_d) is governed by the time constant "*RC*" (i.e., arterial resistance times compliance) [3].

In this way, as the DAP is mainly determined by the diastolic time constant *RC*, one could interpret it as being representative of the arterial tone, while the dependence of systolic arterial pressure on the arterial *RC* is more directly confounded by other parameters—namely, heart function parameters—than the DAP is.

Finally, these observations are not intended to disregard the potential benefits of implementing the DSI as a statistical predictor of some event related to septic shock. Perhaps, its success is explained by the fact that the two quantities somehow interrelated, one linked to the heart and the other linked to the vasculature, are simultaneously analyzed reflecting the interaction between the two systems.

Abbreviations

AP: Arterial pressure; C: Compliance; CO: Cardiac output; DAP: Diastolic arterial pressure; DSI: Diastolic shock index; *e*: Base of the exponential function; HR: Heart rate; *P*: Pressure; R: Resistance; TPR: Total peripheral resistance; *t*: Time; t_d: Time at the start of diastole.

[3] For simplicity, higher-order elements of the Windkessel model, as well as complex phenomena of arterial wave reflection, are neglected in the present consideration.

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References

1. Ospina-Tascón GA, Teboul J, Hernandez G, et al. Diastolic shock index and clinical outcomes in patients with septic shock. Ann Intensive Care. 2020;10(1):41. https://doi.org/10.1186/s13613-020-00658-8.
2. Liu YC, Liu JH, Fang ZA, et al. Modified shock index and mortality rate of emergency patients. World J Emerg Med. 2012;3(2):114–7. https://doi.org/10.5847/wjem.j.issn.1920-8642.2012.02.006.
3. Westerhof N, Lankhaar JW, Westerhof BE. The arterial Windkessel. Med Biol Eng Comput. 2009;47(2):131–41. https://doi.org/10.1007/s11517-008-0359-2.

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