Intensive Care Med
https://doi.org/10.1007/s00134-022-06798-8

SPECIAL ISSUE INSIGHT

Invasive arterial pressure monitoring: much more than mean arterial pressure!

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Arterial pressure monitoring

Invasive arterial pressure monitoring is a standard practice in critically ill patients [1] since it allows accurate and beat to beat assessment of mean arterial pressure (MAP), and also delivers valuable information about cardiac function, heart–lung interactions, the arterial system and valvular diseases [2]. Although several guidelines recommend a MAP target during initial hermodynamic resuscitation [3], focusing only on MAP is an oversimplification, since patients with similar values may have considerable differences in underlying pathophysiological conditions [4]. Therefore, all the components derived from arterial pressure monitoring should be considered for diagnostic or therapeutic decisions when facing an acute circulatory dysfunction, as will be addressed in this article.

Systolic arterial pressure

The systolic arterial pressure (SAP) defines the work that the left ventricle has to perform to generate an adequate stroke volume (SV), and is obtained by the interplay between cardiac performance and rate, the buffer mechanical function of the aorta, and peripheral resistances. Due to its strong dependence on SV, a marked decrease of SAP (< 90 mmHg or a fall > 40 mmHg) has been suggested as a diagnostic criterion and as a hemodynamic target, particularly in cardiogenic and hypovolemic shock.

A composite clinical index is the Shock index (SI), which is obtained by dividing the heart rate (HR) by the SAP. A SI > 0.7–0.9 has been used to detect to detect hypovolemia in patients with trauma and hypovolemic shock, and also as a prognostic factor in the former [5, 6]. One of the resuscitation protocols in the ProCESS trial included a SI ≥ 0.8 as a fluid resuscitation trigger [7], extending its use to septic shock. Another composite index is the modified shock index (HR/MAP) but its role has not been yet clarified.

Diastolic arterial pressure

In normal conditions, the diastolic arterial pressure (DAP) is mainly determined by vascular tone and remains nearly constant from the ascending aorta to the peripheral vessels. A low DAP reflects systemic vasodilation as long as the aortic valve is competent, but surprisingly DAP is not considered in current septic shock definitions, and its relationship with clinical outcomes has not been widely described [8]. Nevertheless, evaluation of the loss of vascular tone through the severity of diastolic hypotension could have profound implications on therapeutic decisions, such as early use of norepinephrine [9].

In addition, a low DAP (< 50 mmHg) may impair left ventricle myocardial perfusion especially in the case of tachycardia [8]. Therefore, when DAP is low, as in early septic shock, there is an increased risk of myocardial ischemia, particularly in patients with prior coronary artery disease. Myocardial ischemia could lead to a decrease in SV and systemic flow, thus further impairing tissue and microcirculatory perfusion. Therefore, DAP-guided resuscitation in early septic shock is being tested by an ongoing major clinical trial [10].

The diastolic shock index (DSI) was recently described as heart rate divided by DAP. DSI > 2.2 was associated with higher mortality in septic shock, and may lead to early vasopressors start [11].

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Mean arterial pressure

MAP is the pressure which exerts the greatest influence on blood flow autoregulation within organs, and on whole-body haemodynamic homeostatic mechanisms (such as the baroreceptors). A MAP of 65–70 mmHg is the initial macrocirculatory target to ensure organ perfusion pressure [3]. However, the relevance of organ back pressure as indirectly assessed by the central venous pressure has to be considered when evaluating the net impact of changes in MAP at the organ level, particularly in the liver and kidneys which are highly vulnerable to venous congestion.

MAP levels over the autoregulatory range does not result in further changes in perfusion, but the issue of the optimal value is controversial, and a higher MAP may be beneficial in chronic hypertensive patients with shock and microcirculatory dysfunction. However, there are some contradictory data, particularly in elderly patients. Thus, more research is clearly required on the best way to individualize MAP target by taking into consideration the effects on systemic, regional and eventually microcirculatory flow especially in patients with septic shock [4].

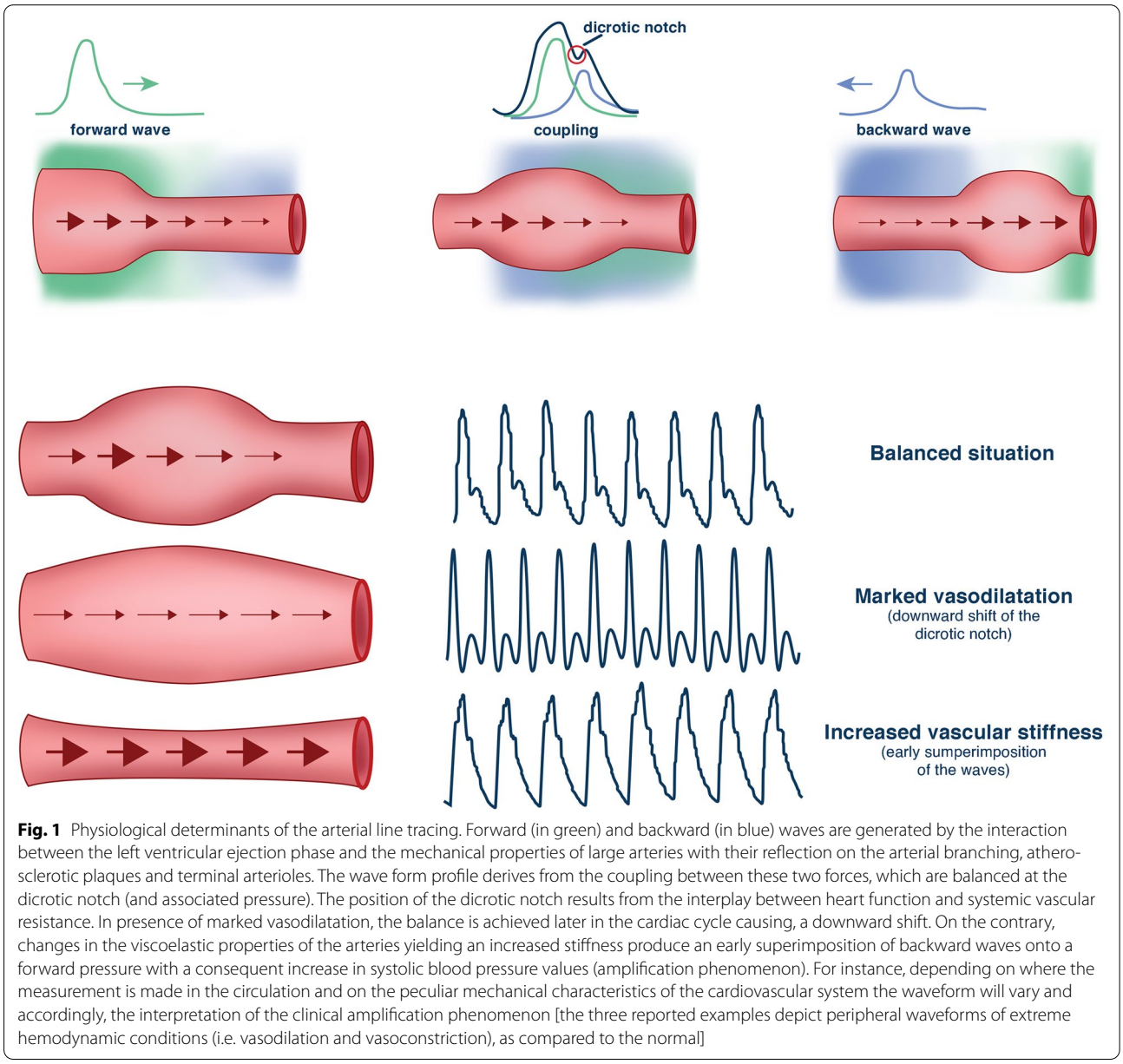

Fig. 1 Physiological determinants of the arterial line tracing. Forward (in green) and backward (in blue) waves are generated by the interaction between the left ventricular ejection phase and the mechanical properties of large arteries with their reflection on the arterial branching, atherosclerotic plaques and terminal arterioles. The wave form profile derives from the coupling between these two forces, which are balanced at the dicrotic notch (and associated pressure). The position of the dicrotic notch results from the interplay between heart function and systemic vascular resistance. In presence of marked vasodilatation, the balance is achieved later in the cardiac cycle causing, a downward shift. On the contrary, changes in the viscoelastic properties of the arteries yielding an increased stiffness produce an early superimposition of backward waves onto a forward pressure with a consequent increase in systolic blood pressure values (amplification phenomenon). For instance, depending on where the measurement is made in the circulation and on the peculiar mechanical characteristics of the cardiovascular system the waveform will vary and accordingly, the interpretation of the clinical amplification phenomenon [the three reported examples depict peripheral waveforms of extreme hemodynamic conditions (i.e. vasodilation and vasoconstriction), as compared to the normal]

Pulse pressure

Pulse pressure (PP) provides a readily accessible monitoring window into the function of the heart and its interaction with the vascular system. The main determinants of PP are SV and aortic impedance [12]. Multiple studies have shown that PP can adequately track stroke volume in different clinical scenarios [13, 14], and the use of PP as a surrogate of stroke volume is a cardinal aspect of multiple bedside fluid responsiveness tests, such as pulse pressure variation. By integrating heart–lung interactions and bedside monitoring, these tests allow clinicians to tailor fluid therapy and avoid unnecessary fluid loading [15].

PP could be used for initial clinical hemodynamic phenotyping in hypotensive patients to individualize further interventions. Broadly speaking, a PP < 40 mmHg is clearly low and reflects a decreased SV, which could be explained either by a decreased preload or a severe systolic dysfunction. In contrast, hypotensive septic patients with maintained PP in general have an impaired vascular tone that may not be corrected by fluid administration alone [10].

Arterial waveform

Continuous beat to beat hemodynamic parameters can be obtained from the shape of the arterial pressure wave. The area under the arterial curve during systole is assumed to be proportional to the stroke volume. This is the basis for several non-invasive continuous cardiac output monitors, that also provide stroke volume variation, an accurate fluid responsiveness test [2].

Even though waveform and arterial notch analysis is more qualitative in nature, and can require certain training, it can be useful as a complementary assessment of the cardiovascular status and allows to identify hemodynamic patterns, as shown in Fig. 1.

Knowledge and application of the different signals provided by invasive arterial pressure monitoring can allow clinicians to understand and interpret hemodynamic derangements in critically ill patients, and eventually guide resuscitation using simple bedside tools.

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Acknowledgements
Arterial line traces have been kindly provided by Mario Romano.

Declarations

Conflicts of interest
None declared.

Publisher's Note

Received: 11 April 2022 Accepted: 22 June 2022
Published online: 08 July 2022

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