Intensive Care Med
DOI 10.1007/s00134-017-4888-8

WHAT'S NEW IN INTENSIVE CARE

The ten principles behind arterial pressure

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Arterial pressure is a fundamental measure of patient's cardiovascular status, but assessment of the values must be interpreted also taking into account its relationship to cardiac output [1].

What is arterial pressure?

Arterial pressure is the result of the interactions between ventricular contraction, the ventricular hydraulic load and the arterial system [1]. Extravascular mechanical forces, such as intra-thoracic and intra-abdominal pressures, acting on the vessels further affect arterial pressure.

Aortic pulse pressure

When the left ventricle (LV) ejects blood into the aorta, the aortic pressure increases to its maximum value, which is the systolic pressure (P_{sys}). During relaxation and subsequent refilling of the LV, the pressure in the aorta decreases to its lowest value, which is the diastolic blood pressure (P_{dias}). The difference between the systolic and diastolic pressures is the pulse pressure. The major determinants of P_{sys} are the pattern and duration of ventricular ejection (stroke volume), the distensibility (compliance) of the arterial vessels, the velocity of the pressure wave in large arteries and the degree of pressure wave reflection in peripheral arteries, the latter being influenced by vasomotor tone [1, 2]. An increase in transmission velocity of both the forward and reflected pressure waves leads to arrival of the reflected wave in the central aorta during systole, thus augmenting pressure [1, 2]. The P_{dias} depends on arterial compliance, which decreases as arterial stiffness increases, as well as on heart rate and the resistance related to the length and diameter of vessels and the distribution of the vascular network (series and parallel circuits). Both P_{sys} and P_{dias} are further influenced by laminar or turbulent flow characteristics and blood viscosity [1]. As the aortic pressure pulse travels away from the aorta to the distributing arteries, P_{sys} rises and P_{dias} falls as a result of reflected waves in the branching vessels and the decreased arterial compliance of the distributing arteries. Consequently, the pulse pressure increases from the aorta to the systemic arteries. Increased stiffness of the central elastic arteries is the major cause of augmented systolic and pulse pressure in aged and hypertensive patients [1, 2]. The amplitude of pulsation progressively decreases in the smaller arteries and arterioles and is minimal in the capillaries because of increased resistance and reduced compliance in smaller vessels [1].

Mean arterial pressure

The mean aortic pressure (MAP) is the average pressure value during the aortic pulse cycle. The relatively low resistance in the aorta and in the distributing arteries means that there is only a small decrease in MAP as the aortic pressure pulse travels away from the aorta to small arteries [1]. MAP can be therefore be used as a reference value along the arterial system to estimate arterial pressure. MAP can be obtained by measuring the area under the pressure curve and dividing this area by the time interval involved. An accurate value can only be obtained by using specific electronic analog or digital techniques, but in common clinical practice formulas are used to calculate MAP.

Arterial pressure and cardiac afterload

The sympathetic nervous system and baroreflex functions play pivotal roles in coupling the left ventricle to the arterial circulation. Sympathetic modulation of afterload allows the left ventricle to generate stroke volume at the

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lowest energy expenditure [1]. Ventricular afterload can be defined as the hydraulic input impedance presented to the ventricle and can be expressed as ascending aortic impedance [1, 3–5] that includes both static (total peripheral resistance) and pulsatile (stiffness and reflection) components [1, 3–5]. Therefore, MAP does not represent the true afterload of the left ventricle. Ideally, central aortic pressure can be considered to be a surrogate of ventricular wall tension and is thus the closest estimate of afterload. Changes in vascular tone, for example those related to sepsis, affect both the amplitude and timing of the reflected waves so that it is difficult to relate peripheral to central pressures in critically ill patients [1, 6].

Pulse pressure variation as a surrogate of volume responsiveness

Intermittent positive-pressure ventilation induces cyclic changes in stroke volume, which in turn leads to cyclic changes in pulse pressure, with increases during inspiration and decreases during expiration. Such pulse pressure variations may be particularly pronounced in preload-dependent conditions and may thus be used to predict the hemodynamic response to a fluid challenge. Notably, neither changes in pulse pressure nor changes in arterial pressure reflect changes in cardiac output during a fluid challenge [7].

Arterial pressure and microvascular perfusion

Modulation of local vascular resistance at the microcirculatory level is the main determinant for blood flow distribution according to metabolic needs [8]. Capillary perfusion depends on the difference between inflow pressure and outflow pressure, and vasodilation is the main mechanism for increasing local blood flow. Hence, a sufficient inflow pressure must be maintained to preserve organ perfusion [8]. The inflow pressure is regulated by the vasomotor tone of arterioles and pre-capillary sphincters along the vascular system and depends on the pressure difference between the central arterial pressure (i.e. MAP) and arterial critical closing pressure (P_{cc}, the pressure threshold that coincides with the stop of organ blood flow) [8, 9]. The outflow pressure is the pressure difference between the mean systemic filling pressure (P_{msf}) and the right atrial pressure (RAP) (Fig. 1). The arterial and venous resistances determine the "vascular waterfall" that contributes to the maintenance of organ perfusion even in low-flow conditions [8–11]. Even if MAP decreases to P_{cc}, a pressure gradient between P_{cc} and P_{msf} is still maintained. The presence of the vascular waterfall explains why changes in RAP minimally affect capillary flow [8–11]. Inflow pressure thresholds (MAP − P_{cc}) and outflow pressure vary between organs and may be affected by interstitial pressure [8–11].

Arterial pressure and organ perfusion during sepsis

In contrast to acute low flow states in which MAP decreases and P_{cc} remains unaffected, the massive loss of smooth muscle tone in sepsis decreases both MAP and P_{cc}. The P_{cc} may thus become equal to the downstream pressure (P_{msf}), rendering the vascular waterfall ineffective [8–12]. When MAP is below the autoregulation threshold and the vascular waterfall is eliminated, tissue perfusion becomes solely dependent on perfusion pressure and thus MAP [8]. If the vascular waterfall is absent, an increase in RAP may further worsen capillary perfusion. Vasopressor agents increase MAP but the effect on P_{cc} remains to be better elucidated [12]. Increasing MAP above the autoregulatory threshold in septic patients with microcirculatory impairment have variable effects on microvascular perfusion.

How to measure arterial pressure

Oscillometry is the most common non-invasive method to measure arterial pressure. However, it does not provide reliable measurements at extreme MAP values. Tonometric and volume clamp devices measure the arterial pressure waveform non-invasively, but their value in extreme conditions may be limited. Arterial catheterization is the gold standard for accurate arterial pressure measurements. Invasive arterial pressure is measured at the radial, brachial and femoral sites, with the radial site being the most common. As the arterial waveform is determined by both incident and reflected waves, different arteries (radial, brachial, femoral) produce different waveforms within the same individual. Invasive arterial pressure measurements require fluid-filled transduction systems. Alterations in the physical characteristics of such systems and irregularities in the conducting tubes (incorrect damping) may affect the accuracy of waveforms obtained.

How to treat arterial hypotension

The treatment goal for vasopressors in hypotension is to increase MAP above the autoregulatory threshold to preserve tissue perfusion. Different vascular beds have different autoregulatory thresholds [8]. Current recommendations suggest targeting a MAP of at least 65 mmHg, with higher MAP cutoff of 80–85 mmHg in patients with chronic hypertension [8]. Nevertheless, higher doses of vasopressors to maintain such elevated pressure thresholds are associated with an increased risk of adverse effects. MAP should be therefore individually targeted to tissue perfusion endpoints, with evaluation of the response to vasopressor agents.

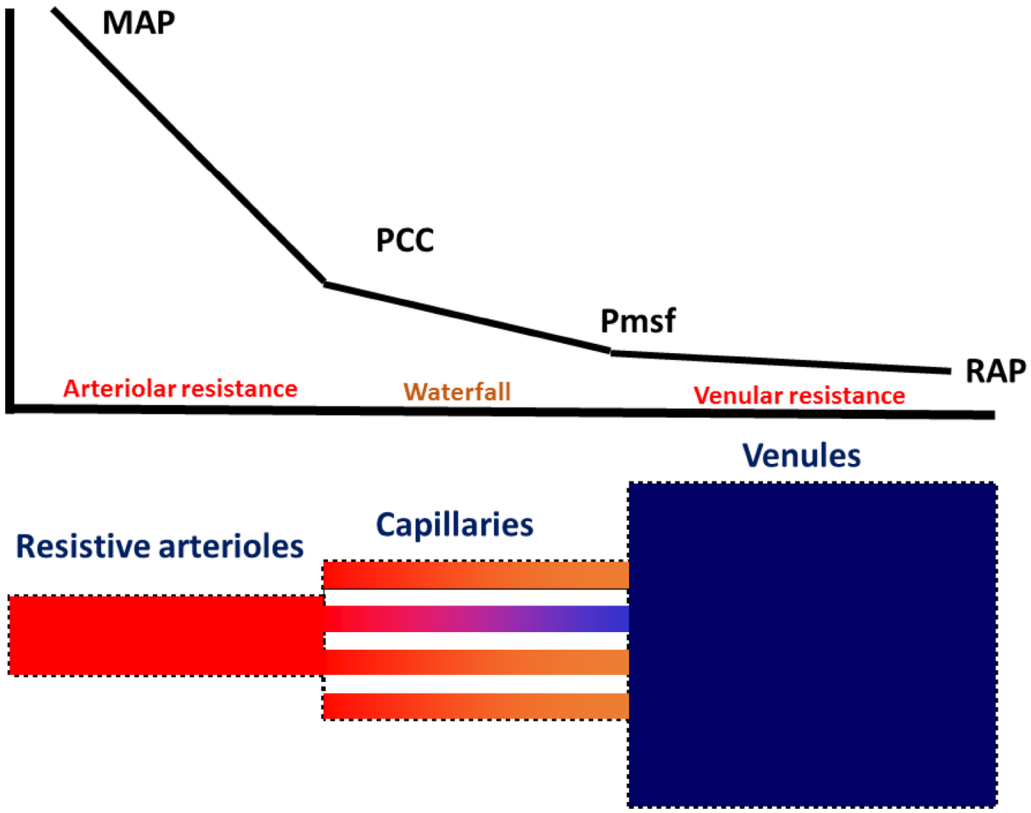

Fig. 1 Schematic representation of the pressure drop along the vascular system. Graphic representation of the pressure drop from the mean arterial pressure (*MAP*) to the critical closing pressure (P_{cc}), and from the mean systemic filling pressure (P_{msf}) to the right atrial pressure (*RAP*). The pressure gradient between P_{cc} and P_{msf} constitutes the "vascular waterfall". The precise location of P_{msf} is not well defined. The presence of a vascular waterfall indicates there are two separate but sequential vascular resistances (arteriolar resistance and venular resistance) which determine the pressure drop, rather than a unique total systemic vascular resistance

Prognostic implications

Hypotension is associated with outcome, especially in septic shock patients. In their study on arterial blood pressure in patients with early sepsis, Dünser et al. reported that an increasing number episodes of MAP of <60 mmHg increased the risk of death by threefold, with a relationship between the total time spent below a MAP level of 60 mmHg and 28-day mortality [13]. Even non-sustained hypotension increases the risk of death during hospitalization, and the more severe the hypotension, the higher the risk of death [14]. Delaying introduction of vasopressor agents is associated with increased mortality. These findings highlight the importance of maintaining MAP within the autoregulatory threshold.

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Compliance with ethical standards

Funding
None.

Conflicts of interest
The author declares that he has no conflict of interest.

Ethical approval
This article does not contain any studies with human participants performed by any of the authors.

Received: 16 May 2017 Accepted: 8 July 2017
Published online: 19 July 2017

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