Reflecting on posture

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See original paper on page 682

Cardiovascular parameters are most often assessed at rest, and in many cases in the supine position. This generally facilitates measurement procedures and obviates the need to account for external and usually complex confounding factors, other than those related to the inherent physiology being investigated. Although the supine resting position may be relevant for some part of circadian living, a large part of human activity is characterized by locomotion and the upright posture. Physiological adaptations to locomotion are studied under controlled conditions and at varying degrees of exercise. However, although primary measures of basic physiological quantities (e.g. stroke volume) can be obtained with some level of confidence at rest in supine position, it becomes extremely difficult to measure them during exercise, and so they are generally derived from other measurable quantities, with the associated errors. Notwithstanding this, the large body of work done so far on the physiological response to exercise has been extremely beneficial in elucidating both underlying and adaptive mechanisms for the function of the circulatory system. On the other hand, assessment of the changes occurring in cardiovascular variables when shifting from supine to standing is the procedure most commonly adopted to explore the response of cardiovascular control mechanisms to gravitational challenges, both when investigating physiological adaptations to the upright posture in healthy individuals and when assessing alterations in cardiovascular regulation in patients with a variety of diseases.

Indeed, studies related to changes in posture can be considered as a subset of exercise studies. That is, whereas conventional exercise studies involve assessment of physiological response to dynamic changes, postural alterations generally involve static changes between supine to active or passive standing, such that measurements are done at different positions, but with no locomotion or movement in any of them. However, these static changes in response to postural shifts involve sensory and neurogenic responses to changes in static pressure of different fluid compartments, due to fluid shifts and gravity effects on fluid columns (such as those in the aortic trunk). In the intact circulation, these effects produce changes in cardiac function due to gravity effects on venous return with the corresponding reaction of autonomic control mechanisms, such as arterial and cardiopulmonary baroreflexes [1]. They also induce changes in the function of large conduit arteries in terms of pressure-dependent effects of wall properties, as well as changes in the function of the peripheral vasculature in terms of alterations in resistance to blood flow.

Because of the inherent pulsatile nature of the cardiac pump and the distributed features of the arterial tree, wave propagation phenomena contribute to the generation of the arterial pressure pulse [2]. These include the speed of wave travel along the arterial wall, determined essentially by the wall stiffness and vessel geometry, and the relative magnitudes of the forward and backward waves. Wall stiffness is dependent, among other factors, on distending pressure, and timing and magnitude of wave reflection depend on pulse wave velocity (and distance to reflecting sites) and on the amount of impedance mismatch at the periphery, relating mainly to peripheral resistance [2]. In physiological and clinical investigations, this wave propagation is assessed noninvasively by measurement of pulse wave velocity (usually in the supine position) and by pulse wave analysis using waveform features to determine relative magnitude of stiffness and effects of wave reflection. Thus, given the physiological effects of postural changes on the intact circulation, the static postural changes from supine to standing would result in significant fluid shifts and gravity-related pressure gradients affecting cardiac contractility, stiffness of large conduit vessels and peripheral resistance, with consequent effects on arterial pulse waveform characteristics.

Postural effects on wave propagation characteristics are being acknowledged in many recent studies, which are conducted at varying degrees of head-up tilt [3,4]. In addition to assessment of changes in reflex control of arterial pressure with age [5,6], such studies are generally aimed at uncovering intrinsic effects of arterial stiffness on orthostatic hypotension [3,7–9].

The study reported by Davis *et al.* [10] in this issue of the Journal offers a further contribution to the discussion in this field, by assessing the changes in peripheral resistance and wave reflection in a group of healthy young (mean age 29 years) and older (mean age 53 years)

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individuals from supine to active standing. Quantities are determined from measurements of continuous pulsatile pressure in the finger and from aortic flow estimated from a vascular model adapted for mean arterial pressure and the individual's age, sex, height and weight. Aortic pressure is obtained from a mathematical transformation of the finger pressure and aortic pressure and flow are used to calculate peripheral resistance and to determine forward and backward waves and augmentation index. From these parameters, a reflection magnitude is calculated.

The study found essentially similar relative changes in both young and older individuals, with an increase in peripheral resistance being associated with a reduction in wave reflection [10]. The authors conclude that the changes in reflected waves between supine to standing are not importantly determined by aging, but appear to be rather related to processes involving predominantly changes in pressure distribution over large arteries and vasoconstriction of the microcirculation.

Compared to studies in the supine position or in arterial models of wave propagation, the findings from this study would seem somewhat paradoxical: an increase in peripheral resistance would be expected to be associated with an increase in wave reflection. However, one plausible explanation offered by the authors is that while the increase in peripheral resistance relates to the resistive component (i.e. the real part) of the impedance of the terminal vascular beds as determined by values of mean pressure and flow, active standing may also change the reactive component (i.e. the imaginary part) and so produce a frequency-dependent phase change. This effect would be manifest as reduced reflection as determined from waveform features of the central aortic pressure.

The study by Davis *et al.* [10] relied on model estimations of aortic flow waveforms, which were also used to estimate stroke volume and, thus, peripheral resistance. This technique of determining stroke volume has been shown to be valid under conditions of orthostatic stress [11]; however, the validation study [11] did not explicitly evaluate the flow waveform. The consistency of the flow waveform is only inferred from the consistency of the stroke volume. This is, of course, only one of many possibilities in which similar area under the curve can be obtained by different wave shapes. And the change in ventricular ejection wave shape would indeed be a plausible effect of standing wherein changes in gravity-related venous return would affect cardiac contractility in accordance with the Starling mechanism. Thus, changes in ejection waveform would affect the aortic waveform, independent of peripheral wave reflection phenomena.

Due to the gravity effect on the blood column in the aortic trunk and lower limbs, it would be expected that the graded increase in distending pressure would produce an increase in regional arterial stiffness resulting in increase in pulse wave velocity with standing. This was not measured by the study of Davis *et al.* [10]; hence, it is not possible to ascertain whether the reduction of wave reflection was also associated with an expected increase in pulse wave velocity as has been found with peripheral resistance. In a previous study by Xu *et al.* [12], an average 21.5% increase in carotid-femoral pulse wave velocity from the supine to the upright position was observed in a group of nine healthy male participants (age 25–37 years) independent of changes in arterial pressure and heart rate. This was associated with a mean increase in the gravity-related pressure of 21.1% at the mid-thoracic level with reference to the position of the femoral artery. For a mean value of supine pulse wave velocity of 6 m/s and an average path length of 50 cm, this gravity-related increase in pulse wave velocity would have the effect of an earlier return of reflected waves of 18 ms. For an unchanged flow waveform, this effect would be manifest as a change in pressure waveform, and an earlier return during systole would be manifest as a possible increase in the augmented pressure component. However, if it is not detected, it may indicate a possible change in the ventricular ejection wave.

Given the complexity of the mechanisms involved in the changes in arterial properties associated with a posture shift from supine to standing, the above-mentioned limitations of the study by Davis *et al.* [10] do not allow to reach a final conclusion on this issue. Indeed, as previously underlined, this study is limited to the magnitude and the effect of changes in total peripheral resistance on wave reflection, with no information on concomitant changes in arterial stiffness through assessment of pulse wave velocity. The latter changes are likely to play an important role, however, in explaining changes in arterial pulse waveform, given both the known increase in sympathetic activity associated with shift to the upright posture, and the recent demonstration that sympathetic nerve activity is related to pulse wave velocity [13]. Moreover, aortic pressure and flow were not measured but were determined from distal, noninvasive pressure measurement. This is another potential limitation, given the peripheral site of blood pressure measurement and its sensitivity to changes in the hydrostatic height difference between finger cuff and the heart level on going from supine to standing posture. The authors minimize this problem based on the results of a previous study, which demonstrated that from the supine to standing position the bias for systolic finger pressure did not change significantly [14]. They also claim that the reconstruction of aortic pressure from finger pressure using a finger to aortic transfer function provides reliable results [15], based in particular on the data collected by Sharman *et al.* during exercise [16] and on a previous validation study [17]. Finally, the effect of heart rate on augmentation index was accounted for by using a relation

that was determined in supine participants [18], and no information is available on whether postural stress modifies such relationship.

In conclusion, although the study by Davis *et al*. provides a stimulating contribution to the discussion in this field, its results cannot be taken as the last word on this issue, given that a number of important variables were not measured in the conditions of this study and because of the above-mentioned potential methodological limitations. The findings by Davis *et al*. should, thus, be considered as important background observations for future studies on the effects of standing on arterial pulse waveform, which should be based on a further refined methodological approach.

Indeed, as concluded by the authors, the occurrence of orthostatic changes in pulse wave characteristics is an issue that deserves additional investigation, given that humans spend a large part of their time in either the seated or standing postures, that is, under conditions that influence wave reflection.

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