Impact of body position on central and peripheral hemodynamic contributions to movement-induced hyperemia: implications for rehabilitative medicine

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1Geriatric Research, Education, and Clinical Center, George E. Whalen Veterans Affairs Medical Center; 2Division of Geriatrics, Department of Medicine; and 3Department of Exercise and Sport Science, University of Utah, Salt Lake City, Utah; and 4Department of Neurological, Neuropsychological, Morphological, and Movement Sciences, University of Verona, Verona, Italy

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Trinity JD, McDaniel J, Venturelli M, Fjeldstad AS, Ives SJ, Witman MA, Barrett-O’Keefe Z, Amann M, Wray DW, Richardson RS. Impact of body position on central and peripheral hemodynamic contributions to movement-induced hyperemia: implications for rehabilitative medicine. Am J Physiol Heart Circ Physiol 300: H1885–H1891, 2011. First published February 25, 2011; doi:10.1152/ajpheart.00038.2011.—This study used alterations in body position to identify differences in hemodynamic responses to passive exercise. Central and peripheral hemodynamics were noninvasively measured during 2 min of passive knee extension in 14 subjects, whereas perfusion pressure (PP) was directly measured in a subset of 6 subjects. Movement-induced increases in leg blood flow (LBF) and leg vascular conductance (LVC) were more than twofold greater in the upright compared with supine positions (LBF, supine: 462 ± 6, and upright: 1,084 ± 159 ml/min, P < 0.001; and LVC, supine: 5.3 ± 1.2, and upright: 11.8 ± 2.8 ml·min⁻¹·mmHg⁻¹, P < 0.002). The change in heart rate (HR) from baseline to peak was not different between positions (supine: 8 ± 1, and upright: 10 ± 1 beats/min, P = 0.22); however, the elevated HR was maintained for a longer duration when upright. Stroke volume contributed to the increase in cardiac output (CO) during the upright movement only. CO increased in both positions; however, the magnitude and duration of the CO response were greater in the upright position. Mean arterial pressure and PP were higher at baseline and throughout passive movement when upright. Thus exaggerated central hemodynamic responses characterized by an increase in stroke volume and a sustained HR response combined to yield a greater increase in CO during upright movement. This greater central response coupled with the increased PP and LVC explains the twofold greater and more sustained increase in movement-induced hyperemia in the upright compared with supine position and has clinical implications for rehabilitative medicine.

Passive movement, unlike voluntary exercise, occurs without a marked increase in skeletal muscle metabolism (15, 36). By the removal of the increase in metabolism associated with exercise, the independent effect of movement on central and peripheral hemodynamic mechanisms governing exercise hyperemia can be identified and studied. This approach has been used by our laboratory to identify that movement-induced hyperemia is transient in nature (22, 40), that central hemodynamics and afferent feedback are integral parts of the hyperemic response (14, 36), and that aging results in a reduction in movement-induced hyperemia that is independent of metabolism (23). At the onset of passive movement, the mechanical deformation of the vascular bed coupled with stimulation of group III afferent fibers initiates a cascade of events resulting in both peripheral vasodilation and a heart rate (HR)-driven increase in cardiac output (CO) and a subsequent increase in leg blood flow (LBF) (36). With the use of high temporal resolution data acquisition (i.e., second by second), this combination of both peripheral and central responses has proven to be an interesting and robust phenomenon.

Understanding the hemodynamic response to passive movement may be useful for clinicians involved in rehabilitative medicine, as individuals with severe peripheral arterial disease, lower limb or spinal cord injuries, and the frail elderly could benefit from the response to passive movement. However, discrepancies exist regarding the magnitude and duration of movement-induced hyperemia during passive exercise. Alterations in body position may be responsible for these discrepancies since we have reported a transient increase in LBF during supine passive movement (14, 22, 23), whereas others have reported that the elevated LBF was sustained for the duration of passive movement when performed in the upright seated position (15, 16). This concept is supported by central and peripheral hemodynamic differences due to manipulation in body position as rest and during exercise (2, 7, 10, 20, 25). Identifying the potentially body position-dependent mechanisms that maximize the increase in LBF may be of utmost importance for the aforementioned individuals, since passive movement performed for an extended period of time or repeatedly performed over the course of weeks appears to be an angiogenic stimulus (15, 16). This adaptation, characterized by enhanced vascular endothelial growth factor, endothelial cell proliferation, and capillary growth (15, 16), is likely highly dependent on the magnitude and duration of the stimulus (i.e., LBF).

The purpose of this study was twofold: 1) use alterations in body position to identify differences in the magnitude and duration of central and peripheral hemodynamic responses to passive leg movement and 2) determine whether the magnitude of the central (HR, SV, and CO) response dictates the peripheral response. We hypothesized that the upright position would result in a greater hyperemic response to passive movement and that the magnitude of the CO response would be greater in the upright compared with the supine position.
METHODS

Subjects

Fourteen healthy subjects volunteered to participate in this research study (men = 11, and women = 3; age, 34.6 ± 2.8 yr; body mass, 72.6 ± 3.3 kg; and stature, 176.7 ± 3.3 m). Subjects were not taking any prescription medications and were free of overt cardiovascular disease, as indicated by health history and physical exam. Written informed consent was obtained from each participant, and all procedures were approved by the Institutional Review Board of both the University of Utah and the Veterans Affairs Medical Center of Salt Lake City, Utah. The study conformed with the Declaration of Helsinki.

Experimental protocol. Before the experiment all subjects reported to the laboratory for a familiarization trial. During this session, passive knee extension and Doppler ultrasound imaging of the femoral artery were performed to both familiarize the subjects and ensure that acceptable images could be obtained at rest and during movement. Eight of the subjects participated in the noninvasive trials only, whereas the remaining six had their right femoral artery and vein catheterized (18-gauge central venous catheter, Arrow International, Reading, PA) using the Seldinger technique to measure arterial, venous, and perfusion pressure (PP) at the level of the limb. Upon arrival at the laboratory, body mass and height were recorded and the subject was positioned in either the supine or upright seated position.

Subjects performing only the noninvasive trials performed upright seated passive movement followed by supine passive movement, whereas the remaining six subjects performed supine passive movement followed by upright seated passive movement. After the subject was situated in their respective position for at least 20 min and instrumentation was complete, baseline hemodynamic measurements were made. Before the commencement of passive movement, stable baseline central and peripheral hemodynamic measures were attained. Single leg passive movement was achieved by a member of the research team moving the leg through 90° range of motion at 1 Hz. The starting position of the leg was the full extension at the knee (i.e., 180°) and the first movement served to passively flex the knee (i.e., move to 90° knee joint angle). Real-time feedback was provided to the researcher by a digital display of cadence. Before the start and throughout the protocol, subjects were encouraged to remain passive and resist the urge to assist with leg movement. To avoid the startle reflex and active resistance to the passive movement, subjects were made aware that passive movement would take place in ~1 min, but to minimize the chance of an anticipatory response, they were not informed of exactly when this movement would initiate. Passive movement was performed for 2 min.

Measurements

Femoral blood flow. Measurement of femoral arterial blood velocity and vessel diameter were performed in the passively moved leg distal to the inguinal ligament and proximal to the deep and superficial femoral bifurcation with a Logic 7 ultrasound system (General Electric Medical Systems, Milwaukee, WI). The ultrasound system was equipped with a linear transducer operating at an imaging frequency of 10 MHz. Vessel diameter was determined at a perpendicular angle along the central axis of the scanned area. Blood velocity was measured using the same transducer with a frequency of 5 MHz. All blood velocity measurements were obtained with the probe appropriately positioned to maintain an insonation angle of 60° or less. The sample volume was maximized according to vessel size and was centered within the vessel. Arterial diameter was measured, and mean velocity (V̄mean) [angle corrected, and intensity weighted area under the curve (AUC)] was automatically calculated (Logic 7). With the use of arterial diameter and V̄mean, blood flow in the femoral artery was calculated as blood flow = V̄mean²(vessel diameter/2)² × 60, where blood flow is in milliliters per minute. Leg vascular conduc-
tance (LVC) was calculated as LBF divided by mean arterial pressure (MAP).

Central hemodynamic variables. HR, SV, CO, and MAP were determined with a finometer (Finapres Medical Systems, Amsterdam, The Netherlands) positioned at heart level. SV was calculated from beat-by-beat pressure waveforms assessed by photoplethysmography using the Modelflow method (Beatscope, version 1.1; Finapres Medical Systems), which in combination with HR has been documented to accurately estimate CO during a variety of experimental protocols (1, 4, 5, 34, 39). In the subset of six subjects, intravascular systolic and diastolic arterial and mean venous pressures (MVP) were determined from in-line pressure transducers (Baxter, Deerfield, IL) placed at the level of the catheters. MAP was calculated as diastolic + 1/3(systolic − diastolic). PP was calculated as arterial minus venous blood pressure.

Data acquisition. Throughout each protocol HR, SV, CO, MAP, MVP, and ECG signals underwent analog-to-digital conversion and were simultaneously acquired (200 Hz) using a data acquisition system (AcqKnowledge; Biopac Systems, Goleta, CA).

Data and Statistical Analysis

The data acquisition software allowed second-by-second analyses of HR, SV, CO, MAP, and MVP. All analyses were performed using a 5-s moving average. The second-by-second velocities were analyzed on the ultrasound system (GE Logic 7) for the first 60 s of movement, and 12-s averages were assessed from 60 to 120 s of movement. Two-way repeated-measures ANOVA was used to determine significant differences between upright seated and supine conditions. When a significant main effect (interaction of body position by time) was observed, further analysis was performed to determine whether a significant change over time occurred within a treatment. Cumulative AUC [or area above the curve (AAC)] was calculated as the summed second-by-second response of a given variable during the first 60 s of passive movement and used to identify how differences over time were affected by the treatment (23). Significance was set at an α-level of 0.05, and data are presented as means ± SE.

RESULTS

Central Responses to Passive Movement

Body position had a significant main effect on HR, as HR was higher at rest and throughout movement in the upright compared with supine position (P = 0.008) (Fig. 1). Baseline HR was elevated in the upright (62 ± 4 beats/min) compared with the supine position (56 ± 3 beats/min, P = 0.03); however, the absolute change in HR from baseline to peak was not different between positions (supine, 8 ± 1 vs. 10 ± 1 beats/min, P = 0.22), indicating that the degree of tachycardia induced by passive movement was similar across positions. Despite a similar absolute change in HR due to passive movement, the cumulative HR AUC was greater during the upright compared with supine position (P = 0.001), demonstrating that the increase in HR was maintained for longer in the upright seated position (Fig. 1).

The interaction of body position and time revealed a significant difference for SV (P < 0.001) (Fig. 1). Baseline SV was numerically higher during the supine (100 ± 6 ml/beat) than the upright (91 ± 7 ml/beat) position; however, this difference was not significant (P = 0.15). SV increased over time during passive movement in the upright position and reached a peak of 104 ± 9 ml/beat and remained unchanged in the supine position. The cumulative SV AUC response was greater in the upright compared with supine position (P = 0.003) (Fig. 1).
Fig. 1. Central and peripheral hemodynamic responses to 2 min of passive limb movement in the supine and upright positions. Values are means ± SE for heart rate [in beats/min (bpm)], stroke volume, cardiac output, mean arterial pressure, and leg blood flow. The left column represents the average temporal responses of all subjects (n = 14). The right column represents the cumulative area above (AAC) or below (AUC) the curve calculated as the summed second-by-second response of these variables for the first 60 s of passive movement. *P < 0.05, significant difference between the supine and upright positions. One minute of baseline data was collected before passive movement. The transition from baseline to passive movement occurred at time 0 on the x-axis.
The contrasting baseline offsets in HR and SV caused by body position resulted in a baseline CO that was not different between conditions (supine, 5.6 ± 0.4 vs. upright, 5.7 ± 0.7 l/min, \( P = 0.8 \)) (Fig. 1). During passive movement, the increase in CO was greater in the upright position as indicated by the greater cumulative AUC (\( P < 0.001 \)) and the significant interaction of body position and time (\( P < 0.001 \)). During upright passive movement, CO remained elevated above baseline throughout the second minute of movement, whereas CO returned to baseline values following the transient increase during the first minute of movement in the supine position (Fig. 1).

Overall, there was a main effect of body position on MAP (\( P = 0.01 \)) (Fig. 1). Baseline MAP was lower in the supine (90 ± 2 mmHg) compared with upright (99 ± 3 mmHg, \( P = 0.01 \)) position. Throughout passive movement, MAP was higher in the upright position compared with supine position despite a transient drop in MAP during the first minute of movement. The reduction in MAP in the upright compared with the supine position is clearly documented by comparing the cumulative AAC between the positions (Fig. 1). MAP, as measured by the arterial catheter in 6 of the 14 subjects, paralleled the changes observed using photoplethysmography (Fig. 2). In this subset of subjects, the transient reduction in MAP was significant during both upright and supine passive movement. MVP before passive movement was higher in the upright (20 ± 2 mmHg) than the supine position (8 ± 2 mmHg, \( P = 0.01 \)) (Fig. 2). During supine passive movement MVP displayed an abrupt transient increase of 0.8 ± 0.2 mmHg and then returned to baseline values. During upright movement, MVP displayed a similar sharp increase at the onset of movement yet continued to increase for the remainder of the passive movement reaching a maximal change of 3.0 ± 0.5 mmHg.

PP, measured directly in the passively moved limb, exhibited a significant body position by time interaction (\( P < 0.001 \)) (Fig. 2). Baseline PP tended to be higher in the upright position (supine, 88 ± 5 vs. upright, 101 ± 5 mmHg, \( P = 0.06 \)), and this elevated PP was greater throughout passive movement in the upright position despite a transient reduction during the first minute of movement. The greater reduction in PP in the upright position is evident as the cumulative AAC is greater in the upright compared with supine position (data not shown, body position by time, \( P < 0.001 \)).

**Peripheral Responses to Passive Movement**

Before passive movement, baseline LBF was not statistically different between positions (supine, 270 ± 44 vs. upright, 358 ± 73 ml/min, \( P = 0.14 \)) (Fig. 1). The movement-induced increase in LBF was more than twofold greater in the upright (1,084 ± 159 ml/min) compared with the supine (462 ± 62 ml/min, \( P < 0.001 \)) position. Analysis of the AUC of the LBF response revealed a similar finding in that the LBF AUC in the upright position was greater than that in the supine position (position \( \times \) time, \( P < 0.001 \)). LBF remained elevated above baseline at the end of the 2-min period of passive movement in the upright (524 ± 107 ml/min, \( P = 0.02 \)) but not the supine (299 ± 57 ml/min, \( P = 0.43 \)) position (Fig. 1).

LVC was similar between positions at rest (supine, 3.0 ± 0.7 vs. upright, 3.5 ± 0.9 ml-min\(^{-1}\)mmHg\(^{-1}\), \( P = 0.35 \)) (Fig. 2). As with LBF, the increase in LVC was more than twofold greater in the upright (11.8 ± 2.8 ml-min\(^{-1}\)mmHg\(^{-1}\) ) compared with the supine (5.3 ± 1.2 ml-min\(^{-1}\)mmHg\(^{-1}\), \( P =

Fig. 2. Leg vascular conductance and peripheral pressure measurements during 2 min of passive limb movement in the supine and upright positions. Values are means ± SE for leg vascular conductance, mean arterial pressure, mean venous pressure, and perfusion pressure. Invasive measures of pressure were determined in 6 subjects. One minute of baseline data were collected before passive movement. The transition from baseline to passive movement occurred at time 0 on the x-axis.
0.002) position, LVC remained elevated above baseline at the end of the 2-min period of passive movement in the upright (5.2 ± 1.0 ml·min⁻¹·mmHg⁻¹, P = 0.017) but not the supine (3.3 ± 0.7 ml·min⁻¹·mmHg⁻¹, P = 0.48) position (Fig. 2).

**DISCUSSION**

We have identified that body position plays an integral role in the magnitude and duration of the peripheral and central hemodynamic responses to passive movement. During limb movement in the upright seated position, LBF and LVC were more than twofold greater and, although decaying significantly, were less transient in nature than that observed in the supine position. Given that the passive movement itself, and therefore the initial stimulus for mechanical vasodilation, was identical between positions, the higher PP due to the increased hydrostatic column in the upright position likely played a key role in augmenting LBF and LVC when upright. Concomitant to the enhanced peripheral hyperemic and LVC response, the essential central hemodynamic reaction was also greater in the upright position, characterized by a progressive increase in SV and a sustained HR response that combined to yield a greater increase in CO. Therefore, at the onset of movement, elevated hyperemia and its sustainability in the upright seated position appears to be driven by two distinct, yet intertwined, mechanisms: peripherally, an elevated PP initiates an increase in LBF and LVC, which is likely magnified and sustained by increased peripheral vasodilation, whereas centrally, CO is augmented by an increase in HR and SV, potentially explained by the baroreflex and muscle pump, respectively. The recognition of these central and peripheral hemodynamic differences between upright and supine positions has implications for the clinical application of passive movement in rehabilitative medicine.

**Reconciling Differences in the Hyperemic Response to Passive Movement**

Passive movement serves as an experimental model for the systematic analysis of the mechanisms governing exercise hyperemia without the confounding factors of skeletal muscle metabolism and central command typically associated with voluntary exercise (11, 15, 36). Hyperemia is a robust consequence of passive movement, and the temporal (second by second) examination of central and peripheral hemodynamic responses reveals that limb movement triggers a series of events, largely independent of metabolism and central command (22, 36). Despite this reductionist approach, discrepancies exist regarding the magnitude and duration of the hyperemic response to passive movement (15, 16, 22, 23, 36, 40). Based on the current findings, the discrepancies appear to be primarily related to body position and secondarily due to the temporal resolution used to determine the time course of the hemodynamic changes. Indeed, in the supine position, all hemodynamic measures returned to baseline by the end of passive movement (Fig. 1). However, in the upright position, SV, CO, LBF, and LVC were maintained above baseline values for the duration of the passive movement (Figs. 1 and 2). Therefore, it is our contention that studies which have not documented a significant increase in LBF or contribution of CO to the hyperemic response simply did not have adequate time resolution for the measurement of LBF and CO as the first measurements were made several minutes after the onset of passive movement (11, 24, 35). With such high time resolution as used in this study and our previous investigations (14, 22, 23, 36, 40), it is clear that the peripheral hyperemic response is closely linked to the central hemodynamic response and that adequate time resolution must be employed to reveal this association.

**Contributions of Peripheral and Central Hemodynamics to the Hyperemic Response**

In general, understanding the series of events involved in limb movement-induced hyperemia may provide insight into how and why the response is amplified in the upright compared with supine position. At the onset of movement, group III muscle mechanoreceptors feedback to the cardiovascular control center to increase HR (9) and CO, which contribute to the increase in LBF (Fig. 1) (21, 30, 36). Blocking this afferent signal attenuates the HR and CO response to passive movement, resulting in a reduction in hyperemia (36). In the present study, the magnitude of cardioacceleration predominantly due to group III mechanoreceptors in this passive model was similar between conditions, suggesting that afferent feedback was not altered. In conjunction with the peripherally stimulated increase in HR, limb movement results in vessel deformation, likely leading to increased LBF flow via mechanically induced vasodilation (3, 18). Although a similar mechanically induced dilatory stimulus occurs in the upright position, the net driving force for blood through the limb (27) is elevated in proportion to the greater hydrostatic column (greater PP). This likely magnifies the subsequent hyperemia via a cascading set of vasodilatory events, such as increased shear stress and nitric oxide bioavailability, beyond what might be expected due solely to the moderate increase in PP.

Following the initial movement-induced afferent feedback and mechanical vasodilation, the central hemodynamic differences between body positions become apparent. In the upright position, SV begins to increase steadily in the face of a transient drop in MAP (Fig. 1), presumably because of the translocation of blood from the periphery to the heart (muscle pump) and to a reduction in afterload (8). This translocation helps to facilitate the continued increase in CO, which remains elevated for the duration of the passive movement. The reduction in MAP may act to stimulate the baroreceptor reflex leading to the sustained increase in HR, further contributing the augmented CO response. In contrast, SV and MAP remain unchanged in the supine position. Therefore, CO is likely driven solely by the transient increase in HR because of afferent feedback (Fig. 1) since the contribution of the muscle pump is reduced and the baroreflex is not invoked while supine. The lack of change in SV in the supine position can be explained by a greater central blood volume, central venous pressure, and left ventricular end-diastolic volume (2, 7, 10, 25).

During the period of passive movement in which LBF was elevated during both the upright and supine positions, a transient reduction in MAP and PP occurred (Fig. 2). Theoretically, with all other variables held constant, these reductions in MAP and PP should act to reduce LBF. However, mechanical vasodilation preceding additive LBF-mediated dilation appears to outstrip the drive to raise pressure that would be expected to
occur as a result of the increase in CO (22, 23, 36). A similar increase in LBF occurring concomitantly with a reduction in MAP has been previously demonstrated during ATP infusion (11). Despite the transient reductions in MAP and PP in the present study, these variables were always greater in the upright compared supine position, certainly contributing to the increased LBF in this position. Additionally, LVC mirrored the twofold greater increase in LBF, indicating enhanced vasodilation in the upright position contributed to the superior hyperemia.

In support of the integration of the central and peripheral hemodynamic changes is the finding that, on average, in both upright and supine positions, the increase in LBF (supine, 1.0 ± 0.2; and upright, 0.4 ± 0.1 l/min) is nearly fully matched by the increase in CO (supine, 1.1 ± 0.3; and upright, 0.5 ± 0.2 l/min). While the increase in the ratio of CO to LBF is not a perfect 1:1 ratio, perfusion of other tissues such as the unmoved limb (22), not accounted for with the current methods, may account for these small 0.1 to 0.2 l/min discrepancies.

Body Position and the Contribution of the Muscle Pump to Cardiovascular Control

The existence and contribution of the muscle pump during exercise are controversial since arguments have been made both in favor (19, 26, 37) and against (6, 13) such a phenomenon. The muscle pump is thought to play an important role in the regulation of central venous pressure, end-diastolic volume, and SV (12, 28, 31, 33). The contribution of the muscle pump may help explain the sustained increases in SV, CO, LBF, and LVC documented in the upright position in our passive model (Fig. 1). Indeed, it has been argued that CO could not be maintained at an increased level during exercise without the translocation of blood from the peripheral vasculature to the heart by an increase in venous return that transiently exceeds CO (12, 28, 29, 33). An abrupt, albeit slight, increase in MVP occurred during both supine and upright movement; however, in the upright position, this increase in MVP was maintained for the duration of movement, whereas in the supine position, it quickly dissipated (Fig. 2). Radegran and Saltin (26) report a similar increase in MVP as well as an increase in pressure during the transition from rest to passive movement, and they too argue that these changes are evidence of the muscle pump. In addition to the muscle pump adding to the increase SV in the upright position, the distribution of CO to stiff regional circulations such as skeletal muscle may also have helped preserve cardiac filling pressure (11, 32, 41).

These increases in downstream venous pressure (26) indicate that passive movement appears to facilitate the movement of blood across the limb and may eventually contribute to increasing venous return, SV, and CO. Given that arterial inflow and the PP gradient are higher at rest and during movement in the upright position, the contribution of the muscle pump would be expected to be more pronounced in this position since the greater hydrostatic column would facilitate an increase in the driving pressure for the movement of blood across the vascular bed (27). Conversely, under supine conditions in which the hyperemic response is attenuated relative to the upright position and the PP gradient is reduced, the venous circulatory system may be able to accept this increase in blood flow without a sustained rise in pressure, thereby limiting or perhaps removing the potential role of the muscle pump. It appears that in the current study, the maintenance of SV, presumably because of the preservation of central venous pressure and end-diastolic volume, was far less challenging in the supine position because of the small limb movement-induced hyperemia.

Clinical Implications for the Application of Passive Movement

In addition to the mechanistic insight into exercise-induced hyperemia acquired from the passive movement model, clinically relevant information regarding the capacity to invoke hyperemia in populations in which the voluntary limb movement is reduced (frail elderly, severe peripheral artery disease, orthopedic injury) or not possible (spinal cord injury) may also be gained. In such populations vascular health is reduced and appears to deteriorate over time since the positive benefits of physical activity and the associated shear stress are absent (17, 38). Hellsten et al. (15) and Hoier et al. (16) have documented that acute and chronic passive exercise training in healthy able-bodied individuals resulted in an angiogenic stimulus characterized by enhanced vascular endothelial growth factor, endothelial cell proliferation, and capillary growth. Passive movement in these investigations (15, 16) was routinely performed for 90 consecutive minutes, a protocol, which based on our current observation of large transient increases in LBF, may not be the most efficient approach to maximize the hyperemic response. Although these investigations performed the passive movement in a upright seated position that does afford a greater and more prolonged hyperemic response (Figs. 1 and 2), a more efficient countermeasure to retard the progressive reduction in vascular health may be to perform intermittent passive exercise (i.e., a series of 1- to 2-min bouts separated by a brief period of rest). Further research is required to determine the efficacy of such an “interval” approach and the effect of passive movement in individuals that are unable to perform voluntary exercise in terms of this potential angiogenic stimulus.

Conclusions

The magnitude and duration of both central and peripheral hemodynamic factors involved in movement-induced hyperemia are altered, dependent on body position. Specifically, a twofold larger increase in LVC, coupled with prolonged increases in HR and SV, yields a greater CO response, which contributes to the elevated and more sustained hyperemia in the upright compared with supine position. Additionally, elevated MAP and PP, as well as a potential role of the muscle pump in the upright position, further explain the observed differences in movement-induced hyperemia. The recognition of these hemodynamic differences between upright and supine positions has implications for the clinical application of passive movement in rehabilitative medicine.

DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the author(s).

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