Hemodynamic consequences of rapid changes in posture in humans

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The aims of the present study were to characterize the hemodynamic consequences of brief HDT on subsequent HUT and to test the hypothesis that an elevation in leg vascular conductance induced by $-G_x$ stress contributes to the exaggerated fall in ELAP. Young healthy subjects ($n = 3$ men and $4$ women) were subjected to $30$ s of $0^\circ$ HUT from a horizontal position and to $30$ s of $30^\circ$ HUT when HUT was immediately preceded by $20$ s of $-15^\circ$ HDT. Four bouts of HDT-HUT were alternated between five bouts of HUT in a counterbalanced design to minimize possible time effects of repeated exposure to gravitational stress. One minute was allowed for recovery between tilts. Brief exposure to HDT elicited an exaggerated fall in ELAP during the first seconds of the subsequent HUT ($-17.9 \pm 1.4$ mmHg) compared with HUT alone ($-12.4 \pm 1.2$ mmHg, $P < 0.05$) despite a greater rise in stroke volume (Doppler ultrasound) and cardiac output over this brief time period in the HDT-HUT trials compared with the HUT trials (thereafter stroke volume fell under both conditions). The greater fall in ELAP was associated with an exaggerated increase in leg blood flow (femoral artery Doppler ultrasound) and was therefore largely (70%) attributable to an exaggerated rise in estimated leg vascular conductance, confirming our hypothesis. Thus brief exposure to $-G_x$ stress leads to an exaggerated fall in ELAP during subsequent HUT, owing to an exaggerated increase in estimated leg vascular conductance.

The reduction in $G_z$ tolerance elicited by push-pull gravitational stress is associated with an exaggerated reduction in eye-level blood pressure in response to $+G_x$ (2, 10, 11, 14, 15, 16, 26, 27), and this hypotension is graded to the magnitude of the “push” stimulus (14). The push-pull effect has been demonstrated in human subjects (1, 2, 3), as well as in conscious dogs (26) and both conscious (27) and anesthetized (14, 15, 16, 27) rats. The axis of rotation appears to be an important factor in human subjects (3) but not in rats (15), and sex does not appear to be a factor in rats (15). A push-pull effect is observed following autonomic blockade in dogs (26), indicating that the regional hydrostatic component of blood pressure likely elicits local vasomotor responses (4, 17, 18, 21) and/or a venous emptying effect in the lower body that could contribute to the push-pull effect. A push-pull effect is not seen in rats following autonomic blockade likely because the changes in regional pressure that occur in rats when they are tilted may simply be too small to elicit functionally important alterations in myogenic stimuli or regional venous pressures. Although the mechanism(s) responsible for the push-pull effect are incompletely understood, it has been speculated that the arterial baroreflexes play a role (3, 5, 10, 14, 15, 16, 27). Support for this hypothesis stems from the observation that peripheral vasoconstriction to head-up tilt is impaired by prior head-down tilt (11). Further support for this hypothesis stems from the observation that inhibition of autonomic ganglionic neurotransmission eliminates the push-pull effect in rats (16) and that the magnitude of the push-pull effect is coupled to the magnitude of the carotid hypertension that occurs during the “push” phase of push-pull gravitational stress (14).

The aims of the present study were to characterize the hemodynamic consequences of push-pull gravitational stress in conscious humans and to test the hypothesis that an exaggerated rise in leg vascular conductance induced by $-G_x$ stress contributes to the exaggerated fall in eye-level arterial pressure during head-up tilt. The rationale for selecting the legs as a probable contributor to the push-pull effect is that the expected changes in regional pressure at this body location, and the local vascular responses to these pressure changes, are expected to work together in a manner to lead to an exaggerated rise in leg blood flow and conductance in push-pull trials compared with control trials. For example, the fall in lower body arterial pressure that occurs during head-down tilt is expected to elicit myogenic relaxation; e.g., even relatively brief (10 s) “unloading” of rat hindlimb arterioles induces a nifedipine-sensitive reactive hyperemia presumably of myogenic origin (25). If such vasodilation persists early on during head-up tilt, an exaggerated rise in conductance would result. Also, head-down...
tilt reduces lower body venous blood pressure, and this could contribute to the push-pull effect in two ways. First, head-down tilt will work to unload the venous vasculature, and this is expected to induce local arteriolar vasodilation by the venoarteriolar mechanism. Again, if such vasodilation persists early on during head-up tilt, an exaggerated rise in conductance would result. Second, the reduction in pressure in the small veins in the lower body (the effective back pressure governing arterial inflow to the lower body) induced by head-down tilt is expected to persist early on during the subsequent head-up tilt, owing to the venous valves. Thus, compared with control trials, there would be a relatively greater arterial-venous pressure gradient driving flow into the lower body during the head-up tilt phase of push-pull gravitational stress, and this could lead to an increase in arterial inflow to the lower body even in the absence of locally produced changes in vessel diameter. This greater pressure gradient would persist until the veins are refilled. In essence, we propose that head-down tilt exerts a muscle pump-like venous emptying effect, which is known to increase the local arterial-venous pressure gradient through dependent tissues during exercise (8, 9). In this setting, if vascular conductance is calculated on the basis of heart-level arterial and venous pressure, the local rise in the arterial-venous pressure gradient induced by the emptying of the veins manifests as a rise in “virtual vascular conductance” (28).

METHODS

Subjects. Seven volunteers (3 men and 4 women) between the ages of 19 and 33 yr, ranging in height from 167 to 198 cm and body mass from 57 to 75 kg, were studied. All were healthy students, nonsmokers, and in good physical shape, with none being highly trained. None were taking any medications or had any signs of cardiovascular disease. Written informed consent was obtained from all participants, and the study was approved by the regional ethics committee.

Experimental design. Subjects were lightly dressed, and the ambient temperature was kept between 20 and 24°C. To minimize cardiovascular variations induced by digestion, all experiments were run after at least 3 h after a light meal (34). Subjects were familiarized with the test situations in several pilot experiments, and none reported any discomfort during tilting. During a period of 2–3 wk, two to three 1-h sessions consisting of one to two experimental runs were held with each subject. Subjects rested supine on the table for 20–30 min before data were collected.

Experiments were carried out using a manually driven tilt-table bed. To minimize any muscular activity, the subject was stabilized supine (+1 Gv) on the tilt-table bed by a vacuum mattress preformed to the body. Control gravitational stress consisted of rotating the table and thus the subject about the pitch (v) axis from supine to a 30° (0.50 Gv) head-up position in <2 s. This position was maintained for 30 s, and the subject was then tilted back to a supine position in <2 s. Push-pull gravitational stress consisted of 30 s of 30° head-up tilt immediately preceded by 20 s of −15° (0.26 G) head-down tilt. Control and push-pull bouts were alternated in a counterbalanced design to minimize the potential time effects of repeated gravitational stress, and 1 min was allowed between tilts for recovery. Between tilting sessions, subjects rested on the tilt table for ~10 min. This experimental protocol is illustrated in Fig. 1.

Cardiovascular variables were continuously recorded during the 16 min it took to complete the protocol. Great care was taken to ensure that the fingers of the left hand, where arterial pressure was recorded, stayed exactly at the level of the right atrium (at the 4th intercostal space, 5 cm below the sternal angle) throughout the tilting procedures.

Instrumentation. Beat-to-beat stroke volume was recorded by using an ultrasound Doppler velocimeter (model SD-100, GE Vingmed Ultrasound, Horten, Norway) was operated in pulsed mode at 2 MHz with a handheld transducer. The ultrasound beam was directed from the suprasternal notch toward the aortic root. The sample volume range was adjusted so that measurements were made 1–2 cm above the aortic valve. The sample volume was positioned centrally in the aorta by searching for the highest obtainable velocity signal. An angle of 20° between the directions of the sound beam and the bloodstream was assumed in the calculations. To remove vessel wall and valve motion artifacts, together with any recorded diastolic movement of blood, the built-in high-pass filter in the SD-100 was set to remove signals originating from velocities <0.3 m/s. The output of the SD-100 maximal velocity estimator and a three-lead surface electrocardiogram were on-line interfaced to a recording computer running a dedicated data collection and analysis program (program for real-time data acquisition, written by Morten Eriksen, Oslo, Norway).

In a separate session, the diameter of the rigid aortic ring was determined by parasternal sector-scanner imaging (model CFM-750, GE Vingmed Ultrasound). On the assumption that the orifice was circular, this diameter was used to calculate the area of the aortic valvular orifice. Stroke volume was calculated by multiplying the value obtained by numerical integration of the recorded instantaneous mean velocity during each R–R interval by the area of the orifice. The calculation is based on the assumption that the velocity profile in the aortic valvular orifice is rectangulart and that this velocity is conserved as the central maximal velocity of a jet 3–4 cm downstream (7).

The ultrasound Doppler transducer was handheld in the suprasternal notch by one of the authors (KT), who sat beside the subject and followed the movement of the tilt table during tilt up and tilt down. Blood flow velocity in the ascending aorta was thus continuously recorded before, during, and after tilt. Great care was taken to ensure that the backscattered ultrasound signal was of good quality and that the angle of insonication did not vary much during the experiment. The quality of the signal was monitored and was good in all the experiments included.

Beat-by-beat femoral artery blood flow was recorded using an ultrasound Doppler method. A bidirectional ultrasound Doppler velocimeter (model SD-100, GE Vingmed Ultrasound) was operated in pulsed mode at 3 MHz. The ultrasound Doppler transducer was handheld over the femoral artery by one of the authors (IHN), who sat beside the subject and followed the movement of the tilt table during tilts. The ultrasound beam was directed at the femoral artery distal to the inguinal ligament at an angle of 45°. The instantaneous intensity-weighted mean velocity was calculated by the velocimeter and on-line interfaced to the recording computer. The diameter of the femoral artery was determined by ultrasonic imaging (model CFM-750, GE Vingmed Ultrasound), and area was calculated assuming that the vessel was circular. Importantly, little or no change in femoral artery diameter is seen in going from supine to 70° head-up tilt (12). The beat-to-beat “stroke volume” in the femoral artery was calculated by multiplying the value obtained by numerical integration of the recorded instantaneous mean velocity during each R–R interval by the area of the femoral artery.
Instantaneous heart rate was obtained from each R-R interval of the electrocardiogram signal, and beat-to-beat cardiac output was calculated from the corresponding heart rate and stroke volume values. Beat-by-beat femoral flow was calculated in a similar manner.

Finger arterial pressure was recorded continuously from the third finger of the left hand (model 2300 Finapres blood pressure monitor, Ohmeda, Madison, WI), which was continuously supported on a board held at heart level. Instantaneous pressure output was transferred on-line to the recording computer, and beat-to-mean arterial pressure was calculated by numerical integration. Arterial pressure obtained by this method has been shown to be in accordance with central, intra-arterial pressure in various situations (19, 20, 24). Distortion of the pressure waveform caused by pulse-wave transmission and reflection in the brachial arteries will be canceled in the process of beat-synchronous averaging for mean arterial blood pressure calculation.

Total peripheral conductance was calculated beat-by-beat by dividing cardiac output by heart-level mean arterial pressure. The beat-to-beat calculation of total peripheral conductance presumes that the beat-to-beat averaged flow into the aorta is equal to the averaged flow through the resistance vessels. This is not the case during rapid changes in arterial pressure, because the amount of blood stored in the aorta varies, depending on the changes in arterial pressure and the compliance of the aorta and the large arteries (22, 29). However, changes in mean arterial pressure were relatively small (e.g., see Fig. 3F). Femoral artery conductance was calculated beat-by-beat by dividing femoral flow by mean arterial pressure. The conductance so derived constitutes a “virtual” conductance during non-steady states because the location and magnitude of the venous back-pressure is unknown and changing, e.g., a rise in flow stemming from a reduction in venous pressure with no change in vessel caliber would manifest as a rise in the calculated conductance (28).

All recorded signals, including the instantaneous angle of the tilt table, were on-line transferred to the recording computer running a dedicated data collection and analysis program (program for real-time data acquisition, written by Morten Eriksen, Oslo, Norway).

Data analysis. Data analysis was performed on 1-s averages of the digitized data. Eye-level arterial pressure, an index of cerebral perfusion pressure, was calculated from the measured heart-level arterial pressure, heart-eye distance, and the angle of tilt. Baseline values were established by averaging the signals over the 10-s period commencing 30 s before the onset of head-up tilt and terminating 20 s before the onset of head-up tilt (i.e., over the 10-s period immediately preceding the onset of head-down tilt in the push-pull trials and over the corresponding period for the control trials). The magnitudes of the responses to −G, stress (ΔPush) were calculated as the difference between the peak or nadir value observed over the 20-s period of head-down tilt, or the corresponding period in the control trials, and the baseline value. The magnitudes of the responses to +G, stress (ΔPull) were calculated as the difference between the peak or nadir value observed during the period of head-up tilt and baseline pressure. Figure 2 shows a primary recording of the cardiovascular variables during one typical experiment. Throughout the recording period, there is considerable beat-to-beat variation in the recorded variables. These variations have previously been reported (6, 13) and are primarily due to the influence of respiration (13, 30).

Statistical analysis. ΔPush and ΔPull values between control and push-pull trials were compared statistically by paired t-tests and, differences were considered significant if P < 0.05. Data are presented as means ± SE.

RESULTS

Figure 2 depicts typical responses of the primary measured variables to control (left) and push-pull (right) gravitational stress from a single subject. Shown are the tilt profile (Fig. 2, A and B); eye-level arterial pressure (Fig. 2, C and D), in which the black lines denote phasic arterial pressure and the white lines denote mean pressure; aortic flow (Fig. 2, E and F), in which the black lines denote phasic aortic flow velocity and the white lines denote mean aortic flow (cardiac output); heart rate (Fig. 2, G and H); and femoral arterial flow (Fig. 2, I and J), in which the black lines denote phasic femoral artery flow velocity and the white lines denote mean femoral flow. As expected, eye-level arterial pressure fell during head-up tilt during control gravitational stress (Fig. 2, A and C). Aortic flow velocity and cardiac output initially rose during head-up tilt and then returned to baseline values, as did mean femoral flow as well as heart rate in this example. Also, as expected, the head-down tilt phase of push-pull gravitational stress led to a rise in eye-level arterial pressure (Fig. 2, B and D), and there was little influence of this maneuver on the other variables. When head-up tilt followed head-down-tilt, eye-level arterial pressure underwent an exaggerated fall in the first seconds following head-up tilt (Fig. 2, B and D). There was also an exaggerated rise in mean femoral arterial flow over this period.

The time courses of the hemodynamic responses to control and push-pull gravitational stress, averaged from the seven subjects, are shown in Fig. 3. The group mean values of the derived variables at the time points of interest are presented in Table 1. Head-down tilt raised eye-level arterial pressure (Fig. 3A, thick line), and this change achieved statistical significance (P < 0.05 for the ΔPush values of eye-level arterial pressure for control vs. push-pull in Table 1). Eye-level arterial pressure was partially restored toward baseline by the end of head-down tilt. Head-up tilt reduced eye-level arterial pressure, and there was a greater initial fall in eye-level arterial pressure when head-up tilt was immediately preceded by head-down tilt (Fig. 3A, thick vs. thin lines), and this difference achieved statistical significance (P < 0.05 for the ΔPull values of eye-level arterial pressure for control vs. push-pull in Table 1).

Head-down tilt slowed heart rate (Fig. 3B, thick line), and this change achieved statistical significance (P < 0.05 for the ΔPush values of heart rate for control vs. push-pull in Table 1). Head-up tilt raised heart rate, and there was a greater rise in rate when head-up tilt was immediately preceded by head-down tilt (Fig. 3B, thick vs. thin lines), and this difference achieved statistical significance (P < 0.05 for the ΔPull values of heart rate for control vs. push-pull in Table 1). Stroke volume rose at the onset of head-up tilt, and there was a greater rise in stroke volume when head-up tilt was immediately preceded by head-down tilt (Fig. 3B, thick vs. thin lines), and this difference achieved statistical significance (P < 0.05 for the ΔPull values of stroke volume for control vs. push-pull in Table 1). Stroke volume thereafter fell for the remainder of head-up tilt. Note that the ΔPull values of stroke volume reflect the rise to the early peak during head-up tilt, which occurred at approximately the same time as the nadir in heart-level arterial pressure (Fig. 3A); that is, ΔPull for stroke volume was not calculated based on the nadir in stroke volume that occurred at the end of the period of head-up tilt. Cardiac output (Fig. 3D) followed the overall pattern exhibited by heart rate and stroke volume, falling during head-down tilt, rising to an early peak soon after the onset of head-up tilt that was greater for the push-pull treatment (P < 0.05 for the ΔPull values of cardiac output for control vs. push-pull in Table 1), and then falling during the remainder of head-up tilt.
Femoral flow (Fig. 3H) followed the overall pattern exhibited by cardiac output, falling during head down tilt ($P < 0.05$ for the $\Delta$Push values of femoral flow for control vs. push-pull in Table 1), rising to an early peak soon after the onset of head-up tilt that was greater for the push-pull treatment ($P < 0.05$ for the $\Delta$Pull values of femoral flow for control vs. push-pull in Table 1), and then falling during the remainder of head-up tilt. Again, the $\Delta$Pull values for femoral flow reflect the rise to the early peak during head-up tilt, not the nadir in flow that occurred at the end of the period of head-up tilt.

Total peripheral conductance (Fig. 3E) followed the overall pattern exhibited by cardiac output, falling during head-down tilt, rising to an early peak soon after the onset of head-up tilt that was greater for the push-pull treatment ($P < 0.05$ for the $\Delta$Pull values of total peripheral conductance for control vs. push-pull in Table 1), and then falling during the remainder of head-up tilt. Femoral conductance (Fig. 3I) followed the overall pattern exhibited by femoral flow, falling during head down tilt ($P < 0.05$ for the $\Delta$Push values of femoral conductance for control vs. push-pull in Table 1), rising to an early peak soon after the onset of head-up tilt that was greater for the push-pull treatment ($P < 0.05$ for the $\Delta$Pull values of femoral conductance for control vs. push-pull in Table 1), and then falling during the remainder of head-up tilt.
after the onset of head-up tilt that was greater for the push-pull treatment ($P < 0.05$ for the $\Delta$Pull values of femoral conductance for control vs. push-pull in Table 1), and then falling during the remainder of head-up tilt. Once again, the $\Delta$Pull values for conductance reflect the rise to the early peak during head-up tilt, not the nadir in conductance that occurred at the end of the period of head-up tilt.

DISCUSSION

The major new findings of this study are that a push-pull effect occurs in human subjects subjected to tilting and that over one-half of this effect is attributable to an exaggerated rise in leg vascular conductance.

Response to head-up tilt. The central hemodynamic responses to head-up tilt and tilt back from the present study are in complete agreement with a previous report (32). For example, Toska and Walloe (32) found that stroke volume initially rose with head-up tilt before falling below baseline values with more prolonged head-up tilt, and others have observed gradual responses (23, 35). Two factors likely contribute to the initial rise in left ventricular stroke volume in the two studies. First, the pulmonary circuit is expected to discharge blood volume...
from apical regions of the lungs to the left heart, and this is expected to increase stroke volume. Second, the fall in right atrial pressure with head-up tilt will reduce right ventricular filling, which can lead to increased left ventricular filling via a rightward shift of the intraventricular septum, i.e., ventricular interdependence (13). Furthermore, we extend the findings of Toska and Walloe (32) with the observation that leg blood flow exhibits a pattern of response similar to that of cardiac output. The response of leg conductance to head-up tilt was similar in pattern to total peripheral conductance. In fact, the changes in leg flow and conductance appeared to meet or exceed the changes in cardiac output and total peripheral conductance. The observation that leg blood flow rose greater than did cardiac output at the onset of head-up tilt (0.82 l/min vs. 0.73 l/min) indicates that blood flow elsewhere must have fallen. The fall most likely occurred in the upper body inasmuch as the changes in regional pressure in the upper body during head-up tilt would mimic the changes in the lower body that occur with head-down tilt, where leg blood flow was observed to fall (Fig. 3I at second − 20). The great extent to which the changes in leg conductance drive the changes in total peripheral conductance likely stems from two factors. First, this region constitutes a sizable fraction (20%) of the total conductance so changes in leg conductance would be expected to alter total conductance. Second, this region lies a sizable distance from the hydrostatic indifferent point, meaning that local tissues are subjected to large changes in pressure during tilting.

What causes leg conductance to rise with head-up tilt? The flow of blood through the legs is governed by the product of the pressure gradient across the legs (arterial luminal pressure minus venous luminal pressure) and vascular conductance (inverse of resistance). When the legs are at heart level, the pressure gradient (aortic minus right atrial) driving flow through the legs is provided by the hydrodynamic pressure attributable to the pumping action of the heart. With head-up tilt, a hydrostatic pressure is added to the hydrodynamic luminal pressure owing to the effects of gravity on the fluid. The magnitude of the hydrostatic pressure is proportional to the height of the column of fluid, the density of the fluid, and the gravitational constant. The hydrostatic pressure will develop immediately with the change in posture in the arteries because they contain no structures (e.g., valves) that could interrupt the formation of a continuous column of blood along the length of the arterial system. The same is not true on the venous side, owing to the venous valves, which are expected to close and thereby interrupt the formation of a continuous column of blood along the large veins. That is, with the closure of the venous valves at the onset of head-up tilt, the pressure within the distal venous compartment is suddenly locked (held constant) at the prevailing pressure. Thus there is expected to be a transient increase in the pressure gradient across tissues in dependent regions owing to the greater local arterial pressure. This will work to augment the flow of blood through these tissues, a factor that will persist until the veins are filled enough to open the venous valves. Once a continuous column of blood is established on the venous side of the circulation, the hydrostatic pressures on the arterial and venous side will cancel each other such that the pressure-flow relationship across dependent tissues will be largely unaffected by gravity (33).

Our leg blood flow data fit with the foregoing reasoning in that leg blood flow rose transiently at the onset of head-up tilt before decaying to a level below baseline at the end of head-up tilt. When heart-level pressures are used to analyze pressure-flow relationships, the local change in pressure owing to the hydrostatic effects of tilt is missed and the “cause” of the increase in flow is manifest as an increase in calculated vascular conductance or as a rise in “virtual conductance” as has been proposed for the action of the muscle pump (28).

How might prior application of head-down tilt modify this effect? Head-down tilt is expected to lower arterial and venous pressures in the legs just as head-up tilt lowers eye-level pressures. With the subsequent transition to head-up tilt, leg arterial pressure is again expected to rise immediately (no valves). However, the reduced pressure in the small veins in the legs (the effective back-pressure governing arterial inflow to the lower body) would be expected to persist early on during the subsequent head-up tilt, owing to the venous valves. Thus, compared with control trials, there would be a relatively greater arterial-venous pressure gradient driving flow into the legs during the head-up tilt phase of push-pull gravitational stress, and this could lead to an increase in arterial inflow to the legs in the absence of locally produced changes in vessel diameter. That is, head-down tilt is expected to lower venous pressure similar to muscle contraction and relaxation in upright subjects (33).

Myogenic relaxation induced during head-down tilt is another potential contributor to the increase in leg conductance during head-up tilt (21), as is the venoarteriolar response (17, 18). Evidence that these mechanisms are likely active in the present study is provided by a study in which a potent push-pull effect was observed in dogs following autonomic blockade (26). The notable difference from this previous study is that autonomic reflex function remained intact in the present study. How might reflex function further modify the responses to tilt?

The responses at the end of the 30 s of head-up tilt in the control trials and the responses at the end of the 20 s of head-down tilt were as expected. In response to head-up tilt, eye-level arterial pressure initially fell and then was partially restored toward baseline. This partial restoration of pressure was attributable to a reduction in total peripheral conductance that partially offset the decrease in cardiac output that followed the initial rise in output. These adjustments caused a rise in heart-level arterial pressure during head-up tilt. Head-down tilt induced opposite responses. Mean arterial pressure during head-down tilt was decreased to a level slightly below resting level, in accordance with the fact that carotid baroreceptors were now slightly lower than in the supine state (meaning the hydrostatic distending pressure was elevated). The responses to head-down tilt were similar to the changes observed in response to a rise in arterial pressure induced by inflation of thigh cuffs in the supine posture (31).

In agreement with previous studies (1, 3, 10, 11, 14, 15, 16, 26, 27), we found an exaggerated fall in eye-level arterial pressure in the transition to head-up tilt when head-up tilt was preceded by head-down tilt. The exaggerated fall in eye-level pressure was attributable to an exaggerated rise in calculated total peripheral conductance, which in turn was largely (70%) attributable to an exaggerated rise in leg conductance. Thus our hypothesis was answered in the affirmative. The causes of the exaggerated changes in conductance likely include the mechanical and myogenic responses outlined above. Since leg conductance is under the influence of multiple factors, it is
unclear whether the sympathetic influence on leg conductance works to protect against the push-pull effect by restricting the rise in leg conductance or whether any sympathetic influence contributes to the push-pull effect via residual reflex vasodilation initiated during the head-down tilt.

In response to push-pull gravitational stress imposed in the present study, we found exaggerated heart rate and cardiac output responses that worked to protect against or minimize the push-pull effect. That is, we found that head-up tilt preceded by head-down tilt was associated with greater initial increases in heart rate, stroke volume, and cardiac output compared with head-up tilt alone. Thus the greater initial increase in cardiac output worked to match some of the exaggerated rise in total peripheral conductance such that eye-level arterial pressure fell to a lesser extent than it otherwise would have fallen without these changes.

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REFERENCES


