

Transient Cardiovascular Responses to Passive Up and Down of Lower Extremities in Supine Position

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Abstract

Objective: The purpose of this study is to clarify the characteristics of the phasic component of cardiovascular responses to the passive raising and lowering of the legs in the supine position.

Methods: Cardiovascular variables were measured continuously using a noninvasive finger blood pressure measurement system in 8 healthy male subjects, and transient responses to leg-up and leg-down in supine position at 20° and 40° leg lifted angles were compared.

Results and Discussion: Phasic cardiovascular responses lasting tens of seconds to leg-up and leg-down were observed. Responses to leg-up at 20° consisted of transient increases in heart rate (HR) and total peripheral resistance (TPR), with concomitant responses in other cardiovascular variables. The transient increase in TPR was slightly delayed to that of heart rate and followed by a slower transient decrease from the base level. The transient responses to the leg-down were similar to the leg-up with slight differences, if any. Dependencies on the elevation angle were also small, except for lowering the legs from 40°, where TPR decreased immediately without an initial transient increase. These transient responses triggered by the passive movement of the legs were probably caused by the autonomic nervous reflex, although it is not clear whether they are triggered by the hemodynamic alteration by the movement of the legs or by the sensory stimulation of the body surface. The change in the response pattern of the 40° leg-down may indicate the existence of an additional factor contributing to the TPR response.

Conclusion: Passive elevation and re-flattening of the legs caused transient cardiovascular responses in addition to tonic responses during leg lift. The pattern of transient cardiovascular responses was relatively indifferent to the direction of upward or downward movement, nor to the lifting angle of the legs, except in the leg-down from a 40° angle of the leg lift.

Keywords: Cardiovascular function, Passive leg-raising, Total peripheral resistance

Abbreviations

CO: Cardiac Output; DBP: Diastolic Blood Pressure; HR: Heart Rate; MBP: Mean Blood Pressure; PLR: Passive Leg Raising; SBP: Systolic Blood Pressure; SV: Stroke Volume; TPR: Total Peripheral Resistance

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Introduction

Passive leg raising (PLR) is commonly used in primary care to expect an increase in cardiac output by increasing venous return [1]. We previously reported that during the PLR, even healthy adults showed considerable sustained responses including significantly increased stroke volume during whole leg-raising period [2]. In addition to sustained responses, we noticed that transient responses to the raising and to the re-resting of the legs were also observed. Here, we report characteristics of these transient responses.

Methods

The data analysed here was part of the data from previously reported research [2]. Among leg elevation angles of 20°, 30° and 40° analysed in the previous report, only the PLR of 20° and 40° were chosen for the analysis in this study as cases of sufficient mild PSR and slightly excessive PLR.

Subjects

The subjects were 8 healthy adult males. Their age, height and weight (expressed on average ± SD) were 32 ± 5 years, 173.5 ± 6.1 cm and 76.8 ± 13.9 kg, respectively. All were fully informed of the experiment procedure, including possible damage and provided written consent for participation. This study was approved by the Ethics Committee for Research on Human Subjects at Kokushikan University.

Measurement device

A continuous finger blood pressure measurement system (Portapres®, Medical Systems B.V.) was used for non-invasive measurements of cardiovascular dynamics [3-5]. Using calculations based on the mathematical model (Modelflow®) constructed by Wesseling and co-workers [5], Portapres can estimate major cardiovascular parameters, including stroke volume (SV), total peripheral resistance (TPR), systolic (SBP), diastolic (DBP) and mean arterial blood pressure (MBP) and HR where TPR is calculated as the ratio of MBP and CO.

Procedures

Initially, each subject rested in a supine position on a horizontal board with a movable lower section that could be raised to an angle of 20° or 40°. A finger cuff for Portapres was fitted on the third finger of the right hand. At the beginning, SBP and DBP at the right upper arm were also manually measured, and the cuff was readjusted when the discrepancy between the reconstructed and manually measured values of either SBP or DBP was more than or equal to 10 mmHg. After 300 sec of rest, the subject’s legs were passively raised to 20° or 40° and maintained in the raised position for 600 sec. Subsequently, the legs were passively returned the horizontal position, and the subject rested for 180 sec, making a total 1080 sec sequence (Figure 1). For each subject, the sequences for 20° and 40° were repeated on 3 different days. Therefore, 24 PLR data sets were obtained from 8 subjects for the 20° and 40° PLR. The lifting and returning of the legs was carried out smoothly by 2 trained experimenters within 1 sec. To minimize the psychosomatic influences on the cardiovascular dynamics, each subject was kept silent, with the eyes masked and free of unnecessary stimuli, except for the lifting and returning of the legs during the experiment. The room temperature and humidity were maintained at 23.0 ± 4.6°C and 44.0 ± 5.1%, respectively.

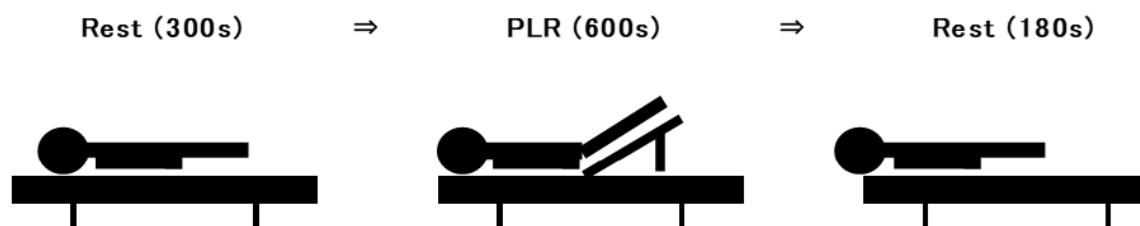


Figure 1: Design of the measurement procedure. One sequence of PLR experiment consisted of 300s rest, 600s PLR and 180 s post-PLR rest. Each of 20° and 40° leg raising was performed in randomized order at a day.

Data processing

Among cardiovascular variables obtained by Portapres, we analysed heart rate (HR), stroke volume (SV), total peripheral vascular resistance (TPR), systolic (SBP), diastolic (DBP) and mean (MBP) arterial blood pressures and cardiac output (CO). The values of each variable of each trial were normalized by taking the average value of the 5-min period immediately before the start of PLR as 100%. The values of the data points less than 50% or greater than 150% after normalization were discarded as artifacts after visual confirmation. To obtain a clear transient response, we took medians of 24 trials rather than averages, since although both the transient responses taken median and average showed a similar level of clarity for most cases, the median data were more robust than the average data when the considerable outliers existed in original traces. To clarify the timing of the phasic responses of different variables, the trajectories of the variables were plotted on a two-dimensional plane.

Results

Arterial blood pressure (BP) responses are shown in figure 2. In response to leg-up of 20° PLR, BPs increased transiently with more than 5% of peak heights and duration of about 10 sec or more. In DBP, the spiky increasing response was followed by trough to 95% level and gradual recovery followed till the end of 50 sec from the leg-up. Transient responses to leg-down of 20° PLR were quite identical to leg-up of 20° PLR. Responses to leg-up of 40° PLR were also similar except a slightly higher peak. Transient responses to leg-down of 40° PLR appeared to be quite different from others. SBP decreased abruptly without the initial increase and recovered slowly to another stable level. Curiously, the timing and amplitude of this abrupt decrease response was almost identical to the dropping phase of transient increase response to leg-up of 20° PLR. DBP responded to leg-down of 40° PLR with a slight, if any, transient increase followed by about 15% rapid decrease which also similar to the dropping phase in leg-up of 20° PLR, despite the start to drop being several seconds earlier.

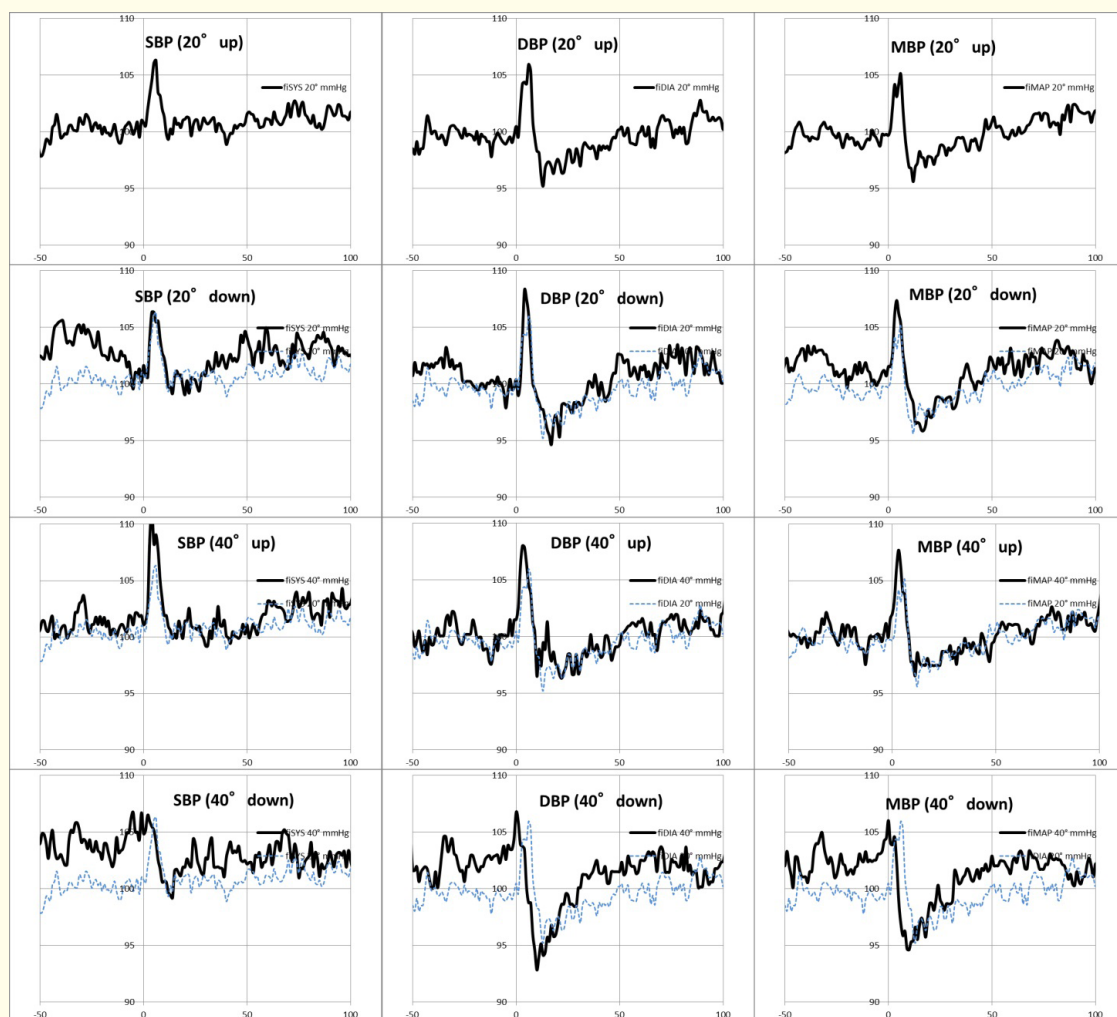


Figure 2: Transient blood pressure responses: The blood pressure responses to the leg-up and leg-down during passive leg raising (PLR) are shown. The 1st row shows the responses of systolic (SBP), diastolic (DBP) and mean (MBP) blood pressure to leg-up in 20° PLR. The 2nd, leg-down in 20° PLR, the 3rd, leg-up in 40° and the 4th, leg-down in leg in 40° PLR. The data are normalized by the values during 50s just before the leg-up and the median of 24 sequences (8 subjects, three times each). The fine (blue) dotted lines in the 2nd to 4th rows showed the same data as the 1st row.

In figure 3, other cardiovascular variables are shown. The HR response to leg-up of 20° PLR was a transient increase of about 8% of the peak height with a duration of less than 10 sec which was slightly shorter than that of SBP. The TPR response to leg-up of 20° PLR was biphasic and consisted with an initial transient increase slightly delayed to that of HR and a following rapid drop and gradual recovery. The Response of HR to leg-down of 20° PLR was similar to that to leg-up, but with a higher peak. That of TPR was a monophasic transient increase with a slight delay to that of HR. In the 40° PLR, the increasing response of HR to leg-up became broader due to slower recovery phase than that in 20° PLR. The increasing responses of HR to leg-down in 40° PLR also became lumpy rather than the spiky peak in 20° PLR. While the TPR response to leg-up in 40° PLR was biphasic similar to those in 20° PLR except for a much higher peak and deeper trough, the response HR to leg-down appeared quite different from that in 20° PLR and consisting of an abrupt decrease of more than 15% and a following gradual recovery. The SV responded to leg-up with transient decreases of 3 to 4 sec duration, followed by a sustained increase during leg lift as previously reported [2]. The transient SV response to leg-down in 20° PLR was similar to that of leg-up. The transient negative SV response in 40° PLR appeared smaller than other SV transient responses. The CO transient response to leg-up was not clear, if any. While the CO response to leg-down in 20° PLR was biphasic with a spike followed by a trough, the response in 40° PLR showed quite an obviously lumpy transient increase.

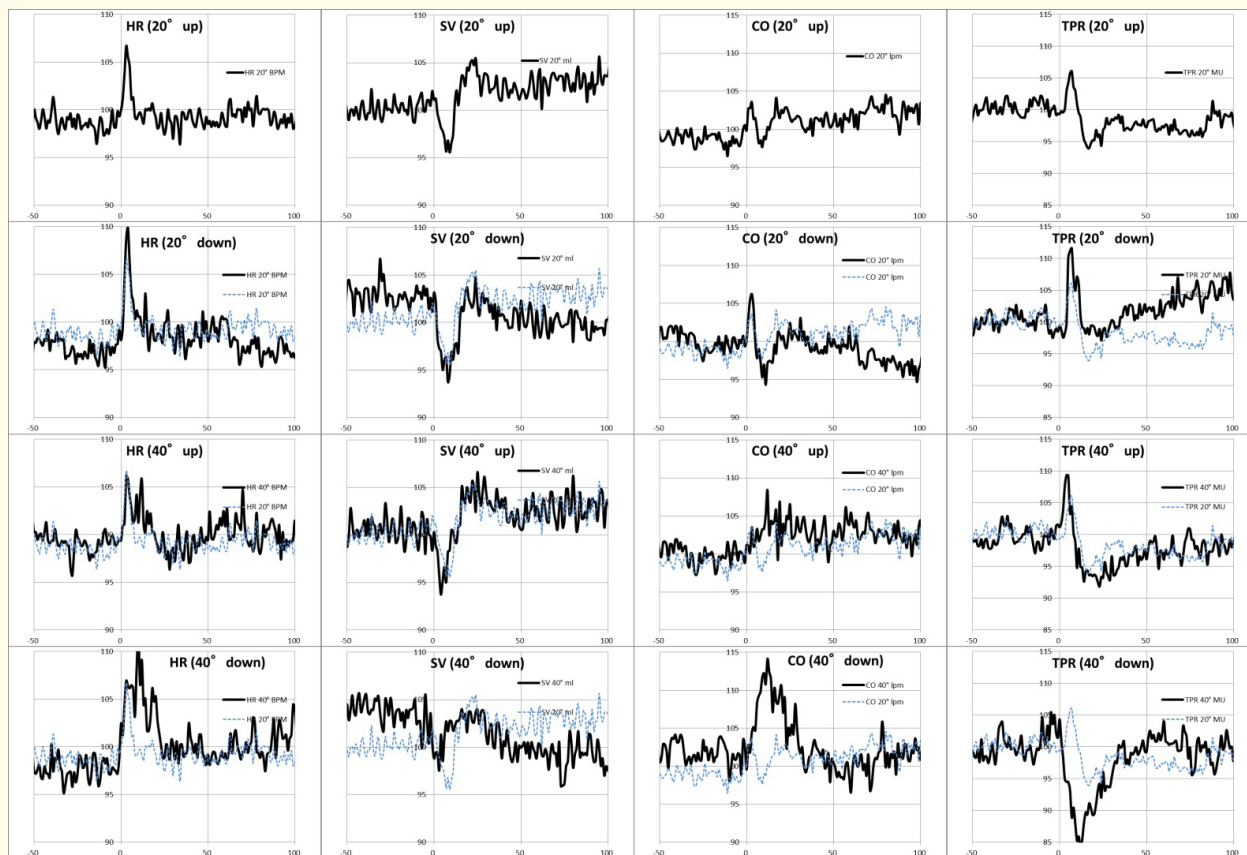


Figure 3: Transient responses of heart rate (HR), stroke volume (SV), cardiac output (CO) and total peripheral vascular resistance (TPR): the responses of HR, SV, CO and TPR to the leg-up and leg-down are shown. The rows are in the same order as figure 2.

In order to clarify the mutual temporal relationship, the trajectories of SV, CO and TPR with respect to HR were drawn on a two-dimensional plane (Figure 4). Except for leg-down in 40° PLR, the trajectory of TPR against HR started in a horizontal right direction, indicating that the HR increase started earlier than the TPR increase, and then it changed the direction upward and the passing highest peaks of TPR rapidly returned near to the starting point within 15 sec showing a left turning loop in the 1st quadrant. In leg-down in 40° PLR, the trajectory started to a right-down direction indicating that the TPR almost simultaneously began to decrease with the HR increase and took more than 30 sec to return to near the starting point. SV started to decrease simultaneously with the initial increase in HR, except for leg-down in 40° PLR, where the decreases in SV was small, probably because the early dropping of TPR compensated the contribution of the HR increase. The CO responses to leg-up initially increased with HR. The trajectories showed right turning loops, indicating a slightly earlier returning of CO than HR, and they were small to leg-up and large to leg-down. The trajectory of CO transient responses to leg-up in 40° PLR was small and rather complicated. The trajectory of CO to leg-down in 40° PLR showed a roughly left turning loop in the 1st quadrant, indicating a slightly delayed recovery of CO compared to HR.

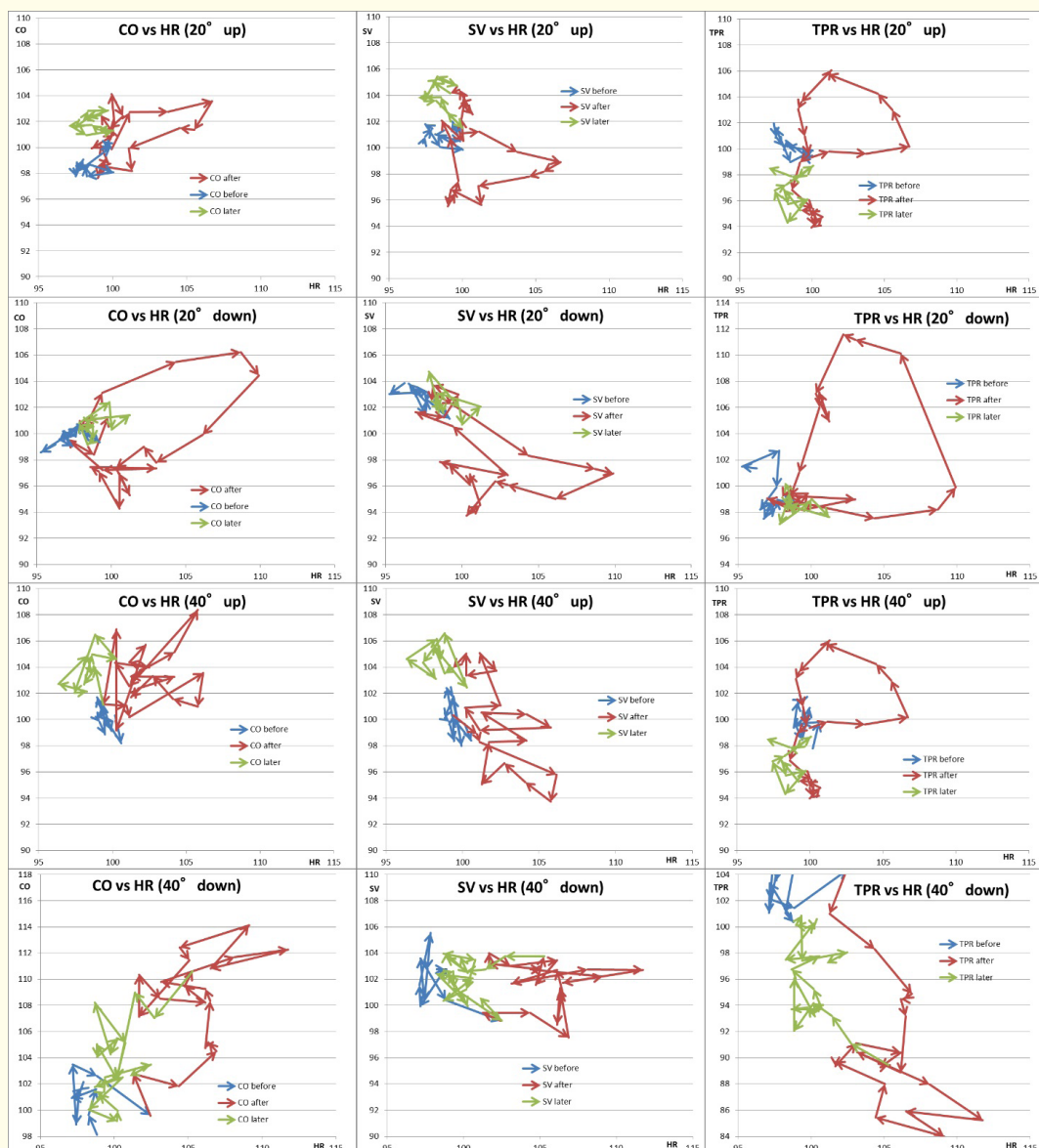


Figure 4: CO, SV and THR trajectories against HR in the transient responses: trajectories of values during the transient responses of CO, SV and THR are plotted against HR. Blue arrows show the 10 sec movements before the leg shift, red arrows 20 sec trajectories after the leg shift and green arrows 10 sec (in the 1st, 2nd and 3rd rows) and 20 sec (in the 4th row) of later phases.

Discussion

It is well known that the variables of the cardiovascular regulatory system change with the change in posture mainly through the homeostatic mechanism to maintain the BP in a healthy person at a constant level (see, for example, to Ganong's textbook [5]). There, however, exist transient responses of the cardiovascular system before the sustained tonic responses adjusted for the shifted posture appear. Orthostatic hypotension is a typical pathological example [6-8] and in diving reflex there have been reported transient components of the cardiovascular responses [9,10]. The developers of the volume clamp continuous finger blood pressure monitoring system Finapres themselves have reported some results on transient cardiovascular responses in body repositioning [11]. In any cases, these transient responses were not merely smoothly connecting two steady states, but also showed some excessive responses in the middle of the reaction even in healthy subjects.

Cardiovascular dynamics is, if simplified, the relationship between the output power of the heart as a pump and the impedance of the vascular system as a flow tube. Homeostasis works to set blood pressure to normal values to maintain blood flow to vital organs. Among the variables analysed in this study, HR and TPR are considered actively controllable by the autonomic nervous system and SV and CO are controlled by HR and TPR, if other contributing factors including changes in the contractile force of the heart and venous return are neglected.

In leg-up and leg-down in PLR, both HR and TPR showed transient responses. The HR responses were monophasic transient increases very narrow to leg-up and leg-down in 20° PLR and wider in 40° PLR, starting immediately with leg shifts. The TPR responses seemed to consist of a narrow spiky transient increase and a following negative trough component. This negative component was clearly seen in leg-up in 20° and 40° PLR but most prominent in leg-down in 40° PLR, in which the positive transient increase seemed to be almost cancelled by the negative component. This negative PLR response to leg-down in 40° PLR was so prominent that all the initial positive transient increases of the BP were concealed and considerable lumpy increase of CO could be also seen in leg-down in 40° PLR. Except for leg-down in 40° PLR, the starting timing of transient PLR responses slightly delayed to the HR response as seen in figure 4. Therefore, it may not be clear whether the TRP responses were triggered by leg shift or by transient HR increase. It also may not be clear whether these transient responses were triggered by change in blood flow dynamics due to the displacement of the legs or simply by stimulation of the somatosensory or proprioceptive receptors.

From these considerations, it is speculated that the transient responses seen in this study may have two distinct components. One acts by obeying all-or-none laws triggered by both up and down movement of the legs and consists of a transient spiky HR increase and a slightly delayed transient TPR increase. The other acts in a direction- and intensity-dependent manner and consists of a slight increase in HR and a rapid decrease and slow recovering of TPR, whose intensity and duration depend on modality and intensity of the stimulus. All-or-none features of cardiovascular transient responses have been found in other studies [6-12] and can be quite interesting both from a physiological and pathological point of view.

Conclusion

Passive elevation and re-flattening of the legs caused transient cardiovascular responses. They may have two distinct components: one consists of a transient spiky HR increase and a slightly delayed transient TPR increase and the other consists of a slight increase in HR and a rapid decrease and slow recovering of TPR.

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Conflict of Interests

Authors declare they have no conflict of interests.

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