Central and peripheral responses to static and dynamic stretch of skeletal muscle: mechano- and metaboreflex implications

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Venturelli M, Cè E, Limonta E, Bisconti AV, Devoto M, Rampichini S, Esposito F. Central and peripheral responses to static and dynamic stretch of skeletal muscle: mechano- and metaboreflex implications. J Appl Physiol 122: 112–120, 2017. First published November 17, 2016; doi:10.1152/japplphysiol.00721.2016.—Passive static stretching (SS), circulatory cuff occlusion (CCO), and the combination of both (SS + CCO) have been used to investigate the mechan- and metaboreflex, respectively. However, the effects of dynamic stretching (DS) alone or in combination with CCO (DS + CCO) on the same reflexes have never been explored. The aim of the study was to compare central and peripheral hemodynamic responses to DS, SS, DS + CCO, and SS + CCO. In 10 participants, femoral blood flow (FFB), heart rate (HR), cardiac output (CO), and mean arterial pressure (MAP) were assessed during DS and SS of the quadriceps muscle with and without CCO. Blood lactate concentration ([La]−) in the lower limb undergoing CCO was also measured. FFB increased significantly in DS and SS by 365 ± 98 and 377 ± 102 ml/min, respectively. Compared with baseline, hyperemia was negligible during DS + CCO and SS + CCO (+11 ± 98 and +5 ± 87 ml/min, respectively). DS generated a significant, sustained increase in HR and CO (~40%), while SS induced a blunted and delayed cardioacceleration (~20 s). After CCO, [La]− in the lower limb increased by 135%. Changes in HR and CO during DS + CCO and SS + CCO were similar to DS and SS alone. MAP decreased significantly by ~5% during DS and SS, did not change in DS + CCO, and increased by 4% in SS + CCO. The present data indicate a reduced mechanoreflex response to SS compared with DS (i.e., different HR and CO changes). SS evoked a hyperemia similar to DS. The similar central hemodynamics recorded during stretching and [La]− accumulation suggest a marginal interaction between mechan- and metaboreflex.

NEW & NOTEWORTHY Different modalities of passive stretching administration (dynamic or static) in combination with circulatory cuff occlusion may reduce or amplify the mechan- and metaboreflex. We showed a reduced mechanoreflex response to static compared with dynamic stretching. The lack of increase in central hemodynamics during the combined mechano- and metaboreflex stimulation implicates marginal interactions between these two pathways.

stretching; metaboreflex; mechanoreflex

CARDIOVASCULAR RESPONSES to physical exercise result mainly from the interaction among three major neural mechanisms: 1) the activation of cardiovascular-related network of neurons (central command), 2) the afferent input from arterial baroreceptors (baroreflex), and 3) the afferent input from skeletal muscle receptors (11, 13, 21, 29, 31). Skeletal muscle feedback during exercise can be partitioned into two major pathways: the mechanoreflex (group III thinly myelinated fibers), and the metaboreflex (group IV unmyelinated fibers) (2, 5, 6, 9, 10, 23, 26, 34).

In previous investigations, passive static stretching (SS) of skeletal muscle, circulatory cuff occlusion (CCO), and the combination of both procedures (SS + CCO) have been used to selectively stimulate group III or IV skeletal muscle afferents, as well as the interaction between these two pathways (1, 14). In humans, a relatively long period (1 min) of SS on a small muscle (calf) generates a rapid and sustained increase in blood pressure and heart rate (HR) (12, 14, 15, 22). In addition, the HR response at the onset of muscle contraction and during passive dynamic stretches (DS) has been investigated (14). Interestingly, the DS-induced cardioacceleration was smaller compared with SS. In a recent study, Kruse and coworkers (22) documented no detectable changes in net blood flow, mean arterial pressure (MAP), and vascular conductance (VC) measured at popliteal artery during 5 min of plantar flexors SS. Collectively, these observations suggest a different central and peripheral hemodynamic responses to DS and SS.

Fisher and coworkers (12) demonstrated a pronounced increase in diastolic blood pressure during SS administered together with CCO performed immediately after exercise [postexercise cuff occlusion (PECO)]. Interestingly, the increase in HR during PECO was similar to SS alone, suggesting that HR and blood pressure responses to peripheral metabo- and mechanoreceptor stimulation are likely different. Moreover, relatively short periods (5 s) of passive SS of small muscles (calf or forearm) produce a transient increase in muscle sympathetic nerve activity (7). Contrary to previous work (12), when SS of longer duration (2 min) was administered during PECO, sympathetic outflow, and MAP were further increased (8), suggesting a sensitization of muscle mechanoreceptors when metabolites are accumulated. However, this phenomenon of mechanoreceptor sensitization by the activation of local metaboreceptors during DS is still a matter of debate.

When comparing small and large muscle mass stretching, the increase in HR and cardiac output (CO) during DS of human knee extensors was still present (27, 38). When group III and IV afferent feedbacks were abolished via spinal injection of fentanyl (36), as well as in individuals with complete spinal cord injury (39, 40), this DS-related increase in HR and
CO was diminished or even absent. Noticeably, contrary to the data reported by Kruse and coworkers (22) on hemodynamic response to SS of calf muscle, during DS of human knee extensors a peripheral hyperemia was observed during the first 45 s of DS, which was primarily explained by local nitric oxide (NO) release (30, 37). In concomitance to this transitory reduction in the local vascular peripheral resistance, systemic MAP was also reduced, implying that a portion of the mechano- and metaboreflex (i.e., increase in blood pressure) may be masked during the first 45 s of DS by the reduction of peripheral resistances. To circumvent the DS-induced hyperemia, McDaniel et al. (27) over imposed DS to CCO and observed an increase in CO, HR, and stroke volume (SV) similar to that obtained when DS was executed without CCO.

Consequently, a sensitization of muscle mechanoreceptors via local metabolites accumulation may be evident only during SS and not during DS. Moreover, central and peripheral responses to SS of knee extensors, as well as the interaction between mechano- and metaboreflex triggered by SS and metabolites accumulation, have not been so far fully elucidated.

Therefore, the aim of the present study was to compare the central and peripheral hemodynamic response to SS and DS of the knee extensor muscles. The interaction between mechano- and metaboreflex was also investigated. Specifically, by studying central and peripheral hemodynamics of SS, DS, SS + CCO, and DS + CCO, we tested the following hypotheses: 1) the central responses to SS would be attenuated in comparison to DS; 2) the peripheral hemodynamic response to SS would be reduced in comparison to DS; and 3) the central responses during SS + CCO and DS + CCO, occurring during accumulation of peripheral metabolites, would be greater in comparison to SS and DS alone, implicating an interaction between mechano- and metaboreflex.

METHODS

Participants. Ten young healthy males (age: 25 ± 1 yr; body mass: 77 ± 2 kg; stature: 1.81 ± 0.02 m; means ± SD) participated in this study. None of the participants were smokers and most were physically active. All procedures conformed to the standards set by the Declaration of Helsinki and were approved by the ethical committee of the University of Milan. Participants gave written informed consent before their participation after full explanation of the purpose of the study and of the experimental procedures. Participants reported to the laboratory in a fasted state. They were asked to abstain from caffeine or similar beverages in the 24 h preceding the test and to report to the laboratory without any form of physical exercise of heavy intensity in the previous 48 h.

Experimental design. After a first visit for familiarization purpose, participants reported to the laboratory four times, during which SS, DS, SS + CCO, and DS + CCO protocols were performed in random order.

Static and dynamic stretching. Participants rested in a supine position for 20 min before data collection was started and remained in this position throughout the entire study. DS protocol consisted of 30 s of resting baseline followed by 45 s of passive knee extensions and flexions. DS was always performed by the same operator, who moved the lower leg through a 130° range of motion (180–50° knee joint angle) at 1 Hz. SS protocol consisted of 30 s of resting baseline followed by passive static knee flexion for 45 s (24). During the entire SS protocol the knee extensors were stretched by the same operator to the point of discomfort, which corresponded to the same range of motion obtained during the DS protocol.

To further determine potential interactions between mechano- and metaboreflex, during an additional visit to the laboratory, participants repeated DS and SS protocols with occlusion of circulation to the thigh passively stretched, thus allowing peripheral lactate accumulation and eliminating hemodynamic changes in the lower limb. In detail, a cuff was positioned in the upper thigh (inguinal region) of the participant and inflated to 250 mmHg for 300 s. Subsequently, during the protocols DS + CCO and SS + CCO the cuff remained inflated for additional 45 s. During each protocol, knee joint angle was continuously recorded using a biaxial electrogoniometer (model TSD 130A; Biopac System, Santa Barbara, CA).

Blood lactate concentration. Artialized blood samples (4 μl) from the second toe of the occluded limb were taken and blood lactate concentration ([La−]) was determined using a lactate analyzer (Labtrend LT14187; Bio Sensor Technology, Berlin, Germany) at rest (freely perfused limb) and after 300 s of cuff-induced occlusion of limb circulation before DS or SS.

Central hemodynamics. HR, SV, CO, and MAP were determined using a Finometer device (Finapres Medical Systems, Amsterdam, The Netherlands). The photoplethysmographic cuff of the finger pressure device was placed on the third finger of the left hand. The Finometer signal was calibrated utilizing the procedure indicated by the manufacturer. The height adjustment sensor and reference were positioned according to the manufacturer’s instructions. SV was estimated using the Modelflow algorithm (Beatscope version 1.1a; Finapres Medical Systems) (4). CO was then calculated as the product of HR and SV. The same method has been documented to accurately track CO during exercise (3, 35). Potentially, this noninvasive assessment presents some limitations such as the absolute estimation of CO and SV. However, as reported in previous investigations, the absolute changes from rest values have been demonstrated to be accurate (38–40).

Femoral blood flow. Measurements of arterial blood velocity and vessel diameter were performed in the passively stretched limb, distal to the inguinal ligament and proximal to the superficial femoral bifurcation with Logiq S7pro ultrasound system (General Electric Medical Systems, Milwaukee, WI). The system was equipped with 12- to 14-MHz linear array transducers. Common femoral artery diameter was determined at a 90° angle along the central axis of the scanned area. Blood velocity (v) was measured using the same probe at a frequency of 5 MHz. Measurements of v were obtained second-by-second with the probe position maintained at an insolation angle of 60° or less and the sample volume was centered and maximized according to vessel size. After arterial diameter and mean v (vmean) assessment, femoral blood flow (FBF) was automatically calculated using the Logiq S7pro software as:

\[
\text{FBF} = \frac{v_{\text{mean}} \cdot \pi \cdot (\text{vessel diameter})^2}{2} \cdot 60
\]

where FBF is in milliliters per minute. All scanning and blinded analyses were performed by experienced and skilled sonographers. To account for potential differences in MAP, VC was calculated as: FBF/MAP.

Data collection and analysis. HR, SV, CO, MAP, ECG, and knee joint angle underwent A/D conversion system (model UM150; Biopac System, Santa Barbara, CA) and were simultaneously acquired (200 Hz) by commercially available data acquisition software (AcqKnowledge; Biopac Systems, Goleta, CA). The software allowed second-by-second analysis of HR, SV, CO, and MAP throughout the experimental protocols. The vmean was analyzed with 1Hz resolution on the Doppler ultrasound system (GE Logiq S7pro) for 30 s at rest and during the first 45 s following the initiation of DS, SS, DS + C, and SS + C. From the velocity and femoral artery diameter, net FBF was calculated on a second by second basis. Before analysis, all hemodynamic data were smoothed using a 3-s rolling average. As the responses to passive stretching are transient and vary
between individuals, a peak response was determined for all variables on an individual basis. Maximal absolute (peak), relative changes (Δpeak), and the area under the curve (AUC) were determined for each subject in all measured variables.

Statistical analysis. Raw data were analyzed using a statistical software package (IBM SPSS Statistics v. 22, Armonk, NY). To check the normal distribution of the parameters, a Shapiro-Wilk test was applied. A sample size of 10 participants was selected to ensure a statistical power higher than 0.80. A two-way (time and protocol) ANOVA for repeated measures was used to establish differences among conditions at rest, after the occlusion of circulation in the lower limb, and during stretching procedures. A Tukey’s post hoc test was applied to define the location of the difference, when necessary. The level of significance was set at α < 0.05. Unless otherwise stated, data are presented as means ± SE.

RESULTS

All the participants took part in this experimental protocol without reporting discomfort. Specifically, on a scale from 0 to 10, the mean muscle discomfort was 1.5 ± 1.3, 1.6 ± 1.7, 2.1 ± 2.8, and 2.0 ± 2.9 for DS, SS, DS + CCO, and SS + CCO procedures, respectively.

Central, peripheral hemodynamics and [La−] at rest. All resting hemodynamic and [La−] values, with and without CCO are summarized in Figs. 1 and 2, respectively. The difference in resting CO before DS and SS was negligible (P = 0.8); however, after 300 s of CCO, CO was increased by 5% (P < 0.05; Fig. 1A). For all the experimental conditions, the differences in SV at rest were undetectable (Fig. 1B). HR at rest was similar between DS and SS (P = 0.9). However, after 300 s of CCO, HR was significantly increased (~13%; P < 0.05) compared with the values recorded at rest (Fig. 1C). MAP at rest was similar between DS and SS (P = 0.9) and after 300 s of CCO increased significantly (by ~28%; P < 0.05) in both conditions (Fig. 1D). The participants exhibited equivalent FBF at rest for the DS and SS experimental trials (P = 0.8; Fig. 1E). As expected, 300 s of CCO induced a significant reduction (~15%) in FBF (P < 0.05; Fig. 1E). Similarly, to FBF, VC at rest was similar between DS and SS (P = 0.8; Fig. 1F). However, after 300 s of CCO, VC was reduced by 43% (P < 0.05; Fig. 1F). [La−] at rest was similar between DS and SS (P = 0.9; Fig. 2). However, after 300 s of CCO, [La−] was significantly increased (~135%; P < 0.05) compared with the values recorded at rest (Fig. 2).

Central and peripheral hemodynamics during passive stretching procedures. All hemodynamic outcomes recorded during stretch procedures are summarized in Table 1. Peak, Δpeak, and the AUC of the MAP recorded during DS and SS procedures indicate a similar drop in MAP compared with rest (~6%; P = 0.8; Table 1 and Fig. 3A). Of note, MAP kinetics (Fig. 3A) appear slower during the SS compared with DS procedure. In fact, during SS, after an early transitory, 3-s increase in MAP, a distinct drop of the systemic pressure started from 11 s. Then, MAP remained significantly reduced from resting values for 22 s. Contrariwise, the early rise in MAP was not evident during the DS protocol, during which MAP dropped at 6 s, 5 s earlier compared with SS. Interestingly, the changes in MAP from rest

**Fig. 1.** Central, peripheral hemodynamics at rest and after 300 s of cuff occlusion of limb circulation (CCO). A, B, C, D, E, and F: cardiac output (CO), stroke volume (SV), heart rate (HR), mean arterial pressure (MAP), femoral blood flow (FBF), and vascular conductance (VC). Data are means ± SE. *Significantly different from Rest.

**Fig. 2.** Lactate concentration ([La−]) at rest and after 300 s of cuff occlusion of limb circulation (CCO). Data are means ± SE. *Significantly different from Rest.

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were negligible during DS and CCO (~1%; \( P = 0.9 \); Fig. 3B; and Table 1). The MAP recorded during the SS + CCO was significantly increased (~4%; \( P < 0.05 \)) compared with the values recorded at rest (Fig. 3B; and Table 1), implicating a greater \( (P < 0.05) \) MAP response to SS + CCO with respect to DS, SS, and DS + CCO.

During both DS and SS, FBF was transiently increased from 3 to 25 s (Fig. 3C). The stretch-induced hyperemia response, in terms of peak, \( \Delta \)peak, and AUC, was similar between DS and SS (Table 1). As expected, CCO abolished the stretch-induced hyperemia, and the changes from rest in peripheral blood flow parameters (peak, \( \Delta \)peak, and the AUC) were consequently negligible during DS + CCO and SS + CCO (~3%; \( P = 0.8 \); Fig. 3D; and Table 1). Similar responses in VC were recorded during DS and SS (Fig. 3E, panel E; and Table 1). Interestingly, the two stretch procedures executed with CCO generated different VC responses. Specifically, no changes from the resting values were observed during DS + CCO (~8%; \( P = 0.7 \); Fig. 3F; and Table 1), whereas, SS + CCO caused a significant and sustained drop in VC (~31%; \( P < 0.05 \); Fig. 3F; and Table 1).

Both kinetics and changes in SV from rest (Fig. 4A; and Table 1) appear different between DS and SS. Except for the SV peak values, which were similar between DS and SS, the time response of the increase in SV during DS appears slower but of longer duration compared with SS (Fig. 4A). During SS, after an early temporary increase of 3 s, SV progressively declined leading to a significant reduction from resting values during the last 18 s of SS. No changes from the resting values were observed during DS + CCO and SS + CCO (~0.3%; \( P = 0.9 \); Fig. 4B; and Table 1).

During DS, HR became significantly elevated within 6 s and remained elevated for the other 39 s (Fig. 4C). Contrariwise, SS generated a first, transitory cardioacceleration at the beginning, that was followed by a delayed increase in HR in the last 20 s of SS (Fig. 3C). As a consequence of this delay in cardioacceleration, the AUC of HR was significantly lower in CCO (~8%; \( P < 0.05 \)) compared with SS (~32%, \( P < 0.01 \); and Table 1), whereas, SS + CCO showed a significantly faster and larger increase than SS (~41%, \( P < 0.01 \); and Table 1).

During both DS and SS, HR remained stable for the rest of the trial. However, during DS + CCO, the changes in HR during DS remained stable for the rest of the DS + CCO trial were shorter compared with SS + CCO. Nevertheless, the changes in HR were even more pronounced during CCO (~0.3%; \( P = 0.9 \); and Table 1). Additionally, a brief increase in CO, similar to that in SS (Fig. 4, E and F), was recorded at the beginning of SS + CCO. Thereafter, CO remained stable for the rest of the trial.

**DISCUSSION**

Although passive stretching of skeletal muscle, CCO, and the combination of both maneuvers have been already investigated in animal models in relation to central hemodynamics mediated by the mecha- and metaboreflex, the influence of these procedures on central and peripheral hemodynamics in humans has received so far only little attention. In the present study we investigated central and peripheral cardiovascular responses to DS and SS of the human knee extensor muscles. Potential interactions between mecha- and metaboreflex...
were studied by imposing SS and DS over CCO. The main findings of this study were: 1) DS of the quadriceps muscle generated a sustained increase in HR and CO. According to our first hypothesis, SS produced a delayed increase in HR and CO, implicating a reduced mechanoreceptor response compared with DS. Contrary to our second hypothesis, SS evoked a hyperemia similar to DS and a marked drop in MAP.

2) 300 s of CCO generated a significant increase in [La⁻]. Interestingly, this pronounced (135%) increase in [La⁻] in the lower limb undergoing CCO was matched by a significant increase in resting HR, CO, and MAP, implicating a noteworthy metaboreflex activation. However, HR, CO during the DS and SS CCO, and SS CCO were not influenced by this marked increase in peripheral [La⁻]. These results imply that both dynamic and static activation of skeletal muscle mechanoreceptors are not influenced by the accumulation of peripheral metabolites and suggest a marginal interaction between mechano- and metaboreflex in this model.

Central and peripheral hemodynamics interaction during stretching. The present findings highlight and advance the knowledge on the interactions between central and peripheral hemodynamics during passive stretching. As reported in several previous studies and confirmed by the present outcomes, DS of knee extensors induced a central hemodynamic response that was likely generated via group III afferents (27, 38). In fact, as documented in previous studies (36, 39, 40), central hemodynamic responses recorded during pharmacological block of afferent feedbacks (spinal injection of fentanyl) (36) or in individuals with spinal cord injury appear reduced (39, 40). From the current study, the high-resolution analysis of peripheral circulation revealed a marked hyperemia in the passively stretched limb in response to stretching maneuvers (Fig. 3, C and E). Our results during SS are not in agreement with the data reported in a recent study (22), in which the investigators documented no detectable changes in net blood flow, MAP, and VC measured in the popliteal artery during 5 min of plantar flexors SS. This discrepancy could be potentially explained by the volume of the stretched muscle, which, in turn, can generate different NO release and greater hyperemia in a larger muscle. Moreover, being the stretch-induced hyperemia transient in terms of time (15 s), the second-by-second high-resolution analysis of peripheral and central hemodynamics adopted in the current study may have better detected the hemodynamic changes, whereas a 12-s resolution approach adopted by Kruse and coworkers (22) likely underestimated these changes. Our data are in agreement with previous studies that adopted similar technical approach (16 – 20, 27, 38). The brief increase in MAP (~3 s) at the beginning of SS was likely provoked by the modality of SS maneuver, which was held constant for 45 s, and possibly induced ischemia of the quadriceps muscle. This brief increase, for the nature of the dynamic maneuver (1 Hz), was not present during DS. The following drop in MAP, very similar between SS and DS, was likely mediated by peripheral vasoactive factors (e.g., NO release). Indeed, should this phenomenon have been triggered by the baroreflex, the magnitude of MAP drop would

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![Fig. 3. Changes in blood pressure, femoral blood flow and vascular conductance to DS, SS, DS CCO, and SS CCO over time, respectively. Data are means ± SE. Gray area indicates significantly different values among conditions. †Significantly different from baseline in DS and DS CCO. *Significantly different from baseline in SS and SS CCO.](http://jap.physiology.org/)

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have been definitively larger. Interestingly, when comparing the effect of CCO on SS and DS and looking at the change between occluded and freely perfused conditions, it seems that SS exhibited a larger change in delta and AUC MAP, suggesting that SS may result in reduced microvascular perfusion and in greater mechano/metaboreflex interaction. Indeed, as with bulk flow measurements in the common femoral artery with the quadriceps undergoing significant stretch, the hamstring tension should be reduced; thus a portion of the flow could be redirected to a different region. This may not have been the case with the oscillatory DS that undulates between quadriceps and hamstring stretching. Future studies implementing microvascular measures, such as near infrared spectroscopy technique, in combination with a standard Doppler measurement might confirm this hypothesis.

Mechanoreflex response to DS and SS. Data from the current investigation indicate that DS of knee extensor muscles generated a greater increase in HR and CO with respect to SS, implicating a reduced mechanoreceptor response to passive SS (Fig. 4, C and E, and 5A). Accordingly, a previous study suggests that mechanoreceptors are mainly frequency dependent and likely more activated during DS (25). Moreover, in support of this interpretation, McDaniel et al. (28) demonstrated that slow passive movement (1°/s) of knee extensor muscles evokes no cardioacceleration. Conversely, in other studies SS of human calf generated rapid and sustained cardioacceleration (14, 15). On the contrary, DS of the same muscle led to a reduced central response (14). Altogether, our and previous data indicate a specific muscle-dependent response to DS and SS. Specifically, a greater response in mechanoreceptor discharge to DS seems more evident in locomotor/proximal muscles, such as the human knee extensor muscles. On the contrary, greater cardioacceleration in response to SS appears stronger in postural/distal muscles such as the human calf. These different mechanoreceptor responses to DS and SS can be potentially dependent on the functional evolution of these different muscles. For instance, the anatomical position of mechanoreceptors into the muscle, as well as their threshold of activation, may be more susceptible to dynamic triggers in the knee extensors, because these muscles are dynamically utilized during locomotion. Conversely, the mechanoreceptors located into postural muscles such as calf muscles are likely more inclined to a static stimulation.

Interaction between mechano- and metaboreflex. Cardioacceleration recorded during the DS + CCO was reduced compared with DS alone. Similarly, negligible increases in HR and CO were documented during SS + CCO protocol. Taking together, these data suggest that both dynamic and static activation of muscle mechanoreceptors are not influenced by the increase in peripheral [La−], implying a marginal interaction between mechano- and metaboreflex. It is important to note that [La−] is only one of the several metabolites involved in the metaboreceptor activation. Therefore, additional studies are needed to fill this gap. Moreover, Fisher et al. (12) docu-
mented a similar stretch-induced blood pressure increase during SS and SS applied during PECO. Moreover, a similar stretch-induced cardioacceleration was observed during passive DS and DS + CCO of the lower limb (27). Interestingly, our data and the results of these studies suggest a negligible sensitization of muscle mechanoreceptors via local [La\(^{-}\)] accumulation. Contrary to our interpretation, an increased sympathetic outflow was reported during passive SS applied during PECO (7, 8), suggesting a sensitization of muscle mechanoreceptors when metabolites are accumulated. In accordance with our findings, the observed sympathetic activation due to SS was transient, and consequently the hemodynamic consequences were limited in time.

It is important to mention that this lack of interaction between mechano- and metaboreflex in this model is not in agreement with what has been previously postulated that an interaction between the mechanoreflex (group III thinly myelinated fibers) and the metaboreflex (group IV unmyelinated fibers) may explain why the mechanoreflex is overactivated in patients with heart failure (19, 32, 33). Future studies in this population are needed to better elucidate this interaction.

Conclusions. This study documented that DS of the human quadriceps muscle was associated with a sustained increase in HR and CO, presumably mediated by mechanoreflex activation. On the contrary, SS produced a minimal increase in HR and CO, likely caused by the reduced mechanoreceptor response to SS stimulus. The central hemodynamic responses documented during these stretching procedures superimposed with CCO, which was accompanied by a pronounced peripheral [La\(^{-}\)] increase, suggest that both dynamic and static activation of skeletal muscle mechanoreceptors are not influenced by the accumulation of peripheral metabolites in this model.

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AUTHOR CONTRIBUTIONS


DISCLOSURES

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