

# Cardiovascular responses to static and dynamic contraction during comparable workloads in humans

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Received 11 March 2002; accepted in final form 15 May 2002

**Stebbins, Charles L., Buddy Walser, and Mehrdad Jafarzadeh.** Cardiovascular responses to static and dynamic contraction during comparable workloads in humans. *Am J Physiol Regul Integr Comp Physiol* 283: R568–R575, 2002. First published May 23, 2002; 10.1152/ajpregu.00160.2002.—Previous studies suggest that the blood pressure response to static contraction is greater than that caused by dynamic exercise. In anesthetized cats, however, pressor responses to electrically induced static and dynamic contraction of the same muscle group are similar during equivalent workloads and peak tension development [i.e., similar tension-time index (TTI)]. To determine if the same relationship exists in humans, where contraction is voluntary and central command is present, dynamic (180 s; 1/s) and static (90 s) contractions at 30% of maximal voluntary contraction (MVC) were performed. Dynamic contraction also was repeated at the same TTI for 90 s at 60% MVC. Mean arterial pressure (MAP), heart rate (HR), cardiac output (CO), MAP during postexercise arterial occlusion (an index of the metaboreceptor-induced activation of the exercise pressor reflex), and relative perceived exertion (RPE) (an index of central command) were assessed. No differences in these variables were found between static and dynamic contraction at a tension of 30% MVC. During dynamic contraction at 60% MVC, changes in MAP ( $16 \pm 3$  vs.  $19 \pm 4$  mmHg) and absolute HR ( $92 \pm 6$  vs.  $69 \pm 5$  beats/min), CO ( $7.9 \pm 0.4$  vs.  $6.3 \pm 0.3$  l/min), RPE ( $16 \pm 1$  vs.  $13 \pm 1$ ), and MAP during postexercise arterial occlusion ( $115 \pm 3$  vs.  $100 \pm 4$  mmHg) were greater than during static contraction ( $P < 0.05$ ). Thus increases in MAP and HR, activation of central command, and muscle metabolite-induced stimulation of the exercise pressor reflex during static and dynamic contraction in humans seem to be similar when peak tension and TTI are equal. Augmented responses to dynamic contraction at 60% MVC are likely related to greater activation of these two mechanisms.

exercise pressor reflex; central command; brachial artery blood flow; tension-time index

THE INCREASE IN BLOOD PRESSURE associated with exercise is caused by a reflex originating in contracting muscle (i.e., the exercise pressor reflex) (16, 22) and by feed-forward neural drive in the central nervous system to cardiovascular control sites in the medulla (i.e., central command) (12). Results of previous studies suggest

that static contraction causes a more pronounced pressor response than dynamic contraction (17, 24, 28). The proposed explanation for this differential response is a continuous compression of statically contracting skeletal muscle, which attenuates the local blood flow (14). The end result is a reduction in oxygen delivery and a greater production and accumulation of muscle metabolites that evoke the exercise pressor reflex. During dynamic contraction, where active muscle blood flow is greater, enhanced washout of these metabolites presumably leads to a smaller pressor response.

Earlier attempts to compare cardiovascular responses to static and dynamic contraction have been complicated by the fact that comparisons were not made using the same muscle group performing equivalent workloads (5, 31). Because the magnitude of the reflex cardiovascular response to contraction is dependent on the active muscle mass and the magnitude of force or tension production (16), these factors must be controlled if accurate comparisons are to be made between the two modes of contraction. In a previous study of cats (10), these two factors were controlled by integrating tension over time [i.e., using the tension-time index (TTI)] (1) and using the same muscle group (i.e., the triceps surae) to perform dynamic or static contractions. Results revealed that when peak tension was held constant and contraction time was varied between the two types of contraction to maintain a constant TTI, the pressor responses were not different, even though active muscle blood flow was much greater during dynamic contraction. When contraction time was held constant and peak tension was varied to maintain a constant TTI, dynamic contraction caused greater pressor and blood flow responses. Thus, when muscle mass and peak tension development were held constant, the pressor responses to static and dynamic contraction were similar. However, because these experiments were performed in the anesthetized state and muscle contraction was induced by electrical stimulation of the sciatic nerve, it was not possible to determine effects of voluntary contraction and central command on this relationship. Moreover, it is not known if a similar relationship may exist in humans.

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Consequently, the purpose of this study was to compare the cardiovascular response to static and dynamic exercise at equivalent workloads (i.e., TTI) in humans. To this end, we proposed two hypotheses: 1) during static and dynamic contraction at the same peak tension and TTI, increases in blood pressure and heart rate (HR) are not different, even though active muscle blood flow is greater during dynamic exercise; and 2) compared with static contraction, larger increases in blood pressure and HR occur during dynamic contraction when TTI is held constant by increasing peak tension during dynamic exercise.

## METHODS

The Human Subjects in Research Committee, University of California, Davis, approved all procedures and protocols. Ten normal healthy subjects (7 males and 3 females), ranging in age from 20 to 51 yr, gave informed consent. Subjects were instructed to refrain from alcohol and strenuous exercise for 12 h before each experimental period.

**Protocols.** After the subjects reported to the laboratory, they performed two 1- to 2-s peak forearm contractions using a handgrip dynamometer to determine their maximal voluntary contraction (MVC). After a rest period of at least 20 min, the experimental paradigm was initiated, and the following three handgrip protocols were performed with TTI held constant: 1) static contraction for 90 s at a tension producing a 30% MVC, 2) dynamic contraction (1/s) for 90 s at 60% MVC, and 3) dynamic contraction for 180 s at 30% MVC. In 9 of the 10 subjects, a blood pressure cuff was placed around the contracting arm and inflated to a pressure of 200–250 mmHg during the last 5 s of contraction to occlude the brachial artery. Occlusion was maintained for 1 min after exercise to trap metabolites produced by contraction and assess blood pressure as an index of the magnitude of muscle metabolite-evoked activation of the exercise pressor reflex (1).

The order of contraction was randomized among subjects. At least 15 min of rest were provided between contraction periods to allow the cardiovascular variables to return to baseline values. To assist the subjects in maintaining the appropriate peak tension, they received feedback from a strip-chart recording that displayed their force production in real time. During dynamic contraction, a metronome was used to provide a 1/s cadence.

**Measurement of hemodynamic variables.** HR was recorded continuously throughout the experiment using a three-lead electrocardiogram (ECG). An average HR was determined over at least 10 beats during the last 30 s of each exercise period. Systolic (SAP), diastolic (DAP), and mean arterial blood pressure (MAP) were continuously monitored by a Finapres 2300 (Ohmeda, Madison, WI) device. Finapres measures finger arterial blood pressure. Using a fast servosystem attached to an inflatable finger cuff, blood volume in a finger artery is kept constant during each pulse cycle and detected by a photoplethysmograph. The changes in cuff pressure needed to maintain constant blood volume reflect finger arterial blood pressure and were displayed on a monitor and chart recorder. During at least three 15-s intervals at rest and the last 30 s of each contraction period, beat-by-beat values of SAP and DAP were measured and averaged. Because fingertip blood pressure may differ from standard blood pressure measurements, sphygmomanometry was used to assess brachial artery blood pressure and confirm that this pressure was within a normal range. Rate-pressure product ( $R \times P$ ), an index of myocardial oxygen demand, was calculated as  $SAP \times HR$ .

Impedance cardiography was used to continuously measure stroke volume (29) during the last 10–15 s of each contraction period. Each subject was fitted with four band electrodes, two around the neck and two around the mid-lower torso, and the previously mentioned ECG electrodes. The impedance changes created by the variances in blood flow through the torso during each cardiac cycle were detected and displayed on a monitor and a chart recorder simultaneously. The first derivative of the impedance waveform ( $dZ/dt$ ) during each cardiac cycle was used to determine stroke volume. Cardiac output was calculated by multiplying the mean stroke volume of at least five cardiac cycles by the mean HR during the same period of time. MAP was then calculated using the formula:  $MAP = [(SAP - DAP) \div 3] + DAP$ . Systemic vascular resistance was calculated by dividing MAP by cardiac output.

Brachial artery blood flow (BABF) was assessed using pulsed-Doppler ultrasound (Hewlett-Packard, Sonos 2500 Ultrasound System) in 6 of the 10 subjects that participated in the study. This protocol was performed on a separate day, and each subject repeated the previous three contraction protocols at the same TTI and peak tension. The subjects refrained from eating for 2–3 h before the experiment. With subjects in the sitting position, a pulsed-Doppler 7.5/5.0 MHz vascular probe was placed on the medial side of the arm, ~2 cm proximal to the elbow joint. Brachial artery images and pulse wave velocity were recorded to a videocassette recorder and then evaluated. Mean brachial artery blood velocity (MBV) was determined from the spectra of the pulsed-Doppler ultrasound signal and was calculated as the mean of the instantaneous MBV values over a given cardiac cycle, which was defined as the onset of the QRS complex (identified by simultaneous ECG recording) to the onset of the subsequent QRS complex of the next heart beat. The MBV of four to six cardiac cycles was averaged and used as the MBV value for a given measurement. Brachial artery diastolic internal diameter was assessed using two-dimensional echo-Doppler. Two to three measurements were made and averaged to determine internal diameter. BABF was calculated as the product of brachial artery cross-sectional area [ $\pi \times (\text{arterial radius})^2$ ] and MBV and is expressed in milliliters per minute. Reactive hyperemia was determined by evaluating BABF over the first two to three beats after the end of contraction. At least 15–30 min of rest were provided between contraction periods to allow MBV and brachial artery internal diameter to return to baseline values.

**Measurement of relative perceived exertion.** Relative perceived exertion (RPE) was determined as an estimate of the level of activation of central command (13, 30). The Borg scale (7) was used for rating RPE among the three contraction paradigms. According to the Borg 6–20 scale, levels of exertion are rated as follows: very, very light (6–8), very light (9–10), light (11–12), somewhat hard (13–14), hard (15–16), very hard (17–18), and very, very hard (19–20).

**Data analysis.** All data are expressed as means  $\pm$  SE. Repeated measures ANOVA was utilized to detect significant differences among means across baseline and contraction conditions. The Student-Newman-Keuls multiple comparison test was applied to make simultaneous multiple comparisons of differences between means when *F* ratios from the ANOVA indicated statistical significance ( $P \leq 0.05$ ). Baseline levels for all variables were not significantly different among the three contraction conditions (repeated measures ANOVA). Therefore, these values were pooled and averaged to serve as a single baseline group for each variable for comparison with the contraction groups. RPE, brachial artery reactive hyperemia, and blood pressure during postex-

ercise arterial occlusion were only compared among the three contraction groups because these variables did not have a corresponding baseline value. The blood pressure response (SAP, DAP, and MAP) to exercise is presented both as absolute values and changes from baseline values. Changes from baseline were assessed because the Finapres can measure absolute blood pressures during exercise (particularly SAP) that are higher than those measured in the brachial artery via sphygmomanometry (21). Thus relative changes in these variables are necessary to fully interpret blood pressure responses to exercise.

## RESULTS

When TTI was compared among the three contraction protocols, no statistically significant differences

were found ( $1,013 \pm 124$ ,  $1,007 \pm 133$ , and  $973 \pm 110$   $\text{kg}\cdot\text{s}$  for 30% MVC static, 30% MVC dynamic, and 60% MVC dynamic contraction, respectively). Thus the workloads were judged to be equivalent. The subjects did not find the 30% MVC static and dynamic contractions to be fatiguing. However, three subjects reported some sensation of fatigue during the last 5–10 s of 60% MVC dynamic contraction.

Fingertip SAP, DAP, and MAP, HR, and  $R \times P$  product were not different during the 30% MVC static and dynamic contractions (Fig. 1). However, all five of these variables were higher during 60% MVC dynamic contraction than during the other two (Fig. 1). The pattern for changes in fingertip SAP, DAP, and MAP

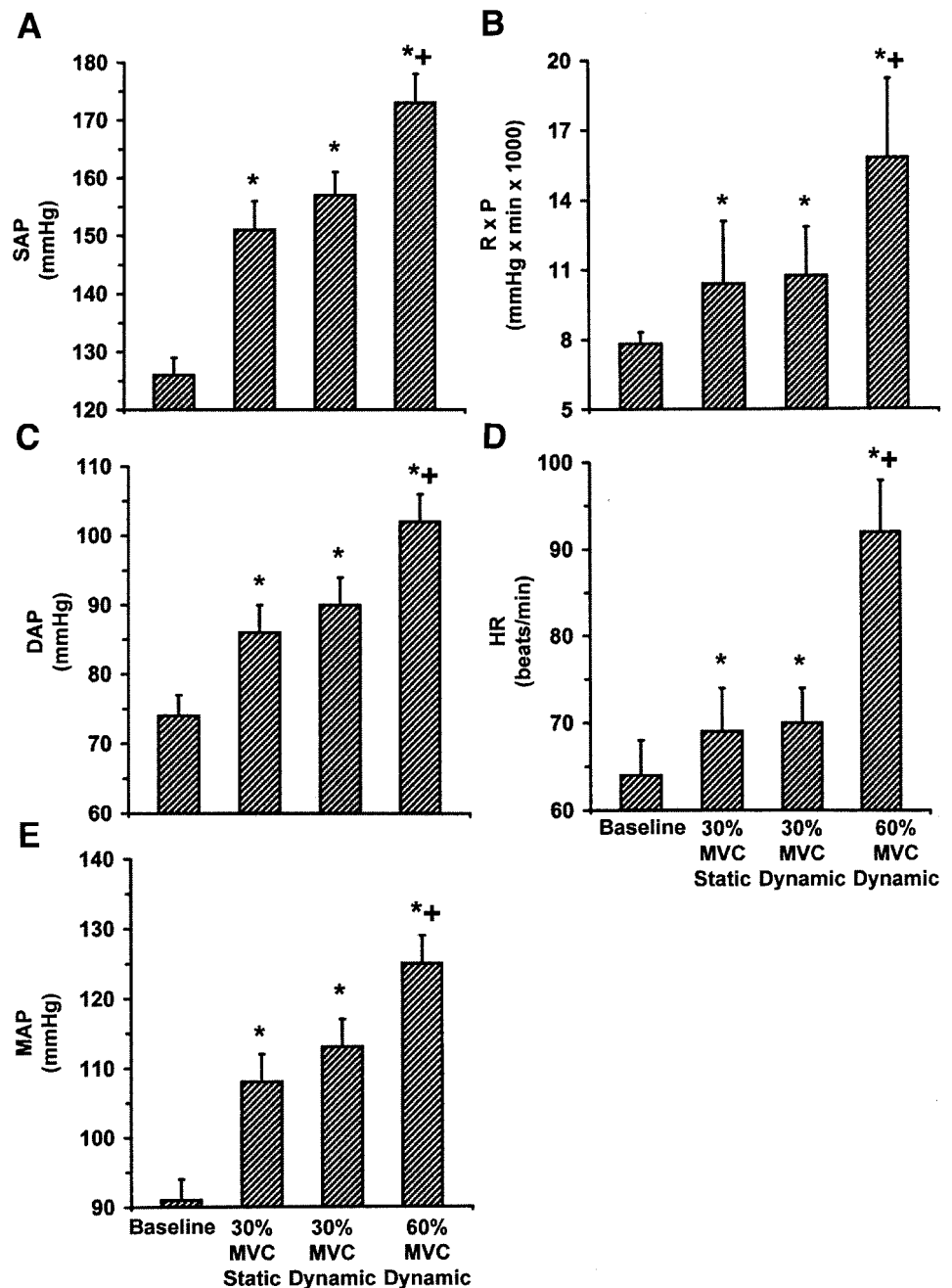


Fig. 1. Peak systolic blood pressure (SAP; A), rate  $\times$  systolic pressure product ( $R \times P$ ; B), diastolic blood pressure (DAP; C), heart rate (HR; D), and mean arterial pressure (MAP; E) at baseline and during 30% maximal voluntary contraction (MVC) static, 30% MVC dynamic, and 60% MVC dynamic contractions. \* $P < 0.05$  vs. baseline; + $P < 0.05$  vs. 30% MVC static and 30% MVC dynamic contractions.

was identical to that seen for the absolute measurements (Fig. 2). Average pooled values of baseline brachial artery blood pressure were  $111 \pm 3$ ,  $69 \pm 2$ , and  $84 \pm 2$  mmHg for SAP, DAP, and MAP, respectively.

Stroke volume and systemic vascular resistance did not increase during any of the contraction protocols (Fig. 3). Cardiac output increased in response to the 30% and 60% MVC dynamic contractions (Fig. 3). Although this variable also increased in each subject during the 30% MVC static contraction, statistical significance was not achieved. The highest cardiac output occurred during dynamic contraction at 60% MVC, while no difference in this variable was found between the other two paradigms (Fig. 3).

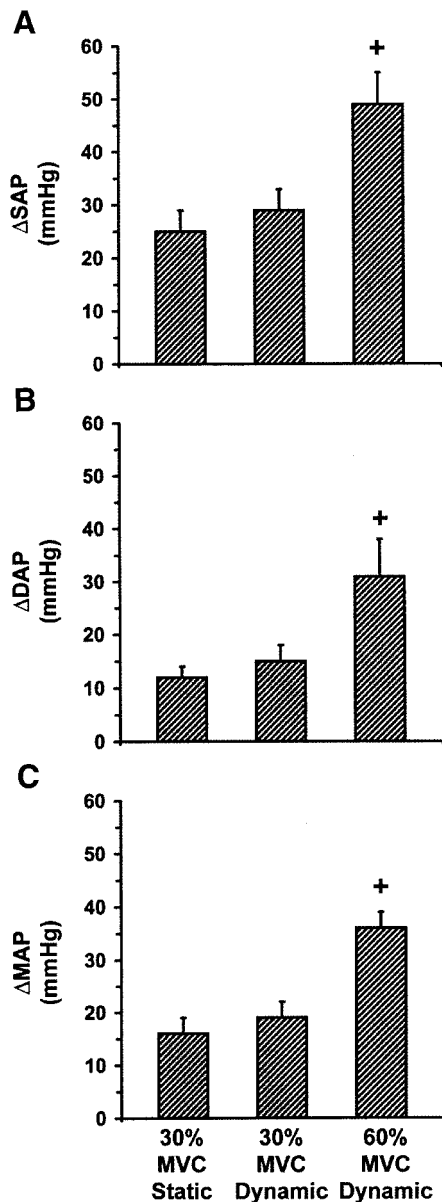


Fig. 2. Changes in SAP ( $\Delta$ SAP; A), DAP ( $\Delta$ DAP; B), and MAP ( $\Delta$ MAP; C) from respective baseline values during 30% MVC static, 30% MVC dynamic, and 60% MVC dynamic contractions. + $P < 0.05$  vs. 30% MVC static and 30% MVC dynamic contractions.

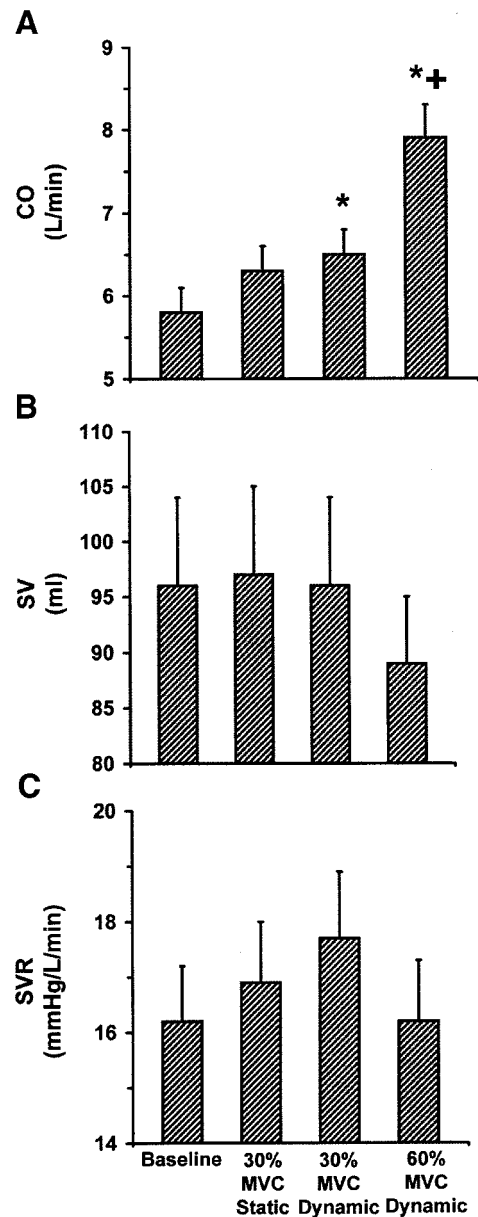


Fig. 3. Peak cardiac output (CO; A), stroke volume (SV; B), and systemic vascular resistance (SVR; C) at baseline and during 30% MVC static, 30% MVC dynamic, and 60% MVC dynamic contractions. \* $P < 0.05$ , vs. baseline; + $P < 0.05$  vs. 30% MVC static and 30% MVC dynamic contractions.

BABF was greatest during the 60% MVC dynamic contraction. Moreover, blood flow during 30% MVC dynamic contraction was higher than during 30% MVC static contraction (Fig. 4A). The highest brachial artery reactive hyperemia was seen after 60% MVC dynamic contraction. After the 30% MVC static and dynamic contractions, reactive hyperemia was similar (Fig. 4B).

During postexercise arterial occlusion, SAP, DAP, and MAP were not different between the 30% MVC static and dynamic contraction protocols (Fig. 5). These three variables were highest in the 60% MVC contraction paradigm (Fig. 5).

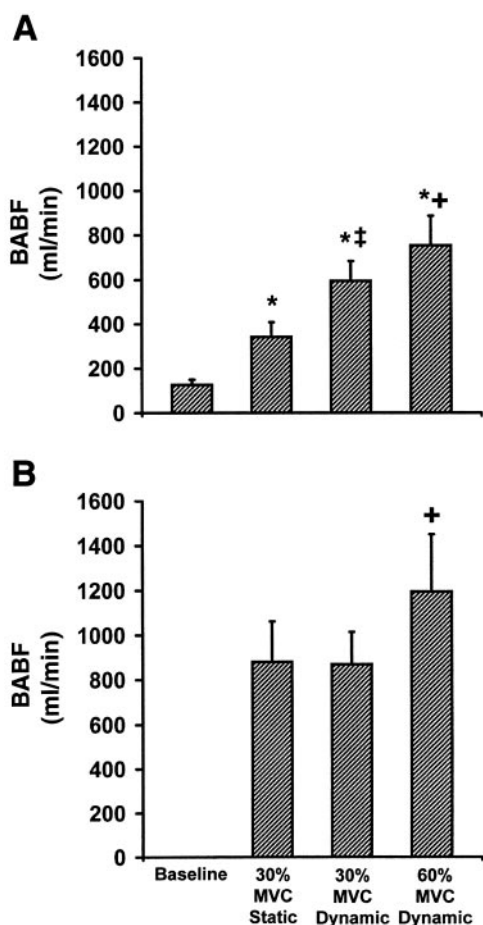


Fig. 4. Brachial artery blood flow (BABF) at baseline and during exercise (A) and reactive hyperemia (B) in response to 30% MVC static, 30% MVC dynamic, and 60% MVC dynamic contractions. \* $P < 0.05$  vs. baseline, + $P < 0.05$  vs. 30% MVC static and 30% MVC dynamic contractions, † $P < 0.05$  vs. 30% MVC static contraction.

Dynamic contraction at 60% MVC evoked the highest level of RPE (Fig. 6). The levels of RPE seen during 30% static and dynamic contraction were not different (Fig. 6).

## DISCUSSION

This investigation confirmed our first hypothesis that the pressor response (both absolute and relative) to static and dynamic contraction is not different when the same muscle group is used and peak tension and TTI are held constant, even though active muscle blood flow evoked by dynamic contraction was higher. Activation of central command (as estimated by RPE) and muscle metabolite-induced stimulation of the exercise pressor reflex (as estimated by the blood pressure response to postexercise arterial occlusion) also did not appear to be different between these two conditions.

It seems paradoxical that the pressor response to both static and dynamic contraction was the same when TTI and peak tension were held constant, because the increase in blood flow during dynamic contraction was over twofold greater. Based on the "wash-out hypothesis," a greater removal of metabolites that

cause the exercise pressor reflex during dynamic contraction should have reduced its magnitude compared with static exercise. However, previous studies in animals have revealed that intermittent contractions actually cause greater increases in muscle metabolism and production of muscle metabolites than those that are continuous. This effect is due to greater energy of activation associated with the onset of each dynamic contraction compared with a single energy of activation during static contraction (9, 15, 27). Because many of these muscle metabolites also cause vasodilation during dynamic exercise, it is possible that any increase in their production during this mode of contraction was offset by a local increase in their removal by the circulation. This contention is supported by our finding that

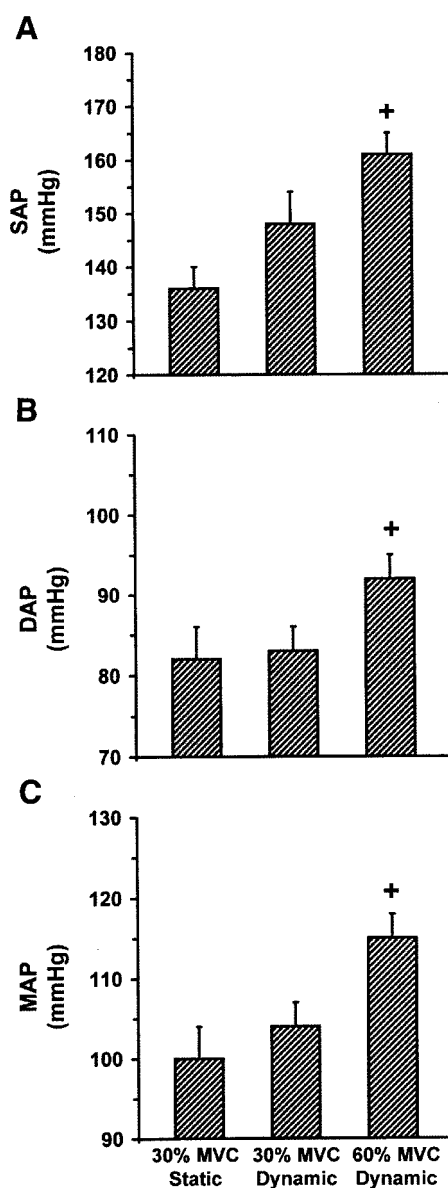


Fig. 5. SAP (A), DAP (B), and MAP (C) during postexercise occlusion of the brachial artery in response to 30% MVC static, 30% MVC dynamic, and 60% MVC dynamic contractions. + $P < 0.05$  vs. 30% MVC static and 30% MVC dynamic contractions.

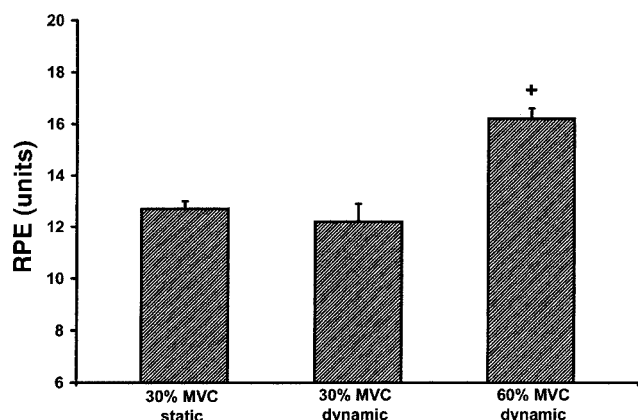


Fig. 6. Relative perceived exertion (RPE) in response to 30% MVC static, 30% MVC dynamic, and 60% MVC dynamic contractions. + $P < 0.05$  vs. 30% MVC static and 30% MVC dynamic contractions.

reactive hyperemia was comparable after the 30% MVC static and dynamic contractions. Moreover, similar blood pressure responses to postexercise arterial occlusion suggest that accumulation of muscle metabolites that evoke the exercise pressor response was also similar during these two types of contraction.

Taken together, our findings lend support to the possibility that central command and muscle metaboreceptor activation of the exercise pressor reflex were similar during both modes of contraction under these conditions. Accordingly, it is reasonable to expect that the corresponding cardiovascular responses would also be analogous.

Our second hypothesis also was confirmed by the results of this study. In this situation, when contraction time was held constant and TTI was equated by increasing peak tension development during dynamic contraction, the blood pressure response (both absolute and relative) and RPE were greater during dynamic than static contraction. What is more, blood pressure during postexercise occlusion of the brachial artery was greater than that seen after static contraction. This occurrence is consistent with a greater dynamic contraction-induced production and/or accumulation of muscle metabolites that cause the exercise pressor reflex.

Compared with static contraction, the higher peak tension produced during dynamic contraction probably also caused a greater activation of skeletal muscle mechanoreceptors that contribute to the exercise pressor reflex. Although we have no data to support this contention, selective activation of muscle mechanoreceptors (using passive muscle stretch in the cat) has been shown to cause a greater blood pressure response during dynamic muscle stretch compared with static stretch at the same TTI (10). This outcome was seen when peak tension produced by dynamic muscle stretch was greater than that seen during static stretch, but not when peak tension between the two modes of contraction was the same. A study in humans also used passive stretch to demonstrate that changes in the level of muscle mechanoreceptor activation con-

tribute to the reflex pressor response to muscle contraction (4). Interestingly, it was also revealed that mechanical stimulation of muscle afferents appears to contribute to the initial blood pressure response during contraction, but metabolic stimuli were necessary to maintain this response.

Our interpretation of the previous data indicates that the augmented pressor response to dynamic compared with static contraction, when TTI is held constant by increasing peak tension development during dynamic exercise, is apparently due to greater stimulation of muscle chemoreceptors and mechanoreceptors and possibly enhanced activation of central command. However, the use of RPE as an index of central command has to be viewed cautiously under these circumstances. Fatigue may affect RPE independently of central command (26), and three of our subjects reported some sensation of fatigue during the 60% dynamic contractions.

Earlier studies in humans have compared the cardiovascular response to static and dynamic contraction by attempting to equate workloads in a variety of different ways that have yielded equivocal results. In studies where peak tension production during static and dynamic contraction were similar, increases in MAP were not different (19), and the HR response was found to be the same or higher during dynamic contraction (11, 19). However, the contraction periods in these studies were either the same or very close to the same during both types of exercise. Consequently, TTI was undoubtedly lower during dynamic contraction, and, as a result, the workloads were not equivalent.

Data from studies where the pressor and HR responses to static and dynamic contraction were compared at the same whole body oxygen uptake suggest that these responses are higher during static contraction (2, 6). Although using oxygen uptake to standardize workloads during static and dynamic contraction seems like a reasonable approach, there are some inherent problems with its application, especially when different muscle groups perform each type of contraction. For example, due to higher active muscle blood flow and oxygen delivery, oxygen consumption ( $\dot{V}O_2$ ) in response to dynamic contraction might be higher than that seen during static contraction. Accordingly, a more intense static contraction would be necessary to match oxygen uptakes. As a result, a given absolute  $\dot{V}O_2$  could represent a lower relative oxygen consumption and relative work intensity during dynamic contraction and would probably result in a smaller reflex-induced pressor response compared with static exercise. A likely explanation for the lower pressor response induced by dynamic contraction is an apparent lack of tight coupling of whole body  $\dot{V}O_2$  to sympathetic nerve activity during exercise. In this regard, Saito and Mano (25) found that static contraction performed by the legs required a lower  $\dot{V}O_2$  than dynamic cycling but evoked a greater increase in muscle sympathetic nerve activity. This observation lead these investigators to conclude that changes in sympathetic nerve activity are primarily determined by metabolic

changes in contracting muscle, not by absolute changes in whole body  $\dot{V}O_2$ . Therefore, matching  $\dot{V}O_2$  to equate workloads may lead to comparisons of cardiovascular responses to dynamic and static contraction that are inaccurate and misleading.

Not all of the cardiovascular variables measured in the present study responded to dynamic exercise in a manner that was consistent with the generally accepted response pattern for this type of activity. Neither systemic vascular resistance, which characteristically decreases during dynamic exercise (8), nor stroke volume, which typically increases (8), changed from resting conditions. In fact, the lack of change in these two variables appears to be more compatible with static exercise (14, 20). The reason for this similar pattern of response to both types of contraction is probably related to the amount of muscle mass involved. Blomqvist et al. (6) revealed that progressive reductions in the mass of dynamically contracting skeletal muscle result in smaller changes in stroke volume and systemic vascular resistance, even though the pressor response may still be large. Eventually, contraction of the smallest muscle mass studied demonstrated a cardiovascular response pattern that was comparable to that evoked by static contraction.

**Summary and conclusions.** The results of this study indicate that pressor responses to static and dynamic contraction of the same small muscle mass, generating the same TTI and peak tension, are equivalent. The fact that blood pressure during postexercise arterial occlusion and relative perceived exertion also were similar suggests that there was no difference in activation of central command or the exercise pressor reflex via muscle metabolites under these conditions. The higher active muscle blood flow during dynamic contraction was probably due to greater energy of activation and enhanced production of vasodilator metabolites compared with static contraction. On the other hand, when TTI was equated by holding contraction time constant and increasing peak tension development during dynamic contraction, a greater increase in blood pressure occurred during this mode of exercise than during static contraction. This type of dynamic contraction also elevated peak tension production, RPE, and blood pressure during postexercise occlusion of the brachial artery, which suggests that there was greater activation of the exercise pressor reflex and central command. If this were the case, it is not surprising that the cardiovascular response was larger compared with static contraction in this situation.

### Perspectives

Although the benefits of resistance (static) training on developing and maintaining muscular strength and endurance have been known for some time (3), application of this type of exercise to individuals with cardiovascular disease has largely been avoided. This avoidance probably relates to fear of exacerbating symptoms of cardiovascular disease due to the higher afterload and stress on the heart compared with dy-

namic contraction of large muscle groups (23) and to a general lack of understanding of how to optimally prescribe this type of exercise (18). Because many patients with heart disease suffer from deconditioning and muscle atrophy and weakness, they stand to benefit from regular resistance activity that improves neuromuscular function (18). Our findings suggest that the level of activation of the mechanisms responsible for the blood pressure response to exercise (i.e., central command and muscle metabolite-induced activation of the exercise pressor reflex) and myocardial oxygen demand (i.e.,  $R \times P$ ) were similar when dynamic and static contractions were performed by the same small muscle mass, producing identical peak tension. Consequently, the magnitude of the pressor response, afterload, and work of the heart also were not different between the two modes of muscular activity. This outcome suggests that paradigms for resistance exercise of small muscle groups could be designed that do not present a greater risk to cardiovascular patients than those prescribed for dynamic exercise.

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