Role of muscle mass and mode of contraction in circulatory responses to exercise

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LEWIS, STEVEN F., PETER G. SNELL, W. FRED TAYLOR, MARY HAMRA, ROBERT M. GRAHAM, WILLIAM A. PETTINGER, AND C. GUNNAR BLOMQVIST. *Role of muscle mass and mode of contraction in circulatory responses to exercise.* J. Appl. Physiol. 58(1): 146–151, 1985.—The roles of the mode of contraction (i.e., dynamic or static) and the active muscle mass as determinants of the cardiovascular responses to exercise were studied. Six healthy men performed static handgrip (SHG), dynamic handgrip (DHG), static two-knee extension (SKE), and dynamic two-knee extension (DKE) to local muscular fatigue in ~6 min. Increases in mean arterial pressure were similar for each mode of contraction, 29 ± 5 and 30 ± 3 mmHg in SHG and DHG and 56 ± 2 and 48 ± 2 mmHg in SKE and DKE ($P > 0.05$) but larger for KE than HG ($P < 0.001$). Cardiac output increased more for dynamic than for static exercise and for each mode more for KE than HG ($P < 0.001$). Systemic resistance was lower for dynamic than static exercise and fell from resting levels by ~⅓ during DKE. The magnitude of the pressor response was related to the active muscle mass but independent of the contraction mode. However, the mode of contraction affected the circulatory changes contributing to the pressor response. Equalization of the pressor responses was achieved by proportionately larger increases in cardiac output during dynamic exercise.

blood pressure; heart rate; cardiac output; systemic vascular resistance; norepinephrine; epinephrine; dynamic and isometric muscular contractions

COMPARISONS OF STATIC HANDGRIP with dynamic treadmill or bicycle exercise have ascribed unique hemodynamic responses to each mode of contraction (i.e., static vs. dynamic) (16). In static handgrip there is a marked increase in mean arterial pressure normally brought about by modest increases in heart rate and cardiac output with little or no change in total peripheral resistance. In contrast, treadmill and bicycle exercise feature larger rises in heart rate and cardiac output, a marked decline in peripheral resistance, and no change or a small elevation in mean arterial pressure. Recent studies have shown that during dynamic exercise with small muscle mass the hemodynamic response is similar to the response to static exercise (5, 14, 15). These findings suggest that the mode of contraction is a minor determinant of the hemodynamic response during small muscle group effort, but there have been few comparisons of the cardiovascular responses to static and dynamic exercise of larger muscle groups (2).

The purpose of the present study was to characterize the roles of the active muscle mass and the mode of contraction as determinants of the circulatory responses to exercise. We compared the systemic circulatory responses to static and dynamic exercise with identical small- and intermediate-size muscle groups. Static and dynamic handgrip and two-knee extension exercise were performed to a common end point of local muscle fatigue. Because of the restricted blood flow in fatiguing static exercise O_2 uptake correlates poorly with energy requirements (2), but the O_2 uptake achieved during maximal dynamic exercise with each muscle group may be used as an estimate of active muscle mass (5, 15). Our main hypothesis was that the mean arterial pressure response to exercise is determined primarily by the active muscle mass. However, the mode of contraction was expected to influence the types of circulatory adjustments contributing to the pressor response.

METHODS

Subjects. The subjects were six young healthy men. Mean values for their physical characteristics were age 27 ± 3 (SE) yr; height 181.8 ± 6.3 cm; weight 74.6 ± 8.7 kg; maximal O_2 uptake (bicycle exercise) 41.7 ± 2.3 ml·kg^{-1}·min^{-1}. No subject performed regular physical training during the period of the study. Four subjects were thoroughly accustomed to the laboratory environment from participation in previous experiments (5, 13, 14). The research protocol was approved by the Institutional Review Board, and each subject gave his signed informed consent to all procedures used.

Procedures. Each participant underwent four to five preliminary sessions during which he was familiarized with the apparatus and experimental protocol. Preliminary sessions were used to establish maximal voluntary contraction (MVC) for static handgrip and static knee extension, to determine work loads which produced local muscular fatigue in ~6 min for each muscle group and mode of contraction, and to establish tidal volumes for acetylene rebreathing cardiac outputs at rest and for

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individual work loads. For static handgrip MVC was measured using a Stoelting dynamometer. For two-knee extension MVC was measured with the subject seated in a specially designed quadriceps exercise chair with both knees at 90° flexion. A bar placed 38 cm below the axis of rotation of the knee joints served as the contact point for a force transducer. Maximal force was displayed on a digital peak detector. For both static handgrip and two-knee extension MVC was determined as the highest value achieved in at least three trials.

In the definitive experiments, measurements were obtained at rest and during separate static and dynamic handgrip and two-knee extension exercise. All measurements were made while the subject sat upright in a quadriceps exercise chair. The subjects were seated with their knees flexed at an angle of 90° and their feet not in contact with the floor. The arms were not used for support. Static and dynamic handgrip were performed with the Stoelting dynamometer and an AMF-Whitley spring-loaded grip device, respectively. Three subjects performed static handgrip with the dominant hand and three performed dynamic handgrip with the dominant hand. The grip width of the Stoelting dynamometer was individually adjusted for each subject's hand size and maintained constant for all sessions. For static handgrip the load was 24 ± 1% of MVC. For dynamic handgrip the grip rate which elicited local muscular exhaustion varied between 33 and 40 contractions/min at a force of 108 N. This grip force was measured by fixing one handle of the AMF-Whitley grip device horizontally in a vice and suspending precalibrated weights in increments from a string attached to the other (above) handle until the handles just touched each other. Complete closure of the handles of the grip device was achieved for each dynamic contraction. The closure distance was 7 cm. Power output (W) was calculated by multiplying the number of contractions/min, the grip force, and the closure distance and using the appropriate conversion factors. Static and dynamic two-knee extension were performed with cuffs fastened around each ankle that were connected to a weight-pulley system. For static two-knee extension the weights were held elevated with both knees at 90° flexion. The combined force generated by both legs represented 25 ± 1% of MVC. For dynamic two-knee extension both knees were fully extended 20 times/min. The combined weight lifted by both legs varied between 22.7 and 45.4 kg among the subjects (mean = 33.0 ± 3.4 kg), and the elevation of the lift was ~40 cm. Power output was calculated by multiplying the number of lifts per minute, the combined weight lifted by both legs, and the elevation of the lift and converting to watts. Dynamic handgrip and two-knee extension were done to the cadence contract-relax-rest according to a metronome. An inability to maintain the designated tension in static handgrip and a failure to keep the weights elevated in static two-knee extension were used as objective criteria for muscular fatigue. In dynamic handgrip muscular fatigue was defined as the inability to maintain a given contraction rate or achieve complete closure of the grip device. In dynamic two-knee extension muscular fatigue was defined as a failure to extend fully both knees in three successive contractions or a noticeable use of accessory muscles. For the definitive protocol no more than two exercise tests were performed each day. If two tests were performed on one day, one handgrip test (static or dynamic) and one two-knee extension test (alternate mode of contraction) always were done. The handgrip was always performed first, and a 15- to 20-min rest was allowed between tests. Measurements at rest were obtained prior to exercise on each testing day. Thus two to four separate sets of definitive rest measurements were obtained for each subject. With the exception of the plasma epinephrine and norepinephrine data, there were no significant differences between sets, and the rest data presented in Table 1 represent the average of the two to four determinations.

The subjects were requested to fast and abstain from smoking for at least 2 h prior to each session and not to perform heavy exercise during the 24 h preceding any test. Resting measurements were obtained after the subject sat quietly for at least 5 min. Expired air for measurement of O_2 uptake was collected in Douglas bags at rest and during the last 2 min of each 5- to 6-min work period. Fractions of O_2, CO_2, and N_2 in expired air were determined with a Searle Medspec or a Perkin-Elmer 1100A mass spectrometer. Expiratory minute volume was measured with a Tissot spirometer. Cardiac output was determined at rest and in the last 15–20 s of each exercise by our version of the acetylene-rebreathing technique (28). During the rebreathing procedures a tape recording of breath sounds was used to induce a uniform respiratory rate of 18/min at rest and during exercise. Detailed descriptions of the O_2 uptake and cardiac output methods used have been given previously (15, 28).

Indirect systolic and diastolic brachial arterial pressures were obtained in an inactive arm at rest, every 30 s during exercise, and simultaneously with cardiac output measurements using a Narco Bio-Systems electrosphygmomanometer (model PE 3000) that provided an analogue output of arterial pulse sounds superimposed on the cuff pressure during the inflation-deflation cycle. Performance data for this apparatus have been published elsewhere (1). Heart rate was determined from continuously recorded Frank orthogonal lead electrocardiographic tracings and R-R interval counting using at least 20 consecutive beats. An Elema-Schonander eight-channel recorder was used for registration of electrocardiogram, blood pressure, and acetylene and helium concentrations during rebreathing. At rest and immediately postexercise a 3- to 5-ml blood sample was drawn from a small catheter placed in an antecubital vein for analysis of plasma epinephrine and norepinephrine by a radioenzymatic technique (21).

The following derived variables were calculated from primary measurements: stroke volume (ml) = cardiac output/heart rate; mean arterial pressure (mmHg) = diastolic pressure + ⅓ pulse pressure; total peripheral resistance (units) = mean arterial pressure/cardiac output; arteriovenous O_2 difference (ml/100 ml) = O_2 uptake/cardiac output.

In four subjects vascular conductance in the inactive forearm was measured during dynamic and static two-

knee extension exercise. These experiments were performed on separate days using work loads and procedures identical to those described above. Forearm vascular conductance was calculated as the ratio of forearm blood flow ($ml \cdot min^{-1} \cdot 100$ ml $tissue^{-1}$) to mean arterial pressure (mmHg) and expressed in conductance units $\times 10^3$. Forearm blood flow was measured by the venous occlusion plethysmography technique (26) using an air-filled latex cuff. Motion artifact was limited by supporting the arm in a crutch-sling.

In three subjects the tension generated during two-knee extension was recorded continuously on the Elema-Schonander recorder using strain gages inserted in series with the pulley cables extending behind each ankle. These studies also were performed on separate days using work loads identical to those described above. The continuous recordings permitted measurement of the transient tensions developed during the initial phase of each contraction, i.e., when inertia had to be overcome to begin lifting the weights.

Data analysis. A one-way analysis of variance (ANOVA) with repeated measures (30) was used to determine the statistical significance of the differences among the cardiovascular data obtained for the five experimental conditions, i.e., rest, static handgrip, dynamic handgrip, static two-knee extension, and dynamic two-knee extension. The significance of specific differences between experimental conditions was determined with a Neuman-Keuls multiple comparison test. A different procedure was used for analysis of the plasma catecholamine data because of the variability of the resting values. Paired *t* tests were used to determine the statistical significance of the differences between the increases in plasma epinephrine and norepinephrine levels in response to exercise and zero. Statistical comparisons of the increases in plasma epinephrine and norepinephrine across the two modes of contraction (static vs. dynamic) and the two active muscle groups (handgrip vs. two-knee extension) were made by a two-way ANOVA with repeated measures (30). For all tests a difference was considered statistically significant if $P < 0.05$.

RESULTS

Tension, work load, and endurance time. For static handgrip the force developed was 128 ± 5 (SE) N, and for static two-knee extension the force developed was 301 ± 19 N. For dynamic handgrip the power output was 4.7 ± 0.2 W, and for dynamic two-knee extension the power output was 43. 2 ± 4.0 W.

The endurance times were 362 ± 7 and 354 ± 6 s for static and dynamic handgrip and 324 ± 4 and 341 ± 5 s for static and dynamic knee extension, respectively. The endurance time was slightly shorter for static knee extension than for dynamic knee extension or static handgrip ($P < 0.05$). The tension developed transiently at the onset of each dynamic knee extension contraction was 36–47% (mean = 42%) greater than that achieved when the same weights were held elevated in a static contraction. The duration of this initial phase was ~0.4 s.

Circulatory responses. O_2 uptake increased by 83 ± 18 ml/min during static handgrip and by 155 ± 66 ml/min during dynamic handgrip (both $P < 0.05$). Increases of 505 ± 61 and 920 ± 54 ml/min were observed in static and dynamic two-knee extension (both $P < 0.001$). For each mode of contraction O_2 uptake increased more for knee extension than for handgrip (both $P < 0.001$; Table 1). These data suggest that the active muscle mass was approximately six times larger during knee extension than during handgrip. A larger increase in O_2 uptake was observed in dynamic than in static knee extension ($P < 0.001$).

Heart rate increased by 15 ± 4 and 23 ± 7 beats/min during static and dynamic handgrip (both $P < 0.05$) and by 58 ± 12 and 52 ± 8 beats/min during static and dynamic two-knee extension (both $P < 0.001$). The mode of contraction did not affect the magnitude of increase in heart rate but there was a greater increase for knee extension than for handgrip ($P < 0.01$; Table 1).

Cardiac output increased by 1.3 ± 0.4 and 1.9 ± 0.3 l/min in response to static and dynamic handgrip (both $P < 0.05$) and by 4.6 ± 0.8 and 7.6 ± 0.8 l/min during static and dynamic two-knee extension (both $P < 0.001$). A larger increase was observed in knee extension than in handgrip ($P < 0.001$) regardless of the mode of contraction. The increase in cardiac output was significantly greater for dynamic than for static knee extension ($P < 0.001$), but for handgrip similar changes were observed for each mode of contraction ($P > 0.05$).

Stroke volume rose by 29 ± 8 ml during dynamic knee extension ($P < 0.001$) and showed little or no change from resting levels in static knee extension or static or dynamic handgrip.

Systolic arterial pressure increased by 32 ± 6 and 33

TABLE 1. *Data at rest and during exercise*

	Rest	Contraction	Handgrip	Two-Knee Extension
O_2 uptake, ml/min STPD	275 ± 17	S	358 ± 23	780 ± 76*
		D	430 ± 73	1,195 ± 64*†
Heart rate, beats/min	76 ± 3	S	91 ± 4	134 ± 11*
		D	99 ± 8	128 ± 8*
Cardiac output, l/min	5.46 ± 0.34	S	6.80 ± 0.28	10.08 ± 0.92*
		D	7.38 ± 0.40	13.06 ± 0.98*†
Stroke volume, ml	72 ± 5	S	76 ± 5	77 ± 8
		D	76 ± 5	101 ± 10*†
Systolic pressure, mmHg	118 ± 3	S	150 ± 6	193 ± 7*
		D	151 ± 4	193 ± 10*
Diastolic pressure, mmHg	67 ± 3	S	94 ± 4	114 ± 2*
		D	95 ± 3	101 ± 2†
Mean arterial pressure, mmHg	84 ± 2	S	113 ± 4	140 ± 3*
		D	114 ± 2	132 ± 3*
Total peripheral resistance, units	15.6 ± 0.6	S	16.8 ± 1.0	14.5 ± 1.3
		D	15.7 ± 1.2	10.3 ± 0.6*†
Arteriovenous O_2 difference, ml/100 ml	5.06 ± 0.15	S	5.33 ± 0.44	7.88 ± 0.62*
		D	5.83 ± 0.85	9.26 ± 0.39*†
ΔPlasma norepinephrine, pg/ml		S	94 ± 32	91 ± 54
		D	121 ± 59†	230 ± 92†
ΔPlasma epinephrine, pg/ml		S	21 ± 10	36 ± 25
		D	25 ± 13	21 ± 10

Values are means ± SE. S, static; D, dynamic; Δ, exercise minus rest values. * Values significantly different from those for handgrip ($P < 0.05$). † Values significantly different from those for static contraction ($P < 0.05$).

± 3 mmHg in static and dynamic handgrip and by 75 ± 5 and 75 ± 7 mmHg in static and dynamic two-knee extension ($P < 0.001$ for each). Systolic pressure increased more in knee extension than in handgrip regardless of the mode of contraction ($P < 0.001$).

Diastolic arterial pressure increased by 27 ± 5 and 28 ± 4 mmHg in static and dynamic handgrip and by 47 ± 3 and 34 ± 4 in static and dynamic two-knee extension ($P < 0.001$ for each). The increase in diastolic pressure in static two-knee extension was greater than that in dynamic two-knee extension ($P < 0.01$) and that in static handgrip ($P < 0.01$).

Mean arterial pressure increased by 29 ± 5 and 30 ± 3 mmHg in static and dynamic handgrip and by 56 ± 2 and 48 ± 2 mmHg in static and dynamic two-knee extension ($P < 0.001$ for each). For each mode of contraction the increase for knee extension was greater than for handgrip ($P < 0.001$), but for each muscle group mean arterial pressure increased to a similar extent ($P > 0.05$) for both modes of contraction.

Total peripheral resistance decreased by 5.3 ± 0.6 units in dynamic knee extension ($P < 0.001$) but did not change significantly from resting levels during static knee extension or each mode of handgrip. Total peripheral resistance was lower during dynamic knee extension than during dynamic handgrip or static knee extension ($P < 0.001$).

Mean forearm vascular conductance in the contralateral inactive limb increased slightly during both static (3.2 ± 5.0 units) and dynamic (2.8 ± 2.1 units) two-knee extension. The mean increases in forearm vascular conductance, respectively, corresponded to 67 and 38%, but there was considerable interindividual variation, and the changes were not significantly different from zero. Arteriovenous O_2 difference did not increase significantly in response to static or dynamic handgrip. During static and dynamic two-knee extension arteriovenous O_2 difference increased by 2.82 ± 0.56 and 4.20 ± 0.37 ml/100 ml, respectively (both $P < 0.001$).

Adrenergic responses. Plasma norepinephrine increased by 94 ± 32 pg/ml for static handgrip and 230 ± 92 pg/ml for dynamic knee extension (both $P < 0.05$). Increases of 121 ± 59 and 91 ± 54 pg/ml for dynamic handgrip and static knee extension were not significant. There was a significant ($P < 0.05$) tendency toward larger increases in plasma norepinephrine for dynamic than for static exercise irrespective of active muscle mass, but there was no significant muscle mass effect. Plasma epinephrine did not increase significantly in any of the four exercises.

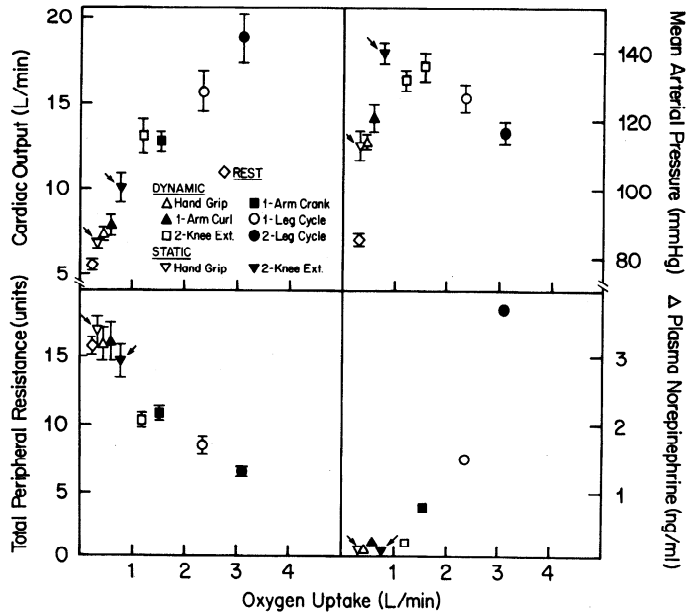

FIG. 1. Means ± SE ($n = 6$) responses of cardiac output, mean arterial pressure, and total peripheral resistance and mean ($n = 6$) responses of plasma norepinephrine to maximal dynamic and static exercise (using work loads leading to fatigue in 4–6 min) over a wide range of active muscle mass. Present handgrip and 2-knee extension data are plotted together with 1-arm curl, 1-arm crank, 1-leg cycle, and 2-leg cycle data from a previous study (15) which included 4 of present subjects. Rest data represent average values from present and previous (15) studies. *Diagonal arrows* highlight static handgrip and 2-knee extension data.

DISCUSSION

The results support our main hypothesis that the magnitude of the heart rate and blood pressure responses to exercise is related to the active muscle mass but independent of the mode of contraction. However, the mode of contraction affects the manner in which the pressor response is achieved. Systemic resistance tended to be lower for dynamic than for static exercise. Virtually identical pressor responses are produced by proportionately larger increases in cardiac output during dynamic exercise.

These data, combined with our previous results (15) from studies of elbow flexion and arm and leg bicycle exercise, provide some insights into the general relationship between muscle mass, the pressor response, and its principal components. During dynamic exercise at a given muscle group-specific relative work load, e.g., maximal, this relationship takes the form of an inverted U (Fig. 1). Mean arterial pressure is highest during exercise involving muscle groups of intermediate size, i.e., two-knee extension and one-arm crank. The significant muscle mass effect also on the pressor response to static exercise is in agreement with recent findings (20, 24). However, the relation between the pressor response and the active muscle mass or developed force may or may not be linear (20, 24).

Mean blood pressure is proportional to the product of cardiac output and peripheral resistance. An outstanding feature of the cardiovascular response to large muscle dynamic exercise is the closely coupled linear relationship between cardiac output and O_2 uptake. The control mechanisms have not been defined, but there is ample evidence for an essentially 1:1 relation between the increase in O_2 transport and the increase in O_2 uptake during two-leg bicycle and treadmill exercise (8, 9, 15). The normal arterial O_2 content is ~200 ml/l of blood, and cardiac output ($\dot{Q}$) increases ~5 l/l of increase in O_2 uptake ($\dot{V}O_2$), i.e., $\Delta\dot{Q}/\Delta\dot{V}O_2$ approaches five. This is true also for one-leg bicycle exercise and one-arm crank (15). However, $\Delta\dot{Q}/\Delta\dot{V}O_2$ ratios as derived from the data in Table 1 were disproportionately large, 12 for dynamic

handgrip and 8 for knee extension. Further studies are required to determine if this is a general feature of small muscle dynamic activity or a consequence of the particular forms of exercise used in the present study. Ratios of blood flow (l/min) to O_2 uptake (l/min) of the active leg of approximately five were found during one-leg quadriceps exercise using a device designed specifically to produce torque levels free from transients (P. Andersen and B. Saltin, personal communication). It is possible that the high $\Delta\dot{Q}/\Delta\dot{V}O_2$ ratios in our series were caused by the brief repetitive heavy efforts that were necessary to overcome the inertia inherent in the exercise devices. The $\Delta\dot{Q}/\Delta\dot{V}O_2$ ratio for static handgrip, 16, and knee extension, 9, were high, as expected from previous studies (17, 29).

O_2 uptake was lower during static than dynamic exercise of the same muscle group. Differences in both contractile energy demands and muscle blood flow are likely to contribute. When static and dynamic exercise each are performed at approximately the same tension development, there is a greater energy expenditure in dynamic exercise due to the external work associated with muscle fiber shortening, i.e., the Fenn effect (10, 22) and because of the energy utilized in the repeated development of tension (6). Furthermore, a large increase in intramuscular pressure during heavy static exercise (25) lowers effective perfusion pressure and limits muscle blood flow and O_2 delivery. Significant increases in both cardiac output and systemic arteriovenous O_2 difference during static knee extension imply that there is considerable nutritive flow, but energy demands must nevertheless partially be covered by anaerobic mechanisms.

The distinct differences between static and dynamic exercise with respect to the metabolic environment in contracting muscle are likely to result in different patterns of afferent impulse traffic from the metabolically sensitive muscle receptors. The group III and IV muscle afferents appear to consist of several different subpopulations including nociceptors that are activated by noxious chemical, mechanical, or thermal stimuli (12). Recent findings support the existence of nociceptors that are activated in response to ischemic muscular contractions but not during contractions with an intact blood supply (11, 19). Maintenance of the pressor response during postexercise occlusion of the blood supply to contracting muscles has been linked to a vasoconstrictor response to skeletal muscle ischemia (23). The persistence of the pressor response during static handgrip or knee extension after abolition of attenuation of the increases in heart rate and cardiac output by combined parasympathetic and β-adrenergic blockade (13, 14) is evidence for a considerable vasoconstrictor potential. However, the pressor responses to static exercise in our series were achieved by an increase in cardiac output without significant changes in peripheral resistance. The measurements of plasma norepinephrine are also consistent with a weaker neurohumoral vasoconstrictor drive during fatiguing static than during dynamic exercise of identical muscle groups.

During dynamic exercise, total peripheral resistance decreases progressively with increasing active muscle mass and metabolic demand (Fig. 1). Metabolically mediated vasodilatation in active muscle has little impact on systemic resistance when the active mass is small as in dynamic handgrip, but when the active muscle mass is large, the metabolic vasodilator drive clearly overrides a systemic α-adrenergic vasoconstrictor drive that appears to become increasingly powerful at higher levels of energy expenditure (Fig. 1). The systemic vasoconstrictor drive can within limits be estimated from the vascular conductance of a nonexercising limb. There was no vasoconstriction in the inactive forearm during static and dynamic knee extension whereas forearm resistance usually increases during two-leg bicycle exercise at higher levels of O_2 uptake but lower mean arterial pressure (4). Plasma levels of norepinephrine, which are thought to represent primarily an overflow from vascular receptors (7), reach a maximum when peripheral resistance is minimal, i.e., during two-leg bicycle exercise.

Considering the significant differences between static and dynamic exercise with respect to cardiac output, systemic resistance, and plasma norepinephrine levels, it becomes difficult to explain why static and dynamic exercise of identical muscle groups carried to a common end point of local fatigue should produce virtually identical responses in terms of heart rate and systolic and mean arterial pressure. Our findings were consistent for two different muscle groups which argues against a mere coincidence. It is tempting to speculate that the similarities of heart rate and pressor responses reflect a modulating effect of the baroreflexes. There is evidence that interventions that affect the afferent limb of the baroreflex alter the hemodynamic response to exercise, and also that exercise alters the stimulus-response characteristics of the carotid baroreflex. In normal humans carotid baroreceptor stimulation by neck suction inhibits forearm vasoconstriction during mild bicycle exercise (3). A recent review by Stephenson (27) provides strong evidence that carotid and aortic baroreflexes continue to operate during exercise and the stimulus (carotid sinus pressure)-response (blood pressure) curve is displaced upward. Data on changes in gain, i.e., effects of exercise on the slope of the stimulus-response curve, are less conclusive (27). However, the degree of displacement of the stimulus-response curve has been quantitatively related to the severity of treadmill exercise (18). It is conceivable that displacement of the baroreceptor stimulus-response curve also is positively related to active muscle mass.

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