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Cardiovascular Response of Human Subjects to Isometric Contraction of Large and Small Muscle Groups (39630)

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The cardiovascular responses to sustained static (isometric) contraction of striated muscle have been previously described (1-6). Tuttle and Horvath (2) reported sharp increases in both systolic (SP) and diastolic (DP) pressures and Hymphreys and Lind (3), using graded handgrip contractions, noted a progressive rise in systemic blood pressure (BP) and heart rate (HR). From further studies of these responses, Lind and McNicol concluded that increases in blood pressure and heart rate were comparable for the same relative tensions in air or leg exercise, irrespective of the size of the muscle mass involved (7).

It is commonly believed that the pressor changes to static exercise result from a combination of (a) a reflex response initiated by the local action of a humoral agent, perhaps potassium, on sensory endings in contracting muscle (8) and (b) a higher neural "central command" factor associated with the subjective effort (9).

Static exercise is being used to an increasing extent for functional evaluation of the circulatory system; such tests frequently involve different-sized muscle masses and various experimental conditions. For example, the circulatory responses in trained subjects may be quite homogeneous (5) but they have a wide range in the untrained subjects (10), and the effect of repetition of static exercise on the cardiovascular responses has received relatively little attention (11, 12).

Our objectives in the current investigation were (1) to study, in untrained normal male subjects, the comparative circulatory effects of static contraction of a small muscle (finger adductor) and that of a large muscle (ankle plantar flexor) and (2) to determine the effect of repetition of these isometric exercises on the cardiovascular responses.

Procedure and methods. Ten normal male subjects (mean age, 25.1 ± 0.7 years) underwent static exercise tests weekly for three successive weeks. All subjects were free of circulatory or other systemic disease as determined by history and physical and cardiovascular examination prior to the study; none of the subjects were athletes or in physical training.

During each test session, static small finger adduction and ankle plantar flexion tests were performed by each subject. In each case, the maximal force output was determined three times (5 min between trials), and the subject was then asked to sustain a contraction equal to 30% of this mean maximal tension to fatigue. The finger adduction was performed first, followed after a 10-min rest by the leg flexion. All the procedures, placements, positioning, and sequences were identical during each of the three weekly visits by the subjects.

A muscle testing table (Keropian Co., San Francisco, Calif., Model 100) was used to indicate the hydraulic isometric force output incident to plantar flexion of the ankle; the table also served to securely support the subject in the sitting position for all tests. A special constructed finger tensiometer served to monitor the adduction force of the little finger and, also, to support the subject's forearm, wrist, and hand. The force output was displayed on a dial visible to the subject and to the operator. To estimate the torque in finger contraction, the tension was multiplied by the distance from the metacarpal-phalangeal joint to the distal interphalangeal joint. From the normal sitting position the knee-raising force exerted by plantar flexion was measured; the force-measuring plate was directly vertical to both the center of the right knee joint and the center of the right ankle joint. The lever arm for

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the leg contraction was taken as the mean distance from the malleoli to the head of the respective ipsilateral metatarsal.

ECG electrodes were placed in a modified Lead II bipolar position. Arterial blood pressure was determined by means of a crystal microphone (Electronics for Medicine Co., White Plains, N.Y.) placed over the right brachial artery under the sphygmomanometer cuff. The pressure in the cuff was altered by an automated inflator-deflator, and the microphone sounds as well as the ECGs were recorded on a Grass Model 7 polygraph. Efforts were made to maintain baseline hemodynamic conditions with a preliminary rest period and the maintenance of a quiet and calm test atmosphere.

Results. The hemodynamic changes are summarized in Table I and indicate significant increases in all variables during isometric contraction in all instances; the mean control SPs and mean ΔSPs (control to peak values) incident to finger and leg contractions were similar. However, sustained contraction of the little finger induced significantly greater ΔDPs (second and third trials) and lesser ΔHRs (all trials) than did leg muscle contraction. The only other difference in circulatory response in the three trials of a single test was a greater control DP in the first trial of finger exercise than in either the second or third trials ($P < 0.01$). There were no differences between recovery values for the circulatory parameters either between the finger and leg contractions or between trials.

Table II indicates the fatigue times and torque outputs for the three trials of the two muscle groups studied. The mean time to fatigue for 30% of maximum voluntary contraction was less for the leg muscle than for the finger in the second and third trials; the

TABLE I. MEAN CIRCULATORY RESPONSES TO STATIC CONTRACTION OF ADDUCTORS OF LITTLE FINGER AND PLANTAR FLEXORS OF ANKLE.[a]

	Finger			Leg		
Trials	1st	2nd	3rd	1st	2nd	3rd
Systolic pressure (mm Hg)						
Control	120.0	122.0	123.8	122.6	124.5	121.8
	±2.2[b]	±3.2	±3.3	±3.5	±3.7	±1.9
ΔSP[c]	29.0	35.0	25.7	33.4	29.0	26.2
	±5.6	±2.8	±4.4	±5.4	±5.5	±4.1
Distolic pressure (mm Hg)						
Control	78.1	74.2	74.1	80.5	80.1	76.4
	±1.32	±1.8	±2.5	±1.6	±4.1	±2.2
ΔDP	28.9	32.3[d]	27.3[d]	23.0	16.9	17.6
	±4.9	±3.4	±3.6	±3.9	±4.8	±3.0
Heart rate (per min)						
Control	72.9	71.9	76.0	72.1	72.7	72.6
	±3.9	±4.3	±2.8	±3.1	±3.9	±3.3
ΔHR	13.7[d]	15.6[d]	11.0[d]	23.3	25.7	18.2
	±3.1	±3.2	±1.6	±4.8	±5.4	±3.2

[a] $N = 10$.
[b] (±) Refers to SE mean.
[c] Δ Values refer to differences from control to peak.
[d] Significantly different from leg values during corresponding trial ($P < 0.05$).

TABLE II. COMPARISON OF FATIGUE TIMES AND TORQUE OUTPUTS DURING STATIC CONTRACTION OF LARGE AND SMALL MUSCLE GROUPS.[a]

	Little finger			Leg		
Trial	1st	2nd	3rd	1st	2nd	3rd
Fatigue time (min)	11.4 ± 4.0	16.8 ± 4.6	15.8 ± 5.0	8.6 ± 1.6	7.2 ± 1.2	6.3 ± 0.8
Torque output (kg-cm)	6.8 ± 0.5	7.8 ± 0.7	8.9 ± 0.7	1576 ± 88	1816 ± 55	1778 ± 106

[a] $N = 10$.

standard errors of the mean fatigue times are large, reflecting the large variations seen in this parameter. The torque values (Table II) indicate the much greater output by the plantar flexors of the foot (about 200-fold). The torque output of the finger was greater on the third trial than on the first ($P < 0.05$), and that of the leg was greater on both the second and third trials than on the first ($P < 0.05$).

Discussion. The marked increases in BP and HR which occur during static exercise have been noted by other investigators (3–5); the bulk of the contracting muscle mass had no apparent effect on the resultant systolic pressure changes as has also been previously reported (7). In our study, the DP response was greater and HR response less when the exercising muscle was smaller; we have no ready explanation for these differences. This divergent effect of the varying muscle mass on the DP and HR does indicate, however, a possible dissociation of the mechanism for HR and BP changes in static exercise. Such dissociation was previously suggested by the observation that relaxation of an isometric handgrip with a tourniquet in place results in a prompt fall of HR but not of BP (8), and, also, by the lack of statistical correlation between individual BP and HR responses of normal subjects and coronary patients to static exercise in a previous study (13). Although the present investigation indicates an effect of the size of contracting muscle on the DP and HR change, it should be emphasized that, by far, the predominant factor influencing the hemodynamic response is the relative intensity of muscle contraction as pointed out by Lind and McNicol (7). In our study, the arterial pressures of fatigue were somewhat less than those previously reported (7, 10), due, perhaps, to the unusual experimental setup and the employment of muscle groups not ordinarily engaged in isometric contraction.

Our results indicate that, in spite of a tendency for an increase in the contractile force exerted by the muscle with repetition of the exercise, the individual hemodynamic responses to a standard sustained isometric contraction were quite consistent. Thus, it appears that static exercise offers a simple, reliable, and repeatable method for hemodynamic evaluation of the circulatory system.

Summary. Sustained static (isometric) contraction of striated muscle to fatigue was carried out in normal, untrained, young (22 to 29 years) male subjects in order to compare the hemodynamic response of large (ankle plantar flexor) and small (little finger adductor) muscle contraction. There were pronounced increases in systolic and diastolic pressures and heart rates in all cases. The increases in systolic pressure were similar during static contraction of the two different muscle groups; however, diastolic pressure increases were greater and heart rate increases less with finger contraction as compared to leg contraction.

Upon three weekly repetitions, the cardiovascular responses were not significantly altered although the force output was increased with repetition in the case of both muscle groups. The results indicate that (i) although the size of the muscle mass influences the diastolic pressure and heart rate response, the relative muscle tension is the major factor determining the hemodynamic result; (ii) the divergence of blood pressure and heart rate responses suggest a dissociation of the mechanisms governing these two variables in static exercise; and (iii) the relative consistency of the hemodynamic changes during these tests indicates that isometric exercise is a simple and reliable method to study cardiovascular response to stress.

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