Journal of Physiology (1991), **435**, *pp.* 573–584
With 5 figures
Printed in Great Britain

EFFECT OF MUSCLE MASS ON THE PRESSOR RESPONSE IN MAN DURING ISOMETRIC CONTRACTIONS

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(*Received 24 July 1990*)

SUMMARY

1. Changes in blood pressure and heart rate were measured in six healthy male subjects during voluntary isometric contractions of the forearm and quadriceps muscles. Arterial pressure was measured directly via a catheter inserted into the radial artery of the non-contracting arm. Each subject exerted two types of contractions: (a) sustained contractions at 70% of the maximum voluntary contraction (MVC) until fatigue occurred and (b) sustained contractions starting at maximum tension (100% MVC), held for a total duration of 1 min.

2. During fatiguing contractions at 70% MVC, there was a progressive increase in blood pressure, reaching a peak level at fatigue. The same level of mean arterial pressure was achieved during contractions of the same relative tension, regardless of the muscle mass. The same trend was observed for the changes in heart rate.

3. During contractions which started with the maximum tension, where tension fell continuously during the 60 s of maximal effort, mean arterial pressure rapidly increased to high levels within a few seconds, and then increased further by 20–30 mmHg during the sustained maximal effort. There was no difference in the initial rapid increases in mean arterial pressure, nor in the final mean arterial pressures reached between contractions of the forearm or quadriceps muscles. There were no differences in the heart rates achieved during these contractions either.

4. There was no significant difference between the mean arterial pressures observed at fatigue of a 70% MVC contraction or at the end of the 60 s maximum effort during handgrip contractions, or during contractions with the quadriceps muscles.

5. These results support the view that muscle mass is not a determinant of the magnitude of the cardiovascular reflexes during fatiguing isometric contractions in man.

INTRODUCTION

The cardiovascular responses to isometric exercise have been well documented (Lind, 1984; Mitchell, Kaufman & Iwamoto, 1983). Typically, fatiguing isometric contractions cause a progressive increase in mean arterial blood pressure and a modest increase in heart rate. The rate at which the blood pressure increases during isometric contractions seems to be dependent on the relative tension of the

contraction. For any given muscle group, the greater the relative tension exerted, the faster the muscle will fatigue and the faster the blood pressure will rise. The role that the muscle mass itself plays in the reflex increase in blood pressure is still unclear. The discussion about its role centres around the relative contributions that two mechanisms (termed 'peripheral reflex' and 'central command') may make. The peripheral reflex theory suggests that the number of 'ergoreceptor' afferent group III and IV fibres (Kalia, Mei & Kao, 1981) activated either by chemical substances presumably released during the contraction or by physical deformation (McCloskey & Mitchell, 1972; Kaufman & Rybicki, 1987) depends on the force of the contraction, since presumably, the greater the number of motor units activated, the greater the amount of muscle activated; therefore, the greater should be the pressor response. The central command theory involves activation of higher brain centres upon the volition and initiation of muscular contraction (Krogh & Lindhard, 1913). Presumably, signals are irradiated to the cardiovascular control centres in the brain stem and possibly elsewhere, and this information contributes to the cardiovascular changes (McAllister, 1979; Mitchell, Reeves, Rogers, Secher & Victor, 1989). It is reasonable to expect, then, that the greater the number of motor units that need to be activated to accomplish a particular contraction, the greater will be the central command signal, and therefore, the greater the contribution should be from this input.

There are a number of studies which suggest that muscle mass does indeed influence the magnitude of the isometric pressor response (Mitchell, Payne, Saltin & Schibye, 1980; Seals, Washburn, Hanson, Painter & Nagel, 1983; Mitchell *et al.* 1989). There are number of studies which suggest that muscle mass does not influence the magnitude of the pressor reflex (Lind & McNicol, 1967; McCloskey & Streatfeild, 1975; Imms & Mehta, 1989). Mostly, these studies have used contractions of submaximal tension, usually at 50% or lower, and usually sustained for a finite time rather than to fatigue. The present study is different because contractions of very high tensions (70% of the maximum) were sustained to fatigue and maximal contractions were sustained until the tension fell to around 50% of the maximum. Muscles of two different sizes, the forearm flexor muscles and the quadriceps were used in this study. These experiments were performed to determine whether muscle mass would affect the cardiovascular responses to these types of fatiguing isometric contractions.

METHODS

Six male subjects, average age 26 ± 3 years, volunteered to participate in these experiments. Each signed an informed consent form before participating in the study. All procedures and protocols were reviewed by the Institutional Review Board of East Tennessee State University.

All subjects underwent an extensive period of training for isometric contractions for both the handgrip and quadriceps muscles. This period lasted about 3 weeks, and basically followed the procedures described previously (Clarke, Hellon & Lind, 1958; Lind & McNicol, 1967; Williams & Lind, 1987). For both types of contractions, subjects were seated in a wooden chair with a seatback angle at 30°; their legs were positioned so that a spine-to-thigh angle of 105° was achieved. They were fixed in this position by a lap belt and a shoulder harness. Fatigue was defined as that point in the sustained contraction when subjects were unable to maintain the targeted tension or unable to exceed the targeted tension. Subjects were considered trained when their maximum efforts (100% maximum voluntary contraction, 100% MVC) were within 5% of each other, and when the

duration of the first of the successive sustained contractions did not vary by ±5–10%. Subjects also received training for performing a sustained maximal effort for 60 s.

On the day of the experiment, each subject exerted two 100% MVC each for the handgrip and quadriceps muscles. These were 3 min apart. The heart rate (HR) was calculated from the recorded ECG during the resting and contraction periods and expressed as beats min^{-1}. Electromyographic activity (EMG) was recorded from the surface of the skin using self-adhesive silver–silver chloride electrodes placed over the following muscle groups: right intercostals between the 5th and 6th intercostal spaces; centre of the left and right quadriceps; the right pectorals, and the inner surface of the contracting forearm. The raw EMG was displayed on a dual channel electromyograph (Teca Model M) and simultaneously fed into a converter (Analog Devices) for root mean square (r.m.s. EMG). Subjects were instructed to remain quiet and relaxed prior to the generation of isometric contractions, and since the EMG signal was negligible during these resting periods, it was taken as the baseline and set at zero signal height. The change in muscular activity was then measured as the change in the height (in centimetres) of the integrated r.m.s. EMG signal. Typically, a 1 cm deflection was equivalent to 7·8 μV.

Arterial blood pressure (BP) was measured directly through a catheter (Medi-cut 20 g) inserted percutaneously under sterile procedures into the radial artery of the non-contracting arm and connected to a Statham-Gould pressure transducer (P23 b) positioned at heart level. The pressure pulse was recorded continuously on a Grass Model 7 polygraph. Mean arterial pressure (MAP) was calculated from the diastolic plus one-third of the pulse pressure.

Subjects were permitted to rest quietly for 30 min after application of the recording devices. Subjects were then given a countdown and instructed to perform the series of fatiguing isometric contractions. Subjects were instructed to breathe freely and not to exert a Valsalva manoeuvre. The order in which the contractions were performed was randomized. There was 30–60 min rest allowed between fatiguing contractions.

The values measured for HR, BP, MAP and r.m.s EMG were averaged for the six subjects and reported as the means ± S.E.M. Since the duration of fatiguing contractions was different for different subjects, the time scale on the diagrams below has been normalized to percentage to fatigue. Significance was determined from analysis of variance for paired data and achieved when $P < 0.05$.

RESULTS

The changes in the systolic and diastolic levels of arterial pressure during isometric contractions are presented in Table 1. The peak systolic pressure attained at fatigue during a sustained handgrip contraction at 70% MVC, 233 ± 9 mmHg, was not significantly different than the peak systolic pressure at fatigue during a sustained leg contraction at 70% MVC, 224 ± 21 mmHg ($P > 0.10$). Likewise, there was no significant difference between the diastolic pressures at fatigue between the handgrip and leg contractions sustained to fatigue at 70% MVC. The average durations of the handgrip contractions at 70% MVC were 46 ± 3·3 s and the average durations of the leg contractions at 70% MVC were 53 ± 3·4 s. Similarly, there were no differences between the systolic pressures at the end of the 60 s of maximum handgrip effort, 255 ± 9 mmHg and the maximum leg effort, 260 ± 29 mmHg, nor were there any statistical differences between the diastolic pressures at the end of the 60 s of maximum handgrip effort of leg effort. There were similar increments in systolic pressures above the resting levels during the sustained contractions at 70% MVC and 100% MVC regardless of the muscle mass (see Table 1), with the following exception. The increment in systolic pressure during the maximal 100% MVC handgrip effort, 102 ± 6 mmHg, was significantly higher than the increment in systolic pressure during the sustained handgrip contraction at 70% MVC, 77 ± 7 mmHg, ($P < 0.025$). This was not the case when comparing the increments in systolic pressures during the

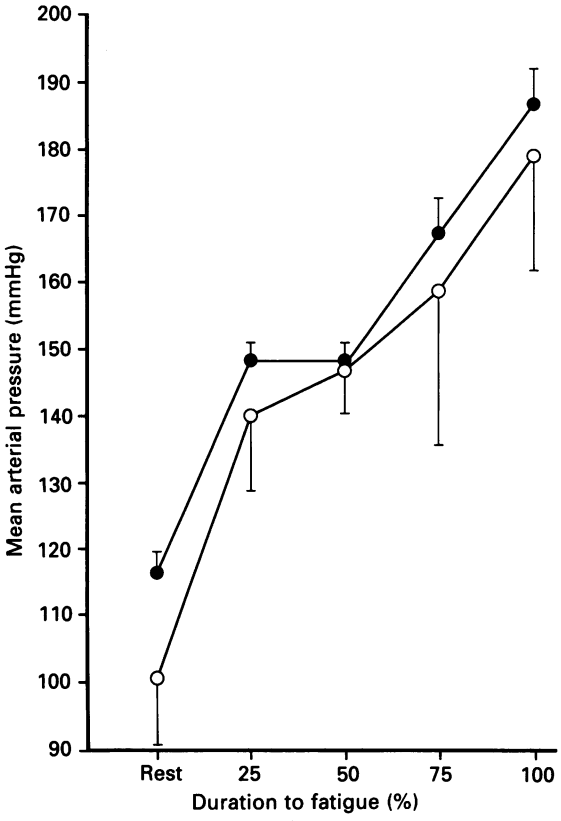

Fig. 1. Changes in mean arterial blood pressure (mmHg) during sustained contractions exerted at 70% MVC by six subjects. Contractions were exerted by either the handgrip (●) or quadriceps (○) muscles and continued to fatigue. Subjects repeated each contraction twice. Each point represents the average for the six subjects ± S.E.M. Since the durations of the fatiguing contractions were different for the individual subjects, the time scale (x-axis) was normalized to percentage to the fatigue time. See text for actual endurance times.

TABLE 1. Changes in blood pressure during isometric contractions

		During sustained contractions at 70% MVC			
		Rest	50% F	100% F	Increment
Handgrip	Systolic	156±7	194±7	233±9	77±7
	Diastolic	96±4	125±3	162±7	66±6
Leg	Systolic	138±17	201±12	224±21	86±4
	Diastolic	82±12	121±4	156±26	74±14
		During sustained maximum effort			
		Rest	30 s	60 s	Increment
Handgrip	Systolic	153±6	228±17	255±9	102±6
	Diastolic	92±3	150±14	169±8	77±5
Leg	Systolic	133±11	220±30	260±29	127±39
	Diastolic	80±10	150±13	178±24	98±19

Values are means ± S.E.M. 50% F indicates 50% to fatigue, 100% F indicates 100% to fatigue.

two types of leg extension contractions. Similarly, the increments in the diastolic pressures above the resting levels were the same for two types of contractions, regardless of muscle mass.

The changes in mean arterial pressure (MAP) during sustained isometric contractions at 70% MVC of both handgrip and quadriceps are presented in Fig. 1.

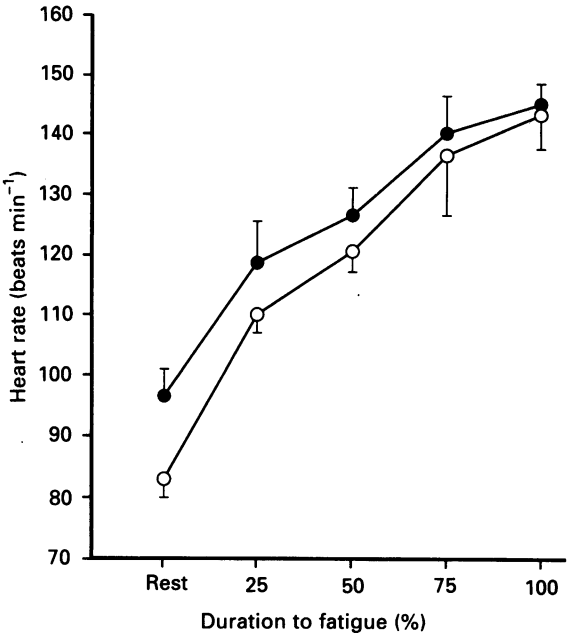

Fig. 2. Changes in heart rate (beats min^{-1}) during sustained contractions exerted at 70% MVC by six subjects. Contractions were exerted by either the handgrip (●) or quadriceps (○) muscles and continued to fatigue. Each point represents the average for the six subjects ± S.E.M. Other details are described in Fig. 1.

There was a significant increase in MAP during both the handgrip and quadriceps contractions. Resting MAP prior to the handgrip contractions averaged 116 ± 4 mmHg and increased to a MAP of 186 ± 6 mmHg at fatigue ($P < 0.001$). Resting MAP prior to the quadriceps contractions averaged 101 ± 14 mmHg and increased to a MAP of 179 ± 25 mmHg at fatigue ($P < 0.02$). There was no difference in the resting MAP prior to either the handgrip or quadriceps contractions ($P > 0.10$). Likewise, there was no difference in the peak MAP achieved at fatigue between the handgrip and quadriceps contractions ($P > 0.25$).

The changes in the heart rates (HR) during the sustained isometric contractions at 70% MVC are presented in Fig. 2. The HR increased from resting levels of 96 ± 5 beats min^{-1} to levels at fatigue of 145 ± 3 beats min^{-1} ($P < 0.01$) during the sustained handgrip contractions. Similarly, the HR increased from resting levels of 82 ± 2 beats min^{-1} to levels of 144 ± 7 beats min^{-1} at fatigue ($P < 0.0005$) during the sustained quadriceps contractions. As with the MAP, there was no difference in the resting HR ($P > 0.10$) nor in the HR at fatigue ($P > 0.10$) between the handgrip and quadriceps contractions.

Figure 3 illustrates the changes in MAP during a maximum effort isometric contraction sustained for a total of 60 s for both the handgrip and quadriceps. During this time, the initial maximum tension falls continuously during the 60 s contraction time, usually reaching levels that are approximately 50% of the original levels of

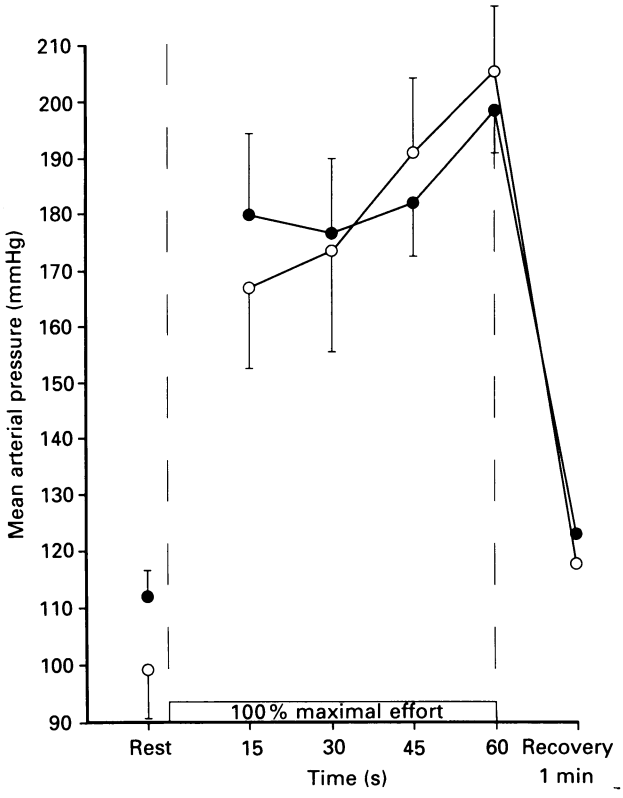

Fig. 3. The changes in mean arterial blood pressure (mmHg) during sustained isometric contractions exerted at 100% maximal effort continuously for 60 s. Contractions were exerted by either the handgrip (●) or quadriceps (○) muscles. Each point represents the average from six subjects ± S.E.M.

force. For example, the maximum force exerted by the handgrip muscles at the beginning of this set of contractions averaged 34·2 ± 2·6 kg and decreased to 20·7 ± 5·5 kg by the end of the maximum effort at 60 s, a 40% reduction in the maximum force. The maximum force exerted by the quadriceps at the start of the maximum effort averaged 87·2 ± 9·0 kg and decreased to 44·5 ± 11·7 kg at the end of the 60 s of contraction, a level that was 51% of the original strength.

Unlike the sustained contractions at submaximal tensions, where the MAP increased steadily throughout the duration of the contraction, reaching peak levels at fatigue (Fig. 1), the MAP during the maximal effort (i.e. 'falling tension') abruptly increased to high levels almost immediately, and then had little further increase during the remainder of the maximal effort, as seen in Fig. 3. During the maximal effort by the handgrip muscles, MAP increased from resting levels of 112 ± 4 to

180 ± 14 mmHg within 15 s ($P < 0{\cdot}0005$). The MAP increased slightly over the remainder of the contraction period, averaging 198 ± 7 mmHg at 60 s, a significant increase above the resting level ($P < 0{\cdot}0005$) but not significantly higher than the levels reached at 15 s ($P \geqslant 0{\cdot}15$). The same pattern occurred when the maximal effort

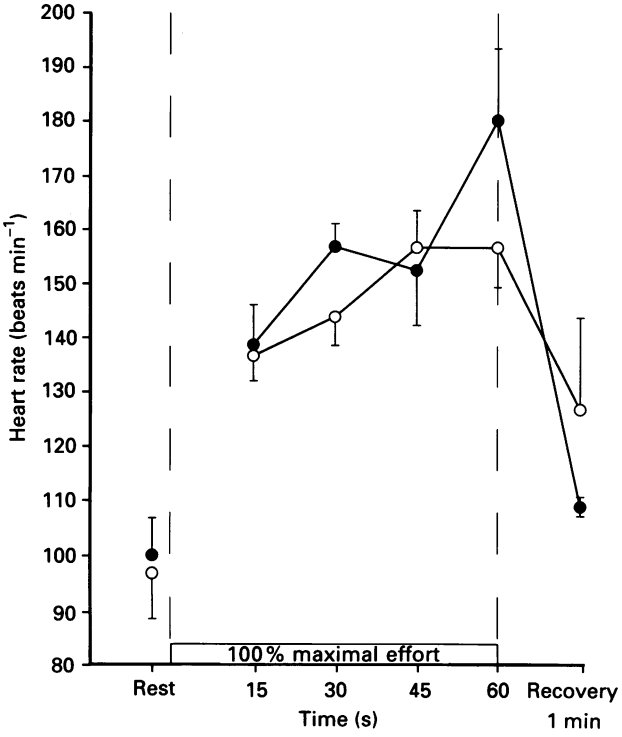

Fig. 4. The changes in heart rate (beats min^{-1}) during sustained contractions exerted at 100% maximal effort continuously for 60 s. Contractions were exerted by either the handgrip (●) or quadriceps (○) muscles. Each point represents the average from six subjects $\pm$ S.E.M.

was exerted by the quadriceps muscles, as shown in Fig. 3. The MAP increased from resting levels of 98 ± 10 to 166 ± 14 mmHg within 15 s ($P < 0{\cdot}01$), and then increased further to 205 ± 22 mmHg by 60 s. This level of pressure was also significantly higher than resting levels ($P < 0{\cdot}005$). The MAP at 60 s was not significantly higher than the MAP achieved within 15 s ($P \leqslant 0{\cdot}10$). In contrast, there was no difference between the resting MAP prior to the maximal efforts exerted by the handgrip or quadriceps ($P > 0{\cdot}10$), nor was there any significant difference between the peak MAP attained at 60 s between the maximal efforts exerted by the two muscle groups ($P > 0{\cdot}35$).

The changes in HR that occurred during the maximal effort contractions illustrated in Fig. 4. The resting HR prior to the handgrip contraction, 100 ± 6 beats min^{-1}, was no different than the resting HR prior to the quadriceps contraction, 96 ± 7 beats min^{-1} ($P > 0{\cdot}25$). In contrast to the pattern for the MAP, the HR did gradually increase during the 60 s duration of maximal effort, reaching peak levels at the end of this time. The HR achieved at the end of the handgrip effort averaged

180 ± 13 beats min^{-1}. This was significantly higher than resting levels ($P < 0{\cdot}005$) and higher than the levels reached after only 15 s ($P < 0{\cdot}01$). The HR achieved at the end of the quadriceps maximal effort, 156 ± 7 beats min^{-1}, was also significantly higher than its corresponding resting level ($P < 0{\cdot}002$) and the level of HR at 15 s (P

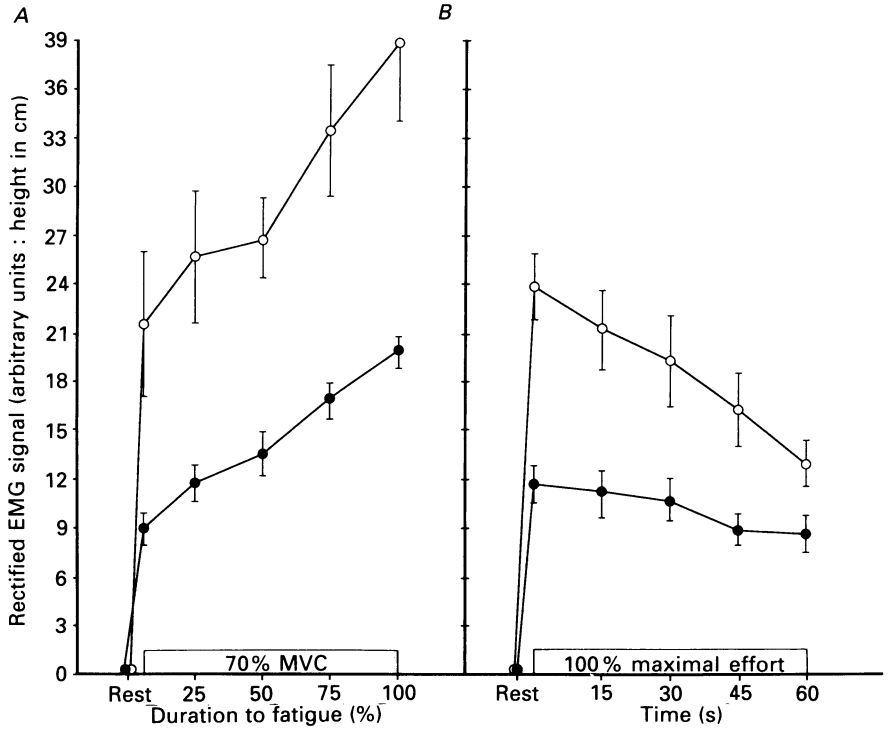

Fig. 5. Rectified EMG changes (given in arbitrary units: height in cm above resting levels) for sustained contractions at 70% MVC taken to fatigue (A) and sustained maximal efforts, continued for 60 s (B). Contractions were exerted by the handgrip (●) and quadriceps (○) muscles. Each point represents the average from six subjects ± S.E.M.

$< 0{\cdot}05$). The peak HR attained at the end of the handgrip maximal effort was not significantly different than the peak HR attained at the end of the quadriceps maximal effort ($P > 0{\cdot}05$).

Figure 5 illustrates the differences in the rectified EMG recorded from the surface of the forearm, during handgrip contractions, and the surface of the thigh, during the quadriceps contractions. The changes in the EMG during fatiguing sustained contractions at 70% MVC are illustrated in Fig. 5A. As can be seen, the height of the EMG signal increases continuously during fatiguing, submaximal isometric contractions, reaching the highest level at the point of fatigue for both the handgrip and quadriceps contractions. In both cases, the EMG signal at fatigue is significantly higher than the signal at the start of the contraction ($P < 0{\cdot}05$). In contrast to this, the EMG signal during the maximal effort, where tension continuously fell during a 60 s contraction period, decreased continuously. For the handgrip maximal effort, the EMG signal decreased by 26% from the starting level ($P < 0{\cdot}05$) while for the

quadriceps maximal effort, the EMG signal decreased by 43% from the starting level ($P < 0.05$). While not shown in this diagram, the EMG signal recorded from the intercostals, pectorals and muscles from non-contracting limbs did not show any significant increase over the duration of the exercise.

DISCUSSION

There were no differences in the peak systolic or peak diastolic pressures or in the mean arterial pressure at the end of fatiguing isometric contractions starting at a submaximal level (70% MVC) between the handgrip and quadriceps muscles. The masses of the muscle groups studied were estimated to be about 500 g for the handgrip and about 2000 g for the thigh extensors (Bischoff, 1863). As a result, the pressor response was not a function of muscle mass, even though the muscle mass differed by about 4-fold. There were no differences in the peak heart rates achieved during these contractions either. The blood pressure results are similar to previous studies (Lind & McNicol, 1967; McCloskey & Streatfeild, 1975; Reindl, Gotshall, Reinke & Smith, 1977; Imms & Mehta, 1989). The endurance time for our subjects for the handgrip contractions at 70% MVC was close to 46 s and for the quadriceps contraction, close to 53 s. Because these endurance times were not significantly different, the contraction time is not a variable in this study. More importantly, unlike other studies where contractions were exerted for a finite period, usually 1–2 min, (Mitchell *et al.* 1980; Kilbom & Persson, 1981; Seals *et al.* 1983; Mitchell *et al.* 1989) the contractions in this study were continued to the same end point for both groups of muscles, fatigue, regardless of the mass of muscle involved. It is important in the interpretation of results regarding changes in cardiovascular function to take into consideration the time it takes a particular group of muscles to fatigue. There is an inverse, non-linear relationship between the relative tension and the endurance time to fatigue (Clarke *et al.* 1958; Funderburk, Hipskind, Welton & Lind, 1974). This applies regardless of the muscle or muscle group performing the isometric contraction. For example, in the group of six subjects participating in our study, the endurance times for contractions of the handgrip at 25% MVC was 284 ± 23 s, at 40% MVC, 116 ± 5 s and at 70% MVC, 46 ± 3 s. In contrast, the endurance times for contractions of the leg extensors at 25% MVC was 252 ± 32 s, at 50% MVC, 95 ± 7 s and at 70% MVC, 53 ± 3 s. Our data do not support the notion that differences in cardiovascular responses related to muscle mass become apparent at the later stages of the exercise (Mitchell *et al.* 1980).

It is possible that because all our contractions are taken to fatigue that most or all of the motor units within a given muscle group are activated during the contraction, especially as fatigue is approached. This is supported from the EMG data (Fig. 5) which showed that the height of the rectified signal during both the handgrip and the quadriceps contractions at 70% MVC approximately doubled above the initial levels at the start of the contractions, suggesting additional recruitment and possibly rate coding during the exercise. This degree of change in the surface EMG agrees with previous reports (Lind & Petrofsky, 1979; Bigland-Ritchie, Johansson, Lippold, Smith & Woods, 1983; Bigland-Ritchie, Cafarelli & Vollestad, 1986). There is no way to determine from the recording of the surface EMG whether the same absolute

number of motor units was activated in both groups of muscles during the course of the fatiguing exercise. However, at fatigue, most of the motor units are presumably activated and those active are presumably firing at maximum frequency at high tensions when fatigue occurs (Merton, 1954; Milner-Brown & Stein, 1975; Bigland-Ritchie *et al.* 1983). One could conclude that during sustained isometric contractions that are *taken to fatigue*, the input signal from the peripheral reflex component (i.e. the activation of peripheral afferent ergoreceptors) will not be a function of the muscle mass because the signal received by the central cardiovascular controlling neurones is presumably maximal.

The finally achieved mean arterial pressure at the conclusion of the maximal effort also was not a function of the muscle mass. The mean arterial pressure achieved at the conclusion of this type of sustained isometric contraction was not different from the mean arterial pressure achieved at the designated fatigue point of the sustained submaximal isometric contraction. This pattern in the blood pressure response is similar to one reported earlier when the blood pressure increased rapidly to a high level and was sustained, even though the tension and integrated EMG decreased (Eklund & Kaijser, 1978). The rectified EMG recorded from our subjects (Fig. 5) also decreased during the maximal effort. During the 'falling tension' maximal effort, presumably the central command input to the brain stem cardiovascular centres is maintained at maximal levels (Bigland-Ritchie *et al.* 1983). Our data however are different from the results reported by Freund, Rowell, Murphy, Hobbs & Butler (1979) where the peak mean arterial pressure in response to maximal isometric contraction of the quadriceps before and after peridural anaesthesia was a function of the maximal force developed. Since their subjects experienced blockade of varying degrees of both proprioception and motor strength, it is difficult to make any direct comparisons between their results and the present ones. It is conceivable that part of the increment in the blood pressure during the later stages of the maximal effort was due to some contribution from straining manoeuvres (Williams & Lind, 1987). While the EMG was recorded from non-contracting muscle groups (e.g. the intercostals and pectorals), and showed no significant increases or patterns of increase during either type of isometric contraction, since the intra-abdominal and intra-oesophageal (intra-thoracic) pressures were not directly measured in this study, the contribution to the increments in blood pressure cannot be completely ruled out. Based on a previous study, the increment in mean arterial pressure due to possible straining manoeuvres would amount to about 20 mmHg (Williams & Lind, 1987). As with the sustained submaximal contractions, the finally achieved heart rates during the falling tension maximal efforts were not a function of the mass of muscle.

The findings from this study are important to the discussion and understanding of the reflex control of cardiovascular function during isometric exercise because of the direct intra-arterial measurement of blood pressures during two types of fatiguing contractions. Because the submaximal contractions were taken to fatigue, and because the mean arterial pressures were not a function of the mass of muscle contracting, it seems reasonable to postulate that the input of the peripheral reflex component and the input from the central command were similar during both types of contractions. Presumably, the integration of such a signal, especially when the muscle is fatiguing, by central cardiovascular controlling neurones is not a function

of the mass of muscle activated. This suggests that there may not be a linear, direct relationship between mass of muscle activated, magnitude of afferent input signal to the central cardiovascular control centres and subsequent degree of cardiovascular (i.e. pressor) response.

The author wishes to thank Professor A. R. Lind for his reading and comments about this manuscript and to dedicate this manuscript in memoriam to him. Ms Leisa Gouge is acknowledged for her assistance in typing this manuscript and Dr John E. Douglas for his assistance with the catheterization during these experiments. This work was supported in part by Systems Research Laboratories and Air Force contract F33615-85-C-0530.

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