Eur J Appl Physiol (1997) 76: 174–180

ORIGINAL ARTICLE

Richard A. Ferguson · Margaret D. Brown

Arterial blood pressure and forearm vascular conductance responses to sustained and rhythmic isometric exercise and arterial occlusion in trained rock climbers and untrained sedentary subjects

Accepted: 24 February 1997

Abstract Cardiovascular responses to sustained and rhythmic (5 s on, 2 s off) forearm isometric exercise to fatigue at 40% maximal voluntary contraction (MVC) and to a period of arterial occlusion were investigated in elite rock climbers (CLIMB) as a trained population compared to non-climbing sedentary subjects (SED). Blood pressure (BP), monitored continuously by Finapres, and forearm blood flow, by venous occlusion plethysmography, were measured and used to calculate vascular conductance. During sustained exercise, times to fatigue were not different between CLIMB and SED. However, peak increases in systolic (S) BP were significantly lower in CLIMB [25 (13) mmHg; (3.3 (1.7) kPa] than in SED [48 (17) mmHg; (6.4 (2.3) kPa] ($P < 0.05$), with a similar trend for increases in diastolic (D) BP. Immediately after sustained exercise, forearm conductance was higher in CLIMB than SED ($P < 0.05$) for up to 2 min. During rhythmic exercise, times to fatigue were two fold longer in CLIMB than SED [853 (76) vs 420 (69) s, $P < 0.05$]. Increases in SBP were not different between groups except during the last quarter of exercise when they fell in CLIMB. Conductance both during and after rhythmic exercise was higher in CLIMB than in SED. Following a 10-min arterial occlusion, peak vascular conductance was significantly greater in CLIMB than SED [0.597 (0.084) vs 0.431 (0.035) ml · min^{-1} · 100 ml^{-1} · mmHg^{-1}; $P < 0.05$]. The attenuated BP response to sustained isometric exercise could be due in part to enhanced forearm vasodilatory capacity, which also supports greater endurance during rhythmic exercise by permitting greater functional hyperaemia in between contraction phases. Such adaptations would all facilitate the ability of rock climbers to perform their task of making repetitive sustained contractions.

Key words Blood pressure · Forearm blood flow · Isometric exercise · Training · Arterial occlusion

R.A. Ferguson · M.D. Brown
School of Sport and Exercise Sciences, University of Birmingham, Edgbaston, Birmingham B15 2TT, UK

R.A. Ferguson (✉)
Neuromuscular Biology Group,
Department of Exercise and Sport Science,
Manchester Metropolitan University, Hassall Road,
Alsager ST7 2HL, UK

Introduction

Physical training results in many well-documented cardiovascular adaptations that serve to make the system more efficient and play a part in the increased ability to perform strenuous exercise.

During exercise, sympathetic vasoconstriction acts to divert blood from areas of non-working tissue to working muscles and contributes to the rise in blood pressure (BP) which may help to increase perfusion to the working muscles (O'Leary and Sheriff 1995). It has been previously demonstrated that sympathetic neural adjustments to isometric exercise are not influenced by the level of aerobic conditioning (Seals 1991). However, it was reported by Somers et al. (1992) that a forearm endurance training protocol resulted in an attenuation of the muscle sympathetic nerve activity (MSNA) during isometric exercise. A reduced output of MSNA following training may be expected to transcribe to a reduced BP response to isometric exercise, yet Somers et al. (1992) failed to observe a reduced pressor response after training. They attributed this to insufficient BP measurements which, since they were made by sphygmomanometry and could only be obtained at minute intervals, may have missed more transient changes. A more recent report by White et al. (1995) described an attenuation of the pressor response during 2 min of involuntary isometric exercise of the calf following a training intervention consisting one-legged heel raises. Therefore, it appears that certain types of training may indeed reduce the magnitude of the pressor response to exercise.

A reduced pressor response after training could be due to an attenuated MSNA. This would reduce opposition to metabolic vasodilatation facilitating functional hyperaemia. It could also be due to a more effective active vasodilatation of the muscle vascular bed. Studies have indeed shown enhanced vasodilator capacity immediately following ischaemic exercise in the forearms of tennis players (Sinoway et al. 1986) and in the lower leg of endurance-trained subjects (Snell et al. 1987) and a greater reactive hyperaemia in the forearms of manual compared to sedentary workers (Smolander 1994).

Modern sport climbers epitomise highly trained athletes. Rock climbing involves intense sustained and intermittent isometric forearm muscle contractions which may provide an effective training stimulus to induce adaptations to the muscle vascular bed and to the muscle sympathetic neural adjustments during exercise. Thus, the present investigation sought to examine the cardiovascular responses to specific stimuli using elite rock climbers as the trained population in comparison to non-climbing sedentary subjects. Arterial BP and forearm blood flow were measured in response to sustained and rhythmic isometric handgrip exercise to fatigue and also following arterial occlusion to produce reactive hyperaemia. Preliminary data have been presented in abstract form (Ferguson et al. 1996/1997).

Methods

Subjects

A total of 15 healthy male subjects took part in the study. Five elite rock climbers (CLIMB), mean (SEM) age 22 (0.8) years, were recruited from a local rock climbing centre. The climbers had an average of 7.4 (1.2) years of climbing experience and climbed an average grade ranging between F6b and F7c+ with the best climbs ranging between F7a and F8a+. These levels of climbing are within the top 10% of graded difficulty in competitive sport rock climbing. Ten matched sedentary subjects (SED), mean age 20 (1.5) years, were recruited from the general University population. Requirements for the sedentary subjects were they had not participated regularly in any form of physical training for at least 12 months prior to the study. All subjects completed a general health screening questionnaire and gave written informed consent. The study had approval from the Ethics Committee of the School of Sport and Exercise Sciences, University of Birmingham, and the South Birmingham Health Authority.

Physiological measurements

Arterial BP and heart rate

Arterial BP and heart rate (HR) were continuously monitored using the Finapres Blood Pressure Monitor (Ohmeda), with a finger cuff positioned around the middle finger of the contralateral hand which was maintained at heart level. This provided a non-invasive beat-to-beat estimate of the arterial waveform from which systolic, diastolic and mean arterial pressures were derived. Pressures and heart rates were evaluated at rest and at intervals corresponding to 25, 50, 75% of exercise duration time and immediately at the end of exercise (100%: END). At each of these time points systolic blood pressure (SBP), diastolic blood pressure (DBP) and HR from ten consecutive waveforms were averaged.

Forearm blood flow

All blood flow measurements were carried out by venous occlusion plethysmography using mercury-in-silastic strain gauges (Whitney 1953), with the strain gauge positioned around the widest portion of the forearm. In order to exclude blood flow to the hand, 30 s prior to blood flow measurements a distal occlusion cuff around the wrist was inflated to 200 mmHg (26.6 kPa). Blood flow was measured by rapidly inflating, using a semiautomatic inflation pump, the proximal occlusion cuff around the arm above the elbow to between 40 and 50 mmHg (5.3–6.7 kPa) for about 3 s. Multiple recordings were made as required with one complete recording (i.e. inflation and deflation) taking approximately 5–8 s. Vascular conductance was then calculated as limb blood flow divided by mean arterial pressure (DBP plus one-third pulse pressure).

Experimental protocol

Three experiments were carried out in a randomised order in a laboratory maintained at a temperature close to 20°C. Each subject arrived in the morning and all experiments were carried out in a single day. The morning session was devoted to habituation of the subject to the procedures of measurement and experimentation. Sufficient periods (> 60 min) between each experiment were given to allow adequate recovery.

Sustained isometric handgrip exercise

All climbers and five of the sedentary subjects participated in this experiment. The subjects rested supine with their left arm supported in a specifically designed forearm ergometer. The forearm was supported at both the wrist and elbow which allowed attachment of occlusion cuffs and mercury-in-silastic strain gauge as described. Maximal voluntary contraction (MVC) was obtained by instructing the subject to pull on the handgrip as hard as possible. MVC was determined as the maximum force the subjects generated during three attempts sustained for 2 s performed 2 min apart. From this the target force of 40% MVC was calculated and displayed on a pen chart recorder for the subject to observe. After a minimum of 20 min rest baseline measurements of BP and blood flow were taken. Three to four resting blood flow measurements were taken and averaged. The subject then performed a sustained isometric handgrip contraction at 40% of MVC which continued until volitional fatigue. This was defined at the point where the subject expressed a desire to stop or was unable to maintain the desired contraction force. Throughout the exercise the subject was encouraged not to perform Valsalva or straining manoeuvres. The distal occlusion cuff around the wrist was inflated upon advice from the subject that termination/fatigue was imminent. When contraction ceased, duration of holding time was recorded and blood flow was measured within 5 and 15 s and further measurements every 15 s for 2 min.

Rhythmic isometric handgrip exercise

Four climbers and four sedentary subjects participated in this experiment. The subject was instrumented, MVC determined and baseline measurements taken as described above. The subject began handgrip exercise at 40% of MVC at a frequency of a 5 s contraction followed by 2 s of relaxation. Timing was made audible by a metronome. Every minute during the exercise the subject was instructed to stop so that a single blood flow measurement could be taken, this procedure lasting a maximum of 5–8 s, after which the subject continued exercising. The distal occlusion cuff around the wrist was not inflated during these measurements. Exercise continued until volitional fatigue, at which point the subject expressed a desire to stop or was unable to maintain the desired contraction force. When contraction ceased, exercise time and blood flow were measured as described above.

Post-occlusion reactive hyperaemia

All 15 subjects participated in this experiment. The subjects rested in a supine position. The left forearm was supported in a pronated position by foam pads. The occlusion cuffs were attached to the wrist and upper arm and the mercury-in-silastic strain gauge positioned and calibrated. After a minimum of 20 min rest baseline levels of BP and blood flow were taken. The proximal occlusion cuff around the upper arm was inflated to 200 mmHg (26.6 kPa) for 1 min followed by deflation and immediate measurement of a single hyperaemic blood flow. This served as a priming stimulus for vasodilatation (Patterson and Whelan 1955) as well as an indication of the subject's post-occlusion blood flow. This allowed time for adjustment of baseline recordings prior to the 10-min occlusion. The arm cuff was then inflated to 200 mmHg (26.6 kPa) for 10 min. After rapid deflation forearm blood flow was measured within 5 and 15 s and then every 15 s thereafter for 2 min.

Analysis

All data is presented as mean (SEM). Statistical analyses were carried out by 2-way analysis of variance (ANOVA) for repeated measures and independent *t*-tests where appropriate. A 5% level of significance was set.

Results

Sustained isometric handgrip exercise

Forearm forces generated during MVCs were not significantly different between climbers and sedentary subjects, being 715 (34) N for CLIMB and 635 (55) N for SED. Holding times to fatigue were also similar for the two groups, being 140 (11.1) s and 122 (14.2) s respectively (not significant, NS).

Resting SBPs were 124 (3) mmHg [16.5 (0.4) kPa] and 136 (6) mmHg [18.1 (0.8) kPa] in CLIMB and SED (NS), respectively, with peak SBPs occurring at fatigue (END) reaching 149 (8) mmHg [19.8 (1.1) kPa] and 184 (8) mmHg [24.5 (1.1) kPa] respectively ($P < 0.05$). These represented increments above rest of 25 (13) mmHg [3.3 (1.7) kPa] and 48 (17) mmHg [6.4 (2.3) kPa], ($P < 0.05$, Fig. 1A). Resting DBPs were 72 (6) mmHg [9.6 (0.8) kPa] and 72 (4) mmHg [9.6 (0.5) kPa] for CLIMB and SED (NS), with peak DBPs also occurring at fatigue reaching 101 (5) mmHg [13.4 (0.7) kPa] and 108 (6) mmHg [14.4 (0.8) kPa] respectively (NS). Although there was an apparent trend for increments in DBP to be lower throughout exercise in CLIMB than SED this did not reach accepted levels of significance (Fig. 1B). Resting HR values were 59 (4) and 60 (3) beats $\cdot$ min^{-1} increasing to 113 (14) and 98 (5) beats $\cdot$ min^{-1} at the end of exercise in CLIMB and SED respectively, which, although slightly higher in CLIMB, did not reach significance.

Resting forearm blood flows were 5.3 (0.8) and 4.4 (1.3) ml $\cdot$ min^{-1} $\cdot$ 100 ml^{-1} in CLIMB and SED (NS). Peak blood flow values attained during the 2-min post-exercise period were 44.1 (3.1) and 28.0 (2.4) ml $\cdot$ min^{-1} $\cdot$ 100 ml^{-1}, respectively ($P < 0.05$). At rest, mean vascular conductances were similar in the two groups [0.06 (0.009) vs 0.048 (0.016) ml $\cdot$ min^{-1} $\cdot$ 100 ml^{-1} $\cdot$ mmHg^{-1}]. Immediately post-exercise, conductance had increased four fold in SED but 7.5-fold in CLIMB (Fig. 2). Thus, with a lower mean arterial pressure at the end of exercise, conductance was much greater in CLIMB [0.439 (0.028) ml $\cdot$ min^{-1} $\cdot$ 100 ml^{-1} $\cdot$ mmHg^{-1}] than SED [0.265 (0.028) ml $\cdot$ min^{-1} $\cdot$ 100 ml^{-1} $\cdot$ mmHg^{-1}] ($P < 0.05$). Conductance remained significantly higher in CLIMB than SED throughout the entire 2-min post-exercise period during which measurements were made (Fig. 2).

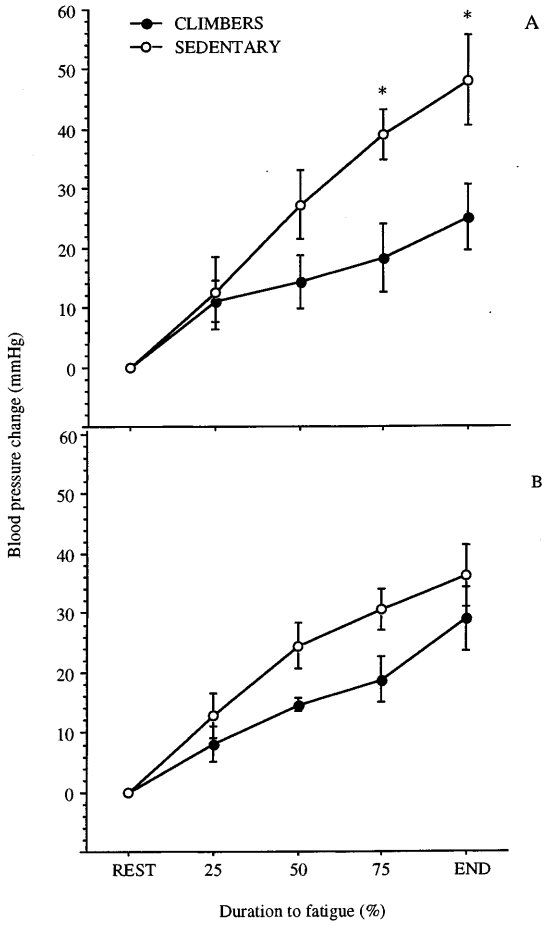

Fig. 1 Changes from resting values in systolic (**A**) and diastolic (**B**) blood pressure during sustained isometric handgrip exercise to fatigue. *$P < 0.05$; climbers vs sedentary

Rhythmic isometric handgrip exercise

MVC forces obtained prior to rhythmic exercise were 730 (7) N for CLIMB and 660 (65) N for SED. These were again not different between groups nor from those obtained prior to the sustained exercise protocol, supporting the repeatability of MVC determination between experiments. However, exercise times to fatigue for rhythmic contractions were almost double in CLIMB [853 (75.6) s] compared to SED [420 (68.9) s] ($P < 0.05$).

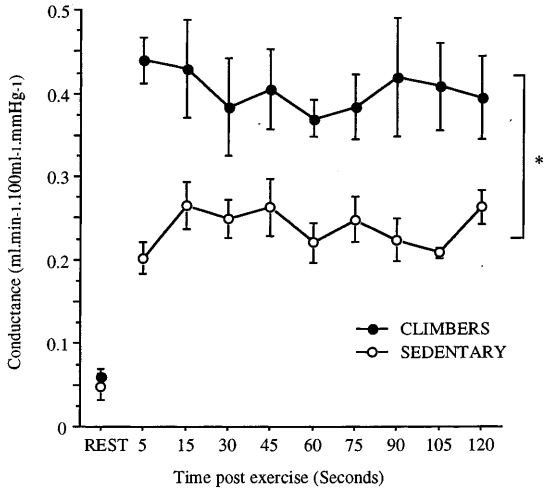

Fig. 2 Forearm vascular conductance at rest and following sustained isometric handgrip exercise to fatigue. $*P < 0.05$; climbers vs sedentary

As with sustained exercise, SBPs and DBPs were taken at time points representing rest, 25, 50, 75 and 100% (END) of the exercise times. With the large differences in exercise times to fatigue between groups it must be emphasised that the percentage duration points represent similar relative, rather than absolute, time points. Resting SBPs and DBPs were similar to those obtained in the sustained exercise experiment. The peak SBPs attained during rhythmic exercise were 158 (10) mmHg [21.0 (1.3) kPa] and 174 (9) mmHg [23.1 (1.2) kPa] in CLIMB and SED respectively, and, although apparently lower in CLIMB, were not significantly different. While peak SBP was reached at 100% of duration to fatigue in the SED group, it occurred at 75% in CLIMB. Thereafter, between 75 and 100% of duration time SBP declined, although this was not significant ($P = 0.08$). Peak DBPs occurred at fatigue in both groups and were not significantly different. The mean increments in SBP and DBP also show these changes (Fig. 3A, B) and demonstrate a trend for increases in DBP, but not SBP, to be lower in CLIMB than in SED throughout exercise. Resting HR were similar to those measured prior to the sustained exercise. Peak HR occurred at the end of exercise, reaching 84 (1) beats $\cdot$ min^{-1} in CLIMB and 107 (4) beats $\cdot$ min^{-1} in SED ($P < 0.05$).

There were large variations in vascular conductance measured during rhythmic exercise, probably arising from difficulties in keeping the forearm still during the brief intervals required to make blood flow measurements. However, as Fig. 4 shows, conductance throughout tended to be higher in CLIMB than in SED. During the post-exercise period blood flows were significantly greater in CLIMB, ranging between 38.7 (4.9) and 48.9 (9.3) ml $\cdot$ min^{-1} $\cdot$ 100 ml^{-1} compared to SED, 24.3 (5.7) to 28.6 (3.4) ml $\cdot$ min^{-1} $\cdot$ 100 ml^{-1} ($P < 0.05$).

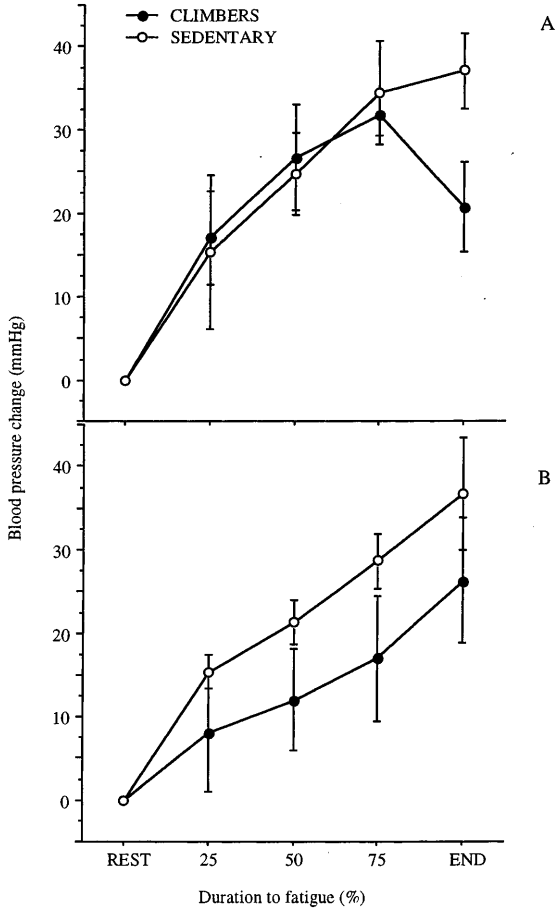

Fig. 3 Changes from resting values in systolic (**A**) and diastolic (**B**) blood pressure during rhythmic isometric handgrip exercise to fatigue

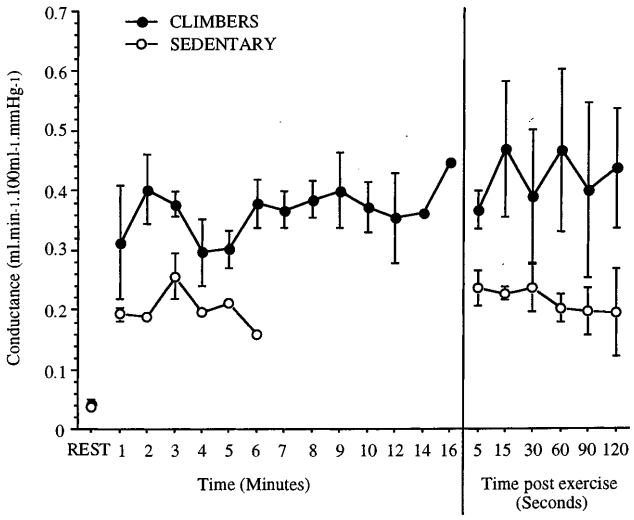

Fig. 4 Forearm vascular conductance at rest, during and following rhythmic isometric handgrip exercise to fatigue

Post-occlusion reactive hyperaemia

Blood flows at rest were 3.8 (0.8) and 3.0 (0.4) ml · min^{-1} · 100 ml^{-1} in CLIMB and SED respectively (NS). The highest level of blood flow attained upon release of the 10-min arterial occlusion was 50.2 (6.7) ml · min^{-1} · 100 ml^{-1} in CLIMB compared with 38.5 (3.6) ml · min^{-1} · 100 ml^{-1} in SED, which did not quite reach significance. Mean arterial blood pressures (MAP), which at rest were 87 (3) and 88 (3) mmHg [11.6 (0.4) and 11.7 (0.4) kPa] in CLIMB and SED, respectively, did not change either during the 10-min occlusion or during the 2-min post-occlusion period during which flow was measured. Because of this, it can be assumed that the changes in flow to the forearm are entirely due to vasodilatation of the muscle vascular bed.

Calculated vascular conductance at rest and at timed intervals during the 2-min post-occlusion period is shown in Fig. 5. There were no differences in resting values but conductance was significantly higher in CLIMB than SED for 60 s following release of the occlusion cuff ($P < 0.05$). The time course of post-occlusion conductance changes as shown in Fig. 5 disguises the fact that peak conductance was attained at different time points after occlusion release for different individuals. For most of the CLIMB group, peak conductance was reached later than that for SED where it occurred almost immediately upon occlusion release. Taking peak conductances for individuals revealed that mean peak conductance for CLIMB was significantly higher [0.597 (0.084) ml A min^{-1} · 100 ml^{-1} · $mmHg^{-1}$] than for SED 0.431 (0.034) ml · min^{-1} · 100 ml^{-1} · $mmHg^{-1}$] ($P < 0.05$).

Discussion

This investigation sought to determine the effects of training status on the arterial BP and vascular conductance responses to forearm isometric exercise and arterial occlusion using elite rock climbers as the trained population compared to non-climbing sedentary control subjects.

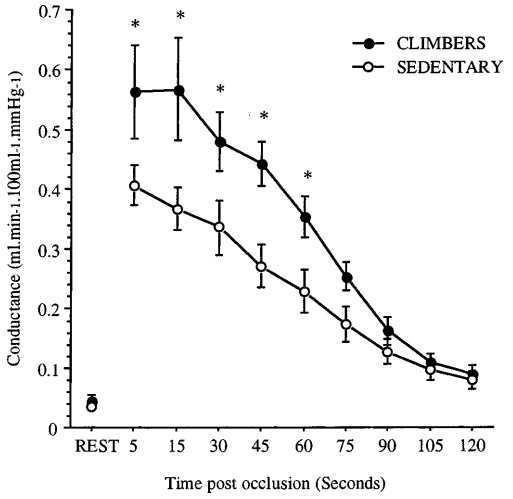

Fig. 5 Forearm vascular conductance following 10 min of arterial occlusion. *$P < 0.05$; climbers vs sedentary

There was no difference in MVC between the CLIMB and SED subjects which may be due to the small number of subjects in each group. However, this is surprising and is in contrast to results of Grant et al. (1996) who reported significantly greater MVCs in their climbing subjects compared to non-climbing controls. Watts et al. (1993), however, suggested that elite climbers may not need to develop high levels of grip strength since it was indicated that the strength to body mass ratio was more important than absolute grip strength. Unfortunately body mass was not measured, and thus it cannot be determined whether any difference in the strength to body mass ratio exists between the two groups of subjects in the present investigation.

There was no significant difference in holding time to fatigue during the sustained exercise between the CLIMB and SED subjects. This is also surprising as it may be thought that climbers would be able to continue to maintain grip for a greater length of time than non-climbers. Previous investigations have demonstrated a negative relationship between MVC and relative endurance such that stronger individuals have the lowest isometric endurance and those with lesser maximum strength possess superior relative endurance (Carlson 1969; Carlson and McCraw 1971). Therefore, as climbers seem to be no stronger than sedentary subjects, there are no potential detrimental effects on the ability to maintain sustained contractions, which would be the consequence of a greater strength. In contrast to sustained exercise, however, times to fatigue during rhythmic isometric exercise were twofold longer in the CLIMB compared to the SED subjects.

There was a clear attenuation of the BP response during sustained isometric handgrip exercise to fatigue in the trained individuals. Exercise intensity and duration are known to have a profound influence on the pressor response. Therefore, since there are no differences in MVC or exercise time between groups, the effects on BP observed cannot be attributed to these factors. There were no significant differences between CLIMB and SED subjects in either SBP or DBP during rhythmic isometric handgrip exercise. There was a trend, however, for SBP to drop during the latter quarter of the exercise. This could be consistent with the sustained exercise SBP response described above. To the author's knowledge, the only other study that has reported a pressor response attenuation in trained individuals was that by White et al. (1995). They measured the BP responses to both voluntary and stimulated contractions of the triceps surae before and after a 5- to 6-week training period. The training resulted in a significant attenuation of the SBP response during the voluntary contractions and also during the 2-min post-exercise circulatory occlusion period in the trained limb. DBP was also reduced during both voluntary and

stimulated exercise as well as during the post-exercise occlusion.

The present data, and results from White et al. (1995), could be consistent with reports describing reduced efferent activity in the form of attenuated MSNA during contractions in trained limbs, as measured by microneurography techniques (Somers et al. 1992). Factors that contribute to the level of MSNA and thus the pressor response include the level of metabolite build-up in the muscle that stimulates muscle chemoreceptors. Therefore, it is conceivable that a reduced afferent input to the pressor response is brought about by a reduction in metabolic by-products causing less stimulation of chemosensitive afferent nerve endings. The peripheral effects of endurance training are well documented (Holloszy and Coyle 1984). Metabolic adaptations include less lactate production and greater muscle buffer capacity. These latter adaptations would result in a lesser accumulation on H^+ ions, one of the many metabolites that stimulate the chemosensitive nerve endings. The present study's subject population were elite rock climbers who are probably not highly aerobically trained due to the relatively low maximal O_2 uptake ($\dot{V}O_{2\,max}$) reported for this group, i.e. $\approx$55 ml $\cdot$ min^{-1} $\cdot$ kg^{-1} (Billat et al. 1995). Although it is difficult to describe the trained status of climbers, the high intensity and ischaemic nature of rock climbing presumably induce adaptations within the skeletal muscle and vascular bed. Whether fewer metabolic by-products are produced as a result of the intense ischaemic contractions of the forearm when climbing can only be speculated upon. It may be possible, however, that even if metabolic adaptations do not occur the attenuated pressor response may be due to the afferent nerve endings themselves being desensitised to the build-up of metabolites.

The present study was unable to distinguish between central and peripheral components of the pressor response since no comparison between voluntary and electrically evoked contractions was made. White et al. (1995), however, suggested that there is no central adaptation to training as BP responses to isometric exercise in untrained contralateral limbs were unchanged. In any case, the pressor response attenuation reported is in contrast with a previous report that demonstrated unchanged BP responses to isometric exercise in trained individuals despite a training-induced reduction in MSNA. Somers et al. (1992) attributed the failure to detect any BP attenuation to their measurement technique, i.e. semi-automated sphygmomanometer recordings whereby only one recording per minute was made.

Two previous investigations have reported that training does not culminate in MSNA attenuation during isometric exercise. Seals (1991) reported the increase in total minute activity of MSNA to be 161% for trained endurance athletes compared to 204% in the untrained subjects during isometric handgrip exercise, this difference being insignificant. Satio et al. (1993) compared MSNA between the dominant and non-dominant forearms of racket sport players and reported no differences in the MSNA between the forearms. Both these studies recruited mainly endurance-trained individuals which may suggest that endurance training, per se, does not influence the MSNA response to isometric exercise, whereas other training paradigms may indeed reduce the magnitude of the MSNA response.

The blood flow and conductance results during and following both sustained and rhythmic exercise clearly demonstrate an enhanced forearm vasodilator capacity in the trained climbers. This supports previous investigations describing enhanced vasodilator capacities following arterial occlusion and ischaemic exercise in trained individuals, i.e. Sinoway et al. (1986), Snell et al. (1987) and Smolander (1994), and also the results from the post-arterial occlusion in the present study. The changes in vascular conductance could be attributed to structural adaptations of the local vascular bed, e.g. increases in the number or cross-sectional area of the capillary bed or the arteriolar vessels having a greater capacity to accommodate blood flow, or functional adaptations such as alterations in the myogenic, metabolic, neural or endothelial control of the muscle vascular bed, or both. There is indeed evidence from animal studies that endothelial-dependent dilator function is enhanced by training (Delp et al. 1995). We have described an attenuated pressor response which may be the result of a reduced MSNA activity. Reduced MSNA ultimately leads to less sympathetic vasoconstrictor activity thus allowing greater dilatation and blood flow. Whatever the mechanisms involved, a greater vasodilator capacity would enable a more efficient substrate supply and metabolite removal during any rest period in a climb, and could contribute to the climbers' ability to maintain sustained and repetitive high-intensity contractions over long periods of time. This has been clearly demonstrated during the rhythmic exercise.

In conclusion it can be seen that elite rock climbers are suitably adapted for the nature of their sport. They are not significantly stronger and thus their submaximal static holding time is not reduced during exercise but they have a twofold improvement in endurance performance during rhythmic isometric exercise. They also have an attenuated BP response to static exercise which may be caused by either a desensitisation of the afferent nerves or a reduced build-up of metabolites causing less stimulation of chemosensitive afferent nerve endings in the muscle. They demonstrate superior peripheral vascular characteristics in that they have an enhanced vasodilator capacity in the forearm, as demonstrated by both post-occlusion and post-exercise hyperaemia conductance. This may in part be due to less active sympathetic vasoconstrictor activity. Furthermore, if there is a reduced metabolite build-up then the enhanced vasodilator capacity may be due to additional mechanisms such as augmented release of endothelial-derived relaxing factor.

Acknowledgements The authors gratefully acknowledge the assistantship of Mr Tom Blakely and Miss Alison Fowler. The research was supported by the School of Sport and Exercise Sciences, Faculty of Science, The University of Birmingham.

References

Billat V, Palleja P, Charlaix T, Rizzardo P, Janel N (1995) Energy specificity of rock climbers and aerobic capacity in competitive sport rock climbers. J Sports Med Phys Fitness 35:20–24

Carlson B (1969) Level of maximum isometric strength and relative load isometric endurance. Ergonomics 12:429–435

Carlson B, McCraw L (1971) Isometric strength and relative isometric endurance. Res Q 42:244–250

Delp MD (1995) Effects of exercise training on endothelium-dependent peripheral vascular responses. Med Sci Sports Exerc 27:1152–1157

Ferguson RA, Brown MD, Blakely T, Fowler A (1996a) Attenuation of the pressor response to sustained isometric handgrip exercise in trained athletes. J Physiol (Lond) 495:136P

Ferguson RA, Brown MD, Blakely T, Fowler A (1997) Forearm maximal vasodilator capacity in elite rock climbers. J Sports Sci 15:49

Grant S, Hynes V, Aitchison T, Whittaker A (1996) Anthropometric, strength, endurance and flexibility characteristics of elite and recreational climbers. J Sports Sci 14:301–309

Holloszy JO, Coyle EF (1984) Adaptations to skeletal muscle to endurance exercise and their metabolic consequences. J Appl Physiol 56:831–838

O'Leary DS, Sheriff DD (1995) Is the muscle metaboreflex important in control of blood flow to ischaemic active skeletal muscle of conscious dogs? Am J Physiol 268:H980–H986

Patterson GC, Whelan RF (1955) Reactive hyperaemia in the human forearm. Clin Sci 14:197–211

Satio M, Watanabe H, Mano T (1993) Comparison of muscle sympathetic nerve activity during exercise in dominant and non-dominant forearm. Eur J Appl Physiol 66:108–115

Seals DR (1991) Sympathetic neural adjustments to stress in physically trained and untrained humans. Hypertension 17:36–43

Sinoway LI, Musch TI, Minotti JR, Zelis R (1986) Enhanced maximal metabolic vasodilatation in the dominant forearms of tennis players. J Appl Physiol 61:673–678

Smolander J (1994) Capacity for vasodilatation in the forearms of manual and office workers. Eur J Appl Physiol 69:163–167

Snell PG, Martin WH, Buckey JC, Blomqvist CG (1987) Maximal vascular leg conductance in trained and untrained men. J Appl Physiol 62:606–610

Somers VK, Leo KC, Shields R, Clary M, Mark AL (1992) Forearm endurance training attenuates sympathetic nerve response to isometric handgrip in normal humans. J Appl Physiol 72:1039–1043

Watts PB, Martin DT, Durtschi S (1993) Anthropometric profiles of elite male and female competitive sport rock climbers. J Sports Sci 11:113–117

White MJ, Carrington CA, Hazell D (1995) Training induced attenuation of the pressor response to isometric exercise in man In: Sutton JR, Thompson MW, Torode ME (eds) Tenth Biennial Conference on Exercise and Thermoregulation. The University of Sydney

Whitney RJ (1953) The measurement of volume changes in human limbs. J Physiol (Lond) 121:1