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BLOOD FLOW IN THE RESTING FOREARM DURING PROLONGED CONTRALATERAL ISOMETRIC HANDGRIP AT MAXIMAL EFFORT

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SUMMARY

1. In earlier studies we have shown that muscle contraction performed as handgrip at constant force, one third of maximal voluntary contraction (MVC), induces a rapid vasodilation in the resting contralateral forearm which in all probability is neurogenically mediated, followed by a relative increase in resistance.

2. The maintenance of contraction at one third MVC for 2 min requires continuously increasing effort because of fatigue, as also evidence by the e.m.g. The biphasic response of the vascular bed may then be related to the increasing *intensity* of somatomotor activation which is needed to maintain contraction force, or, alternatively, to differences in vasomotor activity on *initiation* of and *continued* muscle activity.

3. To elucidate these two possibilities blood flow in the resting forearm was measured during contralateral handgrips at constant maximal *effort* for 6 min (in which case force will drop) and compared to handgrip at constant *force*, one third MVC, for 2 min.

4. The flow reaction during prolonged contraction at maximal effort was similar to that induced by contraction at constant force with a marked transient lowering of vascular resistance, although maximal vasodilation tended to be more pronounced and occur earlier.

5. The results indicate that the rapid decrease in vascular resistance is related primarily to the *initiation* of somatomotor activity, whereas continued muscle contraction produces a relative increase in vasoconstrictor activity irrespective of changes in contraction effort.

INTRODUCTION

Intense muscle activation is accompanied by pronounced and rapidly occurring increases in arterial pressure and heart rate, the initial phase being of neurogenic origin (Tuttle & Horvath, 1957; Lind, Taylor, Humphreys, Kenelly & Donald, 1964; Freyschuss, 1970; Delius, Hagbarth, Hongell & Wallin, 1972; Eklund, Kaijser & Knutsson, 1974). Its effect on vascular resistance in a peripheral vascular bed was analysed by Eklund *et al.* (1974) by measuring arterial pressure and blood flow in the resting forearm during contralateral handgrip. Isometric handgrip at one third of maximal voluntary contraction force (MVC) for 2 min was found to induce an initial decrease in vascular resistance in the resting forearm, followed by a return to precontraction level towards the end of the contraction period. The rapid

occurrence of the increase in blood flow suggested neurogenic mediation, and in a subsequent study it was shown that to a large extent it was mediated by β-adrenergic mechanisms, since it could be attenuated by regional β-adrenergic blockade (Eklund & Kaijser, 1976). The gradual increase in resistance towards the end of contraction, which was found, could be abolished by regional α-adrenergic blockade. Thus it seemed to be the result of a continuous increase in α-adrenergic relative to β-adrenergic effects.

There are several possible interpretations of this biphasic effect on vascular resistance. (1) It may be peripheral in origin. Contraction at constant force requires increased effort due to progressive fatigue and the continuous increase in somatomotor activation which is then developed to maintain force (Clarke, Hellon & Lind, 1958) may be paralleled by an increase in activity in sympathetic vasomotor fibres. A 'dose–response' relationship (Brungardt, Swan & Reynolds, 1974) to the effect that with moderately increased activity predominantly β-receptors whereas with higher activity predominantly α-receptors are stimulated may then explain the biphasic effect on vascular resistance. (2) Alternatively a shift between predominant activity in anatomically distinct β-adrenergic and α-adrenergic fibres might occur at C.N.S. level. The shift may then be the result of increased stimulation of the vasomotor centres in parallel with the increasing somatomotor activation, produced by the increasing effort to maintain tension with fatiguing muscle fibres, or it may be related to time so that initiation of somatomotor activity is paralleled by activity in vasodilator β-adrenergic fibres, while this activity is replaced or dominated by activity in α-adrenergic vasoconstrictor fibres as the muscle contraction is prolonged.

In the present investigation the effect on blood flow in the resting forearm of a prolonged maximal voluntary isometric handgrip, where the effort was kept high and *constant* was compared to the effect of handgrip at constant submaximal force where the effort was continuously *increasing*, to elucidate whether the dual vascular response is determined mainly by the changing intensity of somatomotor activation or by different vasomotor effects of initiation and continuation of activation. To estimate semiquantitatively to what extent the flow changes occurred in skeletal muscle oxygen saturation in the deep vein of the forearm under study was followed.

METHODS

Eight healthy male volunteers of average physical fitness were studied. Their ages ranged between 20 and 40 years. They were carefully informed about the nature and purpose of the experiment before giving their consent to participate.

For recording of arterial pressure a brachial artery was catheterized percutaneously with a teflon catheter, outer diameter 1·4 mm. The deep venous system of the resting forearm was catheterized with the same type of catheter, introduced in the distal direction and manipulated into a deep vein so that the tip could not be palpated. The distance it had been introduced, 4–8 cm, was measured and checked again after the experiment. With the catheter in this position the blood sampled drains mainly muscle tissue if the circulation of the hand is occluded (Coles, Cooper, Mottram & Occleshaw, 1958; Idbohrn & Wahren, 1964).

Blood flow in the resting forearm was measured by venous occlusion plethysmography with an air-filled plethysmograph (Dohn, 1956; Graf & Westersten, 1959), placed around the thickest part of the forearm. During the recording of inflow curves the circulation of the hand was occluded. Blood flow was calculated in ml. min^{-1}. 100 ml^{-1} of forearm tissue.

Arterial pressure was measured by a capacitive transducer (EMT 34 or 35, Siemens–Elema). Mean pressure was obtained by electrical damping, time constant 3 sec.

Oxygen saturation of the deep venous blood was analysed by an Instrumentation Laboratories CO-Oximeter, model 182.

Isometric contralateral handgrip was performed with a strain gauge hand-dynamometer. The force developed was displayed to the volunteer on an oscilloscope (to make it possible to maintain contraction at a predetermined force) and recorded on an ink-jet recorder (Siemens–Elema) together with e.c.g., intra-arterial pressure and plethysmographical inflow curves.

The subjects were studied in the recumbent position with the resting arm at heart level. For the first part of the experiment they were instructed to maintain a handgrip contraction at maximal effort for 6 min, without activation of other muscle groups than those of the arm and without breathholding or performing a Valsalva manoeuvre. The hand with which the grip was performed rested on a pillow, the elbow slightly flexed. Arterial mean pressure and e.c.g. were recorded continuously before, during and for 3 min after the contraction. Plethysmographic inflow curves were recorded with shortest possible intervals, for 4–6 min before, during and after the handgrip. Deep venous blood was sampled at rest, at 1, 2, 4 and 6 min of the contralateral handgrip and again 0·5, 1·5 and 3 min after the contraction.

After about 45 min rest the subject performed a contralateral handgrip at constant force, one third of MVC, for 2 min. MVC was then taken to be the maximal force developed during the preceding 6 min contraction at maximal effort. Arterial mean pressure, e.c.g. and plethysmographic inflow curve were recorded as during prolonged contraction. Deep venous blood was sampled before and after contraction and at 30, 60, 90 and 120 sec of contraction.

RESULTS

Values in the text are given as mean ± S.E. of mean for the group unless otherwise stated. Significances of differences between experimental situations are calculated from paired data.

Contralateral isometric handgrip at one third of MVC for 2 min (Fig. 1)

Most subjects could maintain contraction at the predetermined force for 2 min but in some of them force fell below that level during the last 15 sec, suggesting that 2 min is close to the maximal time for which it is possible to keep one third of MVC with the equipment and arm position used in the present study.

The effects of contralateral isometric contraction at one third of MVC on the resting forearm blood flow, deep venous oxygen saturation, vascular resistance, arterial mean pressure and heart rate were similar to those found in previous series of experiments (Eklund *et al.* 1974; Eklund & Kaijser, 1976). Forearm blood flow showed a maximal increase of 127 ± 25% ($P < 0·01$) reached at 15–75 sec of contraction, and forearm vascular resistance had decreased by 46 ± 4% at 30–60 sec ($P < 0·01$). Deep venous oxygen saturation increased by about 16% from 64·7 ± 3·7 to 74·6 ± 3·4% at 1 min ($P < 0·01$).

Contralateral maximal isometric handgrip for 6 min (Figs. 2 and 3)

The maximal force developed, after about 5 sec of contraction, was 440 ± 35 N (44·5 ± 3·5 kp). It decreased rapidly during the first minute to 168 ± 12 N and then more slowly to level off at about 100 N. The time course of decrease in developed force was similar to that earlier described during prolonged contraction at maximal effort (Stephens & Taylor, 1972) indicating that the subjects kept the handgrip maximally. Heart rate rose rapidly to attain a maximal value at 15 sec and then decreased slightly to a fairly steady level about 15 beats/min above resting level.

Arterial mean pressure increased rapidly during the first minute to remain unchanged at about 125 mmHg from the second minute.

Forearm blood flow increased significantly during the first 15 sec ($P < 0{\cdot}001$) and reached a maximal value at 30–45 sec, the average maximal increase being $182 \pm 20\%$.

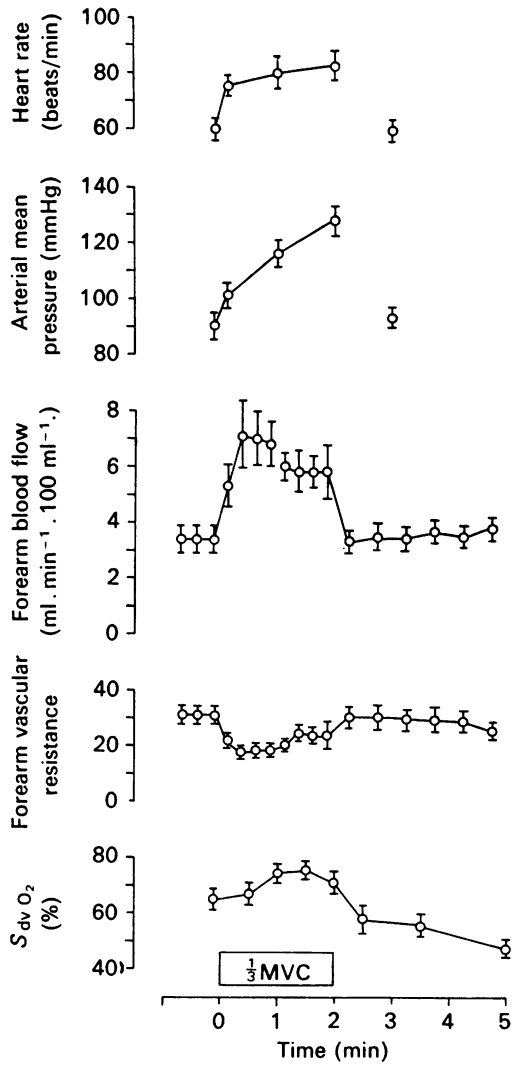

Fig. 1. Heart rate, arterial mean pressure, and blood flow, regional vascular resistance and deep venous oxygen saturation in the resting forearm during contralateral handgrip at one third of maximal voluntary contraction force for 2 min. Mean ± S.E. of mean for eight subjects.

The increase in flow at 30 sec tended to be greater than that produced by contraction at one third of MVC but the difference did not attain significance ($0{\cdot}05 < P < 0{\cdot}10$). The maximal increase was not significantly greater than that produced by handgrip at one third MVC. The time course, however, was different since the blood flow

decreased signficantly from 15–30 sec to 1 min–1 min 30 sec ($P < 0{\cdot}01$), while it was unchanged during that period of contraction at one-third MVC. Up to 3 min it remained at a level significantly above the resting value, but during the rest of the period it did not differ from that level. Forearm vascular resistance decreased significantly during the first 15 sec ($P < 0{\cdot}05$) and decreased further to an average lowest value $54 \pm 4\%$ below the value before contraction at 30–45 sec ($P < 0{\cdot}01$).

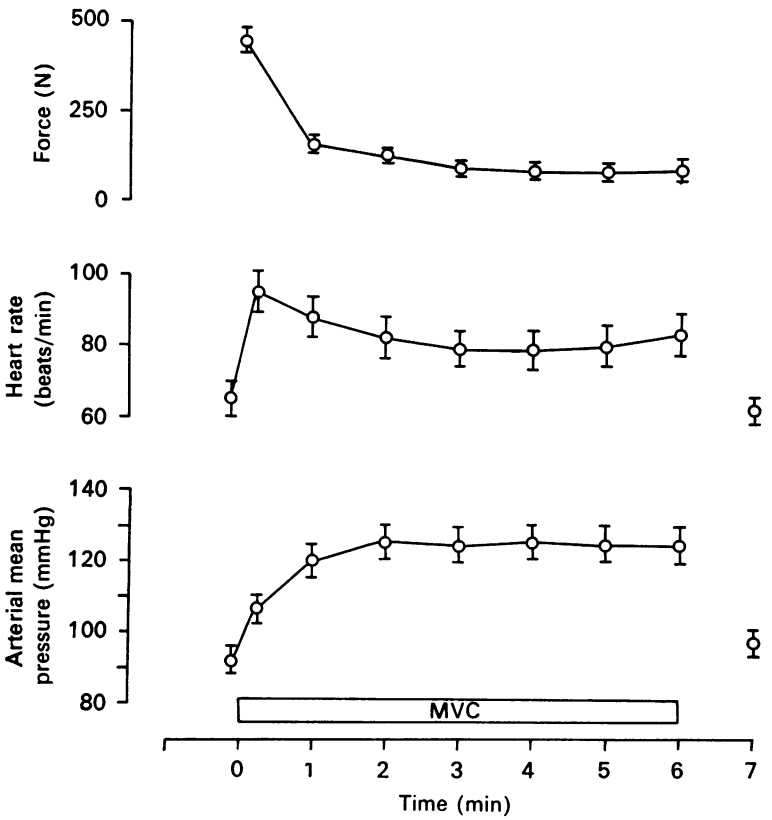

Fig. 2. Developed force, heart rate and arterial mean pressure during handgrip performed at maximal effort for 6 min. Symbols as in Fig. 1.

The maximal decrease was not significantly different from that during handgrip at one third MVC. At 1·5–2 min of contraction it had returned to a value not different from that before contraction.

Deep venous oxygen saturation had increased significantly at 1 min ($P < 0{\cdot}01$), and decreased again to attain a level below the initial value at 4 min ($P < 0{\cdot}01$). At 6 min it was not significantly different from the resting value.

DISCUSSION

The major finding of this study is the similarity in flow reaction (rapid increase followed by relative decrease) produced by constant force (i.e. increasing effort) and constant effort handgrip. Thus the biphasic response in regional vascular resistance

in the resting forearm on muscle activation seems not to be related to the intensity of somatomotor activation to the effect that low grade activity induces predominantly β-adrenergic effects, since similar changes occurred with constant effort and increasing effort contraction. Instead the rapid and transient flow increase at supposedly constant degree of somatomotor activation seems to indicate that the effect on the

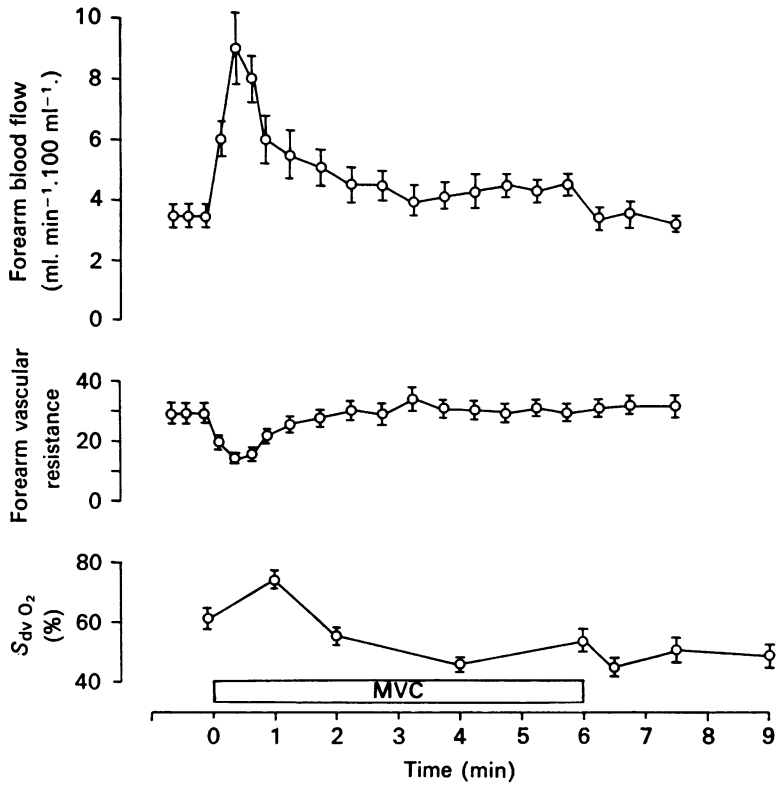

Fig. 3. Blood flow, regional vascular resistance and deep venous oxygen saturation in the resting forearm during contralateral handgrip performed at maximal effort for 6 min. Symbols as in Fig. 1.

vascular bed is related primarily to time so that the *initiation* of somatomotor activity is paralleled by activity in vasodilator β-adrenergic fibres, which is then replaced by or counteracted by activity in vasoconstrictor α-adrenergic fibres. Such a link between somatomotor and vasomotor activity is probably at C.N.S. level although the possibility of modulation on peripheral receptor level cannot be ruled out. Thus an initial predominant activation of β-receptors might gradually be inhibited by decreasing sensitivity to the released transmitter, unmasking α-adrenergic effects. However, on this issue the experiment only permits speculation.

Although the maximal decrease in resistance during the first 2 min was not significantly different between the contraction performed at one third MVC and at maximal effort, it tended to be slightly more pronounced and the maximal change was reached earlier during contraction at maximal effort. This may indicate that

the intensity of somatomotor activation does also have some influence on vasomotor activation and the relation between β- and α-adrenergic effects.

Deep venous oxygen saturation showed similar increases the first minute of both modes of contraction, confirming that a substantial fraction of the flow increase occurred in muscle. Like the total forearm blood flow the muscle blood flow, as judged from the deep venous oxygen saturation, tended to decrease again earlier during the maximal than the submaximal handgrip, but unlike the total forearm blood flow it reached a level significantly below initial at 4 min. This would suggest that the relative resistance increase which followed the transient decrease during prolonged contraction was more pronounced in muscle than in skin and subcutaneous tissue. Similarly, decreased blood flow in resting muscle has been found during steady state dynamic leg work (Blair, Glover & Roddie, 1961) after a transient flow increase at the onset of work (Bevegård & Shepherd, 1966). Comparing isometric and dynamic exercise, however, it must be remembered that in the latter case muscle tension is generally far less and hence the intensity of activity in motor neurones lower, which in all probability means that a possible 'irradiation' to vasomotor centres is weaker. Furthermore, in dynamic leg work where a great proportion of the total body muscle vascular bed is subject to metabolically induced vasodilatation, other mechanisms than in the case of isometric handgrip, to a great extent reflexogenic, must operate to adjust the circulation.

While the initial vascular reactions on muscle activation, as discussed previously, seem to be elicited directly by central nervous mechanisms and mediated neurogenically, blood pressure and flow during prolonged contraction must be affected to a great extent by reflexes from, e.g. the baroceptors and also circulating catecholamines, released from the adrenal medulla. Blood catecholamine concentrations have been shown to increase continuously throughout prolonged forceful handgrip, but since heart rate and arterial mean pressure as well as forearm blood flow remained constant from the third to the sixth min of contraction in the present study, an influence of circulating catecholamines seems to have been of minor importance. The levelling off of the mean arterial pressure after about 3 min of contraction could then suggest an elevated 'set point' of the baroceptors (see Smith, 1974). The gradual pressure increase followed by levelling off at a higher level is then compatible with the assumption that an alteration in set point occurs rapidly at the onset of contraction, but that the new pressure level is attained only slowly due to the competing effect of the initial centrally mediated vasodilation. However, the exact interplay between central and peripheral effects on vasomotion during prolonged muscle contraction cannot be established by the present study.

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