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LOCAL AND CENTRAL CIRCULATORY RESPONSES TO SUSTAINED CONTRACTIONS AND THE EFFECT OF FREE OR RESTRICTED ARTERIAL INFLOW ON POST-EXERCISE HYPERAEMIA

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SUMMARY

1. The cardiovascular responses to sustained contractions at tensions from 5 to 30 % maximal voluntary contraction (MVC) have been examined. At 5 and 10 % MVC blood pressure, heart rate and forearm blood flow all reached a steady state during the contraction; post-exercise hyperaemia did not show peak flows higher than those found during exercise. At tensions of 20 and 30 % MVC, none of the measurements showed a steady state during the contractions, but increased steadily throughout the contraction; post-exercise hyperaemia characteristically showed peak flows in excess of any flow measured during contractions. The results obtained at a tension of 15 % MVC did not show a steady-state during the contraction but the following hyperaemia showed a similar pattern to that seen at the lower tensions.

2. Digital compression of the brachial artery after sustained handgrip contractions for periods of 3 or 6 min after the contraction ended resulted in only a small reduction, on average by 5–15 %, of the post-exercise hyperaemia.

3. Consideration of the evidence leads to the view that in physiological circumstances the post-exercise hyperaemia following sustained contractions bears a close relationship to the metabolism of the active muscles.

INTRODUCTION

The increase in blood flow through a muscle during and after exercise has been considered to be a functional response since Gaskell (1877) first described the phenomenon in detail. A specific and quantitative relationship between the size of the post-exercise hyperaemia and a 'blood debt' (both expressed here in ml. blood/100 ml. tissue) created during the period of exercise was suggested by Freeman (1935) and since then this concept

has been supported by the findings of other groups of workers (McArdle & Verel, 1956; Coles & Cooper, 1957; Clarke & Hellon, 1959; Humphreys & Lind, 1963), all of whom have found a close relationship between the degree and the duration of muscular activity and the magnitude of the ensuing hyperaemia. This relationship seems to be particularly close during and after sustained contractions, possibly owing to the more precise control of that type of muscular activity. Such evidence strongly suggests that the amount of blood flowing to the muscles during and after exercise is related to the metabolism of the exercising muscles.

Evidence that the 'blood debt' in healthy subjects is not necessarily strictly related to the metabolism of the muscles comes from two sources, in both of which the blood supply to the muscle was restricted during or after the contraction. Dornhorst & Whelan (1953) observed a reduction of about 25% in the magnitude of the post-exercise hyperaemia after rhythmic calf exercise when the arm was enclosed in a pressure plethysmograph so that the effective local blood pressure in the enclosed arm segment was reduced by about 50 mm Hg. Blair, Glover & Roddie (1959) restricted the arterial inflow to the forearm by applying digital compression to the brachial artery for variable periods (from 1 to 5 min) immediately after a bout of rhythmic exercise. In these circumstances, the arterial inflow was reduced to resting level, and arterial compression for only 4 min reduced the magnitude of the post-exercise hyperaemia, on average, by 60-65%. The magnitude of this reduction seems surprisingly large in the light of the amount of blood allowed to reach the muscle during the period of brachial arterial occlusion. Although it seems reasonable to expect that, for instance, appropriate amounts of gaseous exchange can occur even when the arterial inflow is greatly restricted, it does not follow that the uptake or release of some solid metabolites into the blood is similarly independent of the volume of blood circulating through the muscle. A further complication in the understanding of the hyperaemic response to exercise is provided by the finding of Barcroft, Greenwood & Whelan (1963), who showed (their Fig. 2) that arterial compression applied to the brachial artery both during and for 5 min after a sustained contraction ended did not result in a decrease of the combined exercise and post-exercise hyperaemia. In consequence of these rather confusing results, it was decided that further systematic study was required into the relationship between exercise and the subsequent hyperaemia. Sustained contractions were chosen as the medium of muscular contraction in the belief that this form of exercise could be more accurately controlled than rhythmic exercise.

The present investigation was intended (1) to establish the extent of hyperaemic responses to systematically graded sustained hand-grip contractions covering a range of tensions which would include both

fatiguing and non-fatiguing contractions, and (2) to find out whether or not the post-exercise hyperaemia following sustained contractions could be substantially reduced by restriction of the arterial inflow for a short period after the contraction ended. The results of some of these experiments have been briefly reported before (Lind, McNicol & Taylor, 1965).

METHODS

Nine healthy males, aged 22–39 yr, volunteered to act as subjects. Four of them were thoroughly trained in the use of the hand-grip dynamometer and were accustomed to all the procedures which they undertook. These four subjects took part in all the series of experiments described below; the remaining five subjects were not trained and were examined in only one of the series of experiments.

In all the experiments reported here the exercise performed by the subjects was a sustained hand grip on a simple strain gauge dynamometer (Clarke, Hellon & Lind, 1958).

Two series of experiments were planned. There were several features of the second series which required additional examination to determine whether the results already obtained were valid. For simplicity of presentation, these subsidiary experiments have been described under separate headings.

Series 1. This series of experiments was required to augment information already available (Humphreys & Lind, 1963) on the blood flow during and after sustained contractions from tensions between 30 and 70 % maximal voluntary contraction (MVC). In this part of the investigation the responses to tensions from 5 to 30 % MVC were examined. Each subject immersed his arm in a water-bath at 34° C for 20 min to stabilize muscle temperature at 36° C (Barcroft & Edholm, 1946). Four successive contractions were then made at 5, 10, 15 and 20 % MVC for 3 min each; 10 min were allowed between each contraction as a recovery period. Also, for each subject, the responses to a tension of 30 % MVC were studied separately to ensure that the responses to it would not be affected by possible residual fatigue from a preceding 3 min contraction at 20 % MVC. All the above experiments were repeated by each subject. Forearm blood flows were measured by the Whitney (1953) strain gauge plethysmograph for 2 min before the contraction, during the 3 min contraction and for 3 min afterwards. The mercury-in-rubber bracelet of the plethysmograph was sited over the most muscular part of the forearm and the position marked to allow of exact replacement in this and in the following series of experiments. Blood pressures, measured by auscultation with a microphone stethoscope, were made every minute in the pre- and post-contraction periods and every 30 sec during the contraction. Heart rates were recorded continuously by e.c.g. and counted over 30 sec periods.

Series 2. 1. Each of the four trained subjects immersed his arm in a water-bath at 34° C throughout each experiment, during which he made three successive contractions at 30 % MVC, all held for 90 sec. There were three separate treatments after the contractions, (*a*) free circulation was allowed through the forearm (control response), (*b*) the arterial flow was restricted by complete compression of the brachial artery against the humerus in the axilla (total compression), (*c*) the blood through the brachial artery was partially occluded by digital compression of the artery as before, with the intention of allowing a forearm blood flow of approximately one third of the peak hyperaemic flow in a control response (partial compression). These three procedures were presented in random order in any one experiment and no more than one experiment was completed by one subject in any 24 hr period. To ensure that no large differences occurred due to the site of compression of the brachial artery, there were several additional experiments in which the arterial compression was applied, in the antecubital fossa. Forearm blood flow was measured for 3 min before, during and after each contraction as well as during and after the compression when this was applied.

The period of compression was constant in any one experiment at 3 or 6 min, durations that were deliberately chosen to approximate the duration (or twice the duration) of the control hyperaemia. Each hyperaemia was assessed from a representation of the results on semi-log paper, plotted against time as suggested by Dornhorst & Whelan (1953). Blood pressure was measured every min or more frequently during periods of expected rapid change. Heart rate was recorded continuously by e.c.g. Blood flows were measured by the Whitney strain-gauge plethysmograph.

The first contraction was made after a 20 min immersion of the forearm in the water-bath. Twenty minutes were allowed between the end of one period of measurement and the start of the next.

In this series of experiments the local and central cardiovascular responses of the five untrained subjects were also measured when they performed one control contraction and when their brachial arteries were compressed both partially and totally for 3 min after contractions.

Series 2. 2. Venous pressure in the forearm was measured in three subjects during experiments which otherwise imitated those in Series 2.1. In addition to the measurements described above, venous pressure was measured in the test forearm by inserting a nylon catheter into the vein at the antecubital fossa. The catheter was attached to a Statham P 23 Db strain-gauge transducer and recording and calibration devices as described in detail elsewhere (Lind *et al.* 1964). The pressure recording was interrupted briefly to withdraw venous samples as required. The catheter was allowed to remain in the antecubital vein for some experiments while in others it was pushed distally until its tip lay in the deep venous plexus that drains the muscles of the forearm.

The influence of the arterial compression on reactive hyperaemia in the forearm was examined by measuring the blood flow for 3 min in the resting forearm, during 6 min of total compression and for the following 3 min.

Series 2.3. The procedure described for Series 2.1 was once more repeated but the forearm blood flow was measured by a conventional, water-filled (34° C) plethysmograph. The arm was not immersed in water except for the segment enclosed by the plethysmograph. Post-exercise hyperaemias were measured in control conditions and following total and partial compression of the brachial artery applied for 3 min after the contraction. Otherwise all procedures and measurements were exactly as in Series 2.1.

Series 2.4. The procedure and measurements described for Series 2.1 were modified only by allowing the arm to be held in air instead of immersing it in water. The subjects sat in a comfortably warm laboratory (23–26° C) throughout the experiment. Hyperaemias were measured after control contractions and following compression applied for 3 min or 6 min.

Series 2.5. To determine whether there was any progressive or systematic change in the magnitude of the post-exercise hyperaemias after three successive contractions in any one experiment, blood flow was measured on each subject before (2 min) during (90 sec) and after (3 min) each of three successive contractions when there was no interference with the arterial blood supply to the muscles after any contraction.

RESULTS

Series 1. *Cardiovascular responses to graded sustained contractions.* Figure 1 shows the blood flow through the forearm at rest and in response to exercise at different tensions ranging from 5 to 30% MVC. Resting blood flow before each contraction was approximately 2·5 ml./100 ml. min on each occasion. Both during and after contractions, the forearm blood flow was obviously graded according to the tension exerted. In addition, there was a clear difference in the pattern of response found at 5 and 10%

MVC compared to that at 20 and 30 % MVC, whereas the results obtained at a tension of 15 % MVC did not clearly follow one pattern or the other.

At 5 % MVC the forearm blood flow increased as soon as the contraction started and reached a steady state at about 7·5 ml./100 ml. min in approximately 1 min. When the contraction ended, the forearm flow fell immediately below the steady state level found during exercise and returned to the

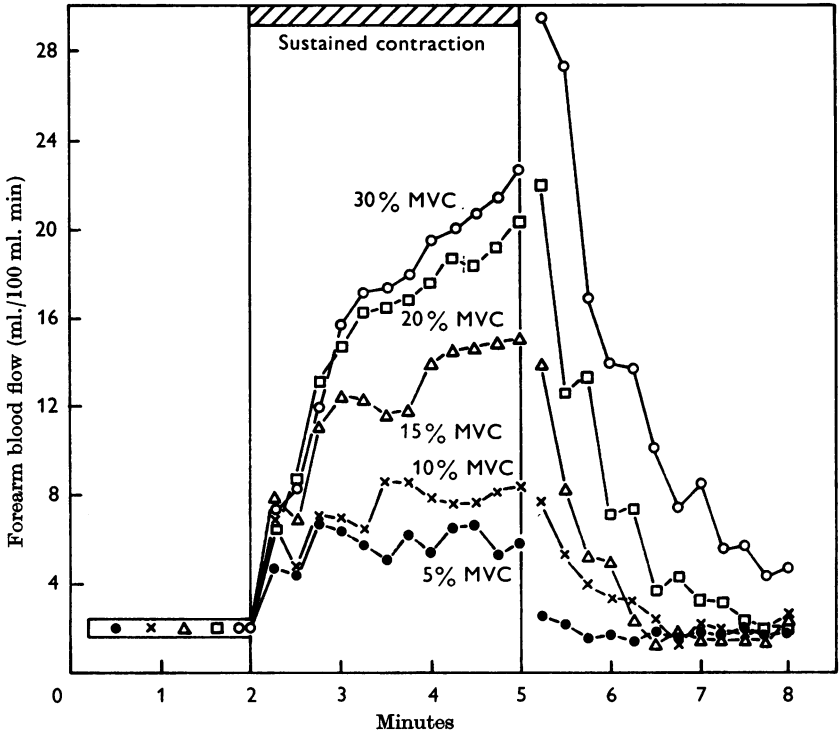

Fig. 1. Forearm blood flows in response to 3 min sustained contractions at tensions from 5 to 30 % MVC. At 5 and 10 % MVC, the responses reached a steady state, whereas at 20 and 30 % MVC they did not. At 15 % MVC, the response did not fit the pattern found at higher or lower tensions.

resting level in about 1 min. At 10 % MVC the pattern of response was similar but it took longer (1·5 min) to reach the steady state. Moreover, the steady state was achieved at a higher level, approximately 10 ml./100 ml min; also, the flows fell immediately after the contraction ended but took about 1·5 min to return to the resting level, slightly longer than it took after a tension of 5 % MVC. By contrast, at 20 % MVC, the forearm blood flow increased steadily throughout the contraction to 21 ml./100 ml. min at the end of the 3 min contraction. After the contractions, the blood flows rose to a peak flow higher than that seen during the contractions

(on these occasions 25 ml./100 ml. min) and then fell in a curvilinear fashion to reach resting level. In the present instance the post-exercise hyperaemia lasted for about 3 min. When the tension was 30% MVC the forearm blood flows were somewhat higher both during and after the contraction, and the post-exercise hyperaemia lasted for about 5 min. These findings at 20 and 30% MVC follow the pattern of results typically seen during sustained contractions that result in muscular fatigue (Humphreys & Lind, 1963). The results obtained at 15% MVC followed neither of the patterns of results described. Blood flows did not reach a steady state during the contraction (and this was true of much longer contractions at the same tension) but, after the 3 min contraction ended, the flows fell at once and reached resting values in about 2·5 min. These results appeared to confirm the belief that a sustained contraction at a tension of 15% MVC is on the borderline between contractions at tensions that result eventually in fatigue and those which do not result in fatigue (Rohmert, 1960; Monod & Scherrer, 1965).

Figure 2 shows the 'blood deficit' during sustained contractions and the resultant 'blood debt' that was incurred. The upper part of the diagram shows, in stylized form, the type of response found in a non-fatiguing and in a fatiguing contraction. During the contraction, the 'blood deficit' is represented by the shaded area over the curve and after the contraction the 'repayment' is shown by the shaded area under the curve. In non-fatiguing contractions the 'deficit' during the contraction was equalled by the 'repayment' and no 'debt' occurred. It appeared that the blood flow through the muscles during the contraction was sufficient for the metabolic needs of the muscle. In contrast, during contractions at tensions which would result eventually in fatigue, the blood flow never achieved a steady state during the contraction. The magnitude of the 'repayment' following such contractions always exceeded that of the 'deficit'—the difference between these two values represents the 'blood debt'. The dimension of the 'blood debt' depends on the tensions exerted and the duration of the contraction. The lower half of Fig. 2 shows the 'blood debts' obtained after 3 min contractions at tensions ranging from 5 to 30% MVC. Above a tension of about 15% MVC the blood flow during the contraction was at no time adequate for the metabolic needs of the muscle.

During the contractions at 5% MVC the blood pressure and heart rate increased to reach a steady state with increments over resting values of 10/8 mm Hg and 4 beats/min. They fell to resting values within a min of the end of the exercise. At 10% MVC the steady state levels were, respectively, 13/12 mm Hg and 6 beats/min above resting values and again resting levels were reached within a min of the end of the exercise. The pattern of central cardiovascular responses at 5 and 10% MVC was,

thereby, similar to that seen for the blood flow. At 15, 20 and 30% MVC neither blood pressure nor heart rate achieved a steady state during the contraction; the increments in blood pressure were 14/13, 18/16 and 27/23 mm Hg respectively, while heart rate was correspondingly increased by 8, 11 and 26 beats/min. After the contraction ended both the blood pressure and the heart rate returned to resting values within 1 min.

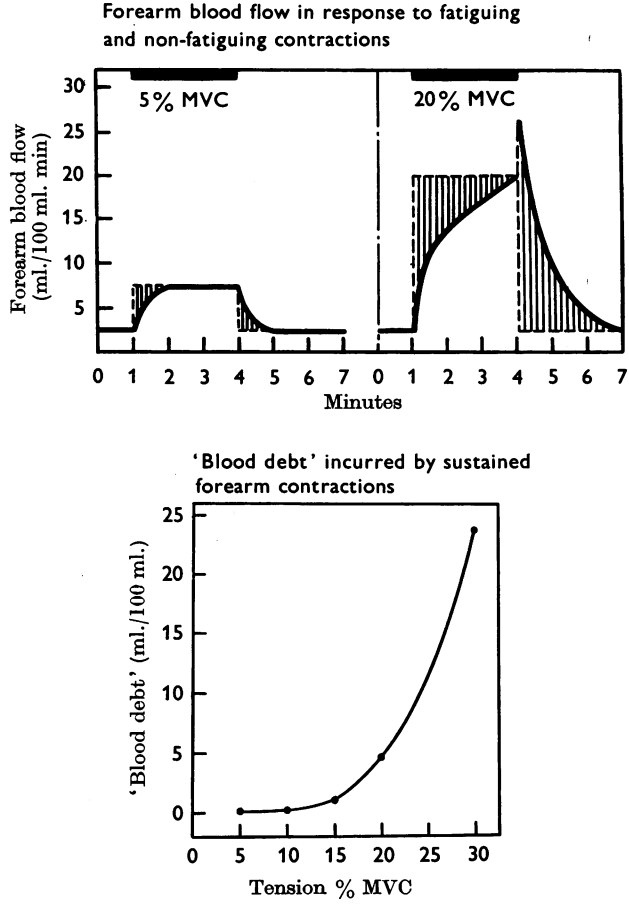

Fig. 2. In the upper part of the diagram is shown the type of blood flow response to a non-fatiguing contraction (5% MVC) and to a fatiguing contraction (20% MVC). At 5% MVC the shaded area above the curve during the contraction ('blood deficit') equals the post-exercise hyperaemia (shaded area below curve). During fatiguing contractions the shaded area above the curve is less than the post-exercise hyperaemia because the blood flow during the contraction is never sufficient for the muscles' metabolic needs, and therefore a 'blood debt' is created.

In the lower part of the diagram the 'blood debt' (the difference between the post-exercise hyperaemia and the 'blood deficit' during exercise) is shown for contractions held for 3 min at tensions from 5 to 30% MVC. At non-fatiguing tensions there is no 'blood debt'.

Series 2.1. *Arterial compression and post-exercise hyperaemia.* All contractions in the second part of this investigation were performed at a tension of 30% MVC and were therefore contractions that would eventually result in fatigue. Consequently, the blood flow throughout all these contractions increased steadily throughout the contraction period, which was kept constant at 90 sec. The blood flow measurements showed that the magnitude of the hyperaemia during the contraction itself was quite reproducible for each subject. This was an important finding, as otherwise, if the results from the first part of the investigation were physiologically meaningful, the post-exercise hyperaemia might be expected to vary reciprocally with the exercise hyperaemia. The average exercise hyperaemia for all subjects was 25 ml./100 ml. and the range for individual subjects was ± 4%.

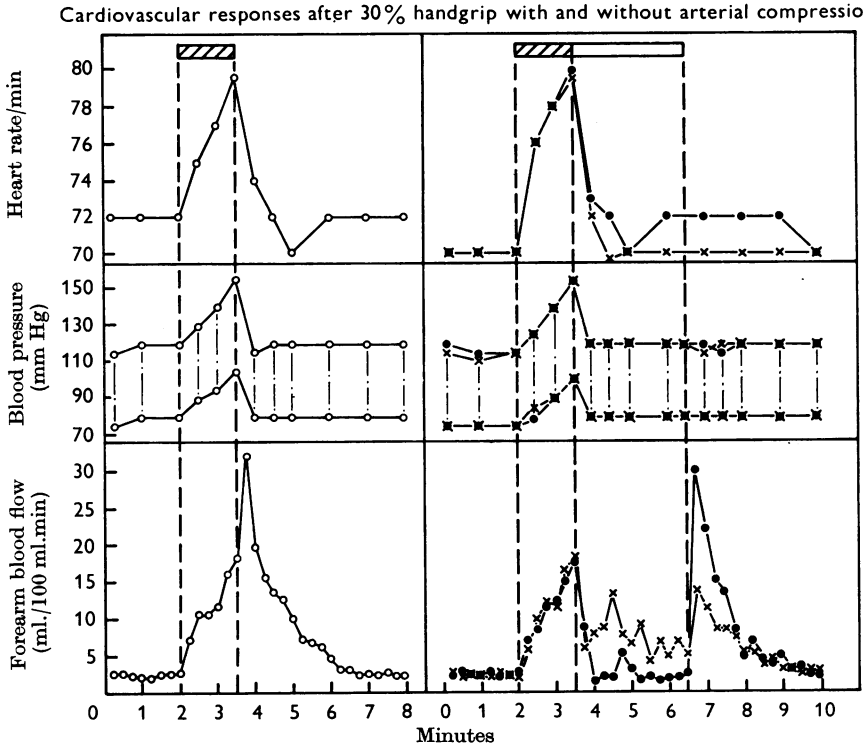

Fig. 3. Details of the blood flow through the forearm of one subject before, during and after three consecutive 90 sec hand-grip contractions at 30% MVC. There was an interval of 20 min between contractions. On the left is seen the control experiment where there was no interference with the local circulation. On the right are seen the blood flow responses to the contraction when 3 min total (●) and 3 min partial (×) compression was applied to the brachial artery immediately after the contraction.

Figure 3 shows a complete set of results obtained from one subject when he performed three successive contractions at 30% MVC for 90 sec each, after two of which either total or partial brachial arterial compression was applied. The arm was immersed in water at 34° C. The control hyperaemic response is shown on the left of Fig. 3 and the responses obtained when total or partial compression was applied for 3 min after the contraction are shown on the right. Blood pressures and heart rates during these manoeuvres were measured but are not shown in Fig. 3; they rose steadily throughout each contraction to reach, on average, levels 35/25 mm Hg and 18 beats/min respectively greater than resting values. They fell rapidly to resting values when the contraction ended, in a manner similar to that described in detail elsewhere (Lind, Taylor, Humphreys, Kennelly & Donald, 1964). Neither response was noticeably affected by the arterial compression; actual values were almost identical before, during and after each contraction, in common with the results obtained from the other subjects. During the control contraction the forearm blood flow rose steadily and was followed by a post-exercise hyperaemia which, in this case, lasted for about 3 min. When total compression was applied to the brachial artery, the collateral arterial vessels supplied a blood flow similar in size to the resting blood flow; it is recognized that because local vasodilatation of blood vessels will occur in the working muscles only, the bulk of this flow must be through these vessels. Since no measurement could be made of the actual flow through these dilated vessels, the procedure used to calculate the post-exercise hyperaemia was the conventional one of subtracting the total forearm flows at rest from those measured after exercise. Thereby, during total compression of the artery when the total forearm flow was the same as the resting flow, there would be a functional hyperaemia of unknown dimension through the previously active muscles. The method of calculating the post-exercise hyperaemia cannot take this phenomenon into account and thereby will underestimate the actual post-exercise hyperaemia. During partial compression of the artery the forearm blood flow was rather variable but was always considerably in excess of the resting flow. Again, a large proportion of this flow must be to the exercised muscles. After the release of compression the blood flow showed the distinctive and characteristic appearance of normal post-exercise hyperaemia, although the magnitude depended on the amount of blood allowed to reach the forearm during the period of compression. The total post-exercise hyperaemias shown in Fig. 3, including those in excess of resting values during the periods of arterial compression, were 28·9 ml./100 ml. for the control condition and 29·5 and 26·5 ml./100 ml. respectively for contractions followed by a total or partial arterial compression.

The results for all the subjects showed a similar pattern although there

was some variation in the extent of the hyperaemia obtained in the different circumstances. Table 1 shows the magnitude of post-exercise hyperaemias obtained from the four trained and also from the five untrained subjects who had their brachial arteries totally and partially

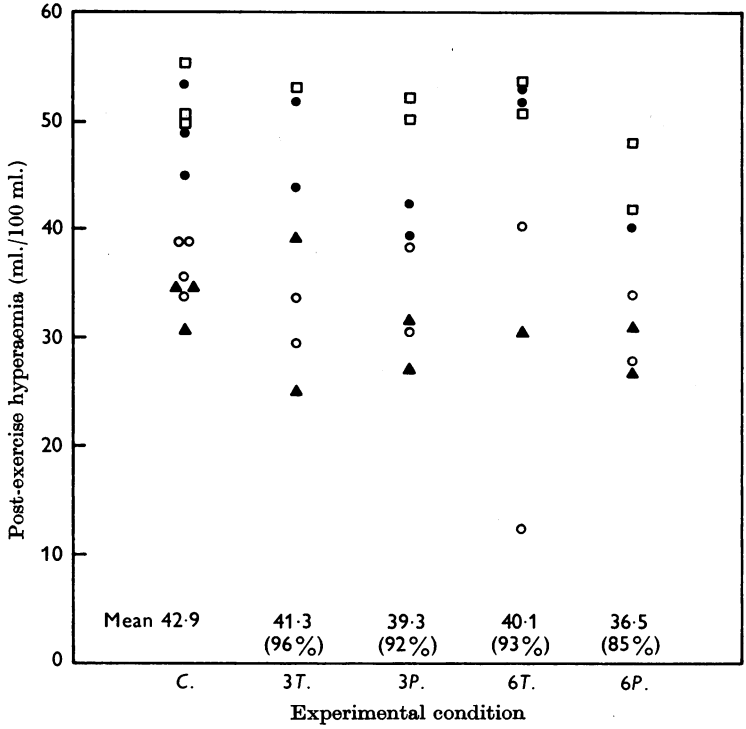

Fig. 4. The post-exercise hyperaemias for the four trained subjects after a 90 sec hand-grip at 30% MVC with the control values (*C*) shown on the extreme left, and during and after 3 min total (*3T*), 3 min partial (*3P*), 6 min total (*6T*) and 6 min partial (*6P*) compression of the brachial artery immediately after the contraction ended. Different symbols represent results from different subjects and the mean values and their percentage relationship with the control values are shown at the bottom of the diagram.

occluded for 3 min after the contraction ended. On average, the post-exercise hyperaemia after 3 min total occlusion was 97% of the control value (individual values ranged from 83 to 103%), while after partial occlusion the average hyperaemia was 94% of the control value (ranging from 79 to 113%). Figure 4 shows the more extended measurements made on the four trained subjects whose brachial arteries were totally and partially occluded for 3 min, and also for 6 min. In all, including the values for the five untrained subjects in Table 1, arterial inflow was restricted on thirty-nine occasions. On only one of these occasions was the magnitude

of the post-exercise hyperaemia less than 79% of the control value; one subject showed a hyperaemia of 35% of the control value after total compression for 6 min. There was no obvious technical reason to question the validity of this result but it was obviously exceptional; a repeated experiment on the same subject under the same conditions resulted in a hyperaemia of 110% of the control value. Averaging all the values obtained

TABLE 1. The post-exercise hyperaemias in nine subjects following hand-grip contractions at 30% MVC for 90 sec. The arm was immersed in water at 34° C. Values in brackets represent percentages of the control values

		Hyperaemia (ml./100 ml.)	
Subject	Control	With 3 min total compression of brachial artery	With 3 min partial compression of brachial artery
1	37·4	36·3 (97)	30·6 (82)
2	73·2	75·1 (102)	83·0 (113)
3	51·0	50·9 (100)	49·0 (96)
4	49·2	47·8 (97)	43·2 (88)
5	36·3	34·2 (94)	35·0 (96)
6	33·1	33·0 (100)	30·9 (93)
7	38·9	32·4 (83)	30·5 (79)
8	36·0	37·2 (103)	37·0 (102)
9	36·1	33·8 (94)	30·1 (83)
Mean	43·5	42·3 (97)	41·0 (94)

(including the low value mentioned) the reduction in post-exercise hyperaemia due to 3 min total and partial occlusion was 4 and 7% respectively below control values, while for 6 min total and partial occlusion it was 8 and 15% respectively. It appeared that partial occlusion of the artery resulted in a greater reduction of the post-exercise hyperaemia than total occlusion; this was a logical finding, since during partial occlusion a greater volume of blood was made available to the muscles. All the values shown in Table 1 and Fig. 4 have taken into account the influence of reactive hyperaemia due to the compression of the artery (see below).

Some experiments were also carried out with the arterial compression applied in the antecubital fossa; the results were not detectably different from those described above when the compression was applied to the axilla.

Investigation of factors which might affect the magnitude of post-exercise hyperaemia. Some features of the experimental procedures used in investigating the effect of arterial compression on post-exercise hyperaemia might have influenced the findings. It was decided that each of these possibilities should be examined.

Series 2.2. Venous pressures were measured in three subjects, both in the antecubital vein and distally in the deep venous plexus draining the forearm muscles. The results were similar for each subject wherever the

site of measurement. The recorded venous pressure was between 7 and 8 mm Hg in the resting forearm. During and after the contraction, variable increases in pressure occurred, related to the muscular tension. Such increases were sometimes as large as 20 mm Hg. When successful arterial compression was applied after a contraction, venous pressures only increased by about 2 mm Hg, whereas deliberate concomitant compression of the veins resulted in a further increase of 15 mm Hg or more. It seemed

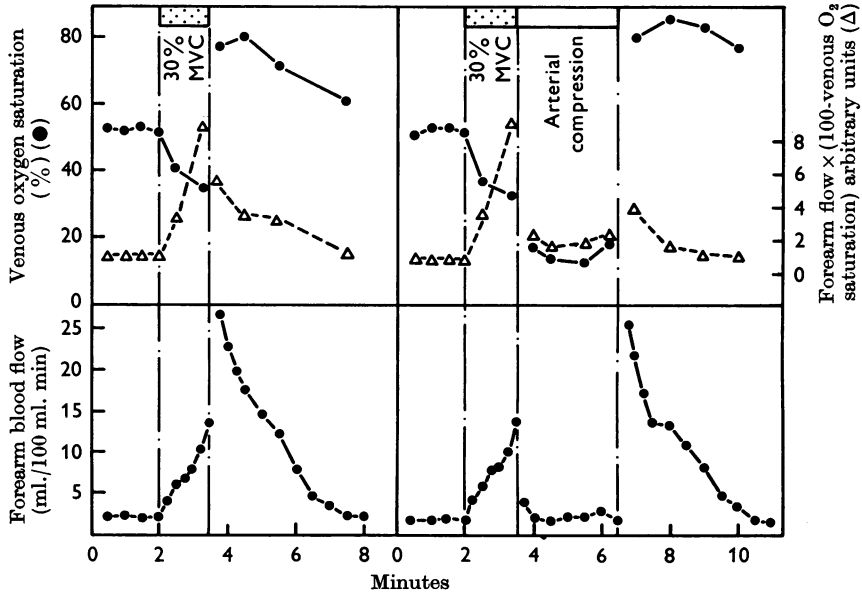

Fig. 5. The blood flow through the forearm, the venous oxygen saturation (●) and the forearm oxygen uptake (Δ) in arbitrary units before, during and after contractions for 90 sec at 30% MVC. In one control contraction, shown on the left, there was no interference with the local circulation. On the right is shown the response to a contraction which was immediately followed by 3 min total compression of the brachial artery.

unlikely that arterial compression with no increase in forearm volume and with an increased venous pressure of only 2 mm Hg would invalidate normal plethysmographic measurements.

Venous samples were drawn from the venous catheter at regular intervals throughout these experiments and oxygen saturations of the samples were determined by an oximeter. Results from one of the subjects are shown in Fig. 5, for a control contraction and for a contraction followed by 3 min partial compression of the artery. The results conformed exactly to the pattern seen by Barcroft *et al.* (1963). During the period of restricted arterial inflow the venous oxygen saturation fell to a very low level so that by the time the compression was released the oxygen requirements of

the forearm had been almost completely repaid. Nevertheless, the subsequent hyperaemia is evident in Fig. 5. As Barcroft *et al.* (1963) have pointed out, the oxygen needs of the tissues can thus be absolved of complicity in post-exercise hyperaemia.

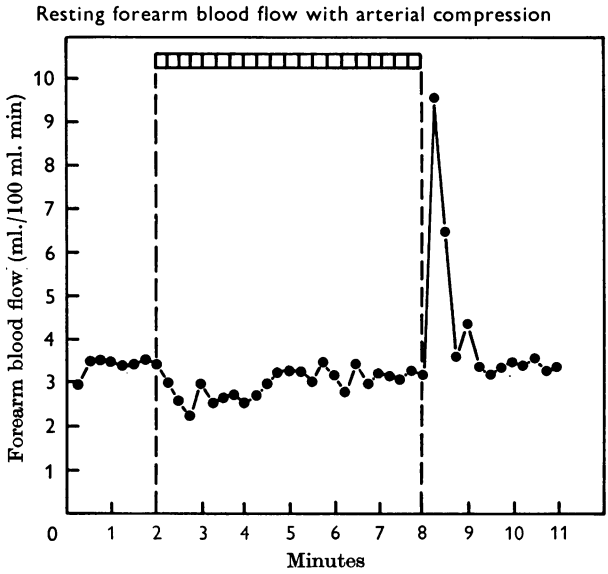

Fig. 6. The blood flow through the resting forearm of one subject before, during and after total digital compression of the brachial artery. There was a small decrease in forearm flow when the compression was applied, thereafter a gradual increase to about the level of resting flows. When the compression was released there was a short-lived but clear hyperaemia.

The possibility that the compression of the artery caused a reactive hyperaemia in the forearm had to be considered. Holling & Verel (1957) found a small hyperaemia in these circumstances while Blair *et al.* (1959) did not. The forearm blood flow was measured before, during and after digital compression of the artery for 3 or 6 min. There was a small fall in the amount of forearm blood flow when total arterial compression was applied (see Fig. 6) but the flow returned to control values during the first few min of the period of compression. Clearly, the condition for production of reactive hyperaemia occurred within the first 3 min of arterial compression. On release of the compression there was a small, transient but distinct reactive hyperaemia. This finding agrees with that reported by Holling & Verel (1957) but differs from that of Blair *et al.* (1959). The magnitude of the reactive hyperaemia varied from subject to subject, from 1·2 to 4·5 ml./100 ml., with an average value of approximately

3 ml./100 ml. In calculating the values for post-exercise hyperaemias seen in Table 1 and Fig. 4 above, appropriate reductions were made for each individual.

Series 2.3. Another factor to be investigated was that in these particular experimental circumstances, where there was interference with the post-exercise arterial inflow, different methods of blood flow measurement might result in different calculated hyperaemias. Accordingly, the conventional water-filled plethysmograph was used to measure post-exercise hyperaemia in four subjects in response to a control contraction and to contractions followed by a 3 min total and partial arterial compression. Blood pressures and heart rates responded in the same way as is described for the earlier experiments. The hyperaemic responses for the four subjects obtained with the different experimental treatments are shown in Table 2.

TABLE 2. Average post-exercise hyperaemias for four subjects after hand-grip contractions of 30% MVC for 90 sec

	Hyperaemia (ml./100 ml.)				
Treatment	Control	3 min total compression of brachial artery	3 min partial compression of brachial artery	6 min total compression of brachial artery	6 min partial compression of brachial artery
Arm immersed in water 34° C Whitney plethysmograph	42·3	41·4	39·2	41·0	40·0
Conventional water-filled (34° C) plethysmograph	35·9	38·3	36·3	—	—
Arm held in air (23–26° C) Whitney plethysmograph	23·7	24·9	—	23·7	—

There was no substantial difference in the size of the post-exercise hyperaemia due to these different treatments. Values for post-exercise hyperaemia were, however, slightly lower than those obtained in the same subjects when blood flow was measured by the Whitney strain-gauge plethysmograph, and when the arm was immersed in water at 34° C. There are two possible reasons for this small difference, (1) the temperature of the muscles may have been slightly different in the two series of experiments; temperature differences in the muscles are known to have a profound effect on the blood flow through the muscles during and after sustained contractions (Humphreys & Lind, 1963), and (2) the relative positions of the conventional plethysmograph and the bracelet of the strain-gauge plethysmograph can result in differences in measurements of forearm blood flow because of differences in the tissue composition of the areas sampled (Clarke & Hellon, 1959).

Series 2.4. Barcroft & Edholm (1946) have pointed out that although immersion of the forearm in water at a temperature of 34° C most closely imitates the temperature of the forearm in normal circumstances, differences in the temperature of some tissues exist. It was conceivable that immersion of the forearm in water might have modified the post-hyperaemic response in experiments described above. The hyperaemia after a control contraction and after those contractions followed by 3 min total or partial arterial compression were measured when the forearm was held in air in a comfortably warm laboratory. The four trained subjects were examined, and the contractions were held, as before, for 90 sec at 30 % MVC. The post-exercise hyperaemias are shown in Table 2 and it is evident that there was, once more, no sign of a large fall in the hyperaemic response when the arterial inflow was restricted after the contraction. The results differed from those obtained in earlier series of experiments in two ways—first, the variability of the results from contraction to contraction (irrespective of the treatment) was greater and, secondly, the magnitudes of the hyperaemias were much lower, roughly two thirds of the values found in exactly the same experimental conditions but for the fact that the arm was immersed in water at 34° C. Both of these differences can be accounted for by differences in muscle temperature. In a water-bath at 34° C the deep muscle temperature is 36° C (Barcroft & Edholm, 1946), the same as in the comfortably clothed forearm. In the bared forearm, in a comfortably warm room, the deep muscle temperature falls steadily to about 32·5° C, even in 1 hr, and may eventually reach a temperature as low as 30° C. A reduction of muscle temperature by these amounts results in the reduction of post-exercise hyperaemia by 30–50 % (Humphreys & Lind, 1963); moreover, quite small changes in the muscle temperature for the three consecutive contractions could very well account for the greater variability of the results between the contractions.

Series 2.5. It was possible that, in any one experiment, the performance of three successive contractions might introduce a progressive change in the magnitude of the post-exercise hyperaemia. To investigate this possibility each subject performed two experiments during which he carried out three successive contractions in the customary experimental protocol, but in no case was the arterial inflow restricted. The results for each subject are shown in Table 3. It is clear that there was no systematic difference in the size of the post-exercise hyperaemia.

In the second part of this investigation, then, the results shown in Table 1 and Fig. 4 represent a consistent picture of the post-exercise response to sustained contractions. The results have shown that in the circumstances of this investigation the restriction of arterial inflow to the forearm for a period of 3 min after the contraction ended reduced the post-exercise

hyperaemia by only 4–7 % on average while after an arterial compression of 6 min it was reduced by only 8–15 % on average. It has been shown that (1) a slight increase in venous pressure when the artery was compressed, (2) the effect of reactive hyperaemia due to the reduction of blood flow when arterial occlusion was applied, (3) the method of measurement of blood flow, (4) the influence of immersion of the arm in water and (5) the effect of making three consecutive contractions, had no clear or large influence on the pattern of results obtained.

TABLE 3. Showing post-exercise responses of four subjects to three successive contractions at 30 % MVC for 90 sec when there was no interference with the local circulation. There was an interval of 20 min between the contractions. The arm was held in air

	Contractions		
Subject	1	2	3
1	10·8	12·9	14·7
2	28·2	24·6	23·7
3	22·8	26·7	27·8
4	21·9	18·0	18·0
Mean	21·9	20·6	20·9

DISCUSSION

Examination of the hyperaemia resulting from sustained contractions offers some advantages over the study of response to rhythmic contractions. The amount of muscular activity can be more closely controlled in sustained contractions and the blood flow during such contractions can be measured easily. For these reasons the present study of post-exercise hyperaemia has involved only sustained contractions. Post-ischaemic hyperaemia has not been considered here since it involves tissues other than muscle, and also because there is reason to believe that the mechanism of reactive hyperaemia differs from that of post-exercise hyperaemia (Dornhorst & Whelan, 1953).

The present investigation can be considered in two parts. The first is concerned with the definition of both central and peripheral cardiovascular responses to sustained contractions at tensions ranging from 5 to 30 % MVC, while the second part examines the question of how these responses are modified by interfering with the arterial blood supply to the contracting muscles.

The results obtained from the first series of experiments performed here, taken in conjunction with information already available on hyperaemic responses to sustained contractions (Clarke *et al.* 1958; Clarke & Hellon, 1959; Humphreys & Lind, 1963; Lind *et al.* 1964), lead inescapably to the conclusion that, in physiological circumstances, the amount of blood

flowing through muscles during and after a sustained contraction is related to the tension exerted and to the duration of the contraction.

The supporting facts are: (1) during contractions of up to 10% MVC (at least) the blood flow through the forearm increases during the contraction to reach a steady state and after the contraction the blood flow does not increase above the values found during work. The small blood 'deficit' incurred during the early part of these contractions before a steady state is reached is similar in magnitude to the post-exercise hyperaemia. These contractions are usually thought to be indefatigable and can certainly be held for a long time; the blood supply during the steady state seems to satisfy the metabolic demands of the muscles. (2) When the tension is 20% MVC or higher, the blood flow through the forearm during the contractions does not reach a steady state but continues to increase for as long as the contraction is maintained. The blood 'deficit' is, therefore, not static as in non-fatiguing contractions, but is accumulative. The local blood supply does not at any time satisfy the metabolic demands of the muscle during the contraction. In the post-exercise hyperaemic phase the initial blood flows are higher than the flow found during the contraction and the total hyperaemia exceeds the blood 'deficit' that can be measured during exercise, thereby demonstrating a blood 'debt'. (3) The increase of forearm blood flow during contractions is confined to the active muscles. (4) The magnitude of the hyperaemia that occurs during a contraction of given tension and duration is steady provided that muscle temperature is constant. (5) There is a linear relationship at any given fatiguing tension between the duration of the contraction and the magnitude of the post-exercise hyperaemia if the muscle temperature is held constant. (6) For a given duration of contraction the magnitude of the post-exercise hyperaemia increases with the tension, if the muscle temperature is held constant. (7) For a given duration of contraction at a given tension the magnitude of the post-exercise hyperaemia increases as the muscle temperature increases by an amount similar to that expected by the increase in metabolism due to the temperature rise.

All these facts taken together present a picture of a steady-state condition during contractions that can be held for a long time and which can therefore be considered as theoretically indefatigable, since the blood supply is sufficient for the metabolic demands of the contracting muscle. In these experiments this includes tensions up to 10% MVC but the position of a tension of 15% is equivocal. During contractions at tensions of 20% MVC and above, in which fatigue eventually intervenes, the blood supply is apparently insufficient for the requirements of the muscles and there is an accumulating blood 'deficit' during the contraction which results in a 'debt' repaid during the post-exercise hyperaemia. The most

reasonable hypothesis to fit all these facts is that in the normal physiological state the blood flow through the muscles during and after contractions is functionally related to the metabolic requirements of the active muscles.

If some interference occurs in the circulation the customary physiological relationship between muscular activity and the subsequent hyperaemia may be altered. For instance, in pathological conditions arteriosclerosis or claudication can upset the relationship, often leading to a reduction but by no means to an abolition of post-exercise hyperaemia (Shepherd, 1963). Since control values are not available on these individuals for comparison and with concomitant reactive hyperaemia as a possible complicating factor, it is not feasible to assess the extent of such an effect accurately. However, if the circulation to the muscles of healthy individuals is artificially reduced, the post-exercise hyperaemia can also be reduced. After rhythmic contractions of the forearm, Blair *et al.* (1959) were able to reduce the post-exercise hyperaemia. After digital compression of the brachial artery for only 4 min or about the duration of the control hyperaemia, they found the hyperaemia was reduced, on average, by 65%; furthermore, they considered that total abolition of the hyperaemia would probably follow compression of the brachial artery for a period of about 8 min, or twice the duration of their control hyperaemia. In the present experiments, total compression of the brachial artery for a period of 6 min (twice the duration of our control hyperaemia) resulted in a reduction of the measured hyperaemia of only 8–15%. In fact, because of the functional hyperaemia that must have occurred through the working muscles when arterial compression had reduced the total forearm flow, it is inevitable that the calculated hyperaemia was underestimated. Therefore the calculated reduction in the hyperaemia due to arterial compression must be overestimated. The main difference in the methods employed in this investigation and that of Blair *et al.* (1959) lies in the type of exercise investigated. If the large differences in responses obtained in the two investigations are a true reflexion of the different types of exercise involved this raises interesting questions, which deserve further investigation, about the role of muscle metabolites in the production of post-exercise hyperaemia. It is generally accepted that post-exercise hyperaemia may be caused by a complex of metabolic stimuli (Hilton, 1953), the accumulation of which presumably reflects the preceding events in the muscle. At first sight it seems unlikely that different metabolites are produced in sustained or rhythmic contractions. However, the concentration of various metabolites at the end of bouts of the two types of exercise might be quite dissimilar, in view of the very different local conditions of capillary circulation and pressure that exist during those exercises.

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