FOREARM BLOOD FLOW DURING AND AFTER ISOMETRIC HAND-GRIP CONTRACTIONS

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SUMMARY

1. A method is described for the measurement of forearm blood flow during sustained voluntary isometric hand-grip contractions that can be maintained constant by the subject’s monitoring of the strength of his contractions.

2. In most subjects it was found that contractions of 5% of their maximal voluntary force could be maintained without fatigue, and without any post-exercise increase in blood flow. More powerful contractions produced both fatigue and a large post-exercise hyperaemia, even though blood flow had reached a plateau during the exercise.

3. An attempt to show that the post-exercise hyperaemia was a response to a steadily accumulating ‘metabolic vasodilator stimulus’ and varied directly in size with the duration of the contraction gave equivocal results.

4. Ischaemic pain in the hand was not the cause of the post-exercise hyperaemia.

Key words: blood flow, skeletal muscle, isometric muscle contraction, fatigue, exercise hyperaemia.

That activity of a muscle influences the rate of blood flow through it has been known since the observations of Chauveau & Kaufmann (1887). Grant (1938), applying the venous occlusion plethysmographic technique to human forearms, observed that exercise produced an increase in blood flow both during and after the exercise. Barcroft & Millen (1939) found that, in the calf muscles, strong contractions led to a cessation of blood flow, thought to be due to a rise in intramuscular pressure consequent upon the changing shape of muscle fibres within a rigid deep fascial sheath. They also studied the hyperaemia that followed contractions. Blood flow rose immediately to very high levels on relaxation, and then declined in an approximately exponential manner. This rise and the exponential decline are compatible with the hypothesis that during ischaemic exercise the concentration of some vasodilator product of metabolism steadily increases and that when blood flow is restored at the end of contraction, this substance is washed away in the effluent blood. It is equally possible that the post-exercise hyperaemia...
is due to the restoration of some material the tissue concentration of which had become de-
pleted during the contraction, to the failure of supply of some blood-borne material, to a
neural vasodilator reflex mechanism with a considerable after-discharge, or to a myogenic
autoregulatory response as in post-occlusional hyperaemia (see Rodbard, 1971, for discussions
of these possibilities).

The systematic study of hyperaemia during muscular contraction in man depended upon
the development by Clarke, Hellon & Lind (1958) of their isometric hand-grip ergometer.
With this instrument Humphreys & Lind (1963) showed that it was possible to maintain and
measure forearm blood flow during a sustained muscular contraction. They further showed
that the hyperaemia was confined to the active muscles. Flow through inactive muscules was
unaffected by the contractions. The success of their technique is attributable to the fact that
external movement (the attempt to flex the fingers into a fist) was only about 1.0 mm with a
maximal grip effort. The alteration of shape of the muscles and thus the compression of blood
vessels were reduced to a minimum.

Since the work of Humphreys & Lind (1963) clearly showed that blood continued to flow
through active muscles and that a constant contraction force produced a constant proportional
flow response, it was possible to plan metabolic studies of exercising muscle in a steady
state of activity and hyperaemia, using the method of simultaneous venous occlusion plenthsy-
mography and sampling of muscle effluent blood (Mottram, 1955). Zierler (1961) has shown
that it is only when such equilibrium conditions exist between tissue metabolism, blood flow
rate, and movement of substances between blood and metabolizing tissues that the Fick
principle can be used to study metabolism.

Before proceeding to such studies it was essential to investigate further the blood flow changes
during and after sustained contractions at varying strengths and durations in order to define
the conditions under which a sustained contraction of constant force produced a constant
hyperaemia that fully met muscle requirements. This paper reports the results of such investi-
gations and discusses their implications.

In some of the studies reported in this paper large post-exercise hyperaemias were seen to
follow contractions during which the blood flow increase had reached a steady level. If this
post-exercise hyperaemia were due to an accumulating metabolic deficit, then it would be
expected to increase with increasing duration of exercise. If, however, compression of blood
vessels by the contracting skeletal muscle were to elicit a myogenic autoregulatory response
due to a low pressure downstream from the point of compression, then the post-exercise
hyperaemia would be unaffected by the length of exercise. Results of studies designed to
investigate this are also reported here. Some of the results have been briefly reported before

**METHODS**

An ergometer for performing quantitated, isometric hand-grips was built similar to the design
of Clarke et al. (1958) the spring-steel bar used having the same dimensions and specification
as theirs. Maximal hand-grip contractions might deflect the free end of the bar by as much as
1 mm. Bar deflection was detected by a strain-gauge dynamometer (J. Langham Thomson,
Type UFl), the electrical output of which was fed to a galvanometer and to one channel of a
pen recorder. By watching the galvanometer the subjects could sustain constant-strength
contractions at pre-determined levels. Each subject’s maximal strength was determined at the onset of the study and the test contractions were always related to the individual’s own maximal effort on a percentage scale (Humphreys & Lind, 1963). They are referred to as % MVC (maximal voluntary contraction) in this paper.

Blood flow was measured by the venous occlusion plethysmographic method, using a 15.2 cm (6 in) long plethysmograph, filled with water at 34°C. Volume change was detected by the electrical conductivity method and recorded on the second channel of the pen recorder. Five 6 s venous occlusions, at a cuff pressure of 60 mmHg, were used per minute to measure blood flow. ‘Blood flow’ was always determined as the rate of change in volume during venous occlusion, this being referred to the horizontal grid lines on the recorder chart. This technique takes no account of the relation between arterial inflow, venous outflow and the size of the blood pool in the limb once the 6 s period of venous obstruction ceases. In most studies the hand was isolated from the circulation for 1 min prior to and throughout the period of recording, but in some the entry of hand effluent blood into the plethysmograph was prevented by simultaneous occlusion of the veins at the elbow and the wrist.

With these techniques for recording the blood flow during voluntarily maintained hand-grips the following were investigated.

1. Site of the increase in blood flow

Scans of forearm skin temperature were made by thermography simultaneously over the skin of both forearms while one hand was making a sustained 10% MVC hand-grip. Twelve subjects were studied, a single pair of thermograms being obtained on each.

2. The greatest strength of contraction that can occur with a constant hyperaemia during contraction and no marked post-exercise hyperaemia

Each one of twelve subjects was studied while performing 2 min contractions at 5, 10, 15 and 20% MVC. The order in which these were undertaken was randomized and each contraction studied twice in each subject at a single experimental session.

3. Effect of wrist occlusion

In four further subjects the effect of recording blood flow by the technique of simultaneous venous occlusion at the wrist and elbow was compared with the normal blood flow recording method while they performed 4 min contractions at 10% MVC. Each technique was used twice in each subject. In two of these subjects 4 min contractions at 15% MVC were also studied.

4. Effect of variation in length of contraction on the post-exercise hyperaemia

The blood flow responses during and after contractions known to produce a post-exercise hyperaemia (usually 10 and 15% MVC) were studied in contractions of 2, 4 and 6 min duration. Seven subjects were studied and at least two different strengths of contraction in each subject, for each of the three durations. Duplicate records were made of each strength and duration of exercise. In the examples shown in Fig. 5 the size of the post-exercise hyperaemia was determined as the volume of blood in excess of resting flow rate that flowed through the limb in the first 3 min of the post-exercise period.

All subjects were young male adult volunteers. They were medical students or laboratory
personnel, with no known cardiovascular or other disability. Throughout all studies the subjects were kept as comfortable as possible, provided they remained awake. They lay on a bed and the arm being studied was carefully positioned and supported at the elbow and wrist. The forearm was in the semi-prone position and the elbow nearly fully extended (Fig. 1).

FIG. 1. An arm in position with plethysmograph and the ergometer. The electrical conductivity signal and that from the ergometer are both displayed on the pen-recorder, while the latter is also shown on a galvanometer so that the subject can monitor his strength of contraction. This figure is reproduced from Shephard, R.J. (Ed.) *Frontiers of Fitness*, 1971, courtesy of Charles C. Thomas, Springfield, Illinois.

RESULTS

1. Nature of the blood-flow response

Fig. 2 is a photograph of the records obtained during 1 min before and the first minute after the onset of 10% MVC in two contractions. The arterial inflow (slope during venous occlusion) rises almost immediately to twice the resting value, and during the remainder of the first minute to about three times the resting flow. During this initial minute of contraction there is a gradual increase in limb volume, which is seen in the plethysmograms as a rise in the 'base line' limb volume between the successive occlusions. Some of this increase may be an increase in capacity of blood vessels and some an increase in tissue fluid volume. In either case, during this phase the rate of arterial inflow, measured as the volume increase during venous occlusion, is greater than the rate of venous outflow, the difference between the rates causing the rise in base line volume. In the examples shown in Fig. 2 this amounts to 9 ml in 1 min for the whole segment, or 1.5 ml 100 ml limb$^{-1}$ min$^{-1}$. These changes in base line accompanied the changes in arterial inflow. When this reached a plateau during a sustained contraction there was no further change in base line. At this time, therefore, arterial inflow equalled venous outflow.
FIG. 2. Portions of two records of blood flow at the onset of contraction. On the upper channel of each is the signal from the ergometer and on the lower that from the water level in the plethysmograph. Venous occlusions occurred for 6 s in every 12 and at the signal the subjects began a sustained contraction of 10% of their maximal efforts (10% MVC).
2. **Site of the increase in blood flow**

Humphreys & Lind (1963) have already shown that inactive muscle plays no part in the hyperaemic response. In the twelve pairs of forearm skin thermograms all skin temperatures of both arms were in the range 33–36°C. In six there was no detectable difference in the thermograms of the two forearms, in two the skin of the exercising arm appeared about 1°C warmer and in four about 1°C cooler than the resting arm. Thus it can be inferred that there was no systematic change in skin blood flow of sufficient size to alter the skin temperature.

3. **Constancy of response to repeated contractions of the same strength**

In no studies of forearm blood flow is exactly the same value found from second to second, from minute to minute, or over a period of 10 min or more. A constant response to repeated contractions of the same strength would not therefore be expected and the results shown in Fig. 3 (the forearm blood flow changes induced by five, 4 min contractions at 10% MVC, with 12 min rests between successive contractions) are typical of the variations normally seen in these studies. In this subject the contractions invariably produced a 3-4-fold increase in blood flow in 2 min, though the rate of flow increase varied from contraction to contraction. Once the plateau was reached, it showed little further variation and was the same in all five contractions. In this study there was a small and inconstant further rise in blood flow immediately after relaxation and the flow thereafter rapidly returned to the resting value.

It can reasonably be assumed that all this increase in flow is passing through the active muscle, which is about 30% of the whole forearm volume. To obtain a value for change in flow through the active muscle, the observed rise in forearm blood flow must therefore be multiplied by 100/30. In the case of Fig. 3, the observed increase in flow is about 12 ml 100 ml forearm⁻¹ min⁻¹. The flow through the active muscle must therefore be about 40 ml 100 ml muscle⁻¹ min⁻¹, a 10-fold increase in flow from the resting state in man.

The between-subject variation in the response to exercise is very large. Contractions of the same absolute force, or of the same % MVC may produce very different changes on the forearm blood flow. Twenty subjects have each had two or three studies made of their blood flow response to 4 min contractions at 5% MVC. The average response was +92% increase in flow, with a SD of 56% and a range of response from +17 to +220% of the resting forearm blood flow. No analysis of the reasons for this variation has been attempted. Differences of a similar magnitude have already been reported in resting muscle blood flow and metabolism (Mottram, 1955), and in the metabolism of exercising muscle (Baker & Mottram, 1973).

4. **Varying the strength of contraction**

Contractions at 5% MVC may produce no increase in blood flow, though a definite hyperaemia is usually seen. Thereafter, increasing the strength of contraction leads usually to an increase in the hyperaemia during exercise. Results like those of Fig. 4 are typical. Fig. 4 also shows the appearance of a post-exercise hyperaemia after the more powerful contraction.

In the series of twelve studies in which development of post-exercise hyperaemia was studied as contraction strength was increased, post-exercise hyperaemia was invariable at 20% MVC, present in nine subjects at 15%, in four at 10% and in none of the 5% contractions.

It was noted that a large post-exercise hyperaemia occurred frequently when the blood flow during contraction had become steady (Fig. 4), indicating perhaps that the circulation had
Exercise and forearm blood flow

Fig. 3. The blood flow response to five, 4 min contractions at 10% of the subject's MVC. Each point on each line is derived from the tangent drawn to the volume change caused by each 6 s period of venous occlusion.

fully responded to the increased metabolism of the active tissue. In all cases in which a large post-exercise hyperaemia was seen, flow declined in an exponential manner, similar to the observations of both Grant (1938) and Barcroft & Millen (1939) for the hyperaemia after exercise.

Fig. 4. Each line represents the average of two similar periods of flow recording, the continuous line being the result of two 4 min contractions at 10% MVC and the dashed line that of two contractions at 15% MVC.
5. Effect of ischaemic pain

Pain, when it occurred, was usually felt in the hand distal to the arterial cuff at the wrist, particularly in the muscles of the thenar eminence, caused by the ischaemia in the hand. In the four subjects in whom blood flow was recorded without wrist occlusion the blood flow responses, both during and after contractions, were similar for the two methods of recording blood flow. This indicates that pain in the hand excluded from the circulation played no part in the responses during and after exercise. Ischaemic pain was never experienced in the forearm, only an increasing weakness at fatigue developed during the more powerful contractions.

6. Varying the length of contraction

The results of these experiments were inconclusive, since in four subjects the post-exercise hyperaemia increased with length of contraction and in three subjects there was little change in its size. Fig. 5 portrays the actual blood flows seen during and after exercise in two of the studies. It was thus not possible by this method to elucidate the cause of the raised blood flow after exercise. While conducting these experiments some other observations were made on blood flow responses to exercise. In some subjects, contractions that produced post-exercise hyperaemiae resulted in fatigue and the subjects failed to complete the studies that had been planned. The following effects on blood flow were seen.

(a) Driving a subject ‘to his limit’ on one occasion may alter the blood flow response to subsequent contractions of the same force. In one subject, contracting at 15% MVC, the blood flow in the first two contractions rose from 6 to 18 ml 100 ml forearm⁻¹. After the second contraction of the study, which lasted 6 min and which the subject was scarcely able to complete the blood flow rose in further contractions of the same strength to 30 ml by the end of the first minute of contraction. This raised response to work persisted for the remainder of the study, even though the subject felt fully rested between contractions and the resting blood flow has returned to its original value.

(b) Exhausting a subject in this way may also affect his response to milder contractions. In one subject blood-flow changes were recorded during 10% MVCs. One record was obtained before and the other after a series of 15% MVCs similar in effect to those just described. Although the resting flow, and the recovery after exercise are the same, the blood flow changed during the second 10% contraction from 6 to 18 ml 100 ml forearm⁻¹ min⁻¹. In the one performed before the 15% contractions, it only rose from 6 to 10 ml.

7. Effect of changing the elbow position

In ten subjects forearm blood flow was measured at rest and during 10 or 15% MVC when the elbow was both flexed and extended. There was no significant difference in the flows at rest in the two positions in any subject. Of the seven subjects who exercised at 10% MVC the hyperaemia was significantly greater when the elbow was flexed in four and lower in two. It was higher with elbow flexion in two of the three subjects who worked at 15% MVC.

DISCUSSION

It is clear from these studies that it is possible to produce and measure an increase in blood flow during sustained forearm muscle activity and that, in the milder contractions, a steady-
Fig. 5. Blood-flow responses to pairs of contractions performed at varying strengths for varying durations in each of two subjects. The figures under the flow records are the number of ml/100 ml tissue of excess blood that flowed through the limb in the 3 min period after each contraction ended.
state hyperaemic response is reached during the contraction with no evidence from post-
contraction hyperaemia that full equilibration between the tissue and the raised blood flow
has not occurred. Further evidence than that ascertainable from purely circulatory studies
(in which the blood flow may be seen to rise during exercise to a plateau and to return promptly
to the resting value on cessation of exercise) would be required before it would be possible to
assert confidently that full metabolic equilibrium had occurred between the metabolizing
tissues and the circulation. This would be supplied by muscle biopsy studies (Hultman, 1971)
and by samples of muscle effluent blood.

One major difference appears when these results are compared with those of Humphreys
& Lind (1963). This is the contraction force at which circulatory insufficiency appears. Marked
post-exercise hyperaemia has been found, sometimes increasing with the length of contraction
and associated with fatigue, at contraction forces as low as 10% MVC. Humphreys & Lind
(1963) seldom saw this state at forces less than 30% MVC. The ergometers are of the same
design, with the same dimensions and specification of the spring-steel bar. The MVCs of the
two groups of subjects were similar, and so were the actual deflections of the bars. Humphreys
& Lind (1963) measured blood flow with a Whitney strain gauge but in the present studies a
water-filled plethysmograph was employed. The main difference in the conduct of the experi-
ments lies perhaps in the fact that the subjects studied by Humphreys & Lind (1963) sat
upright with the upper arm vertical and the forearm horizontal, the elbow being flexed at 90°.
In the present studies the subjects reclined on a bed, the upper arm was beside the body and the
elbow flexed 0–20°. It is possible that when forearm flexor muscles are contracted, extension
of the elbow might lead to a nipping of the brachial artery by deep fascia at the apex of the
antecubital fossa or of the ulnar artery where it passed through the 'common flexor origin'.

An attempt to investigate this possibility has been performed using the injection of a radiopaque
material into the brachial artery and studying its distribution in the forearm arteries (Lynch,
Mottram & Owen, 1973). Although these radiographs confirmed the difference in rate of
blood flow during exercise caused by the differences in posture, there was no evidence that
this was due to narrowing or kinking of the arteries around the elbow. An alternative suggestion
is that extension of the elbow leads to a greater rise of inter- and intra-muscular pressure during
contraction than does the flexed position. Barcroft & Millen (1939) have already commented on
intramuscular pressure as the factor causing restriction of blood flow in contracting calf
muscles. Even though the deep fascia of the forearm is less dense than that of the calf, changes
in intramuscular pressure under the conditions of the present studies must be considered as a
cause of prevention of an adequate blood flow during contraction (Sylvest & Hvid, 1959).
The studies reported here on blood flow measured during contractions with different elbow
positions have failed to supply definite confirmation that these affect the blood flow response
to exercise.

Whatever is the cause of the post-exercise hyperaemia, especially if there is any possibility
that it might be related to the gradual accumulation of products of metabolism, it is clear that
metabolic studies cannot be performed during contractions either in which the blood flow is
continually rising or which are followed by a further rise in blood flow. Zierler (1961) has
analysed mathematically the errors that may occur when metabolism is assessed by use of the
Fick equation (uptake = blood flow × A–V difference) under conditions of changing blood
flow, changing metabolic activity or accumulation of metabolites in the tissue. The errors
are caused by the pools in tissue fluid or within cells which lie between the site of use or
Exercise and forearm blood flow

production of a substance and the blood vessels. Thus the tissue fluid of skeletal muscle contains a pool of glucose which is 10–30 times the amount of glucose that passes through the tissue fluid into the muscle cells each minute, even during the period of high glucose uptake that follows a meal (Mottram & Brown, 1963). Similar considerations apply for CO₂ and O₂, where the tissue-fluid HCO₃⁻ and the myoglobin store may be many times the gaseous exchanges per minute of the resting muscle. In some of the present groups of experiments a stable plateau of blood flow during exercise has been followed by a large post-exercise hyperaemia. An example is shown in the 15% MVC result in Fig. 4. It cannot be presumed that in these studies a complete metabolic equilibrium has been achieved between the circulation and the metabolizing tissue. They would be as unsuitable for metabolic determinations as those in which the blood flow rose continually during exercise. This applies particularly to the work reported by Kontos, Richardson & Patterson (1966) in which this was not appreciated. It is only possible to study mild contractions using the sustained hand-grip contraction method described in this paper. Even in these, however, a 5% MVC produces a 4-fold increase in blood flow through the active muscle. In ‘whole body’ terms, these changes would be equivalent to a cardiac output increase of 4 litres min⁻¹. The respiratory oxygen uptake changes as described in the accompanying paper would be increased by 400 ml min⁻¹ (Baker & Mottram, 1973).

While the work reported here has defined the conditions in which metabolic studies could be performed in future work, the nature of the vasodilator stimulus during exercise has not been studied. The manner in which fatigue has sometimes been found to have altered the response of blood vessels to subsequent exercise, while not affecting resting blood flow, has made the problem more confused. Other workers are currently studying the identity of this stimulus. For example, Forrester & Lind (1969) found an increase in the output of ATP from muscles into the circulation during 10% MVCs in man. Hilton & Hudlická (1971) have found that exercise hyperaemia in cats was related closely to phosphate output, less closely to potassium output and not at all to the osmolarity of the effluent blood. Other workers have studied other components of the venous blood (Rodbard, 1971). Skinner & Costin (1969) have perfused through its arteries a resting muscle vascular bed with blood containing a raised concentration of K⁺ and a decreased O₂ content. They were able to produce changes in resistance of the bed, of the same sizes as those produced by exercise, with blood compositions comparable to those of venous blood during the exercise. Probably, as has been suggested by Kontos, Richardson, Raper & Patterson (1970), only the measurement of concentrations of tissue fluid constituents in exercising muscle [e.g. the measurement of O₂ by the method of Gore & Whalen (1968)] will provide definitive information on the chemical aspects of the vasodilatation.

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REFERENCES


