# SPECIAL COMMUNICATIONS

## Effect of a brief contraction of forearm muscles on forearm blood flow[1]

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CORCONDILAS, ADRIAN, GABRIEL T. KOROXENIDIS, AND JOHN T. SHEPHERD. *Effect of a brief contraction of forearm muscles on forearm blood flow.* J. Appl. Physiol. 19(1): 142–146. 1964.—With the use of a strain-gauge plethysmograph, the effect of a brief (0.3 sec) contraction of the forearm muscles on forearm blood flow has been studied in eight healthy adults. An increase in flow due to dilatation of the muscle vessels could be detected within a second after the completion of the contraction. This increase was proportional to the strength of the contraction. The blood flow was maximal immediately and decreased rapidly. A second contraction of the same magnitude made during the period of increased flow caused an additional increase in flow. The maximal increase in flow caused by a strong brief contraction was only about 25% of that recorded after strong repeated rhythmic contractions or a sustained contraction. Cervical sympathectomy did not change these findings, indicating the local nature of the response. Since breathing oxygen failed to reduce the dilatation for a given strength of contraction, it is unlikely that oxygen lack was the stimulus for vasodilatation. The oxygen saturation of blood that drained the muscles could not be determined accurately immediately after contraction because at this time, muscle venous blood was contaminated by venous blood from the skin.

exercise-induced vasodilatation  local mechanism of vasodilatation  venous $O_2$ saturation during muscle contraction  brachial arterial pressure during vasodilatation  speed of muscle vasodilatation

PREVIOUS STUDIES OF BLOOD FLOW in human muscles have been carried out after repeated or sustained muscular contractions. In such circumstances, the stimulus to vasodilatation has been present for a considerable time before observations of flow are made. Thus, it was of interest to study the effects of a brief (0.3 sec) contraction of the forearm muscles on forearm blood flow in the hope that this might provide additional data on the mechanism of exercise-induced vasodilatation.

### METHODS

Studies were carried out on eight healthy adults, aged 25–40 years. Two of them had undergone bilateral cervical sympathectomy for intermittent episodes of coldness and pallor in the fingers. The completeness of the sympathectomy was verified by the absence of sweating in the upper extremities during body heating. Blood flow in the forearm was measured by venous occlusion plethysmography. Strain-gauge plethysmographs (16) were used to avoid the oscillations of the water column which occur with conventional plethysmographs after contraction of the forearm muscles and which interfere with the recording of immediate changes in blood flow. The subjects sat with their forearms supported at the wrists and elbows on an inclined table; plethysmographs were placed around the upper third of both forearms. Wrist cuffs, inflated to more than the systolic pressure, arrested the circulation to the hands during the measurements of forearm blood flow. Cuffs above the elbow (collecting cuffs) were inflated to a pressure (collecting pressure) that gave the maximal rate of volume increase. This pressure was usually 65–70 mm Hg. Wide-bore taps and tubing connected the collecting cuffs to an air reservoir so that the desired collecting pressure could be reached in less than a second. Flow in both forearms was recorded simultaneously. After two or three observations of the flow in the resting forearm had been made, the subject rapidly squeezed a stiff metallic ring that was held in the right hand. The grasping movement involved the large majority of the forearm muscles, and, because of the stiffness of the ring, the muscle contraction was more isometric than isotonic. Coinciding with the onset of contraction, the collecting cuffs were inflated to record the immediate changes in flow. On other occasions the contraction was made during the recording of an inflow curve.

The strength and duration of the contractions were measured by a strain gauge connected to the metallic ring. This strain gauge was calibrated against known weights.

In two of the subjects, nylon catheters, 0.9 mm in external diameter, were inserted into the veins at the antecubital fossa through thin-walled needles which were then withdrawn. One catheter was inserted into a superficial vein, the other, distal to the valve, into the deep branch of the median cubital vein (7). The oxygen saturation of the blood withdrawn through these catheters was continuously monitored by a cuvette oximeter (17). The wrist cuff was inflated during these determinations. Toward the end of the study, indocyanine green (Cardio-green) dye was injected at a constant rate into the cephalic vein about 5 cm proximal to the wrist cuff; during this time, blood was withdrawn at a constant rate from the deep vein, and the changes in concentration of dye were recorded continuously by a densitometer-galvanometer system.

### RESULTS

Fifteen to thirty single forearm muscle contractions of different strengths were performed by each subject. A typical example of the flow tracings is shown in Fig. 1*A*. Each contraction lasted about 0.3 sec. Immediately after contraction of the muscles of the right forearm, the volume increased more rapidly in that forearm than before contraction when the collecting pressure was applied. The increased rate of

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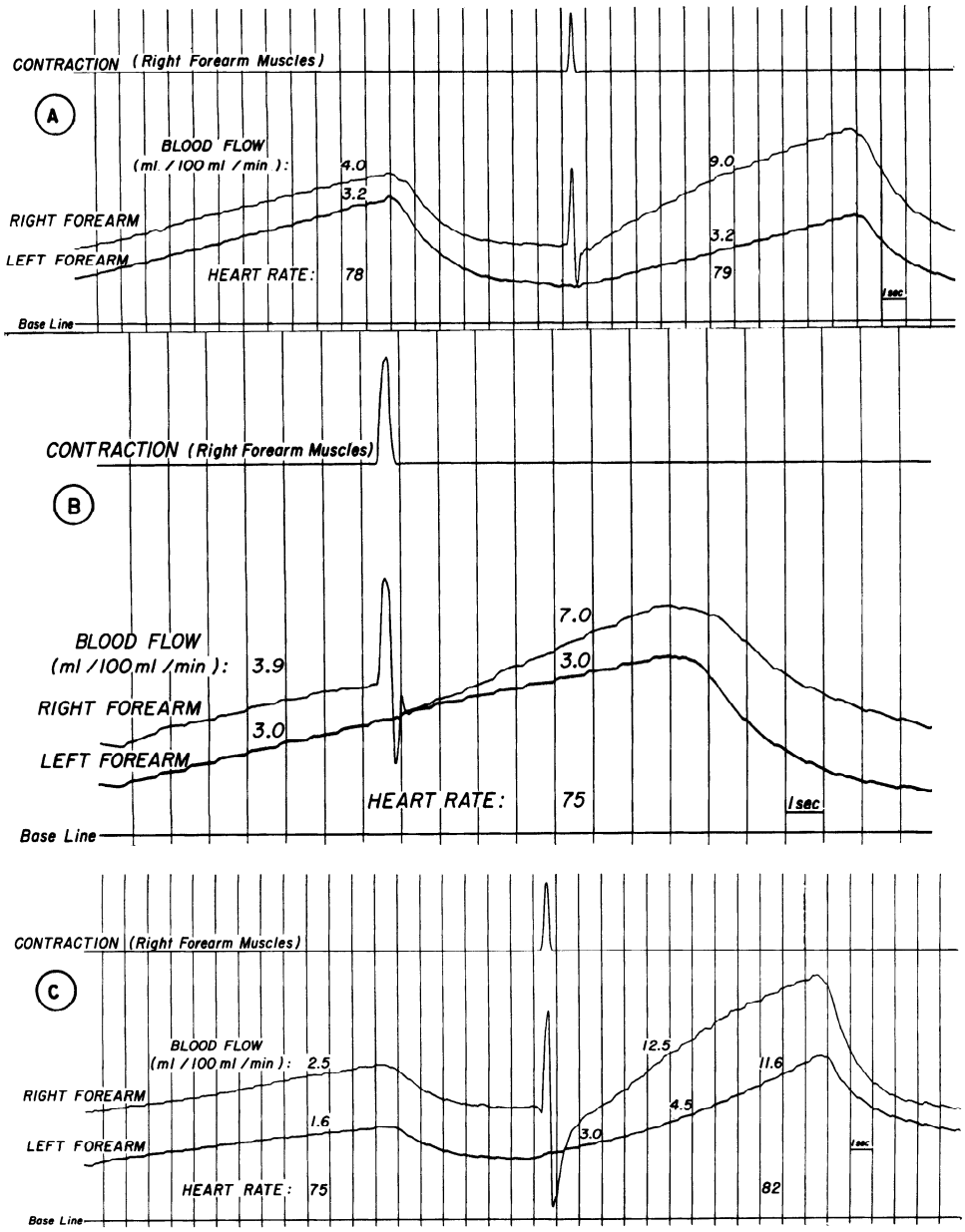


FIG. 1. *A*, *B*, and *C*: plethysmographic records of blood flow through both forearms before and after brief contraction of muscles of the right forearm. At time of contraction there is an artifact on the plethysmographic tracings due to muscle movement. Figures above each plethysmogram are the forearm blood flow (in ml/100 ml min).

filling could be detected within a second from the end of the contraction. No change in flow was noted in the left forearm (control) immediately after the contraction, and little or no change was noted in heart rate.

On some occasions, flow tracings were obtained when the contractions were made in the middle of an inflow curve (Fig. 1*B*). The more rapid increase in volume caused by the contraction of the muscles of the right forearm could be detected within 0.5–1.0 sec after completion of the contraction. There was little or no change in flow in the control forearm. The tracings from the sympathectomized subjects were similar to the others and to the typical tracings shown in Fig. 1, *A* and *B*.

Although flow in the control forearm usually did not change after the muscle contraction, increase in flow on this side was seen during some of the observations in two of the normal subjects. This increase in flow was never as great as that seen in the contracting forearm, nor did it follow the same pattern. In the tracing shown in Fig. 1*C* the flow was maximal in the right forearm immediately after the contraction of that forearm, whereas on the control side the flow continued to increase gradually. The flow in the control forearm was 3.0 ml/100 ml forearm per min immediately after contraction, increased to 4.5 ml within 5 sec, and reached 11.6 ml in 8–10 sec after the end of contraction.

The brachial arterial pressure was measured simultaneously in both arms in one subject with a strain-gauge manometer. Immediately after a strong contraction of the forearm muscles, there was a transient drop in diastolic pressure in the brachial artery on that side limited to the subsequent pulse beat. With a more modest contraction, sufficient to elicit an increase in blood flow, little if any change in pressure occurred (Fig. 2).

With a mild or moderate contraction, the immediate increase in flow could be detected in all subjects, whether normal or sympathectomized, within 0.5–1.0 sec after the end of the contraction. After a strong contraction, it was usually 1.5

sec after the contraction before the increase could be detected; this was due to the longer duration of the artifact caused by the muscle movement.

A linear relationship existed between the immediate increase in flow and the strength of the contraction both in the normal and in the sympathectomized subjects (Fig. 3). Thus, the flow continued to increase up to the maximal single contraction of which any subject was capable.

The flow was always maximal immediately after the contraction, regardless of the strength of the contraction; it decreased rapidly and reached the precontraction value in 15–20 sec. The example shown in Fig. 4 is typical. In this subject, the forearm muscle contractions increased the forearm flow from its control value of 2.0 ml/100 ml forearm per min to values from 2.7 to 9.0 ml, according to the strength of

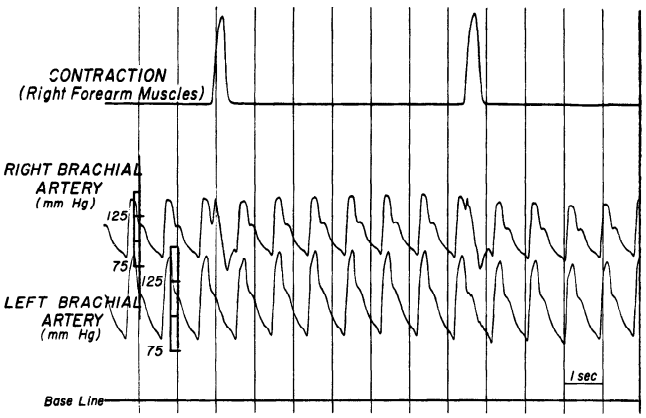


FIG. 2. Simultaneous recording of pressure in brachial arteries. A brief strong contraction of the muscles of the right forearm was made at the times indicated.

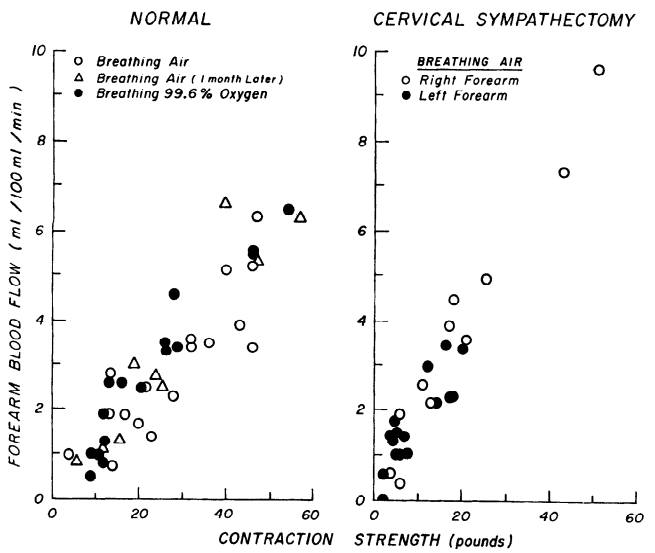


FIG. 3. Increase in flow in forearm immediately after single brief contraction of forearm muscles in normal subject and in a subject who had had cervical sympathectomy 3 years previously. The increase in forearm flow was obtained by subtracting the resting flow before each contraction from the flow measured just after contraction. $O_2$ capacity of radial artery blood was 19.4 vol.%; $O_2$ content was 19.0 vol.% breathing air and 20.8 vol.% while breathing $O_2$.

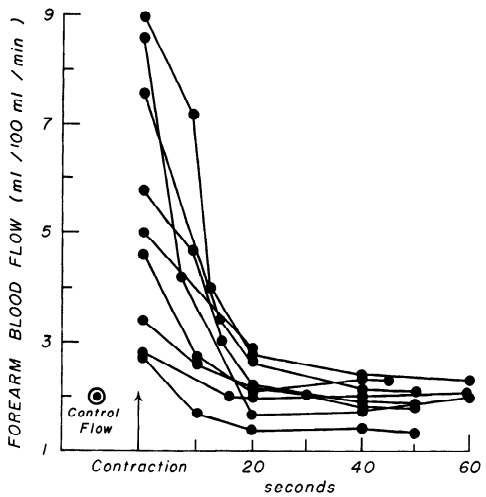


FIG. 4. Changes in blood flow in forearm following single brief contractions of different strengths.

the contraction. The flow then progressively decreased and returned nearly to its control value in about 20 sec. A second contraction of the same strength made during the period of increased flow which followed the first contraction caused a greater increase in flow than that occasioned by the first contraction alone. For example, one subject after an initial contraction of moderate strength had an immediate post-contraction flow of 3.9 ml more than the control value. A second contraction of similar strength made 4 sec after the first caused the flow to increase by 8.1 ml; with a second contraction made 10 sec after the first the increase was 4.7 ml above the control value. If the second contraction was made at the time the flow had returned to the control level, the increase was similar to that seen after the first contraction. When more contractions of equal strength were made in quick succession, a point was reached where further contractions caused no further increase in the immediate post-exercise flow. The number of contractions necessary to achieve this depended on the strength of the contractions and the interval between them.

The increases in flow that followed a strong, brief contraction were modest when compared to those seen after a strong, sustained contraction or repeated rhythmic contractions. For example, the maximal increase in flow in the experiment shown in Fig. 3 (left panel) was about 6–7 ml/100 ml forearm per min. After ten such contractions in 10 sec, the increase was 25 ml; after 1 min of ischemic exercise, flow reached 37 ml.

In four subjects, the magnitude of the immediate post-exercise flow was compared when the subjects breathed air and when they breathed 99.6% oxygen. There was no significant difference in blood flow under these two circumstances (Fig. 3, left panel).

An attempt was made to correlate the flow changes after contraction of the forearm muscles and the oxygen saturation of venous blood obtained from the deep branch of the median cubital vein.

In one subject, ten series of determinations of oxygen saturation of blood from the deep vein were made before and after graded, single, brief (0.3-sec) contraction of the forearm muscles. During withdrawal of the blood samples, the wrist cuff was inflated to a pressure sufficient to occlude the circulation to the hand. When the muscles were at rest, the oxygen saturation of blood from the superficial veins was 88% and

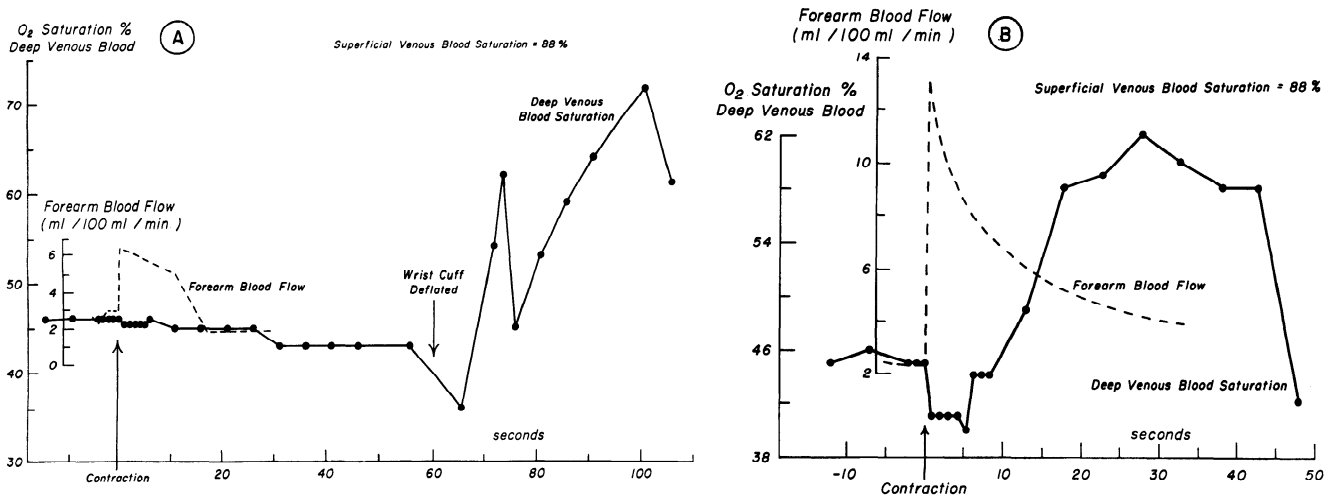


FIG. 5. Changes in $O_2$ saturation of deep venous blood from the forearm. *A:* with a weak contraction. *B:* with a strong brief single contraction of forearm muscle. Dotted lines are averages of three sets of determinations of blood flow in the same subject with the same contraction made at a different time.

from the deep veins it was 44–66%. With a weak contraction, there was little or no change in saturation (Fig. 5*A*). On release of the wrist cuff, the oxygen saturation in the deep vein increased rapidly, coinciding with the reactive hyperemia in the hand (Fig. 5*A*). This emphasizes the importance of inflating the wrist cuff during withdrawal of the blood samples in order to prevent contamination of the blood in the deep forearm vein by blood from the hand veins (7). With stronger contractions (Fig. 5*B*), an increase in oxygen saturation started about 6 sec after the contraction and lasted about 40 sec; within a few seconds after the contraction, however, the results varied—the saturation increased, decreased, or stayed the same. Forearm blood flow was not recorded simultaneously since, during the application of a venous occlusion cuff above the elbow, blood flow from the superficial veins of the forearm enters the deep vein (7, 15). The values of forearm flow, indicated by an interrupted line on Fig. 5, are flow values obtained in the same subject for contractions of comparable strength within 24 hr of the venous saturation determinations.

On a second subject, the six series of determinations made before and after graded contractions lasting 0.3 sec showed a pattern similar to that illustrated in Fig. 5*B*. With the muscle at rest, superficial venous blood was 92% saturated with oxygen and that of the deep vein was 62–72%. The oxygen saturation in the deep vein decreased immediately after the contraction, but the extent of the decrease was not proportional to the strength of the contraction. This decrease was then followed by prolonged increase in saturation above the control value; this increase was less with the weaker contractions and became larger with the stronger contractions.

During infusion of Cardio-green dye at a constant rate into a cutaneous vein (the cephalic vein 5 cm proximal to the wrist cuff), a mild contraction resulted in a small amount of the dye appearing in the deep vein, and a strong contraction resulted in a large amount appearing (Fig. 6). The wrist cuff was inflated throughout. Allowance for the time lag in the sampling system suggests that the dye appeared immediately after the contraction and was present for about the next 10 sec. Repeated contractions at 5-sec intervals caused dye to appear after each contraction. With infusion of dye over the same period in the absence of a contraction, no dye appeared in the blood from the deep vein.

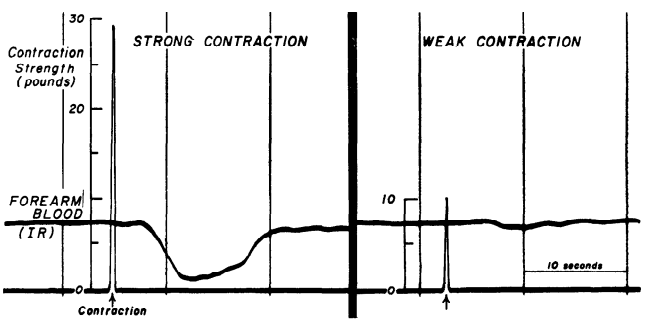


FIG. 6. Continuous injection of indocyanine green dye into a superficial vein of the forearm with continuous sampling from the deep vein of the same forearm during a weak and strong brief, single contraction of forearm muscles. Note changes in optical density of deep venous blood after contraction due to the passage of dye from superficial to deep vein. IR = infrared absorption of the blood drawn from the forearm through a densitometer. No calibration was done; a downward deflection represents an increasing concentration of indocyanine green dye.

## DISCUSSION

Anrep and associates, in 1934 (2), measured arterial inflow after brief tetanic contractions of the gastrocnemius muscle of the dog. They described a transient phase of increased flow lasting 0.1–0.15 sec that ended as soon as the blood vessels, which had been emptied by the contraction, were refilled. This was followed by a phase of true vasodilatation.

In the present study, the drop in diastolic pressure in the brachial artery, which was associated with a strong contraction and limited to the pulse beat following the contraction, may represent the initial transient phase described by Anrep and his colleagues (2). The immediate increase in forearm volume seen on application of the collecting pressure after contraction of the forearm muscles was linear for about 6 sec. During this time the total volume of the right forearm was or became greater than it was while at rest, and the brachial arterial pressure did not change. Thus, the increase in blood flow seen after forearm muscle contraction in the present study represents true vasodilatation.

That vasodilatation could be detected within half a second of the contraction by the relatively insensitive technic used suggests that the dilatation occurs almost instantaneously

with the contraction. Since the magnitude and time of onset of the dilatation were similar in the two persons with sympathectomized forearms and since no change was usually seen in the control forearm, the vasodilatation is mediated through a local mechanism (10). This vasodilatation could result from the direct action of some chemical factor on the wall of the resistance vessels in the muscle or from an axon reflex initiated by a chemical stimulus (11). Since the vasodilatation was maximal immediately, presumably there was no buildup of the stimulus after the contraction. Whatever the mechanism, it not only has a fast response but also is finely adjusted to the strength of the contraction. Since breathing oxygen, which adds 1.5 1.8 vol.% $O_2$ in physical solution to the blood, failed to reduce the dilatation for a given strength of contraction, it is unlikely that oxygen lack plays a major part in the response.

Accurate measurement of blood flow in the muscle and the arteriovenous difference for any substance is of obvious value in the study of muscle metabolism. It is, therefore, important to know if blood from the deep veins of the forearm contains only the blood that drains the muscles. Contamination by venous drainage from the hand (7) can be avoided by occluding the circulation of the hand during the measurements. Whether under these conditions the resting sample represents solely muscle venous blood is unknown. Values reported for oxygen content or oxygen saturation of blood sampled from the deep forearm veins have varied widely in the same subject and from subject to subject. With the forearm at rest, Coles and associates (7) found values ranging from 4.3 to 19.3 vol% $O_2$. Blair et al. (6) found oxygen saturations ranging from 29 to 55%, and Love (12) up to 80%.

Changes in the oxygen saturation of the deep venous blood can occur independently of cutaneous venous blood and vice versa (14, 15). Inflation of a cuff above the elbow to produce venous congestion causes drainage from the superficial forearm veins to the deep vein, and indicates the ever-present possibility of admixtures (7, 14). In the present study, dye passed from a superficial to the deep vein as a consequence of the muscle contraction. This result makes it unlikely that any accurate estimation can be made, for example, of the oxygen saturation of blood leaving the muscles during the 5- to 6-sec period that follows their contraction unless the blood flow through the skin has been suppressed. Admixture of blood from the superficial veins would explain the fact that the deep forearm vein saturation remained the same or increased in the 5 or 6 sec after contraction. When it decreased, presumably it would have diminished further had this admixture not occurred. Since, with moderate or strong contractions, the saturation usually decreased, the muscle appeared to receive its additional oxygen supplies both by increased flow and by increased extraction.

The increase in saturation, which usually commenced about 5–10 sec after the contraction, was noted by Love (12) after both rhythmic and sustained contractions of the forearm muscles. During the continuous withdrawal of the blood samples through the oximeter, a total of 5–6 ml/min was usually withdrawn; thus, the possibility exists that as the withdrawal continues, blood from sites other than muscle might be drawn into the deep vein. However, in the present study, withdrawals from the control forearm at the same rate usually showed no change in saturation. Since no dye passed from the superficial to the deep vein at this time, the oxygen saturation of the veins draining the muscles probably was raised above control values. Thus, the flow at this time probably exceeds that which is necessary to supply any increased oxygen uptake in this period (12).

Reeves and co-workers, in 1961 (13), measured cardiac output and femoral arteriovenous oxygen difference in men at rest and during exercise. Since the femoral arteriovenous oxygen difference increased sharply with mild exercise and showed smaller further increases with heavy exercise, they concluded that, with mild exercise, increased oxygen transport to the muscles depended mainly on increased oxygen extraction, and for heavy exercise it depended mainly on increased blood flow. This interpretation, however, requires the assumption that the change in femoral venous blood with exercise represents change in venous blood of the muscle. Since the opportunity for admixture in the femoral vein is probably even greater than in the deep vein of the forearm, this assumption may not be valid. The present investigation suggests that the increase in blood flow is proportional to the strength of the contraction and that even the mildest contraction which lasts only about 0.3 sec results in an increase.

Vasodilatation in skeletal muscles has been produced in animals by stimulation of the brain stem, and in man by emotional stress and by fainting (1, 3–5, 9). The dilatation is due to activation of sympathetic vasodilator fibers. Abrahams and his colleagues, in 1960 (1), concluded from studies on the cat that this reflex muscle vasodilatation is one component of a coordinated defense reaction; the reflex may be activated whenever a sudden increase in blood flow of the muscle is required for muscular effort (8). If this is true, it could reinforce and even precede the dilatation mediated locally in the active muscles.

The speed of onset of the dilatation from the local mechanism makes it unnecessary to postulate a reflex adjustment of blood flow to the muscles to meet immediate demands at the onset of exercise. Although vasodilator fibers may be excited during exercise, perhaps as a consequence of emotional stress, they are not an integral part of the general vasomotor response to exercise (5).

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