Reactive hyperemia in individual capillaries of skeletal muscle

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BURTON, K. S., AND P. C. JOHNSON. Reactive hyperemia in individual capillaries of skeletal muscle. Am. J. Physiol. 223(3): 517-524, 1972.—Red cell velocity profiles in individual capillaries were studied in the sartorius muscle of the cat during reactive hyperemia using a dual-slit photometric system. The velocity profiles varied considerably from each other and from the simultaneously measured volume flow. The profiles were divided into four groups on the basis of the duration of reactive hyperemia following a 60-sec occlusion and the time required to attain peak velocity. One group showed almost no response. A second group had a long period of increased flow (60 ± 8 sec) and reached a peak value in 35 ± 16 sec. The duration of reactive hyperemia for the third group (22 ± 3 sec) was comparable to that of the gross flow (28 ± 6 sec) and reached its peak value after 10 ± 4 sec. The peak response in the fourth group occurred after 5 ± 3 sec as compared with 6 ± 1 sec in the gross flow and was followed by an interval of zero flow (7 ± 3 sec) before returning to normal. The distributions of the 57 capillaries studied were 28, 14, 21, and 37%, respectively. The question of whether reactive hyperemia was due to the opening of new capillaries or to increased flow in previously open capillaries was investigated. Comparing the ratio of peak velocity to control for the average of all flowing capillaries (1.5:1) with that of the gross flow (1.8:1), we conclude that augmentation of flow in previously open capillaries is the primary source of reactive hyperemia.

Studies of various vascular beds have shown that after an interval of arterial occlusion the blood flow usually increases considerably over the normal flow level for a short time. This phenomenon (commonly known as reactive hyperemia) has been shown to occur in many vascular beds under a variety of conditions. These have included normally perfused tissues such as human leg and forearm and dog myocardium and isolated perfused preparations such as heart, intestine, kidney, and skeletal muscle. In such studies reactive hyperemia has been investigated by measuring the total blood flow through the tissue or perfusion pressure during constant flow. Behavior of individual microvascular vessels in reactive hyperemia has not been examined with quantitative techniques, although qualitative observations have been made on the frog web and tongue (19).

It is commonly believed that increased blood flow is associated with opening of closed or static capillaries as suggested by Krogh in 1919 (13). In studies on frog and hamster muscle during rest and exercise, Krogh found that the increased flow during exercise was apparently associated with a large increase in the number of capillaries carrying the circulating blood. On the other hand, it also seems plausible to suggest that the increased flow may represent, at least in part, an augmentation of flow in capillaries already open and flowing.

In the experiments reported here, we have extended the study of reactive hyperemia with a quantitative investigation of flow in individual capillaries of cat sartorius muscle. The patterns of flow in individual capillaries have been examined during reactive hyperemia and compared with the pattern of total flow through the muscle, and the extent of augmentation of capillary flow has been investigated.

METHODS

Cats in a mass range of 1.5-2.6 kg were tranquilized with 1 mg/kg propiopromazine hydrochloride and anesthetized with 75 mg/kg of α-chloralose. The sartorius muscle was surgically removed from the left hindlimb, with care being taken to preserve as many of the communicating blood vessels from the femoral artery and vein to the muscle as possible. The muscle was kept moist with Ringer solution throughout the dissection procedure. After isolation, the muscle was continuously perfused with blood from the femoral artery in the right hindlimb through 1.6 mm id polyethylene tubing as shown in Fig. 1. Arterial blood pressure was monitored in this circuit by a Statham pressure transducer. Venous outflow passed through a drop counter to monitor the gross flow rate, and the blood was then returned to the animal through the external jugular vein. Venous pressure was measured by a Statham pressure transducer connected to the venous circuit between the muscle and the drop counter. The venous pressure was maintained at about 14 mm Hg throughout the experiment. To insure good transillumination of the muscle, it was necessary to remove as much fascia from the surface of the muscle as possible. This was done with a cautery, with care taken to avoid heating or drying of the muscle during this procedure. The muscle was then mounted on a frame and placed on a heated microscope stage maintained at 37°C. The muscle was bathed with Ringer solution and covered with Saran wrap to prevent drying during the experiment. Muscle weight averaged 4.1 g in these experiments.

The velocities of red cells in the capillaries were measured using the dual-slit photometric method described by Wayland and Johnson (21). The muscle mounted on the microscope stage was illuminated with a mercury-arc lamp, and the magnified image of the capillaries was projected onto a
FIG. 1. Experimental arrangement for on-line velocity measurements of red cells in capillaries. Image of capillaries is projected onto a screen containing dual slits. Photomultipliers A and B are optically coupled to slits by light pipes. Signals from photomultipliers produced by red cells passing through capillary are amplified and displayed by a dual-beam oscilloscope. Two signals are time correlated, and resulting time interval is divided into distance between slits to obtain red cell velocities.

The image was aligned so that the capillary being studied crossed both slits as shown in Fig. 1. For each of the capillaries studied, the inside diameter of the vessel was about 5 μ. Thus the red cells moved through the vessels in single file. The intensity of the light passing through each slit was monitored with RCA 6199 photomultiplier tubes. When a red cell crossed the screen the light level through each slit was momentarily decreased resulting in corresponding pulses in the photomultiplier outputs. The time delay between the pulses in the two photomultiplier signals is inversely proportional to the velocity of the red cell. A Hewlett-Packard 3721A correlator was used to determine the time delay. The method used to obtain the delay is as follows. A correlation function $R_{xy}(\tau)$ for the two photomultiplier signals $x(t)$ and $y(t)$ can be defined as

$$R_{xy}(\tau) = \lim_{T \to \infty} \frac{1}{T} \int_{-T}^{T} s(t-\tau)y(t)\, dt \quad (1)$$

$\tau$ represents the time delay between the signals $x(t)$ and $y(t)$ (12). This function has the property that if there is a correlation between $x(t)$ and $y(t)$, with $y(t)$ delayed by a time $\tau'$ from $x(t)$, then $R_{xy}(\tau)$ will have a peak at $\tau - \tau'$. In practice, $R_{xy}(\tau)$ is calculated for discrete values of $\tau = k\Delta t$ for $k = 1-100$, and the integral is replaced by a finite sum

$$R_{xy}(\tau) = \frac{1}{N} \sum_{k=1}^{N} x(k\Delta t - \tau)y(k\Delta t) \quad (2)$$

with a new value, $R_{xy}(\tau)_{\text{new}}$, being calculated for each new sample of $x(t)$ and $y(t)$. In equation 2, $R_{xy}(\tau)$ is obtained from equation 2, and $R_{xy}(\tau)_{\text{old}}$ is the value of the correlation function previously stored in the memory. In the results quoted in this paper, $N$ and $\Delta t$ were chosen to give a time constant of about 1 sec. To determine the red cell velocity, the value of $\tau$ at which the peak occurs was read after each new sampling of $x(t)$ and $y(t)$, which was every 12.5 msec. The delay $\tau$ was then divided by the distance between the slits using an analog divider circuit. See also descriptions of this technique by Wayland and Johnson (21) and In-
taglietta (7). The velocities thus obtained were recorded along with arterial and venous pressure and drop-counter signals on strip-chart paper and magnetic tape.

The arterial inflow to the muscle was occluded for 1-min intervals. The red cell velocities in the capillary being observed were monitored continuously for at least 1 min prior to the occlusion and 2 min following release of the occlusion. At this time the gross flow through the muscle and red cell velocities in the capillary had returned to control levels. During the study of a particular capillary, the blood flow was frequently occluded 2 or 3 times to test the reproducibility of the red cell velocity pattern and the gross flow pattern. In this series of experiments, data are reported on 57 capillaries from 7 cats.

RESULTS

The average gross flow pattern obtained from sixteen 1-min occlusions is shown in Fig. 2. The average flow through the muscle prior to occlusion was 3.2 ± 1.1 ml/(min·100 g). The gross flow parameters during reactive hyperemia varied somewhat from occlusion to occlusion even in the same muscle. The average time from the end of an occlusion to peak flow was 4.8 ± 1.6 sec, the ratio of peak flow to control flow was 1.8 ± 0.4, and the duration of reactive hyperemia was 28 ± 6 sec. All quoted variances are standard deviations. The average flow debt repayment in these studies was 12 ± 6%.

The velocity patterns observed in capillaries of the sartorius muscle in the period prior to occlusion were of three types: steady flow, periodic flow, and irregular variations in flow. In 65% of the capillaries studied the velocity was reasonably steady, seldom varying more than 95% in any 15-sec interval. Gradual changes in velocity did occur in some of these capillaries, with variations of up to 50% occasionally seen over a 1-min interval. The capillaries with periodic variations in velocity constituted 10% of the capillaries studied. The period of the rhythmic changes ranged from 10 to 25 sec, and the amplitude change varied from a 30% increase in the maximum velocity over the minimum velocity to the extreme case of the minimum velocity being zero. The remaining 25% of the capillaries had irregularly varying velocities with variations of 100% in a 5-sec interval being not uncommon.

The average control velocity of red cells also varied among the individual capillaries. Figure 3 shows histograms of pre- and postocclusion velocities. The mean preocclusion velocity for the 57 capillaries studied was 0.38 mm/sec, with the majority of the capillaries having control velocities between 0.1 and 0.5 mm/sec. The velocity distribution
During the control period is different in some respects from the distribution at the peak of reactive hyperemia. The spread of velocities at peak flow is much broader than in the control phase; individual velocities of 0–1.2 mm/sec are seen with almost equal frequency, with the mean red cell velocity being 0.60 mm/sec. Also, the number of capillaries with flow velocities less than 0.1 mm/sec is increased at the peak of reactive hyperemia as compared with the control distribution.

The red cell velocity patterns in individual capillaries are in general quite different from the changes in total flow through the isolated muscle following occlusion. Among the individual capillaries there is a great deal of variability in the amplitude and duration of the flow overshoot observed during reactive hyperemia, considerably more than one sees in the gross flow patterns. However, the velocity patterns do not vary from one another in a random fashion, but appear to fall into four identifiable groups. We have classified the capillaries examined in this study on the basis of the velocity patterns observed during reactive hyperemia. Figure 4 shows examples of capillary velocity patterns from each of the four groups and illustrates some of the variability seen within each group. Also shown is the average velocity profile for all responses classified in a particular group. The indicated errors on the average velocity profile for all responses classified in a particular group. The indicated errors on the average velocity profile curves were estimated by normalizing the control velocity of each capillary to the appropriate average group control velocity, and calculating the standard deviation for each point. The various parameters describing reactive hyperemia in each of the four groups and in the gross flow are summarized in Table 1.

Most capillaries showed increased flow after release of the arterial occlusion. However this was not invariably the case; in 98% of the capillaries studied, little or no overshoot was seen. Capillaries in which the velocity increased less than 25% over the control velocity or those in which the velocity did not reach or exceed the control flow within 15 sec after release of occlusion were classified as group I.

The second type of flow behavior (group II) was characterized by reactive hyperemia of long duration as compared with the gross flow. While the average duration of reactive hyperemia in the gross flow pattern was 28 sec, the duration of increased flow in this group averaged 60 sec, with peak flow occurring, on the average, 35 sec after the end of the occlusion. The average velocity pattern for this group shown in Fig. 4 indicates a much smaller peak-to-control velocity ratio than is given in Table 1. This is due to the variation in times when the peak velocity was reached in the individual capillaries. The number of examples of group II behavior was rather small with only 14% of the 57 capillaries being of this type.

Capillary velocity patterns which constitute group III were most nearly similar to the gross flow profile. There was little difference in the duration of reactive hyperemia in these vessels and in the gross flow. However, the time required to reach peak flow in these capillaries was about twice as long as in the gross flow, and the ratio of peak-to-control velocity for the capillaries was about double the corresponding ratio for the gross flow. Twenty-one percent of the capillaries studied displayed this type of flow pattern. Velocity profiles characterized as group IV were most numerous with 37% of the 57 capillaries falling in this category. This flow pattern characterized consisted of a rapid rise to a high peak velocity following the occlusion, a precipitous fall to zero flow, and a slower return to control levels. The time at which peak velocity was reached very nearly coincided with the peak in the gross flow pattern. Also present in this group were all seven capillaries whose control velocity profiles were periodic in nature. While the flow patterns of these capillaries were quite different from the remainder of the group in the control state, during reactive hyperemia the response of these capillaries was indistinguishable from the rest of the group. The average velocity profile shown for group IV in Fig. 4 does not indicate an interval of zero flow. This is due to the variability in onset and duration of zero flow among the individual capillaries. The dashed lines in the reactive hyperemia phase indicate the average interval of zero flow.

To assist comparison of the group profiles with the reactive hyperemia observed in the gross flow, these profiles

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**TABLE 1. Parameters describing reactive hyperemia in capillary groups and in gross flow**

<table>
<thead>
<tr>
<th></th>
<th>Gross Flow</th>
<th>Individual Capillaries</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Group I</td>
<td>Group II</td>
</tr>
<tr>
<td>Avg preocclusion value, mm/sec</td>
<td>3.2 ±1.1*</td>
<td>3.0 ±1.4*</td>
</tr>
<tr>
<td>Duration of response following occlusion, sec</td>
<td>9.8 ±1.6</td>
<td>14 ±1.8</td>
</tr>
<tr>
<td>Time following occlusion to reach peak flow, sec</td>
<td>4.8 ±1.6</td>
<td>6 ±1.5</td>
</tr>
<tr>
<td>Peak velocity/control velocity</td>
<td>1.8 ±0.4*</td>
<td>1.5 ±0.9</td>
</tr>
<tr>
<td>Duration of flow during reactive hyperemia</td>
<td>13 ±4</td>
<td></td>
</tr>
<tr>
<td>Duration of zero flow during reactive hyperemia</td>
<td>7 ±3</td>
<td></td>
</tr>
</tbody>
</table>

* Units are ml/(min*100 g). † Peak flow/control flow. ‡ Time to reach control flow following occlusion.
have been superimposed on the gross flow curve in Fig. 5. The control velocities have been normalized so that differences in peak velocity to control velocity can be more clearly seen.

In classifying the velocity profiles into groups, the question arises as to whether the same velocity profile will be observed in a particular capillary following several occlusions, or if the pattern will change spontaneously. To examine this question, the effect of repeated occlusions was studied in 31 capillaries. In Fig. 6 are shown the flow patterns from such a study in two capillaries. The errors indicate the variability in the response with successive occlusions. The upper profile was classified as type IV and is the average of data from four occlusions. The lower velocity profile was classified as type I and was obtained from three occlusions. In general, we found that none of the capillaries changed velocity profiles in terms of group designations, at least during the duration of these studies which did not exceed 15 min per capillary. There did seem to be more variability following repeated occlusions for capillaries showing the type II velocity profiles, but it was not sufficient to shift classification.

In addition to the quantitative studies, the following qualitative observation was made. After release of an arterial occlusion, we frequently observed that many of the capillaries within the microscope field of view (0.5 mm diam) displayed similar flow characteristics. This was most easily seen when a group IV capillary was being studied. The flow in a set of 6–12 capillaries would appear to reach peak velocity at the same time and the duration of the zero flow phase following reactive hyperemia appeared to be identical. This similarity of flow behavior in adjacent capillaries was also observed with other types of behavior, although direct comparison in those instances was more difficult.

Over the time period in which capillary flows were studied in one muscle, and from cat to cat, the arterial blood pressure varied somewhat. The average blood pressure range during all measurements was 70–110 mm Hg. There was no apparent correlation between capillary flow patterns and the blood pressure, at least within the range of pressures found in these studies.

To obtain an estimate of the degree to which reactive hyperemia can be attributed to augmentation of flow in capillaries flowing prior to an occlusion, we summed the individual capillary flow profiles and compared this sum to the average gross flow. This comparison is shown in Fig. 7. The two curves do not exactly coincide, but the summed velocity curve does lie entirely within the experimental errors of the gross flow curve. In addition, it is of interest that the time course of reactive hyperemia as measured by the gross flow is quite similar to that obtained from the sum of individual capillaries. A more quantitative comparison of the two curves can be made by comparing times at which peak flow is reached in each case, and also comparing the ratios of peak flow to control flow for each curve. The time at which the summed peak velocity is reached is 6.1 ± 1.6 sec following the end of occlusion while the maximum gross flow is reached after 4.8 ± 1.6 sec. For the gross flow obtained from venous effluent, the ratio of peak flow to control flow is 1.8, and for the estimated flow from the summed velocity profiles the ratio is 1.48. Thus it appears that about 70% of the increased flow following an occlusion can be accounted for by augmentation of capillary flows.

If one varies the percentage contribution of each group to the total flow, it is possible to obtain a summed velocity curve which agrees very closely with the average gross flow, i.e., the difference between the two normalized curves is less than 10%. The distribution among the various types of responses which would give the best agreement are: group IV, 20%; group II, 7%; group III, 39%; and group IV, 34%. This compares with the distribution of 28% in group I, 14% in group II, 21% in group III, and 37% in group IV in the population studied.

Two possible difficulties in making the above comparison are immediately evident. First, the gross flow measurements were made on the venous effluent and thus may include venous capacitance effects. This should, however, have the effect of delaying the peak in gross flow beyond that in the capillary network. This was not evident in our studies. It might also have the effect of reducing the magnitude of peak flow. But the rise in venous outflow was greater than in the summed red cell velocities. A second limitation on direct comparison is that we have measured red cell velocities in the capillaries rather than plasma and red cell flow. The assumption made in comparing the summed...
velocities with the gross flow is that the velocity of the red cells is proportional to the flow of whole blood in the capillary. To estimate the validity of this assumption, we calculated the flow through each capillary assuming a constant capillary diameter of 4.5 μ and using the results of Hochmuth, Marple, and Suter (6) on plasma layer thickness as a function of red cell velocity in glass tubes. These calculated flows were summed and compared with the summed velocity profiles. The calculated flow differed from the velocity sum by less than 2%. Thus we feel that the red cell velocities give a reasonably accurate picture of changes in flow of whole blood through the capillaries.

Discussion

The cat sartorius muscle was selected for these studies because of its suitability for transillumination coupled with the fact that it can be surgically isolated. Since this muscle has not been used previously, to our knowledge, for reactive hyperemia studies, direct comparison with results of others is of course not possible. On initial examination it appears that the magnitude of reactive hyperemia in this preparation is somewhat less than that usually found in skeletal muscle.

However, Konradi and Levtov (11) have studied the degree of reactive hyperemia in cat gastrocnemius muscle as a function of occlusion duration. Their experiments show that with a 1-min occlusion, the ratio of peak flow during reactive hyperemia to control flow is about 1.82. We found this ratio in cat sartorius to be 1.8 ± 0.4, obviously in very good agreement. Studies of reactive hyperemia in canine skeletal muscle (9, 15, 22) reveal a peak-to-control flow ratio of between 2.3 and 4.0. Further, in plethysmographic studies of reactive hyperemia in human forearm (1, 10), this ratio was found to be 5.5 ± 6.5. Thus the available data suggest that the intensity of the reactive hyperemia response in cat skeletal muscle is somewhat less than that usually found in humans.

The average gross flow of 3.2 ml/(min · 100 g) is somewhat lower than that observed in other cat skeletal muscle preparations (4, 11). In our preparations we usually observed a higher flow rate (5–7 ml/(min · 100 g)) at the beginning of the experiment, with the flow usually dropping within the 1st hr to the average level quoted above. The early flow rates were comparable to those measured by Konradi and Levtov (11).

The various mechanisms which are currently thought to play a significant role in reactive hyperemia can be subdivided into two types: 1) metabolic or chemical mechanisms and 2) myogenic mechanisms. One of the first formulations of a metabolic hypothesis was presented by Lewis and Grant (14) in 1925. They postulated that active vascular changes following a period of occlusion were proportional to the amount of slowly diffusible vasoactive substances which accumulated in the extravascular space during the occlusion. These substances were assumed to have vasodilator effects until they were chemically broken down or washed out when blood flow was restored. An implication of this hypothesis is that the greatest concentration of vasoactive metabolites will exist at the time flow is restored, and thus the resistance vessels will be maximally dilated at this time. Thus one would expect peak blood flow to occur very soon after free flow is restored.

The hypothesis that intravascular pressure can play a role in regulating vascular tone was first proposed by Bayliss (2). According to this hypothesis, the smooth muscle is thought to be sensitive to the transmural pressure, relaxing when the pressure decreases as during an occlusion and constricting when pressure is increased. Follow (3) and Patterson (16) have shown that myogenic effects do indeed seem to play a role in skeletal muscle during reactive hyperemia. If the vascular relaxation during reactive hyperemia is due to the loss of pressure stimulus one would again expect the greatest muscle relaxation at the end of the occlusion period, and peak flow should be reached very soon after the occlusion is released.

We had hoped that by examination of the flow profiles of individual capillaries during reactive hyperemia, it might be possible to see more clearly the role of metabolic and/or myogenic mechanisms in the dilation of the resistance vessels feeding that capillary. Thus each flow profile type has been considered with this in mind.

Capillary flow profiles of the first type were characterized by a return to control flow following the occlusion, with little or no flow greater than the control values. It appears that the vessels controlling flow through these capillaries are relatively unresponsive to changes in intraluminal pressure and to changes in metabolite concentration. Thus there is little evidence of myogenic or metabolic factors playing a role in these vessels. One possibility which might be considered is that these vessels were maximally dilated prior to occlusion since the average control velocity for this group is considerably higher than in the other groups (see Table 1). However, this control velocity is still well below the peak velocity of any of the other groups. An additional difficulty with assuming that the resistance vessels upstream to these capillaries respond passively to pressure is that the return to control flow is much slower than would be expected. On the average the flow in these capillaries had not returned to control levels until well after the gross flow had reached its peak.

Capillaries with flow profiles of the second type are not readily explained in terms of a metabolic or myogenic hypothesis. The reactive hyperemia in these capillaries is characterized by a rather rapid rise to the control red cell velocity followed by a sustained elevated flow with the peak value being reached about 35 sec following the occlusion. In terms of a classical metabolic or myogenic hypothesis, we would have expected a much more rapid rise to peak flow than observed here since vascular relaxation should be at its peak when the occlusion is released. A possible modification of the metabolic hypothesis which might explain the slow increase is that vasoactive forms of the metabolites are not present until sometime after the restoration of flow.

Postocclusion flow profiles of the third type were more nearly similar to the classical gross flow pattern than were the other three types. The average flow in this group was characterized by a fairly slow rise to peak flow (about 10 sec) followed by a gradual return to control flow levels. The return to control flow with no phase of decreased flow following the increased flow may be indicative of either a...
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relatively fast removal of vasoactive metabolites or a moderately strong, critically damped myogenic response. The greatest difficulty in explaining this response in terms of the usual metabolic or myogenic effects is the time required for the development of vasodilation following the occlusion. If the dilation is due to accumulation of metabolites, then the data seem to indicate that the vasoactive agents do not fully affect the resistance until sometime after the arterial occlusion ends, and that there is increasing relaxation of the smooth muscle for some 10 sec. One possibility is that at least some of the vasoactive metabolites are produced by aerobic metabolism which are formed after flow is restored. It is equally difficult to explain this 10-sec period to reach maximal flow purely in terms of a myogenic response since the muscle fibers would be expected to be most relaxed when the intraluminal pressure is reduced (i.e., during the occlusion). Thus the resistance vessels should be most dilated very soon after pressure is restored.

The group IV flow profiles are suggestive of an underdamped response to pressure or flow. A possible explanation of the flow in terms of myogenic effects is as follows: 1) during the control phase, the flow is maintained at a low level by partial constriction of myogenically active muscle cells; 2) during the arterial occlusion, loss of pressure stimulus allows the muscle fibers to relax so that following the occlusion, the flow rapidly increases; 3) the higher than usual pressures existing in the resistance vessels during the increased flow cause the muscle fibers to overreact, completely stopping the flow; 4) the muscle fibers then respond to the reduction in pressure stimulus during zero flow by relaxing sufficiently to allow the flow to return to control levels. In some cases it appears that there is overreaction both to increased and decreased pressures which is repeated several times giving rise to periodic flow patterns. It is possible that the duration of increased flow following occlusion and the duration of zero flow represent the delay in response of the myogenically active vascular smooth muscle fibers to the pressure stimulus.

The data on reactive hyperemia suggest primarily metabolic regulation of flow for groups II and III and myogenic control for capillaries of group IV. However, the data do not prove that this correlation between capillary types and regulatory mechanisms is correct.

It is known that the rate of blood flow through skeletal muscle depends on the kind of fibers which make up the muscle. Reis and Wooten (17) found the flow through red muscle to be greater than through intermediate type muscle and the flow through intermediate muscles to be greater than through white muscles. Also, Folkow and Halicka (4) have reported resting blood flow through cat soleus muscle (red muscle) to be 20 ml/(min·100 g) while the flow rate through gastrocnemius muscle (principally white muscle) was 9 ml/(min·100 g). At low stimulation rates the relative increase in flow through the soleus muscle was much less than through the gastrocnemius muscle. Hilton (5) found a relatively small increase in flow through the soleus muscle during exercise. If a similar relationship exists in respect to reactive hyperemia, then the response seen in an individual capillary might be determined by the type of muscle fibers surrounding the capillary or, more appropriately, its feeding arteriole. For instance, group I capillaries, which have a high control flow rate and very little hyperemic response after release of occlusion, might be associated with the slow twitch, soleus type muscle fibers. Similarly, the group III or IV capillaries might be associated with primarily white fibers since they have a lower control flow rate and much greater hyperemic response after occlusion. However, we have no data to indicate that this is actually the case.

A second goal of these studies was to estimate the contribution of flow augmentation in the capillaries to the reactive hyperemia of skeletal muscle as measured by the gross blood flow. In 1919, Krogh (13) suggested that in normal resting muscle, only a small percentage of the capillaries in skeletal muscle were open, and that this number increased considerably during exercise. In particular, in frog sartorius muscle he found the ratio of capillaries open during exercise to the number open during rest was about 3.4:1. Other studies (14, 27) using capillary filtration and diffusion rates of small ions to assess capillary surface area have also indicated that there is an increase in the number of open capillaries in exercise. These studies have generally indicated smaller increases than suggested by Krogh. Stainsby and Otis (20) measured oxygen consumption during rest and exercise in the dog gastrocnemius-plantaris muscle group. Using Krogh's equation for the diffusion of oxygen out of a cylinder into a second cylinder, they estimated that capillary number increased 15-fold during exercise. The indirect evidence for recruitment of static capillaries in exercise might lead one to suppose that such a mechanism might also be operative in reactive hyperemia. However, our data provide little support for such a mechanism in cat sartorius muscle. It appears that perhaps 30% of the increased flow may be due to the opening of additional capillaries. We have no proof that this is the case, however, since the 30% difference may simply be due to statistical variability. If we assume any newly opened capillaries have flow velocities equal to the average control velocities, then the ratio of capillaries open during reactive hyperemia to capillaries open during control would be about 1.3:1. Several of the capillaries which we studied did have short intervals of zero flow during the control phase. These might appear as "closed" capillaries in Krogh's India ink infusion experiments, even though they were flowing most of the time. It appears that during reactive hyperemia in cat sartorius muscle, the main contribution to the increased flow is by augmentation of the flow in capillaries previously flowing, rather than by recruitment of additional capillaries.

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