Communication Between Feed Arteries and Microvessels in Hamster Striated Muscle: Segmental Vascular Responses Are Functionally Coordinated

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Pressures in the primary arterioles of the cremaster muscle are reported to be approximately 50% of systemic, indicating that arterial resistance proximal to microvessels is high and may limit maximal blood flow. With no change in arterial resistance, increases in perfusion normally associated with muscle work either could not occur or would require increments in systemic pressure far greater than those actually observed in vivo. Therefore, we hypothesized that the small arteries feeding the muscle may participate in the hyperemic response. To test this hypothesis, male golden hamsters (n = 31, 118 g) were anesthetized (pentobarbital, 70 mg/kg i.p.), and the right cremaster was opened to expose its feed arteries, which originated from the iliac artery. Preparations were superfused and maintained at 35 ± 1°C. Feed arteries had substantial tone, as shown by the fact that topical acetylcholine, applied at supramaximal concentration, dilated these vessels from 115 ± 8 μm at rest to 158 ± 9 μm (mean ± SE; n = 38 vessels; p < 0.01), corresponding to an estimated 4.4-fold increase in conductance. Stimulation of the sectioned motor nerve (8 Hz, 30 seconds) induced striated muscle contraction and increased feed vessel diameter from 93 ± 5 μm to 116 ± 5 μm (n = 14; p < 0.01), consistent with a 2.6-fold increase in conductance. A 5-minute occlusion of the iliac artery resulted in feed artery dilation of similar magnitude. Supramaximal doses of acetylcholine applied topically to the distal portions of the cremaster resulted in striated muscle contraction and a dilation that propagated upstream to increase feed artery diameter by 25%. Succinylcholine abolished muscle contraction induced by acetylcholine, but not distal or propagated dilations. Supramaximal doses of adenosine applied distally also produced dilation of feed vessels, but of a smaller magnitude than seen with acetylcholine. Similar results were obtained for feed arteries of the gracilis muscle in response to both muscular contraction and topical acetylcholine. We conclude that the designation “resistance vessels” should include these larger feed arteries and that vasodilation spreads upstream during functional demand. Thus, feed vessels contribute to the integration of muscle blood flow with metabolic requirements. Furthermore, there appear to be separate components involved in coordinating vasomotor responses among segments of the resistance vasculature. (Circulation Research 1986; 59:283–290)

VASCULAR resistance is generally considered to reside in arterioles that are 50 μm or smaller in diameter.1 However, in a variety of organs, including brain, intestine, heart, cremaster, and cheek pouch, the intravascular pressure measured in the largest arterioles has been shown to be 50 to 60% of systemic arterial pressure.2-8 Intravascular pressures this much below systemic arterial pressure indicate that small feed arteries proximal to the microvasculature contribute significantly to vascular resistance and are thus a potential site of flow control. If blood flow is to increase several fold during vasodilation, the diameter of feed arterioles, as well as that of arterioles, must increase significantly.

Conduit arteries of the heart10-11 and skeletal muscle,12-14 which are several millimeters in diameter, have been shown to undergo dilation in response to hyperemic stimuli. However, there has been no systematic investigation of vasomotor responses in small feed arterioles less than 1 mm in diameter. In the present study, we have examined dilation of feed arterioles in response to four types of experimental manipulations: vasoactive agents, functional hyperemia, reactive hyperemia, and alterations in PO₂. In addition, we have tested three hypotheses that have direct bearing on the role of feed arterioles in the control of flow to striated muscle: 1) that feed arterioles of the cremaster and gracilis muscles have significant tone at rest, 2) that the conductance of these feed arterioles increases during functional demand, and 3) that communication exists between feed arterioles and microvessels in striated muscle.
Materials and Methods

Animal Preparation

Male golden hamsters (n = 31, 118 ± 3 g) were anesthetized (pentobarbital sodium, 70 mg/kg i.p.). A tracheostomy was performed to ensure a patent airway, and either the right jugular or a femoral vein was cannulated for continuous infusion of anesthesia and fluid replacement (420 µl/hour). In 17 animals, carotid arterial pressure was followed throughout and was paired to these resistance vessels. Pressure measured at locations indicated by the star are approximately 50 mm Hg. Asterisks indicate feed artery observation sites. In general, due to anatomical variations between hamsters, two to four sites were observable in a given preparation.

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Our hamster cremaster preparation (anterior view, right side). The tissue beyond the dotted line designates the region of the cremaster muscle usually exposed for microcircular studies (e.g., Baez, 15 and that to which ACh or ADO was applied in order to evoke propagated vasodilation of feed arteries. Only the feed arteries and primary and secondary arterioles are illustrated; venules and veins generally run paired to these resistance vessels. Pressure measured at locations indicated by the star are approximately 50 mm Hg. Asterisks indicate feed artery observation sites. In general, due to anatomical variations between hamsters, two to four sites were observable in a given preparation.

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As an estimate of the physiological importance of changes in feed vessel diameter, we assumed that feed vessel conductance was proportional to the fourth power of diameter. To estimate the changes in feed vessel conductance induced experimentally, a conductance ratio was calculated for individual observations as the ratio of $D_{\text{response}}$ to $D_{\text{baseline}}$, where D is the diameter. A total of 54 feed arteries were studied. For a given vessel, the first test was for resting tone; typically, two to three additional interventions were then studied. A
mean value was obtained for responses to each experimental intervention.

**Vasomotor Stimuli**

Four different forms of stimuli were used to assess the *in vivo* reactivity of the feed vessels of striated muscle: topical application of vasoactive agents, functional hyperemia, reactive hyperemia, and altered PO₂ in the superfusion solution.

Adenosine (ADO) and acetylcholine chloride (ACh) (Sigma Chemical Corporation) were freshly prepared in isotonic saline at a concentration of 1 mM; norepinephrine bitartrate (Levophed; Winthrop Laboratories) was prepared at 0.1 mM. Feed vessels were exposed to drugs by applying 3–5 drops of a stock solution through a 30-gauge needle onto the proximal region of the superfused preparation. Although drugs were diluted by the superfusion solution, our observations indicate that affective tissue concentrations were supra-maximal. Additionally, application of drugs could be restricted to the distal portion of the cremaster because of the tissue topography and the direction of superfusate flow (Figure 1). We ascertained that solutes were confined. Distal arterioles were exposed without surgical disturbance of the tissue topography and the direction of superfu-

In six experiments, succinylcholine chloride (0.1 mM; Sigma) was added to the superfusion solution to block striated muscle contraction. These experiments were designed to determine the vasomotor effect of ACh independent of the striated muscle contraction normally accompanying topical ACh application.

Functional hyperemia was studied by inducing contraction of the cremaster and gracilis muscles via electrical stimulation (8 V, 50 μsec pulse duration; WPI Instruments Inc., Model 301 A) of the distal stump of the motor nerve, which was positioned across platinum wire electrodes. Stimulus parameters were determined during initial experiments as being of sufficient intensity and pulse duration to elicit maximal activation of the cremaster striated muscle fibers.

To study reactive dilation of the cremaster feed arteries, the iliac artery was occluded proximal to the origin of feed vessels with an atraumatic vascular clamp; for most experiments the duration of occlusion was 5 minutes.

The sensitivity of cremaster feed vessels and arterioles to oxygen was determined by increasing the PO₂ in the superfusate solution from 0 to 21%; CO₂ was constant at 5%, and the balance was N₂. Oxygen constricts arterioles. Vasoconstriction of vessels in the distal region of the cremaster may be accompanied by an elevation of the upstream pressure in the feeding arteries and could thereby elicit a myogenic response. Therefore, to more directly evaluate the oxygen sensitivity of cremaster feed arteries, in 3 hamsters these vessels were exposed without surgical disturbance of the cremaster muscle.

At the end of each day’s experiments, the hamster was euthanized with an overdose of pentobarbital delivered intravenously.

**Statistics**

The statistical significance of vasomotor responses was determined with paired *t* tests and one-way analysis of variance with a repeated measures design. When significant F ratios were obtained, contrasts were used for specific comparisons. Summary data are presented as means ± 1 SEM.

**Results**

**Anatomical Observations**

The primary feed vessels of the hamster cremaster muscle originate from the iliac artery. The typical configuration of the arterial supply is a vessel 200–250 μm in diameter that runs transversely across the top of the cremaster, branching once and then subdividing into 2, or sometimes 3, smaller (80–120 μm) vessels (Figure 1). These, in turn, course below and through the cremaster parallel to its long axis. Occasionally, a medial feed artery (60–100 μm) arises from a larger vessel that is also derived from the iliac artery. The motor nerve to the cremaster runs parallel to the aorta and enters the top of the muscle as a discrete bundle at the medial border.

The main feed artery of the gracilis muscle is also approximately 250 μm in diameter and originates from the femoral artery. It then divides into branches that are 100–125 μm in diameter. These, in turn, give rise to daughter branches 50–70 μm in diameter, which penetrate the muscle parenchyma. The feed arteries of the gracilis are shorter in length than those of the cremaster, and the majority of their length is external to the muscle.

**Vasomotor Responses**

**Topical Acetylcholine.** The first requirement for the participation of feed arteries in physiological control is that these vessels have tone at rest, a fact demonstrated by the observation that ACh applied topically dilated these vessels significantly (Figure 2 and Table 1). These vessels were also quite responsive to constrictor agents, since application of norepinephrine induced a 70% constriction of feed arteries (Table 2).

Acetylcholine also produced feed vessel dilation by an indirect mechanism. When restricted to distal portions of the cremaster, topical ACh resulted in striated muscle contraction locally and a significant dilation that spread upstream (Table 2). Succinylcholine added to the superfusion solution abolished muscle contraction but not local or ascending vasodilations with ACh (Table 2).

Of the feed arteries tested for ascending vasodilation, only 58% actually dilated in response to topical application of ACh. The variability of ascending vasodilation is similar to earlier observations; however, its basis remains to be established.

**Functional Hyperemia.** Functional dilation of feed arteries was induced by muscular contraction. Stimulation of the sectioned motor nerve elicited vigorous
twitch contractions for both the cremaster and the gracilis muscles and resulted in significant vasodilation of feed arteries supplying both tissues (Figure 2 and Table 1). Functional dilation was absent in 20–25% of the feed arteries studied in both the cremaster and gracilis muscles.

**Reactive Hyperemia.** Feed arteries of the cremaster dilated during reactive as well as functional hyperemia. On occlusion of the iliac artery, feed vessel diameter declined (see Figure 3) from 114 ± 8 μm to 85 ± 7 μm (n = 15, p < 0.01); probably due to concomitant reduction in intravascular pressure. During the occlusion period, a significant dilation of these vessels occurred (from 85 ± 7 μm to 93 ± 7 μm, p < 0.01). Restoration of iliac arterial flow resulted in pronounced dilation of feed vessels, succeeded by a return to control diameter within 5–10 minutes. The reactive dilation of cremaster feed arteries on release of the arterial clamp was dependent on the duration of occlusion (Figure 3). A 5-minute occlusion elicited a reac-

tive vasodilation that was similar in magnitude to that obtained with topical vasodilators (Figure 4, Table 2).

Both flow and diameter are reduced in feed arteries during occlusion of the iliac artery. Therefore, we sought to evaluate reactive dilation in feed artery segments in which intravascular pressure was not reduced during occlusion. For this purpose, we occluded single feed arteries distal to the site of observation. Little or no feed artery dilation was observed on release of

**TABLE 1. Vasomotor Responses of Hamster Gracilis Muscle Feed Arteries**

<table>
<thead>
<tr>
<th>Experimental intervention</th>
<th>n</th>
<th>Diameter (μm)</th>
<th>Conductance ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acetylcholine*</td>
<td>16</td>
<td>112±12</td>
<td>138±14</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2.5±0.3</td>
<td></td>
</tr>
<tr>
<td>Muscle contraction*</td>
<td>11</td>
<td>102±11</td>
<td>121±12</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2.0±0.1</td>
<td></td>
</tr>
</tbody>
</table>

Data are means ± SEM; n = number of vessels. Conductance ratio is defined in "Materials and Methods." The concentration of acetylcholine in working solutions was 1.0 mM; applications consisted of 3–5 drops of solution administered topically through the tip of a 30-gauge needle. Muscle contraction (30 seconds) was induced by nerve stimulation (8 Hz, 8 V, 50 μsecond pulse duration).

*Significant difference between control and response diameters, p < 0.01.

**FIGURE 2. Vasomotor response of the feed arteries supplying the hamster cremaster muscle to topical application of 3–4 drops of 1.0 mM ACh (panel A, n = 38) and 30 seconds of striated muscle contraction at a frequency of 8 Hz (panel B, n = 14).** The conductance ratio was estimated as described in "Materials and Methods." 

**TABLE 2. Vasomotor Responses of Hamster Cremaster Muscle Feed Arteries**

<table>
<thead>
<tr>
<th>Experimental intervention</th>
<th>n</th>
<th>Diameter (μm)</th>
<th>Conductance ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>Local adenosine*</td>
<td>29</td>
<td>113±7</td>
<td>150±8</td>
</tr>
<tr>
<td></td>
<td></td>
<td>3.5±0.3</td>
<td></td>
</tr>
<tr>
<td>Distal acetylcholine*</td>
<td>15</td>
<td>117±14</td>
<td>143±15</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2.7±0.4</td>
<td></td>
</tr>
<tr>
<td>Distal acetylcholine* (+ succinylcholine)</td>
<td>6</td>
<td>88±6</td>
<td>122±7</td>
</tr>
<tr>
<td>Iliac artery occlusion*</td>
<td>15</td>
<td>114±8</td>
<td>148±10</td>
</tr>
<tr>
<td>(5 min)</td>
<td></td>
<td>3.2±0.4</td>
<td></td>
</tr>
<tr>
<td>Oxygen (21%)</td>
<td>11</td>
<td>113±7</td>
<td>111±8</td>
</tr>
<tr>
<td></td>
<td></td>
<td>0.9±0.1</td>
<td></td>
</tr>
<tr>
<td>Local norepinephrine*</td>
<td>22</td>
<td>135±11</td>
<td>42±5</td>
</tr>
<tr>
<td></td>
<td></td>
<td>0.02±0.01</td>
<td></td>
</tr>
</tbody>
</table>

Data are means ± SEM; n = number of vessels. The concentrations of acetylcholine and adenosine in working solutions were 1.0 mM, that of norepinephrine was 0.1 mM. Local applications consisted of 3–5 drops of solution administered topically through the tip of a 30-gauge needle. Distal acetylcholine consisted of 10–15 drops applied to the distal region of the preparation (see "Materials and Methods"); in six experiments this was also done in the presence of 0.1 mM succinylcholine added to the superfusion solution. For oxygen, control values were obtained with 0% oxygen in the gas bubbling the superfusate, response values were recorded in the presence of gas containing 21% oxygen after a 5-minute equilibration period.

*Significant difference between control and response diameters, p < 0.01.
occlusion. However, as indicated by a sausage-like appearance where the clamp had been placed, vessels appeared to be damaged during this maneuver, thus precluding resolution of this issue without further study.

Effects of oxygen. In contrast to the effects of ACh, ADO, muscle stimulation, or vascular occlusion, manipulation of superfusion solution oxygen content caused a distal microvessel response without affecting feed vessels. Application of 21% O2 to the cremaster superfusion solution consistently induced 30-50% constriction of arterioles that were 25-30 μm in diameter, yet feed artery diameter was unaffected (Table 2 and Figure 4A). The lack of oxygen sensitivity of feed arterioles was apparent either with or without surgical exposure of the distal portion of the muscle; therefore, these data were pooled (Table 2).

We also tested whether feed arterioles consistently respond in parallel with microvessels during consecutive perturbations (Figure 4). Raising superfusate PO2 to 21% was followed by arteriolar constriction, and returning PO2 to 0% was accompanied by arteriolar dilation. However, elevation and reduction of superfusate PO2 was without effect on feed artery diameter (Figure 4A). In contrast to the results obtained with oxygen, muscular contraction and transient vascular occlusion resulted in dilation of feed arterioles (Figure 4B and 4C) as well as arterioles.12 Additional sensitivity of arterioles to oxygen was present at times when either ascending or functional vasodilation of feed arterioles could not be elicited.

Dilation of cremasteric feed arteries in response to distal application of vasodilators was separable into two components (Table 3). Application of either ADO or ACh to the distal region of the cremaster produced maximal dilation of those arterioles, shown by the observation that superposition of one dilator on the other was without further effect on arteriolar diameter. In contrast, the effect of distally applied ADO on feed vessel diameter was less than that of distally applied ACh; application of ACh in the presence of ADO induced a further dilation of feed vessels. Feed vessel response to distal application of ACh alone was not different from that obtained with ACh superimposed on ADO.

Discussion

Several aspects of our present work strongly support a role for the feed arteries in the physiological control of blood flow in striated muscle. First, the presence of basal tone in these vessels indicates that active control is present and that changes in feed vessel conductance can significantly influence tissue perfusion. Second, dilation of feed vessels in response to muscular contraction or vascular occlusion implies that changes in their resistance is one of several coordinated elements in the integration of tissue perfusion with metabolic demand. Third, dilation of these vessels in response to distal application of ACh or ADO indicates that communication exists between the microvasculature and the feed arteries. Similar results were obtained for the gracilis muscle with two experimental manipulations, indicating that such responses are not unique to the cremaster muscle. Our observations are therefore consistent with the general idea that feed arteries dilate in response to muscular exercise and thereby contribute functionally to the regulation of blood flow.

Many other observations support the conclusion that flow control is not confined to arterioles. Significant increases in the resistance of limb arteries occurred during vasomotor nerve stimulation.21 In the heart4 and brain,2 pressure measurements indicate that a major fraction of vascular resistance lies proximal to the microvasculature. In the brain, blood flow has been shown to be controlled by both arterial vessels and microvessels.22-23 Combined with observations of pressure distribution in the gut5 and cheek pouch,3,8 our present observations suggest that the control of feed vessel conductance may be a general component of cardiovascular regulation.

![Figure 3. Vasomotor responses of a hamster cremaster feed artery to progressively longer occlusions of the iliac artery. Successive panels were traced directly from a stripchart record. The duration of occlusion is given at the top of each panel; the onset and release of occlusion are designated by the first and second arrows, respectively. The horizontal reference line in each panel indicates 100 μ.](http://circres.ahajournals.org/)
FIGURE 4. Typical vasomotor response of a hamster cremaster muscle feed artery. Successive panels were traced directly from a stripchart record. A. Changes in oxygen content of the superfusion solution. Oxygen was initially 21%; then changed to 0% and 21% at the first and second arrows, respectively. Five minutes of equilibration elapsed after each change in oxygen content before vessel diameter was recorded. B. 30 seconds of striated muscle contraction at 8 Hz; the arrows designate the onset and cessation of stimulation, respectively. C. 5-minute occlusion of the iliac artery; arrows designate the onset and cessation of occlusion, respectively. D. Successive topical application of supramaximal doses of ACh (first arrow) or ADO (second arrow). The horizontal reference line in each panel indicates 100 μ.

It should be noted that not all tissues show high upstream resistance under resting conditions. In the central feed artery (diameter = 70–100 μm) of the cat tenuissimus muscle, intravascular pressure was not substantially below arterial pressure.18 However, even in this muscle, stimulation of vasoconstrictor nerves resulted in significant constriction of feed arteries as large as 200 μm in diameter.23 Therefore, although the role of feed arteries portrayed in the present study applies particularly to tissues in which the resting tone of feed vessels is high, during neurogenic vasomotor activity, feed vessel resistance can increase markedly and thereby limit flow.21–22

We used a calculation of vascular conductance to estimate the potential effect of changing feed vessel diameter on tissue perfusion. With this approximation, the values obtained for conductance ratios in response to our experimental manipulations ranged from two- to fivefold and suggest an important role for feed vessels in controlling flow to the working muscle. The potential significance of feed vessel dilation can be appreciated from the simple model shown in Figure 5. We can assume that 50–60% of vascular resistance lies proximal to the primary arterioles (i.e., $R_{PV} \approx R_{MV}$). Were the feed vessel resistance ($R_{PV}$) to remain fixed at resting levels while the more distal microvessel resistance ($R_{MV}$) decreased to minimum levels, microcirculatory perfusion pressure in the primary arterioles would fall toward venous values (solid line; $R_{MV} = 2$), and tissue perfusion (broken line) could increase less than twofold above rest before becoming limited by the upstream resistance. If feed vessels then dilate, larger increases in tissue blood flow would occur as microcirculatory perfusion pressure returned toward resting levels (solid line; $R_{PV}$ decreasing from 12 to 2 while $R_{MV} = 2$). Alternatively, if both $R_{PV}$ and $R_{MV}$ declined during stimulation such that the ratio of these resistances remained constant, tissue perfusion would increase in proportion to the ratio of total resistance during stimulation to that during control, without affecting microcirculatory perfusion pressure.

The responses that we observed during vascular occlusion (Figure 3) may have been due to either metabolic or myogenic factors. The increase in magnitude and duration of reactive dilation of cremaster feed arteries as occlusion duration was extended is consistent with progressive accumulation of vasoactive metabolites in the muscle.24 However, since feed vessel diameter (and presumably pressure) decreased consistently on occlusion of the iliac artery, vasodilation on

| Table 3. Responses of Cremaster Muscle Arterioles and Feed Arteries to Distal Application of Adenosine and Acetylcholine |
|---------------------------------|----------------|----------------|----------------|
|                                |                 | Distal ADO     | Distal ACh     |
| Arterioles ($n = 8$)*           | 28 ± 2          | 49 ± 4         | 50 ± 4         | 28 ± 2         |
| Feed arteries ($n = 8$)*†        | 78 ± 3          | 94 ± 4         | 111 ± 4        | 80 ± 3         |

Data are means ± SEM; $n$ = number of vessels. ADO = adenosine, ACh = acetylcholine. Distal applications consisted of 10–15 drops of a 1 mM solution on to the distal region of a preparation as described in "Materials and Methods."

*Significant different between control and vasodilator response diameters, $p < 0.01$.
†Significant different between ADO and ACh response diameters, $p < 0.05$. 

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restoration of flow may well have been the result of a myogenic mechanism. To address whether a myogenic response could contribute to reactive dilation, we applied either ACh or ADO to feed arteries during the period of iliac occlusion. These vasodilators were without effect on feed artery diameter, suggesting that these vessels had relaxed in response to a reduction of intravascular pressure. Subsequent restoration of flow and pressure may have thereby induced a transient distension.

In the cremaster muscle, distal application of ACh alone was accompanied by striated muscle contraction locally and by vasodilation that spread upstream into feed arteries. Absorption into the blood of either ACh or vasoactive metabolites produced during muscular contraction could, by diffusing from an adjacent vein or the parenchyma, directly cause feed vessel dilation. However, we think that this possibility is unlikely because of the continual movement of superfusion solution over the preparation. Furthermore, in several preparations, feed arteries were either not embedded in the parenchyma of the muscle or were not located adjacent to a countercurrent vein. Therefore, in response to nerve stimulation, muscular contraction itself may have induced the spread of vasodilation into feed arteries supplying the cremaster and gracilis muscles. Furthermore, since the nerve bundle of both muscles was sectioned proximally prior to electrical stimulation, it seems unlikely that feed vessel dilation during these experiments was mediated through an afferent reflex loop.

The nature of the stimulus inducing the spread of dilation into the feed arteries remains elusive in spite of over 25 years of research. A flow-dependent dilation has been reported for the femoral artery and coronary arteries. Propagated vasodilation, involving a conducted stimulus acting independent of flow, has also been proposed. Additional studies are required to ascertain the mechanisms that underlie a coordinated dilatory response between feed arteries and microvessels.

For both functional and reactive hyperemia, feed arteries and arterioles dilated in parallel. However, our data show that the resistance vasculature does not simply function as a syncitium. In the cremaster muscle, oxygen was without effect on feed artery diameter, yet it consistently constricted arterioles. Furthermore, in response to successive distal application of ADO and ACh, incremental dilation of feed arteries occurred without further dilation of arterioles. We cannot explain the differential responses of arterioles and feed arteries to these experimental manipulations; nevertheless, these differences may prove useful in future studies addressing the mechanism(s) of intravascular communication.

In conclusion: Feed arteries of both the cremaster and gracilis muscles have substantial tone at rest. These vessels dilate in response to both striated muscle contraction and vascular occlusion, demonstrating that feed vessels participate in functional and reactive hyperemia. Topical application of either acetylcholine or adenosine to the distal portion of the cremaster induced vasodilation that spread upstream into feed arteries, indicating that communication exists between microvessels and feed vessels.

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References

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KEY WORDS • muscle blood flow • exercise • oxygen delivery • functional hyperemia • reactive hyperemia • propagated vasodilation • ascending vasodilation • acetylcholine • adenine
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