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Flow-induced dilation of skeletal muscle feed arteries: relevance to exercise hyperemia

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The purpose of this study was to determine if flow-induced dilation potentially contributes to exercise hyperemia in rat extensor digitorum longus muscle, primarily composed of fast-glycolytic fibers, and rat gastrocnemius, a muscle of mixed fiber types. The differences in fiber type of each muscle may be a factor in how the feed arteries dilate during exercise.

The study was designed to determine if flow-induced dilation potentially contributes to exercise hyperemia. In rat extensor digitorum longus muscle, flow-induced dilation was not a plausible mechanism to explain the increase in blood flow during exercise. Furthermore, the soleus muscle is primarily composed of slow-oxidative fibers. This data led to the conclusion that flow-induced dilation is not a plausible mechanism to explain the increase in blood flow during exercise.

Methods

- Rat EDLFA and GFA were isolated and cannulated on two glass micropipettes. The arterial feed was measured using an inverted microscope and video camera (Figure 1).
- After cannulation, feed arteries were bathed in warm physiological saline solution (WPS) at 37°C and pH of 7.4. WPS with albumin (WPSA), closely resembling blood plasma, was used inside the artery.
- EDLFA had a maximum diameter of 142.4 ± 1.0 μm and developed spontaneous tone of 33.4 ± 0.7% (N=12). GFA had a maximum diameter of 274.0 ± 1.7 μm and developed spontaneous tone of 35.7 ± 0.8% (N=16).
- Each feed artery was allowed to develop spontaneous tone (a minimum of 20%) during the equilibration period. Once tone was established, flow was induced by elevating one reservoir while lowering the other an equal distance (see Figure 1). This induces flow through the vessel, allowing increased intraluminal arterial pressure (p). The arteries were allowed approximately 3 minutes at each flow step. Corresponding flow (μl/min) and internal diameter were measured. Shear stress values were calculated using the equation: 

\[ \tau = 4Q / \pi r^2 \]

where Q is blood viscosity (0.008 poise at 37°C), is perfuse flow (μl/min), and r is internal radius of each feed artery.

- Pressure gradients of 2, 6, 8, 10, 15, 20, and 40 cmH O (L) were used.

Discussion

Although our findings show that the range of EDLFA shear stress values were higher than the range of shear stress observed at various activity levels (1,2,3,7,10,12,14,15) leading to the assumption that flow-induced dilation could contribute to exercise hyperemia—EDLFA did not show significant dilation to increasing levels of flow. In fact, the artery dilated only 7.0 μm to a flow range of 40 μl/min. Thus, flow-induced dilation does not account for the dilation seen during exercise.

Furthermore, our findings show that the values of shear stress at which dilation occurred in gastrocnemius feed arteries were much lower than in vivo values found in previous studies (1,2,3,7,10,12,14,15) at different activity levels. With the exception of anesthetized rat skeletal muscle, these data demonstrate that flow-induced dilation does not contribute to the dilation that occurs during exercise. These data are consistent with that of Jasepulis and Laughlin (8) who found that shear stress at which dilation occurred in soleus feed arteries was lower than the shear stress values during exercise. Clifford et al. (5) extended the findings of Jasepulis and Laughlin, finding that rabbit femoral arteries contracted at higher levels of flow, which indicated that flow-induced dilation is not a plausible mechanism for the dilation occurring during exercise. Our data, taken together with those of Jasepulis and Laughlin (8) and Clifford et al. (5,9), strongly support the hypothesis that flow-induced dilation does not contribute to exercise hyperemia in muscles of different fiber types.

There have been other proposed mechanisms to explain the dilatory response of feed arteries during exercise. Segal and Duling (7) propose that propagated dilation, in which signals are conducted through gap junctions between cells, can pass signals from arterioles within contracting muscle to feed arteries. Additionally, Salto et al. (10) found evidence that venous arterial communication can explain the increase in arterial diameter during exercise. Lastly, Clifford et al. (5) proposed that compression from muscular contraction can cause arterial dilation. They found that external pressure on feed arteries elicited an immediate dilatory response that may explain dilation during exercise. In short, the mechanism by which feed arteries dilate during the onset of exercise is still unknown. Perhaps a satisfactory explanation will include a combination of multiple mechanisms.

References


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