Among muscular contraction in healthy animals, the skeletal muscle arterioles typically dilate to increase blood flow and sustain the elevated metabolic requirements of the tissue. The general consensus is that during such periods of increased tissue metabolism, local control mechanisms are capable of overriding any existing constrictor influences to cause arteriolar dilation. In hypertension, these local mechanisms may not be strong enough to fully overcome the background of increased constrictor stimuli and decreased vascular distensibility. Under such circumstances, functional dilation would be impaired.

Microvascular pressure measurements indicate that vessels throughout the arteriolar network as well as small resistance arteries contribute substantially to the increased skeletal muscle resistance in spontaneously hypertensive rats (SHR). To determine whether skeletal muscle arterioles in hypertensive animals have a normal or impaired ability to respond to local metabolic stimuli, it is first necessary to consider how arteriolar network resistance is increased in the hypertensive state. Various studies suggest two hemodynamic causes of this increased resistance: a reduction in arteriolar diameter, although this has not been observed in all studies, and a temporary or permanent decrease in the number of small arterioles open to blood flow. Even in cases in which the arterioles of SHR have near-normal diameters, a greater than normal constrictor tone would be required to maintain those diameters against the elevated distending pressures that have been measured in these vessels.

A permanent loss of vessels from the arteriolar network could reduce its ability to increase blood flow during periods of increased tissue metabolism.
However, the effect of temporary arteriolar closure on active hyperemia is less predictable. If local metabolic stimuli are strong enough to open the arterioles to blood flow, the flow increase may be normal. However, if these vessels are not readily opened by local metabolic stimuli, nutrient delivery to the tissue could become limited and result in an unusually low tissue oxygen tension (PO2).

We tested the hypothesis that the microvascular abnormalities associated with hypertension reduce the ability of local vascular control mechanisms to dilate the arterioles, such that nutrient delivery during contraction is compromised. The rat spinotrapezius muscle was chosen for this study because it normally exhibits well-developed local vascular control. For example, even during muscular contractions at 12 Hz, arteriolar dilation maintains the tissue PO2 at about 80% of the resting value in normotensive rats.3 The inner diameters of arterioles of comparable branch order were measured in adult normal rats and SHR to determine if vasoconstriction was present in hypertensive animals either at rest or during contractions. Contraction frequencies of 1 to 8 Hz were used so that vascular behavior could be evaluated over a wide range of metabolic conditions. The spinotrapezius muscle of SHR does not exhibit a permanent loss of arterioles,17 but to our knowledge, the possibility of temporary arteriolar closure in this muscle has never been evaluated. Therefore, discrete tissue regions were also observed to determine if there were any arterioles that opened to blood flow during either muscle contractions or maximal dilation with adenosine. In addition, tissue PO2 was measured during contractions and used as an index of the overall ability of the vasculature to supply nutrients for the contracting tissue through vasodilation and related processes that influence extraction. The distribution of tissue PO2 at rest was also determined to ascertain whether the hypertensive state in any way limits the availability of oxygen to the resting tissue.

Materials and Methods

Adult SHR (16-20 weeks old) were used in this study, and age-matched Wistar-Kyoto rats (WKY) served as controls. Animals were obtained from both Taconic Farms (Germantown, NY, USA) and Harlan Sprague Dawley (Indianapolis, IN, USA). All animals were anesthetized with sodium thiopental (100 mg/kg) intraperitoneally, with supplemental anesthetic (20% of initial dose) administered intramuscularly as needed during the experimental period. The trachea was intubated to ensure a patent airway, and the right femoral artery was cannulated for measurement of systemic arterial blood pressure with a Gould P23Db pressure transducer (Cleveland, OH, USA). The animal was placed on a thin Lucite chamber through which warm (34–37 °C) water was circulated to maintain a 37 °C rectal temperature. The right spinotrapezius muscle was prepared for microscopic observation as described by Gray.18 Throughout the operation and subsequent experimental period, the muscle was superfused with a bicarbonate-buffered physiological solution (119 mM NaCl, 25 mM NaHCO3, 6 mM KCl, 3.6 mM CaCl2) warmed to 35 °C and equilibrated with a mixture of 95% N2, 5% CO2 (pH = 7.35–7.40). The animal was placed on its left side, and the right spinotrapezius muscle was reflected away from the body wall. The muscle was extended with the ventral surface facing up and draped over a circular Lucite pedestal. The muscle was secured in this position by four 6-0 silk ligatures tied along its lateral edge. Throughout these manipulations, care was taken to keep the muscle at a normal resting length and width. In rats in which the muscle was inadvertently stretched beyond the resting dimensions, we observed a partial or complete loss of arteriolar tone. Data were not collected from such preparations.

A Lucite chamber was placed over the muscle and sealed to the underlying pedestal with Dow-Corning stopcock grease (Corning, NY, USA). The physiological saline solution was then allowed to flow through the chamber, with the flow rate adjusted as needed to maintain a solution PO2 of 10 to 15 mm Hg over the muscle. This condition was achieved at rates between 4 and 6 ml/min.

Metal stimulating electrodes made from small suture needles (<0.2 mm diameter) were inserted through the rostral and caudal edges of the muscle at least 10 mm away from the viewing area. The electrodes were connected to a Grass S9 stimulator (Quincy, MA, USA). Square-wave stimulating pulses of 0.2 msec in duration, 3 to 6 V in amplitude, and 1, 2, 4, or 8 Hz in frequency were used to elicit muscle contraction. The stimulus voltage was adjusted to produce the largest possible tissue movement with each contraction. The duration and voltage of the stimulating pulses were chosen because they have been reported to be below the threshold of excitability of skeletal muscle cells and autonomic neurons.19, 20 In addition, Lash and Bohlen have demonstrated that during neuromuscular blockade, stimulation at these parameters has no effect on arteriolar diameter.3 Stimulation of the muscle in the current study produced no changes in heart rate or arterial blood pressure, and contraction was limited to the spinotrapezius muscle.

The muscle was transilluminated with a 100-W quartz-iodine lamp, and infrared and ultraviolet filters were placed in the light path. The microvasculature was visualized using an Olympus BHMJ intravital microscope (New Hyde Park, NY, USA) with a 10 x eyepiece and Nikon (Garden City, NJ, USA) W10 and W20 water immersion objectives (numerical aperture, 0.22 and 0.33, respectively). The microscope was fitted with a Dage-MTI SIT-66 video camera (Michigan City, IN, USA), and the image was viewed on a 17-in. Ikegami video monitor (Tushink, Tokyo, Japan). Total video magni-
Epithelialization was 430 × with the W10 objective and 860 × with the W20 objective. The magnification of the videomicroscope system was calibrated after each experiment using a stage micrometer marked in divisions of 10 and 100 μm. The video image of the micrometer and all vessel images were stored on a Mitsubishi HS-329UR videocassette recorder (Garden City, CA, USA). The arteriolar diameters were measured from the video screen during tape playback.

Vessels were classified according to location within the arteriolar network. Blood is supplied to the spinotrapezius muscle through two or three large (first-order) arterioles. These vessels enter from both the rostral and caudal boundaries of the muscle and in turn give rise to the second-order arterioles, which anastomose to form an arcing network throughout the muscle. The third-order arterioles branch directly from the second-order arterioles.

Tissue P02 was measured with recessed-tip oxygen microelectrodes21 fabricated in our laboratory. Lash and Bohlen21 recently reported that electrodes with outer tip diameters of 4 μm or less frequently break during contraction of the spinotrapezius muscle. Therefore, electrodes with tip diameters sharpened to between 5 and 8 μm were used for this study. Electrodes of this size are strong enough to withstand the forces associated with muscle contraction yet flexible enough to freely move with the contracting tissue. Only electrodes with currents less than 10⁻¹⁰ A at a PO2 of 140 mm Hg were used. Electrodes were calibrated over a PO2 range of 0 to 70 mm Hg immediately before and after each experiment. For calibration, the electrode was placed in a tonometer, as designed by Proctor and Bohlen,22 and the current output was recorded after equilibration with 5 and 10% oxygen gas mixtures. Zero-level PO2 was then determined by placing the electrode tip in an actively respiring yeast mixture attached to the muscle chamber.

Experimental Protocol

After a postsurgical equilibration period that typically lasted 45 to 60 minutes, a vessel was chosen for study, and its location within the arteriolar network was determined to permit classification by branching order. Following a 2-minute control period, the muscle was stimulated to contract at a given frequency for 3 minutes. In preliminary experiments, a 5-minute contraction period was used. However, repeated diameter measurement throughout this period revealed that steady state diameter is reached in all arterioles by the third minute of contraction for both normal and SHR. The contraction period was therefore shortened to 3 minutes to reduce the total demand made on the muscle over the typical 3-hour experimental period. A postcontraction recovery period of at least 5 minutes was allowed before the sequence was repeated for a different contraction frequency. Each vessel was studied at all contraction frequencies (1, 2, 4, and 8 Hz), and the order in which each frequency was used was randomized. The muscle sulfation solution was then changed to one containing 10⁻⁴ M adenosine to elicit maximal vasodilation, and vessel diameter was remeasured. Suffusion with the control solution was then resumed, and the next vessel was chosen for study after normal vascular tone had returned. Data were discarded if the vessel under study did not regain its resting diameter after each period of contractions. The experiment was terminated if mean arterial pressure fell below 90 mm Hg for WKY or 140 mm Hg for SHR.

Parenchymal tissue PO2 was measured near the venous end of capillary networks. Typically, all capillaries perfused by a single terminal arteriole join a small collecting venule that is oriented perpendicular to the capillaries and skeletal muscle fibers. The microelectrode tip was placed 25 to 50 μm from the collecting venule equidistant between two capillaries. Proximity to other vessels was avoided. The tip of the microelectrode was inserted between the skeletal muscle cells at tissue depths ranging from 20 to 50 μm. Tissue PO2 was measured continuously throughout the 2-minute control period, the 3 minutes of muscle contractions at 8 Hz, and the subsequent recovery period. At 30-second intervals throughout the stimulation period, the contractions were stopped for 3 to 5 seconds to confirm that the position of the electrode tip relative to the tissue had not changed and that no tissue or vascular damage had occurred. Data were discarded if damage was present or if the electrode was observed to interfere with capillary perfusion during contractions. Data were also discarded if tissue PO2 did not return to precontraction levels within 5 to 10 minutes after contractions had ceased. Data obtained from electrodes having more than a 5% change in gain during the experiment was discarded as well.

Statistics

All data are expressed as means ± SEM. Comparisons between two dependent or independent sample means were made with paired or unpaired Student's t tests, respectively. Multiple comparisons were made with one-way analysis of variance and the Newman-Keuls multiple-range procedure. Significance for all tests was assessed at a p level below 0.05.

Results

A total of 25 WKY and 27 SHR were successfully evaluated in this study. Mean WKY body weight (325 ± 6 g) was slightly greater than that for SHR (317 ± 6 g), but the difference was not statistically
significant. The mean arterial blood pressure was 100.4 ± 1.5 mm Hg for WKY and 157.4 ± 3.3 mm Hg for SHR.

Arteriolar Diameter

Table 1 lists the mean arteriolar inner diameters measured at rest and immediately following contraction periods for WKY and SHR. The maximum diameters as measured in the presence of 10^{-4} M adenosine are also included. For any given branching order, resting arteriolar inner diameter in hypertensive animals was not significantly different (p < 0.05) from that in normotensive animals. In addition, the diameters of comparable arterioles in WKY and SHR immediately following 3 minutes of muscular contractions were remarkably similar for a given contraction frequency. The only significant difference between the two groups was in the second-order arterioles after 4-Hz contractions, where the vessels in SHR were smaller than those in WKY. At all other contraction frequencies for this order of arteriole and at all contraction frequencies for the first-order and third-order arterioles, the mean post-contraction diameters for SHR and WKY were not significantly different. Similarly, there was no significant difference between WKY and SHR in mean diameters during adenosine-induced vasodilation for any arteriolar branching order.

The responses of arterioles to contractions and adenosine application are expressed as percentage of control diameter in Figure 1. The normalization was used to compare the relative ability of different branch order arterioles to dilate in a given strain of rats as well as to compare the relative dilation of equivalent types of arterioles in normal and hypertensive rats. In both normotensive and hypertensive animals, muscle contractions caused significant dilation of all branch orders except for the first-order vessels in WKY at 1 Hz. In agreement with the absolute diameter data, for a given branch order of arterioles, we found no significant difference between WKY and SHR in percent dilation during muscular contractions or suffusion with adenosine. In both groups, the magnitude of dilation in any arteriolar branch order was progressively greater as the contraction frequency was increased up to 8 Hz. In addition, for any given contraction frequency above 1 Hz, the response increased distally in the arteriolar network, with the third-order arterioles consistently showing the greatest percent dilation in both normal and hypertensive rats. Contraction at 8 Hz and application of 10^{-4} M adenosine caused the same degree of dilation in all vessels except for the second-order arterioles in SHR (see Figure 1 and Table 1).

As temporary arteriolar closure is a characteristic of the vasculature in some muscles of the SHR, the third-order and fourth-order arterioles open to blood flow in the field of view were monitored at rest, immediately after 3 minutes of muscle contraction (8 Hz), and during maximum vasodilation with topically applied adenosine. Opening of previously closed third-order or fourth-order arterioles by either the application of adenosine or vigorous muscle contractions occurred in less than 5% of the observations in both WKY and SHR.

Tissue Oxygen Tension

The PO2 of the solution bathing the muscle surface was measured periodically throughout each experiment. At middepth in the solution layer approximately 1 mm above the muscle surface, the PO2 averaged 14.1 ± 0.9 mm Hg. Tissue PO2 was always measured at the venous end of the capillaries and as far from arterioles as possible. The distributions of resting tissue PO2 in normotensive and hypertensive animals are shown in Figure 2. Mean tissue PO2 for WKY was 25.5 ± 1.3 mm Hg, with measured values ranging from 7 to 46 mm Hg. Mean tissue PO2 for SHR was 26.1 ± 2.1 mm Hg, with measured values ranging from 4 to 53 mm Hg. The mean tissue PO2 for hypertensive animals was not significantly different from that for the normotensive controls (p < 0.05). However, it is evident from the data in Figure 2 that there was greater variation in resting tissue PO2 in the hypertensive than in the normal animals. The coefficient of variation (100 × SD/mean) was 51.4% for the SHR compared with 36.8% for the WKY.

### Table 1. Arteriolar Inner Diameters at Rest, Immediately After Muscle Contractions, and When Maximally Dilated by Adenosine (10^{-4} M)

<table>
<thead>
<tr>
<th>Vessel</th>
<th>Diameter (μm)</th>
<th>Rest</th>
<th>1 Hz</th>
<th>2 Hz</th>
<th>4 Hz</th>
<th>8 Hz</th>
<th>ADO</th>
</tr>
</thead>
<tbody>
<tr>
<td>1A</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>WKY (n = 7)</td>
<td>42.3 ± 7.5</td>
<td>45 ± 12.5</td>
<td>50.1 ± 11.2</td>
<td>66 ± 13.1</td>
<td>68.6 ± 13.5</td>
<td>77.3 ± 12.2</td>
<td></td>
</tr>
<tr>
<td>SHR (n = 7)</td>
<td>42.3 ± 3.6</td>
<td>40.4 ± 4.6</td>
<td>44.7 ± 4.3</td>
<td>56.7 ± 5.3</td>
<td>67.9 ± 6.9</td>
<td>82.3 ± 10.0</td>
<td></td>
</tr>
<tr>
<td>2A</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>WKY (n = 9)</td>
<td>23.2 ± 4.4</td>
<td>24.1 ± 3.9</td>
<td>26.9 ± 4.3</td>
<td>37.2 ± 4.9</td>
<td>36.2 ± 4.4</td>
<td>42.5 ± 3.5</td>
<td></td>
</tr>
<tr>
<td>SHR (n = 9)</td>
<td>16.8 ± 1.7</td>
<td>22.2 ± 3.1</td>
<td>24 ± 3.3</td>
<td>27.2 ± 2.1*</td>
<td>30.2 ± 3.1</td>
<td>43.9 ± 2.9</td>
<td></td>
</tr>
<tr>
<td>3A</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>WKY (n = 10)</td>
<td>10.2 ± 1.3</td>
<td>10.2 ± 1.1</td>
<td>13.3 ± 1.5</td>
<td>18.3 ± 1.8</td>
<td>20.8 ± 2.2</td>
<td>23 ± 2.6</td>
<td></td>
</tr>
<tr>
<td>SHR (n = 10)</td>
<td>10.3 ± 0.8</td>
<td>11.5 ± 1.0</td>
<td>13.0 ± 0.9</td>
<td>16.3 ± 1.4</td>
<td>22.1 ± 2.3</td>
<td>23.4 ± 2.7</td>
<td></td>
</tr>
</tbody>
</table>

Values are means ± SEM. 1A, 2A, and 3A denote first-order, second-order, and third-order arterioles, respectively. Muscular contractions were at 1, 2, 4, and 8 Hz. ADO denotes diameter under 10^{-4} M adenosine.

*p < 0.05, compared with corresponding value in WKY.
**FIGURE 1.** Arteriolar diameter (expressed as percentage of control) following 3 minutes of muscle contraction at frequencies of 1, 2, 4, and 8 Hz and during suffusion with $10^{-4} \text{M}$ adenosine (ADO). Top panel: First-order (supply) arterioles; middle panel: second-order (arcade) arterioles; bottom panel: third-order (transverse) arterioles. Data are expressed as means ± SEM. *n* = number of vessels studied in each branching order. WKY data obtained from 15 animals; SHR data obtained from 16 animals. For each branching order, the SHR value was not significantly different from the corresponding WKY value for any of the treatments.

Tissue $P_O_2$ during control, 8-Hz muscular contractions, and the first 2 minutes of the ensuing recovery period are shown for both WKY and SHR in Figure 3. Whereas tissue $P_O_2$ fell quickly in both groups with the onset of contractions, the decrease in $P_O_2$ was greater in the hypertensive than in the normal animals. Thirty seconds into the contractions period, tissue $P_O_2$ had reached $48.4 \pm 12.2\%$ of control (9.6 ± 1.8 mm Hg) in WKY and 21.3 ± 2.3% of control (4.8 ± 1.5 mm Hg) in SHR. The difference between WKY and SHR both in terms of percentage of control and actual $P_O_2$ values at this time was statistically significant (*p* < 0.05). In normotensive animals, tissue $P_O_2$ began to return toward control after 90 seconds of contractions (time = 210 seconds in Figure 3) and reached what appeared to be a steady state level at 150 seconds into the contraction period (time = 270 seconds). At the end of the contraction period, tissue $P_O_2$ had returned to 81.9 ± 12.7% of control in normal animals and was not significantly different from control. In hypertensive animals, tissue $P_O_2$ recovered to about 40% of control by the end of the first minute of contractions (time = 180 seconds), with no significant further recovery. At the end of the contraction period, tissue $P_O_2$ for the hypertensive rats averaged only 41.2 ± 13.0% of control and was significantly less than the end-contraction value for normotensive rats. There was also a difference between the two groups in the pattern of tissue $P_O_2$ restoration during the recovery period. Following the cessation of contractions in WKY, tissue $P_O_2$ usually rose quickly above control and remained elevated for 2 or 3 minutes before returning to the precontraction tissue $P_O_2$. In contrast, such a $P_O_2$ "overshoot" occurred in only one SHR. In this group, tissue $P_O_2$ typically returned directly to the control $P_O_2$ during recovery.

**FIGURE 2.** Distribution of $P_O_2$ values measured at the venous end of capillary networks in the resting spinotrapezius muscle of WKY (top panel) and SHR (bottom panel). *n* = number of individual measurements. WKY data obtained from 10 animals; SHR data obtained from 12 animals. The mean tissue $P_O_2$ for SHR was not significantly different from that for WKY.

**Discussion**

The results of this study do not support the initial hypothesis that the hypertensive process compromises the ability of local vascular control mechanisms to dilate the arterioles in the spinotrapezius muscle. Not only are the inner diameters of arterioles of comparable branch order equivalent at rest in adult normal rats and SHR, but also the vessels dilated to comparable actual and percentage-of-
control diameters for every frequency of contraction used (see Figure 1 and Table 1). The observation of normal arteriolar diameters in the spinotrapezius muscle of SHR is in agreement with other studies that report little difference in resting arteriolar diameters between normal rats and SHR in this muscle. However, to our knowledge, the observation in this study that a physiological increase in metabolism causes normal arteriolar dilation in SHR has not been previously reported.

There is ample evidence of substantially elevated microvascular pressure in the arterioles of the spinotrapezius muscle of SHR and in virtually every other vascular bed studied to date in adult SHR. Based on this information, it is reasonable to assume that for the arterioles of hypertensive animals to maintain a normal resting diameter (see Table 1), either a greater force development by the vascular smooth muscle or a larger amount of smooth muscle (or both) is required. On the other hand, the higher microvascular pressure in the spinotrapezius muscle of the SHR may assist the dilation of the arterioles when local control mechanisms suppress constrictor tone as tissue metabolism is increased. Such an effect could play a role in offsetting the increased constrictor influences associated with hypertension and could therefore be at least partly responsible for the near normal dilation of SHR arterioles.

The finding of virtually identical mean resting tissue \( \text{PO}_2 \) in normal and hypertensive rats was not surprising (see Figure 2). Earlier reports that the resting blood flow in rat hindlimb and spinotrapezius muscles is near normal in adult SHR suggest that at least the blood flow contribution to tissue oxygenation would favor a near-normal mean tissue \( \text{PO}_2 \). In addition, Gray has shown that the anatomical capillary density in the SHR spinotrapezius muscle is normal in animals slightly younger than those used in the current study. However, as is demonstrated by the data in Figure 2, the distribution of tissue \( \text{PO}_2 \) at rest was substantially broader in hypertensive rats than in normotensive rats. The larger variation in SHR might be explained by either a greater percentage of temporarily closed small arterioles (functional rarefaction) or a frank loss of small arterioles (anatomical rarefaction). However, Engelson et al. have actually reported a denser network of arcade (second-order) arterioles and almost twice as many transverse (third-order) arterioles per unit tissue volume in the spinotrapezius muscle of adult SHR as in normal rats during maximal vasodilation. Therefore, anatomical rarefaction of arterioles does not seem to explain the wider distribution of tissue \( \text{PO}_2 \) values in hypertensive than in normotensive rats. In both groups, we looked for but did not find any evidence of significant functional rarefaction of small arterioles. Muscle contractions at frequencies of up to 8 Hz or maximal vasodilation with adenosine opened previously closed arterioles only on very few occasions. This finding is markedly different from observations made in the cremaster and gracilis muscles, in which temporary closure of small arterioles occurred in hypertensive rats at about twice the frequency as that in normotensive rats. The absence of functional rarefaction of small arterioles in the present study cannot be attributed to low vascular tone. Upon abolition of vascular smooth muscle tone with adenosine, the vessels studied in both normal and hypertensive rats dilated proportionately as much or more than arterioles observed in other studies on the spinotrapezius and other rat muscle vascular beds. The greater variation of resting tissue \( \text{PO}_2 \) about a normal mean in SHR therefore does not appear to be the result of temporary or permanent closure of small arterioles or a frank loss of capillaries.

One remaining possibility is that differences in the number of actively perfused capillaries may be responsible for this variation. Prewitt et al. have reported that the density of perfused capillaries in the resting adult gracilis muscle with normal vascular tone is approximately 60% lower in SHR than in WKY. To our knowledge, data of this type have not been obtained for the spinotrapezius muscle with normal vascular tone. However, the lower incidence of functional arteriolar rarefaction in the spinotrapezius as compared with the gracilis muscle would suggest that functional capillary rarefaction due solely to temporary arteriolar closure is rare in this muscle.
half that at rest but was restored to about 80% of control once the functional dilation was fully established (see Figure 3). In contrast, tissue PO2 in the SHR demonstrated substantially less recovery during 8-Hz contractions, returning on the average to about 40% of the control PO2 after an initial fall to about 20% of control. The absence of a normal recovery of tissue PO2 during muscle contractions was somewhat surprising. The actual diameters of arterioles of comparable branch order were equivalent both at rest and during muscle contractions in normal and hypertensive rats (see Table 1). In addition, the responses (percentage of control diameter) for individual arterioles were equivalent in normal and hypertensive rats at virtually every contraction frequency used (see Figure 1). This lack of relationship between arteriolar diameter and tissue PO2 highlights the complex nature of the numerous factors that determine tissue PO2. The lower steady state contraction PO2 in the SHR might be attributable to structural or hemodynamic consequences of hypertension arising either proximally or distally to the vessels studied here. A recent study by Segal and Duling32 has established that the small feed arteries proximal to the microcirculation contribute substantially to blood flow regulation in the hamster cremaster and gracilis muscles. This may be true for the rat spinotrapezius muscle as well. Pressure in the largest arterioles of the rat spinotrapezius muscle is only about 60% of systemic arterial pressure,11 indicating an important contribution of the small feed arteries to the muscle’s total vascular resistance. Schmid-Schonbein et al.31 have reported that some of these feed arteries have thickened vascular walls in the SHR. These anatomical changes could prevent a normal increase in luminal diameter during contraction, thereby limiting the blood flow increase to the muscle. Alternatively, the lower contraction PO2 in the SHR could reflect an impaired dilation of the terminal (fourth-order) arterioles, which we did not study. An abnormal response at this level could compromise tissue oxygen delivery during contraction by preventing a normal increase in the number of perfused capillaries.

It is also possible that the difference between WKY and SHR in steady state contraction PO2 is completely unrelated to differences in vascular behavior. At any given level of contraction, skeletal muscle fibers in the SHR may consume more oxygen than those in WKY. To our knowledge, there are no published data addressing this possibility.

Whatever the cause, the lower tissue PO2 in SHR during 8-Hz muscle contractions is of debatable functional significance. Based on in vitro data obtained from isolated mitochondria, Duling32 has estimated critical tissue PO2 to be in the range of 2 to 6 mm Hg. In both WKY and SHR, the steady state tissue PO2 during 8-Hz contractions exceeded this estimated range. In both groups, the steady state arteriolar diameter during 8-Hz contraction typically ranged between 82 and 94% of the maximal diameter under adenosine (see Table 1). Because of the limited ability for additional dilation, larger increases in tissue metabolism caused by contractions at higher frequencies would undoubtedly cause a greater reduction in tissue PO2.

These considerations aside, the key issue of this study is that, during an increase in tissue metabolism great enough to cause near-maximal dilation, the arterioles in the spinotrapezius muscle of SHR exhibit a normal ability to dilate. Although normal arteriolar dilation has not been found in all muscles of the SHR,33, 34 it may be germane to this issue to point out that, in humans with mild to severe essential hypertension, there are rarely any symptoms of vascular dysfunction (such as pain or impaired organ function) during activities that require increased blood flow to support elevated tissue metabolism. The on-demand capability for vasodilation through local metabolic control mechanisms may therefore be sufficiently preserved to prevent a marked deterioration in microvascular nutritional characteristics during hypertension.

References


19. Folkow B, Halicka HD. A comparison between “red” and “white” muscle with respect to blood supply, capillary surface area and oxygen uptake during rest and exercise. Microvasc Res 1968;1:1–14


Arteriolar diameter and tissue oxygen tension during muscle contraction in hypertensive rats.
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