Effects of Potassium Ion on the Microcirculation of the Hamster

By Brian R. Duling

ABSTRACT

The vasoactive properties of potassium were assessed in the microcirculation of the hamster cremaster muscle and the muscular and epithelial portions of the hamster cheek pouch. Tissues were transilluminated and suffused with a physiological salt solution whose potassium concentration varied from 0 to 20 mM. Vessel diameters were measured and normalized as a percent of the control diameter (± SE) observed during exposure to 4.7 mM K+. Altering the potassium concentration in the suffusion solution caused a transient vascular response. The peak changes in the vascular diameter of the arterioles supplying striated muscle varied directly with the suffusion solution potassium concentration from a minimum of 78 ± 3% in 0 mM K+ to 155 ± 15% in 15 mM K+. Vascular diameter increases were sustained for the full 5-minute test period only in 15 mM K+. In the epithelial portions of the cheek pouch, only the constrictor component of the potassium response was observed. The data indicate that potassium is sufficiently potent to participate in initiating functional hyperemia in striated muscle and might cause as much as a 6.3-fold increase in flow. Functional hyperemias exceeding approximately 3 minutes cannot be due to potassium ion, since the dilation induced by this agent is transient.

I have therefore studied the responses of the arterioles of the hamster cremaster muscle and cheek pouch to changes in the potassium concentration of a superfusion solution. The aims of the experiment were to: (1) establish dose-response relations for in situ arteriolar smooth muscle, (2) determine the temporal stability of the responses, and (3) compare responses in two different tissues, epithelium and striated muscle.

Methods

Male golden hamsters weighing 90–120 g were studied. They were anesthetized with sodium pentobarbital (60 mg/kg, ip), and their tracheas and femoral veins were cannulated. The cheek pouches were prepared as described by Duling (33), and the cremaster muscles were prepared as described by Baez (34). Tissue surfaces were suffused with a bicarbonate-buffered salt solution whose millimolar composition was: NaCl 131.9, KCl 4.7, CaCl2 2.0, MgSO4 1.2, and NaHCO3 18. After equilibration with 5% CO2, the solution pH was 7.35. The temperature of the microvessel preparations was maintained at a normal temperature for the cremaster muscle of 33.5°C by heating the suffusion fluid. The rectal temperature of the hamster was maintained at 37.5°C.

The oxygen tension (Po2) and the carbon dioxide tension (Pco2) of the suffusion solution were normally maintained at levels of 10–15 mm Hg and 32–35 mm Hg, respectively, by equilibrating the solution in the supply reservoir with 95% N2-5% CO2. When desired, the equilibration gas could be changed to 5% O2-5% CO2-90% N2 to elevate the suffusion solution Po2 and thereby reduce the requirement for tissue oxygen supply via vascular sources. The hamsters were allowed to stabilize for 30–60 minutes after preparation. Vessel reactivity, tone, and maximum vessel diameter were assessed by application of a drop of adenosine (1 × 10−5 M) to the suffusion solution. Vessels suitable for study showed brisk dila-
tions and returned to base-line diameters within 1 minute.

Internal vascular diameter measurements were made using a Vickers image-splitting eyepiece in conjunction with objective lenses between 20x and 50x magnification.

Early experiments were performed by applying potassium iontophoretically and topically using Pasteur pipettes. Iontophoretic application was found to be unsatisfactory because of the high currents required and the exceedingly large gradients generated at the tip of the micropipettes (35). Topical application from Pasteur pipettes also gave erratic results due to temperature and oxygenation differences between the control solution and the test solution. Low temperatures caused dilation and high PO2 levels caused constrictions. These methods were replaced by one in which two separate heated, aerated reservoirs were used. In preliminary experiments, it was observed that large responses to potassium were obtained only when the fluid delivery lines were short. This finding is probably related to the fact that the magnitude of the response of vascular smooth muscle to potassium is related to the rate of change of the ion concentration (29).

Desired potassium concentrations were obtained by mixing solutions containing 0 and 20 mM K+ in appropriate ratios; sodium and potassium were varied reciprocally to maintain osmolality constant. This procedure was possible because of the relative insensitivity of vascular smooth muscle to sodium (29). Prior to each experiment, the system was tested by switching between two reservoirs filled with identical solutions and ascertaining that no responses were induced by the switching operation itself. This method required that great care be taken to ensure that no heat loss or gas flux occurred across the tubing in the reservoirs prior to switching.

**Results**

**CREMASTER MUSCLE**

Elevations of potassium concentration above 4.7 mM caused vasodilation, and reductions of potassium concentration caused vasoconstriction. The changes were both dose and time dependent. Figure 1 shows a typical record of diameter changes obtained on a 12μ arteriole during exposure to different potassium concentrations. At zero time, the potassium concentration was changed from 4.7 mM to the value indicated in the upper right corner of each section of the figure. Decreases to 0 and 2 mM K+ from 4.7 mM caused small, transient constrictions. When 0 mM K+ was applied, there was usually a return to the control diameter within 2 minutes; this change was frequently followed by a marked vasodilation as shown at the arrow in the upper left section. Usually, although not always, this secondary vasodilation was accompanied by contracture or fasciculation of the striated muscle fibers. Therefore, it is probable that the dilation was caused by increased muscle metabolism. The secondary dilation was not seen during exposure to 2 mM K+.

When the solution potassium level was elevated above 4.7 mM, the vessel dilated, reached a peak diameter within 2 minutes, and then returned slowly toward the control size. In 15 mM K+, the response was frequently more sustained than that shown in Figure 1. Also, about one-third of the vessels showed some oscillatory behavior associated with the change in potassium concentration, as shown in the later portion of the bottom section of Figure 1.

Exposure to potassium levels between 15 and 25 mM caused highly variable vascular responses (both dilations and constrictions) which were frequently not reversible during observation periods of 30 minutes. Fasciculations were commonly induced in the striated muscle, and it was not possible to determine how much of the vasomotor effect was due to this phenomenon and how much was due to an effect of potassium on the vascular smooth muscle cells. In several cases, transition from 4.7 to 20 mM K+ appeared to exert no effect on the vessels. When the solution was returned to 4.7 mM K+ after a 5-minute exposure, however, a rapid and profound constriction ensued which frequently caused closure of all of the vessels in the field of view.

Attempts were made to correlate the magnitude of the vascular responses with vessel sizes and with vessel branching order. Over the range of 4 to 34μ, no size correlation could be seen. Therefore, all data were combined and are presented in Table 1 as the averaged, normalized responses expressed as

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TABLE 1
Effect of Altered Potassium Concentration on Cremaster Muscle Arterioles

<table>
<thead>
<tr>
<th>Time (minutes)</th>
<th>Potassium concentration (mM)</th>
<th>0.0</th>
<th>2.0</th>
<th>4.7</th>
<th>8.0</th>
<th>10.0</th>
<th>15.0</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.5</td>
<td>78 ± 3*</td>
<td>97 ± 8</td>
<td>100</td>
<td>127 ± 8*</td>
<td>119 ± 5*</td>
<td>134 ± 14*</td>
<td></td>
</tr>
<tr>
<td>1.0</td>
<td>86 ± 10</td>
<td>93 ± 6</td>
<td>100</td>
<td>132 ± 9*</td>
<td>121 ± 5*</td>
<td>144 ± 10*</td>
<td></td>
</tr>
<tr>
<td>2.0</td>
<td>97 ± 9</td>
<td>101 ± 5</td>
<td>100</td>
<td>127 ± 11*</td>
<td>115 ± 5*</td>
<td>155 ± 15*</td>
<td></td>
</tr>
<tr>
<td>5.0</td>
<td>131 ± 16</td>
<td>99 ± 4</td>
<td>100</td>
<td>116 ± 14</td>
<td>103 ± 4</td>
<td>138 ± 16*</td>
<td></td>
</tr>
</tbody>
</table>

Data are normalized as a percent of the diameter in 4.7 mM K⁺ solution. All values are means ± SE, and the number of experiments is given in parentheses. * Significantly different from 100%, P < 0.05.

CHEEK POUCH

The effects of potassium on the microvessels of the epithelial portion of the hamster cheek pouch were also studied. Table 2 illustrates the results of these experiments. The initial responses following reductions in potassium concentrations were identical in the cremaster muscle and the cheek pouch—a transient constriction with a return to baseline within a few minutes. Surprisingly, however, I was not able to induce any significant vasodilation in the cheek pouch vessels by elevating the potassium concentration. The difference between epithelial portions of the cheek pouch and the cremaster muscle are particularly obvious if the data in Tables 1 and 2 at 1 minute are compared. The similarities of the responses during exposure to low potassium concentrations and the differences during exposure to high potassium concentrations are obvious. The striking absence of the vasodilatory component in the potassium response of the epithelial vasculature might be due either to some difference between cremaster muscle and cheek pouch or to a basic difference between epithelial vessels and striated muscle vessels. The portions of the cheek pouch more proximal to the head contain striated muscle fibers, and we took advantage of this fact to separate the two possibilities. The responses of the arterioles in the proximal (muscular) and distal (epithelial) portions of the cheek pouch were compared 1 minute after exposure to 10 mM K⁺ (Table 2). Ten mM K⁺ applied to the muscular portion of the cheek pouch induced a dilation whose magni-

TABLE 2
Effect of Altered Potassium Concentration on Cheek Pouch Arterioles

<table>
<thead>
<tr>
<th>Time (minutes)</th>
<th>Potassium concentration (mM)</th>
<th>Nonmuscle</th>
<th>Muscle</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.5</td>
<td>81 ± 8*</td>
<td>89 ± 7</td>
<td>100</td>
</tr>
<tr>
<td>1.0</td>
<td>87 ± 1*</td>
<td>90 ± 5</td>
<td>100</td>
</tr>
<tr>
<td>2.0</td>
<td>91 ± 7</td>
<td>97 ± 3</td>
<td>100</td>
</tr>
<tr>
<td>5.0</td>
<td>125</td>
<td>102 ± 4</td>
<td>100</td>
</tr>
</tbody>
</table>

Data are normalized as a percent of the diameter in 4.7 mM K⁺ solution. All values are means ± SE, and the number of experiments is given in parentheses. * Significantly different from 100%, P < 0.05.
tude was larger than that induced by any dose of potassium in the epithelial portion of the cheek pouch and approximated that seen in the cremaster muscle with 15 mM K⁺.

Oxygen has been reported to alter the response of the peripheral vasculature to potassium (19, 20, 26). Since the solution Po₂ was rather arbitrarily set at low levels, the effect of potassium at higher solution Po₂ levels was also investigated. The solution reservoir was bubbled with 5% O₂-5% CO₂-90% N₂, this procedure raised the Po₂ in the solution over the tissue surface to an average of 47 mm Hg. Diameter measurements made 2 and 5 minutes after the potassium change are shown in Table 3. Data in this table are presented as absolute diameter measurements, since the size range of vessels studied was small. The mean diameter of the vessels in 4.7 mM K⁺ was decreased 29% by elevating the solution Po₂, but the absolute change in diameter in response to potassium was unaffected by altering the Po₂ in the suffusion solution.

Discussion

The necessary and sufficient conditions for demonstrating that potassium is active in blood flow regulation during functional hyperemia are: (1) that potassium concentrations in the environment of the smooth muscle rise as fast or faster than the vascular resistance changes, (2) that vascular smooth muscle of the arterioles are capable of giving a sufficiently large response to potassium to cause the observed changes in flow, and (3) that the smooth muscle show a temporal response pattern compatible with the observed potassium concentration and resistance changes. My data have direct bearing on the latter two of these three requirements.

After an initial rise at the onset of exercise, the venous concentration of potassium either declines during prolonged exercise (7) or remains constant (8). Thus, the decay of the potassium-induced vasodilation which we observed (Tables 1 and 2) and that which other investigators have observed inperfused tissues (10) and isolated vessels (26) clearly exclude the participation of this ion in the sustained phase of functional hyperemia.

There may, however, be two separable components to exercise hyperemia, an early transient phase and a later sustained phase (36). Moreover, there seems to be a good correlation between the early phase of functional hyperemia and the estimated tissue potassium levels (37), Mohrman and Sparks (37) and others (10, 12) have therefore proposed that this ion is important in initiating functional hyperemia but that some other substance is required to sustain the hyperemia. My data support this concept in that they show that potassium can produce a dilation approximating that seen in exercise within the time required. In Table 4 the diameter changes measured in my experiments have been translated into conductance changes for perfused tissues, assuming that conductance varies with the fourth power of the diameter. Conductances were calculated from the fourth power of the mean change in diameter (column 3) or from the mean of the fourth power of the individual diameter changes (column 4).

### Table 3

<table>
<thead>
<tr>
<th>Reservoir Po₂</th>
<th>0 mm Hg</th>
<th>35 mm Hg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time (minutes)</td>
<td>0 mm Hg</td>
<td>35 mm Hg</td>
</tr>
<tr>
<td>2</td>
<td>3.3 ± 1.3</td>
<td>5.1 ± 1.0</td>
</tr>
<tr>
<td>5</td>
<td>0.5 ± 0.9</td>
<td>4.1 ± 1.3</td>
</tr>
</tbody>
</table>

All values are means ± se for 12 experiments. Changing from 5% CO₂-96% N₂ to 5% O₂-5% CO₂-90% N₂ in the suffusion solution caused a 29% decrease in mean vascular diameter from 17.9 ± 1.8µ to 11.1 ± 1.6µ. These data reflect the increases in diameter induced by potassium.

### Table 4

<table>
<thead>
<tr>
<th>[K⁺] (mM)</th>
<th>% Change in diameter*</th>
<th>Conductance of mean†</th>
<th>Mean of conductances‡</th>
</tr>
</thead>
<tbody>
<tr>
<td>8</td>
<td>132 ± 9 (6)</td>
<td>264.2 ± 24.8</td>
<td>353.4 ± 140.1</td>
</tr>
<tr>
<td>10</td>
<td>124.8 ± 9.9 (23)</td>
<td>242.5 ± 19.2</td>
<td>540.0 ± 250.0</td>
</tr>
<tr>
<td>15</td>
<td>145.8 ± 7.9 (20)</td>
<td>451.8 ± 24.5</td>
<td>627.0 ± 172.0</td>
</tr>
</tbody>
</table>

All values are means ± se.

* Peak values were at 1, 1, and 2 minutes for 8, 10, and 15 mM K⁺, respectively. Data for 10 and 15 mM K⁺ in 0-mm Hg Po₂ solution shown in Tables 1-3 were combined, and the total number of experiments is indicated in parentheses.

† This value is calculated from the data in column 2 by raising the diameter change to the fourth power. The standard deviations were calculated according to σ = (σdiameter change)² × (diameter change)² as shown by Bevington (47).

‡ Conductances were calculated for each observation, and the value shown is the mean for the individual values.

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though the conductance changes calculated by the latter method are substantially larger, using either method the dilations produced by potassium correspond well with the vascular responses occurring with modest to heavy exercise (6). Only when conductance is calculated as the sum of individual observations is the potassium-induced hyperemia large enough to produce dilations equivalent to maximum exercise. Thus, potassium ion appears to be capable of inducing a substantial fraction of the dilation observed during modest exercise.

It remains to be shown that potassium levels in the environment of the smooth muscle cells rise to sufficiently high levels during exercise to induce relaxation; at this time there are no precise data available on interstitial potassium concentrations during hyperemias of more than a few seconds duration. However, interstitial fluid potassium concentration can be estimated if one knows arterial and venous potassium concentrations, flow, and the permeability–surface area product of the tissue; fortunately, such measurements are available during rest and exercise for the dog gracilis muscle (7, 20, 38). These data have been used to estimate interstitial potassium levels, and the calculated values are shown in Table 5. There is some question as to the appropriate value for the permeability–surface area product, since it increases during exercise, but a range between a resting value of 4.2 and a contracting value of 12.6 seems appropriate based on the findings of Sheehan and Renkin (38). Thus, the two columns in Table 4 for estimated interstitial potassium concentration should be reasonable upper and lower bounds on potassium during exercise as long as one considers only a simple model with a homogeneous capillary population and interstitial fluid potassium distribution. (Other assumptions would only increase the disparity between venous and interstitial potassium concentrations.) It is apparent from Table 5 that the interstitial potassium concentrations are in all cases higher than the venous level, and at all frequencies above 1/sec they approach those used in the experiments reported in the present paper. Note that, at the same time, venous potassium levels are substantially below those needed to induce vasodilations as large as those seen during functional hyperemia. The comparison between my data and these calculations is particularly impressive in view of the fact that the control tissue levels of potassium in the dog gracilis muscle were probably 1–1.5 mM below those used in my experiments, since the concentration of potassium in dog plasma is less than that in hamster plasma. Mohrman and Sparks (37) have performed similar but more elegant calculations estimating tissue concentrations during and immediately after 1-second tetanic contractions in dog skeletal muscle. Their conclusions are qualitatively the same as those just cited.

Interstitial potassium concentrations can also be estimated using potassium microelectrodes. Gebert (1) and Hnik et al. (2) have measured extracellular potassium concentration during functional hyperemia; they observed differences of 2–4 mM between venous and tissue potassium concentrations. That is, they found extracellular levels in the range of 8 to 9 mM. This level is less than the largest calculated value but still high enough to give very substantial increases in flow. In fact, the calculated conductance changes shown in Table 4 at 8 mM K⁺ correlate very well with the observed response of both flow and extracellular potassium concentration reported by Gebert (1) and Hnik et al. (2).

### TABLE 5

<table>
<thead>
<tr>
<th>Stimulus frequency (sec⁻¹)</th>
<th>Plasma flow* (ml/min 100 g⁻¹)</th>
<th>Plasma [K⁺]†</th>
<th>Estimated interstitial [K⁺]‡</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Control</td>
<td>Hyperemia</td>
<td>Artery</td>
<td>Vein</td>
</tr>
<tr>
<td>1</td>
<td>5.4</td>
<td>9.1</td>
<td>3.2</td>
<td>3.6</td>
</tr>
<tr>
<td>5</td>
<td>5.8</td>
<td>20.5</td>
<td>3.2</td>
<td>4.1</td>
</tr>
<tr>
<td>6</td>
<td>6.7</td>
<td>43.7</td>
<td>3.6</td>
<td>4.8</td>
</tr>
<tr>
<td>10–20</td>
<td>5.0</td>
<td>30.8</td>
<td>3.2</td>
<td>5.9</td>
</tr>
</tbody>
</table>

* Plasma flow was calculated from reported blood flow and an assumed hematocrit of 44%.
† Arterial potassium concentration was assumed to equal control venous potassium concentration.
‡ Interstitial potassium concentration was calculated according to the equation \((C_a - C_t)/(C_v - C_t) = 1 - \exp(-PS/Q)\), where \(C_a\), \(C_v\), and \(C_t\) are potassium concentrations in arterial plasma, venous plasma, and interstitial space, respectively, PS is the permeability–surface area product for the capillaries, and Q is the plasma flow.
§ PS values are based on reference 38 and an assumed threefold increase in PS during exercise.

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It is not clear how my data may relate to other types of local blood flow control such as autoregulation and reactive hyperemia, since the literature contains conflicting reports on the tissue and venous levels of potassium during ischemia and anoxia. However, the results do seem consistent to the extent that they indicate that cerebrospinal potassium concentration is elevated during anoxia (9) whereas striated muscle shows little or no elevation of venous potassium concentration during ischemia or anoxia (7, 8). Potassium ion is therefore an unlikely candidate for vasodilation during these manipulations in striated muscle but warrants further study in the brain.

Previous results of experiments on the vascular reactivity to potassium ion are confusing and at times contradictory. Recent evidence indicates that this situation is probably attributable to the fact that the net effect of potassium ion may be the result of several different factors which sum to give a final response. This complexity is highlighted in my data by the difference in the responses of microvessels in the muscular and epithelial portions of the cheek pouch. These two groups of vessels were prepared in identical ways, were covered with the same solution, received identical blood supply, and developed from the same arterial supply. The strikingly different responses of the arterioles in the two regions of the cheek pouch, combined with the observed behavior of the cremaster arterioles, suggest that the difference is somehow due to the presence of striated muscle fibers.

The origin of the diverse responses in the arterioles of epithelial and muscular beds is not known; it might simply reflect the inherent diversity of vascular smooth muscle. However, a consideration of the available literature suggests that the effects of potassium ion are sufficiently varied to permit this degree of variation in response. A change in potassium concentration may produce an effect on arterioles via at least four different mechanisms: two different direct effects on the vascular smooth muscle, an effect secondary to altered catecholamine release from nerve terminals, and, finally, an effect involving a change in the metabolism of the striated muscle cells. The direct effects of elevated potassium concentration on vascular smooth muscle appear to result from two separate actions of this ion: the well-known depolarizing effect and a second, hyperpolarizing effect resulting from stimulation of an electrogenic pump. The net effect of potassium on the smooth muscle cell has, in fact, been interpreted quantitatively as the sum of opposing actions of the ion (27, 30, 39). In support of this concept is the fact that the vascular smooth muscle relaxation induced by potassium can be eliminated or converted to a constriction by the administration of ouabain (10, 12, 25). Furthermore, at least part of the apparent differences among various tissues and preparations seems to be due to the experimental conditions used, since in some preparations consistent smooth muscle relaxations can be induced by potassium in in vitro preparations only when the tissues are incubated in 2 mM $K^+$ (27, 31).

The membrane effects induced by potassium are also time dependent, and this characteristic is an additional source of potential variability in the response to this ion (29, 40). In my experiments, slow changes in potassium ion induced by the use of a long supply line for the suffusion solution (approximately 1 meter) severely blunted the potassium-induced arteriolar dilation and, indeed, eliminated it altogether in many cases. Thus, experiments purporting to show small responses to this ion must be interpreted with regard to the possibility that the rate of change of potassium concentration may have been low due to large volumes or slow mixing in the experimental apparatus.

A possible interaction between potassium and tissue neural elements is suggested by the fact that the accumulation of various tissue amines is altered in a complex fashion by potassium ion (41-43). As an indication that this effect may be a real factor in the microvascular response to potassium, Sudak and Fulton (44) found that the cheek pouch vasculature constricts when the plasma potassium concentration is increased. This finding appears to be in conflict with my findings; however, these authors also found that either adrenalectomy or a-receptor blockade abolished the constrictor effect of potassium and that there was no direct effect at the dose levels they studied. This finding, of course, is in agreement with the observations reported in the present paper on the epithelial portion of the cheek pouch. It should be emphasized that this discussion is not meant to imply that either the dilator or the constrictor component of the response to potassium is solely due to catecholamine release, as there is clearly an effect of potassium which is not blocked by adrenergic blockers and tetrodotoxin (13, 18, 30, 31). In addition to the various possible effects of potassium on arteriolar smooth muscle and nerve terminals, the potential exists that some portion of the responses that I and others have observed is...
secondary to altered striated muscle cell metabolism. Elevations in extracellular potassium ion concentrations cause increases in metabolic rate (Solandt effect). In frog muscle, the threshold for this effect appears to be about 10 ISM K⁺ (45), and the experiments reported in the present paper were performed at solution concentrations in excess of this level. Thus, some part of the dilation resulting from elevated potassium concentration may have been due to metabolic feedback from striated muscle to the vasculature. This effect might well be absent in the epithelial portions of the cheek pouch.

Another possible metabolic component of the net potassium response is the late vasodilation observed during exposure of the cremaster muscle to 0 mM K⁺. The frequent correlation with muscle fasciculation and contracture strongly suggests that the dilation is due to metabolic activity of the striated muscle cells. The induction of a contraction by potassium-free solutions is not unexpected, since Hodgkin and Horowicz (46) observed such behavior in striated muscle and attributed it to a decrease in the muscle cell membrane potassium conductance. A similar mechanism might be responsible for the contraction of vascular smooth muscle subjected to low potassium concentrations (27, 29, 32).

In summary, my findings strongly support the involvement of potassium in the early phase of functional hyperemia and they emphasize the need for more measurements of tissue potassium levels and further study of the mechanism of action of potassium on the vasculature.

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