Stimulation of Renal Sympathetic Activity by Static Contraction: Evidence for Mechanoreceptor-Induced Reflexes From Skeletal Muscle

Ronald G. Victor, Diane M. Rotto, Susan L. Pryor, and Marc P. Kaufman

Static muscular contraction in anesthetized animals has been firmly established to reflexly increase arterial pressure. Although group III and IV muscle afferents are known to be responsible for this reflex pressor response, there is no evidence that the stimulation of muscle mechanoreceptors, many of which are supplied by group III fibers, plays a role in causing this contraction-induced reflex effect. To provide this evidence, we recorded renal sympathetic nerve activity in chloralose-anesthetized cats while contracting the triceps surae muscles. We found that static contraction tripled renal nerve activity within three seconds of its onset, an increase that was abolished by cutting the L6 and S2 dorsal roots. On average, the contraction-induced increase in renal nerve activity was observed 0.8±0.1 seconds after the onset of this maneuver. In addition, intermittent tetanic contractions synchronized renal nerve discharge so that a burst of activity was evoked by each contraction. A similarly synchronized renal nerve discharge was evoked in paralyzed cats by electrical stimulation of the tibial nerve at five times motor threshold, a current intensity that activates group III afferents. We conclude that, in anesthetized animal preparations, mechanoreceptors with group III afferents contribute to the reflex stimulation of renal sympathetic outflow evoked by muscular contraction.
properties of the groups III and IV muscle afferents, we reasoned that stimulation of metaboreceptors during static contraction would evoke a slow and progressive increase in sympathetic activity, whereas stimulation of mechanoreceptors would evoke an abrupt increase in sympathetic discharge beginning at the onset of contraction.

Materials and Methods

General
Cats weighing 1.8–4.0 kg were anesthetized with sodium thiopental (25 mg/kg i.p.) and α-chloralose (60–80 mg/kg i.v. followed by 10 mg/kg i.v. per hour). The right common carotid artery, right jugular vein, and cervical trachea were cannulated. Arterial pressure was measured through the carotid artery cannula connected to a Statham P23ID transducer (Gould Instruments, Cleveland, Ohio). Heart rate was derived from the arterial pressure pulse using a Gould Biotach. Tension generated by the right triceps surae muscles was measured by connecting the calcaneal tendon to a force-displacement transducer (FT-10, Grass Instruments, Quincy, Massachusetts). The lungs were ventilated with room air. Arterial blood gases were measured periodically (Radiometer ABL-3) and were kept within normal limits.

The L₆–S₁ spinal roots were exposed, after which the cat was placed in a Kopf spinal unit. The skin overlying the exposed spinal roots was tied to stainless steel bars to form a pool, which was filled with warm (37°C) mineral oil. The dura was then incised. The right L₆–S₁ ventral roots were identified and cut. Each of the cut peripheral ends of the ventral roots was placed on a separate shielded and grounded stimulating electrode. The right hind limb was clamped to prevent its movement.

Sinoaortic Denervation
Sinoaortic denervation was performed by cutting the aortic depressor nerves at the junction of the superior laryngeal nerves and by stripping the arterial walls in the region of the carotid sinuses. The cervical sympathetic, superior laryngeal nerves, and cervical vagi were also cut. The effectiveness of sinoaortic deafferentation was confirmed by demonstrating the failure of phenylephrine-induced (6–12 µg/kg i.v.) increases in mean arterial pressure (+20 to +40 mm Hg) to decrease heart rate and renal sympathetic nerve traffic.

Recording of Sympathetic Nerve Activity
The left kidney was exposed with a retroperitoneal dissection. The sympathetic nerve branch close to the aorta was dissected free and placed on thin bipolar platinum electrodes and covered with silicone rubber (Wacker Siligel 604) according to the technique of Schad and Seller.¹⁰ In every case, renal sympathetic discharge was shown to be inhibited by the pressor response to phenylephrine infusion (6–12 µg/kg i.v.).

The electrode wires were connected to a high impedance probe (Grass HIP511). Action potentials were amplified 20–50 thousandfold by a Grass P511 bandpass amplifier with a bandwidth of 100 to 1,000 Hz. The filtered neurogram was routed through a storage oscilloscope (model S111, Tektronix, Beaverton, Oregon) and an amplitude discriminator to an audio amplifier. For permanent recording and analysis, the filtered neurogram was routed through a nerve-traffic analyzer (model 66C-2, University of Iowa Bioengineering, Iowa City, Iowa), which counted nerve spikes exceeding a threshold voltage set just above the noise level. This level remained constant throughout the experiment. The nerve spikes were analyzed by an integrator which reset after each 100 spikes. Data were recorded on both a Gould ES-1000 electrostatic recorder and on a Hewlett-Packard (model 7404A, Palo Alto, California) recorder.

Experimental Protocols
After surgery, the cats were allowed to stabilize for at least 1 hour before beginning the experimental protocols. In five cats, we examined the effect of static contraction of the hind-limb muscles on renal nerve activity, arterial pressure and heart rate both before and after cutting the L₆ and S₁ dorsal roots. The hind-limb muscles were statically contracted for 60 seconds by stimulating the cut peripheral ends of the L₆ and S₁ ventral roots (40 Hz; 0.1 msec; 2.5 times motor threshold). The tension developed by the triceps surae muscles was used as an index of the level of static muscular contraction. In three other cats, we examined the effects of static contraction on renal nerve activity, arterial pressure, and heart rate before and after sinoaortic denervation, which removed cardiac and arterial baroreceptor input to the medulla.

In five cats, we recorded renal nerve activity while we caused the hind-limb muscles to contract in an intermittent tetanic (i.e., static) manner. To accomplish this type of contraction, we electrically stimulated the cut peripheral ends of the L₆ and S₁ ventral roots with a 40-Hz train of pulses (0.1 msec) for 500–750 msec. The train of pulses occurred once every 3–4 seconds, and the current intensity of the pulses were 2.5 times motor threshold.

In four cats, one of which was also used in the experiments using intermittent tetanic contraction, we examined the renal nerve response to graded electrical stimulation of afferents in the tibial nerve. We first determined the minimum current intensity, which when applied as a single pulse to the tibial nerve, caused the triceps surae muscles to twitch (i.e., motor threshold). We then paralyzed the cats with gallamine triethiodide (2–4 mg/kg i.v.). Next, the tibial nerve was stimulated with 40 Hz trains of pulses (0.1 msec) once every 3–4 seconds. Each train lasted for 500–750 msec. The current intensity applied to the tibial nerve varied between motor threshold and 140 times motor threshold.
TABLE 1. Responses to Static Contraction Before and After Dorsal Root Section

<table>
<thead>
<tr>
<th>Period (seconds)</th>
<th>RSNA (%)</th>
<th>MAP (mm Hg)</th>
<th>HR (beats/min)</th>
<th>Tension (kg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>30</td>
<td>100</td>
<td>88±12</td>
<td>139±18</td>
<td>0.3±0.2</td>
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<tr>
<td>Contraction</td>
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</tr>
<tr>
<td>3</td>
<td>332±58*</td>
<td>88±12</td>
<td>144±16</td>
<td>3.4±0.3</td>
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<tr>
<td>15</td>
<td>190±28*</td>
<td>113±10*</td>
<td>152±17</td>
<td>3.9±0.4</td>
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<tr>
<td>30</td>
<td>160±13*</td>
<td>118±11*</td>
<td>149±16</td>
<td>3.2±0.3</td>
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<tr>
<td>45</td>
<td>145±13</td>
<td>117±11*</td>
<td>152±17</td>
<td>2.5±0.3</td>
</tr>
<tr>
<td>60</td>
<td>154±16</td>
<td>112±10*</td>
<td>150±17</td>
<td>2.1±0.3</td>
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<tr>
<td>Recovery</td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>15</td>
<td>120±17</td>
<td>90±10</td>
<td>148±18</td>
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<td>30</td>
<td>112±13</td>
<td>86±10</td>
<td>145±17</td>
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Dorsal roots intact

<table>
<thead>
<tr>
<th>Period (seconds)</th>
<th>RSNA (%)</th>
<th>MAP (mm Hg)</th>
<th>HR (beats/min)</th>
<th>Tension (kg)</th>
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<tbody>
<tr>
<td>Control</td>
<td>100</td>
<td>79±11</td>
<td>120±20</td>
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<td>126±9</td>
<td>79±11</td>
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<td>78±11</td>
<td>120±21</td>
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<td>94±11</td>
<td>75±11</td>
<td>119±21</td>
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<td>60</td>
<td>93±9</td>
<td>74±10</td>
<td>120±20</td>
<td>1.7±0.2</td>
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<td>Recovery</td>
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</tr>
<tr>
<td>15</td>
<td>114±14</td>
<td>70±9</td>
<td>120±20</td>
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<td>100±18</td>
<td>71±10</td>
<td>120±20</td>
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</table>

Dorsal roots sectioned

Renal sympathetic nerve activity (RSNA), mean arterial pressure (MAP), and heart rate (HR) during control, contraction of the triceps surae muscle, and recovery before and after section of L6 and S1 dorsal roots. Nerve activity is expressed as a percentage of the control value. Entries are mean±SEM for five cats.*Statistically different from control (p<0.05).

Data Analysis

All renal nerve activity was expressed as a percentage of control values because the actual voltages recorded varied greatly from cat to cat. Control values were measured for the 30-second period immediately preceding the onset of contraction and were assigned a value of 100%. Heart rate and arterial pressure responses to contraction are reported as peak effects. All values are expressed as mean±SEM. Repeated measures analysis of variance with the Bonferroni adjustment for multiple comparisons was used to analyze the data. The criteria for statistical significance was p<0.05.

Results

Responses to Static Contraction (Tables 1 and 2 and Figures 1 and 2)

In each of the five cats with intact sinoaortic nerves, static contraction of the triceps surae muscles increased renal sympathetic activity as well as

TABLE 2. Responses to Static Contraction After Sinoaortic Denervation

<table>
<thead>
<tr>
<th>Cat 1</th>
<th>Control period</th>
<th>3</th>
<th>15</th>
<th>30</th>
<th>45</th>
<th>60</th>
<th>Recovery period</th>
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<th>30</th>
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<tbody>
<tr>
<td>RSNA (%)</td>
<td>100</td>
<td>158</td>
<td>128</td>
<td>128</td>
<td>119</td>
<td>113</td>
<td>113</td>
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<tr>
<td>MAP (mm Hg)</td>
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<td>67</td>
<td>92</td>
<td>97</td>
<td>80</td>
<td>75</td>
<td>65</td>
<td>60</td>
<td></td>
</tr>
<tr>
<td>HR (beats/min)</td>
<td>150</td>
<td>150</td>
<td>150</td>
<td>155</td>
<td>155</td>
<td>155</td>
<td>152</td>
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<tr>
<td>Tension (kg)</td>
<td>0.2</td>
<td>2.4</td>
<td>2.6</td>
<td>1.6</td>
<td>1.2</td>
<td>1.0</td>
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<th>Control period</th>
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<th>15</th>
<th>30</th>
<th>45</th>
<th>60</th>
<th>Recovery period</th>
<th>15</th>
<th>30</th>
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<tbody>
<tr>
<td>RSNA (%)</td>
<td>100</td>
<td>177</td>
<td>166</td>
<td>140</td>
<td>130</td>
<td>114</td>
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<td>MAP (mm Hg)</td>
<td>78</td>
<td>78</td>
<td>102</td>
<td>140</td>
<td>145</td>
<td>137</td>
<td>103</td>
<td>93</td>
<td></td>
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<tr>
<td>HR (beats/min)</td>
<td>156</td>
<td>156</td>
<td>156</td>
<td>162</td>
<td>162</td>
<td>162</td>
<td>160</td>
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<tr>
<td>Tension (kg)</td>
<td>0.2</td>
<td>2.9</td>
<td>3.2</td>
<td>3.0</td>
<td>2.4</td>
<td>2.2</td>
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<th>Cat 3</th>
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<tbody>
<tr>
<td>RSNA (%)</td>
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<td>282</td>
<td>159</td>
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<td>102</td>
<td>105</td>
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<td>102</td>
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<tr>
<td>MAP (mm Hg)</td>
<td>117</td>
<td>119</td>
<td>145</td>
<td>148</td>
<td>130</td>
<td>121</td>
<td>96</td>
<td>95</td>
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<tr>
<td>HR (beats/min)</td>
<td>153</td>
<td>153</td>
<td>153</td>
<td>153</td>
<td>153</td>
<td>153</td>
<td>153</td>
<td>153</td>
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<tr>
<td>Tension (kg)</td>
<td>0.4</td>
<td>6.6</td>
<td>7.6</td>
<td>6.7</td>
<td>5.2</td>
<td>4.3</td>
<td>0.3</td>
<td>0.3</td>
<td></td>
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</table>

Mean±SEM

| RSNA (%) | 100 | 206±39 | 151±12 | 124±11 | 117±8 | 111±3 | 100±7 | 100±2 |
| MAP (mm Hg) | 88±15 | 88±16 | 113±16 | 128±20 | 118±20 | 111±19 | 88±12 | 83±11 |
| HR (beats/min) | 153±2 | 153±2 | 153±2 | 157±3 | 157±3 | 157±3 | 155±3 | 154±1 |
| Tension (kg) | 0.3±0.1 | 4.0±1.3 | 4.5±1.6 | 3.8±1.5 | 2.9±1.2 | 2.5±1.0 | 0.3±0.1 | 0.3±0.1 |

Entries are data for renal sympathetic nerve activity (RSNA), mean arterial pressure (MAP), heart rate (HR), and tension during static contraction of the triceps surae muscles in three cats with sinoaortic denervation.
mean arterial pressure. In each of the cats, the renal nerve response to contraction was quite rapid and had a shorter onset latency than that of the pressor response to contraction (0.8±0.1 seconds vs. 6.5±1.0 seconds, respectively; p<0.05). Furthermore, the contraction-induced renal nerve discharge was not sustained throughout the 60-second period of ventral root stimulation. Specifically, the renal nerve response to contraction peaked after 3 seconds of ventral root stimulation and then rapidly declined toward control. By contrast, the pressor response to contraction peaked after 30 seconds of ventral root stimulation and remained well above control levels for the 60-second period. The contraction-induced increases in renal sympathetic activity and arterial pressure were abolished by dorsal root section.

In cats with sinoaortic deafferentation, the renal sympathetic activity displayed the same pattern of discharge as that in the cats with intact baroreceptors, that is, an abrupt burst of sympathetic discharge at the onset of contraction followed by a rapid return to the control level of sympathetic
activity during sustained contraction. Two of the three cats with sinoaortic denervation also demonstrated a burst of sympathetic activity immediately after the offset of contraction.

Responses to Intermittent Tetanic Contractions (Figures 3 and 4)

Intermittent tetanic contraction of the hind-limb muscles caused the renal nerve to discharge synchronously with each increase in tension development. During this synchronization, the overall level of renal sympathetic activity increased by 61 ± 4% and mean arterial pressure increased by 19 ± 4 mm Hg over control (p < 0.05). In addition, the synchronization of the renal nerve discharge appeared to depend on the tension developed by the contractions. The effect occurred when the triceps surae developed 3–5 kg of tension, but it did not occur when this muscle group developed <2 kg of tension. Section of the L6 and S2 dorsal roots abolished the synchronization of renal nerve discharge to intermittent tetanic contraction and abolished the pressor response associated with this maneuver.

Responses to Graded Electrical Stimulation of Afferents in the Tibial Nerve (Figure 5)

In four paralyzed cats, we determined the minimum current needed to reflexly increase renal nerve activity when the tibial nerve was stimulated electrically with intermittent trains of pulses. This minimum current was two times motor threshold in one cat, three times threshold in two cats, and five times threshold in the other cat. The renal nerve responses to electrical stimulation at five times threshold consisted of a burst of impulses having an onset latency of 200 msec. This burst lasted for about 200 msec and was followed by inhibition, lasting about 500 msec. In each of the four cats, the renal nerve response to electrical stimulation of the tibial nerve increased steadily as the current intensity increased. Thus, as the intensity was increased from 5 to 20 to 70 to 140 times threshold, the amplitude of the renal nerve burst steadily increased and the subsequent inhibition became more profound.

Intermittent electrical stimulation of the tibial nerve had graded effects on mean arterial pressure that were dependent on the current intensity used. For example, stimulation at one to three times threshold had no effect on mean arterial pressure, whereas stimulation at five times threshold increased arterial pressure by 7 to 11 mm Hg. Stimulation at 20 times threshold increased mean arterial pressure by 12 to 16 mm Hg.

Discussion

Many previous studies have examined the reflex activation of sympathetic outflow produced by electrical stimulation of peripheral nerves. This study provides the first direct measurements of sympa-
The principal new findings are threefold: 1) static contraction of hind-limb muscles reflexly increases renal sympathetic nerve discharge; 2) the onset of the renal sympathetic response to contraction is rapid, occurring with a latency of less than 1 second, and always precedes the onset of the blood pressure response to this maneuver; and 3) intermittent tetanic contractions synchronize the renal sympathetic discharge to the muscle tension, and this synchronization is a reflex arising from the working hind-limb muscles.

The muscle afferents responsible for causing these reflex changes in renal sympathetic discharge must belong to a subtype of group III and IV afferents. In our experiments, mechanoreceptors supplied mainly by group III fibers were the afferents most likely to be responsible for both the reflex increase in renal nerve discharge evoked by static contraction and the reflex synchronization of this discharge by intermittent tetanic contraction. Mechanoreceptors with group III afferents have been shown to respond vigorously to static contraction with an average onset latency of about 220 msec. Likewise, group III afferents have been shown to discharge synchronously with both intermittent tetanic contractions and repetitive twitch contractions. Unlike group III afferents, most group IV afferents respond too slowly (i.e., with an average latency of 10–15 seconds) to static muscular contraction to be responsible for the rapid onset of the renal nerve discharge observed in our experiments.

The decline in the renal nerve response during 60 seconds of static contraction offered further support for our hypothesis that mechanoreceptors in working muscle played an important role in reflexly evoking this sympathetic response. The most probable explanation for the rapid decline in sympathetic activity despite sustained muscle tension during the first 15 seconds of contraction is that the response is mediated mainly by rapidly adapting mechanoreceptors. The most likely explanation for the further decline in renal nerve activity during the last 45 seconds of static contraction is that muscular fatigue decreased the intensity of the stimulus causing the reflex. When stimulated by static contraction, Group III mechanoreceptors in the triceps surae muscles also demonstrate rapid adaptation and decrease their discharge as the muscle fatigues. An alternative explanation might be that the pressor response to this maneuver stimulated sinoaortic baroreceptors, which, in turn, reflexly decreased sympathetic outflow. This explanation is unlikely because sinoaortic denervation had no effect on the decline in the renal nerve response to static contraction.

The finding that dorsal root section abolished the renal sympathetic nerve response to contraction of the triceps surae provides strong evidence that this sympathetic effect was caused by a reflex arising in these muscles. Furthermore, the reflex response was caused by muscle contraction per se and not by direct electrical stimulation of afferents in the L7 and S1 ventral roots because the current intensity applied to the ventral roots in our experiments (i.e., 2.5 times motor threshold) was far too low to stimulate ventral root afferents and to fire the dorsal horn cells that receive synaptic input from these afferents. Chung et al have demonstrated that the minimum current intensity needed to elicit reflex autonomic responses from electrical stimulation of ventral roots in paralyzed cats is 200 times motor threshold, a level of stimulation that is 80 times greater than that which we used to elicit muscle contraction.

Our experiments, therefore, provide the first evidence that group III mechanoreceptors, when stimulated by muscular contraction, reflexly increase sympathetic discharge. Previously, group III mech-
anoreceptors have been thought to be responsible for the heart rate response to static muscular contraction evoked by electrical stimulation of a peripheral nerve. This reflex response, however, could not be evoked when the static contraction was caused by electrical stimulation of the ventral roots, and therefore the origin of the response is uncertain.

The renal sympathetic response to electrical stimulation of the tibial nerve in paralyzed cats provides additional clues about the subtype of afferents responsible for the sympatoexcitatory responses to actual contraction. Two to five times motor threshold was the minimum current applied to the tibial nerve required to reflexly increase renal sympathetic activity. This current intensity is well below that which is necessary to activate group IV afferents. Furthermore, tibial nerve stimulation at one time motor threshold, which activates only group I afferents, had no effect on renal nerve activity. Therefore, the sympathetic activation observed in these experiments could have been caused only by electrical stimulation of group II or III afferents in either muscle or skin.

Previous studies have demonstrated that sympathetic activity is increased by electrical stimulation of group II afferents in skin nerves but not by stimulation of group II afferents in muscle nerves. Stimulation of cutaneous afferents could not have contributed to the reflex sympathetic response to actual contraction because the triceps surae was skinned and the hind limb was immobilized. We therefore suggest that group III muscle afferents were primarily responsible for the reflex stimulation of renal sympathetic outflow evoked by sustained and intermittent contraction in our experiments.

Although increases in renal nerve discharge have provided us with important information about the rapidity of the reflex autonomic response to static muscular contraction, we cannot assign a specific function to this sympathetic effect. Increases in renal sympathetic discharge have been shown to cause renal arterial vasoconstriction and renin release, as well as increased sodium reabsorption. Nevertheless, our finding that renal sympathetic activity increased in response to static contraction might be viewed as providing an electrophysiological basis for the finding that static contraction of the hind-limb muscles of anesthetized dogs reflexly decreased blood flow to the kidney. In addition, the data suggest that stimulation of sympathetic outflow to the kidney may contribute to the initiation of the rise in arterial pressure during muscle contraction. During static contraction, renal sympathetic excitation always preceded the pressor response to this maneuver. During intermittent contractions, the minimum tension required to reflexly increase arterial pressure was the same as that required to increase renal sympathetic activity.

Our findings in anesthetized cats contrast at first glance with recent findings in conscious humans. In the cats, static and intermittent tetanic contractions reflexly increased renal sympathetic discharge with a latency of less than 1 second, whereas in humans, static and dynamic exercise increased skeletal muscle sympathetic discharge with a latency of almost 1 minute. One interpretation of these human studies is that stimulation of group III mechanoreceptors does not reflexly increase sympathetic outflow during exercise. However, in light of our new findings in cats, an alternative possibility might be that mechanosensitive muscle afferents mainly govern sympathetic outflow to the kidney, whereas chemosensitive muscle afferents mainly regulate sympathetic outflow to skeletal muscle.

In summary, we have applied direct measurements of sympathetic nerve activity to the animal model that previously has been used to define the discharge properties of groups III and IV muscle afferents. The principal new concept arising from this study is that, in addition to chemically sensitive afferents, mechanically sensitive muscle afferents also play an important role in the reflex stimulation of sympathetic neural outflow evoked by static muscle contraction.

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References

KEY WORDS • sympathetic nerve activity • muscle afferents • mechanoreceptors • exercise • circulation, neural control of
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