Effects of static muscular contraction on impulse activity of groups III and IV afferents in cats

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KAUFMAN, MARC P., JOHN C. LONGHURST, KENNETH J. RYBICKI, JEFFREY H. WALLACH, AND JERE H. MITCHELL. *Effects of static muscular contraction on impulse activity of groups III and IV afferents in cats.* J. Appl. Physiol.: Respirat. Environ. Exercise Physiol. 55(1): 105–112, 1983.—Static contraction of the hindlimb muscles, induced by electrical stimulation of the ventral roots, reflexly increases arterial blood pressure and heart rate. Although stimulation of groups III and IV muscle afferents is believed to cause these reflex increases, the responses of these afferents to a level of static contraction that increases arterial pressure have not yet been determined. Therefore, in barbiturate-anesthetized cats, afferent impulses arising from endings in the gastrocnemius muscle were recorded from the L_7 or S_1 dorsal roots, while the cut peripheral end of the L_7 ventral root was stimulated. In addition, the effects of capsaicin (100–200 μg) and bradykinin (25 μg) on the activity of the groups III and IV afferents stimulated by contraction were examined. Contraction of the gastrocnemius muscle to a level equal to or greater than that needed to cause a pressor response stimulated 12 of 19 (63%) group III afferents and 13 of 19 (68%) group IV afferents. However, the discharge patterns of the group III afferents stimulated by contraction were very different from those of the group IV fibers. No relationship was found between those fibers stimulated by contraction and those stimulated by chemicals. Our results suggest that although both groups III and IV muscle afferents contribute to the reflex cardiovascular increases evoked by static exercise, group III fibers were likely to be stimulated by the mechanical effects of muscular contraction, whereas at least some group IV fibers were likely to be stimulated by the metabolic products of muscular contraction.

exercise pressor reflex

ALAM AND SMIRK (1) were among the first to show that exercise can produce increases in cardiovascular function by a reflex mechanism. Subsequent support for this concept has come from studies performed on anesthetized animals. For example, in anesthetized cats, static contraction of the hindlimb muscles, induced by electrical stimulation of the cut peripheral ends of the ventral roots, increased arterial pressure and heart rate, effects which were abolished by section of the dorsal roots innervating the exercising hindlimb (5, 15).

Muscular contraction has been shown to be necessary to evoke the exercise pressor reflex induced by ventral root stimulation, because when cats are paralyzed with gallamine triethiodide, the reflex is prevented (5). Furthermore the exercise pressor reflex has been shown to be unaffected either by cutting the nerves supplying the ankle and knee (5) or by skinning the contracting hindlimb (3, 15). These findings indicate that the afferent arm of the exercise pressor reflex arises from endings in contracting skeletal muscle.

To identify the afferent fibers responsible for evoking the exercise pressor reflex, McCloskey and Mitchell (15) used anodal direct current and local anesthesia to block reversibly the dorsal roots that received input from endings in exercising skeletal muscle. These investigators found that when groups I and II afferents were blocked with anodal current, the exercise pressor reflex was unaffected. However, when groups III (Aδ) and IV (C) afferents were blocked with a local anesthetic, the exercise pressor reflex was abolished.

The findings of McCloskey and Mitchell (15) suggested that groups III and IV afferents were responsible for the exercise pressor reflex. However, the responses of these afferents to a level of static muscular contraction that evokes a pressor reflex have not yet been characterized. We therefore recorded the impulse activity of groups III and IV muscle afferents from the dorsal roots while we statically contracted the gastrocnemius muscle by electrically stimulating the L_7 ventral root. In addition, we examined the effects of capsaicin and bradykinin on the firing of the groups III and IV afferents found to be stimulated by muscular contraction. Our purpose was to test the hypothesis that these substances stimulate the same afferents as those stimulated by static muscular contraction.

METHODS

General. Twenty-three cats, weighing 1.6–3.0 kg, were anesthetized with pentobarbital sodium (35 mg/kg ip). The right common carotid artery, right external jugular vein, and cervical trachea were cannulated. Blood pressure in the aortic arch was measured through the carotid arterial cannula, which was connnected to a Statham P23ID transducer. Tension, generated by the right triceps surae muscles, was measured by connecting the calcaneal tendon to a force-displacement transducer (Grass, FT-10). Anesthesia was maintained by additional intravenous injections of pentobarbital sodium.

The skin of the right hindlimb was incised from the

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hip to the calcaneus bone, and the sciatic nerve was exposed. All visible branches of the sciatic nerve were cut except for those supplying the triceps surae. In addition, the right gracilis and femoral nerves were cut. The skin overlying the right thigh was closed with suture, and the exposed triceps surae was covered with gauze soaked in warm (37°C) Ringer solution.

The L_4-S_1 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 curved stainless steel bars to form a pool, which was filled with warm (37°C) mineral oil. The dura was then incised. The right L_7 ventral root was identified and cut; the peripheral cut end was placed on a shielded and grounded electrode assembly (Fig. 1). The right hindlimb was clamped to prevent movement.

Recording of afferent impulse activity. We recorded afferent impulses from either the right L_7 or S_1 dorsal roots. For filaments containing either spontaneously active or silent fibers, we either lightly squeezed or vigorously pinched the gastrocnemius muscle with blunt-edged forceps to stimulate the receptive fields of the afferents. The latter maneuver did not cause any observable damage, such as bleeding, to this muscle. We discarded all fibers whose endings (receptive fields) we could not locate in the gastrocnemius muscle. We discarded afferents with endings in the soleus muscle, because static contraction of this muscle does not reflexly evoke a pressor response (19). To measure the conduction velocities of the fibers having endings in the gastrocnemius muscle, we electrically stimulated the lateral and medial gastrocnemius nerves through a pair of electrodes fixed in a shielded assembly (Fig. 1) and calculated conduction velocity by dividing the conduction distance between the stimulating and recording electrodes by the conduction time.

Experimental protocol. With the dorsal roots intact, we electrically stimulated (20–40 Hz; 0.01 ms) the peripheral cut end of the right L_7 ventral root at three times motor threshold for 30–45 s and measured arterial blood pressure, heart rate, and triceps surae tension. Our purpose was to find a level of static contraction (tension) that caused an increase in mean arterial pressure, which based on past experiments (5, 15, 19) was presumed to be a reflex arising from the gastrocnemius muscle.

Next we examined the responses of groups III and IV muscle afferents to both static muscular contraction and to the injection of bradykinin and capsaicin. The order of these three maneuvers was varied randomly. Static contraction was induced by electrical stimulation (20–40 Hz; 0.01 ms) of the right L_7 ventral root for 30–45 s, the voltage being adjusted so that peak triceps surae tension equaled or exceeded that previously shown to increase arterial blood pressure. Onset latencies were measured from the start of ventral root stimulation. Using a rack and pinion, we set the resting tension of the triceps surae at 0.5–1.0 kg. The effect of ventral root stimulation on triceps surae tension is reported as developed tension, i.e., peak tension minus resting tension. In some experiments we examined the effects of different amounts of tension development by the triceps surae on the impulse activity of group III fibers. The amount of tension development was varied by changing the frequency and the voltage of the pulses applied to the L_7 ventral root.

Capsaicin (100–200 μg) and bradykinin (25 μg) were injected into the abdominal aorta through a catheter inserted into the left femoral artery. Capsaicin was dissolved as previously described (4). Bradykinin triacetate

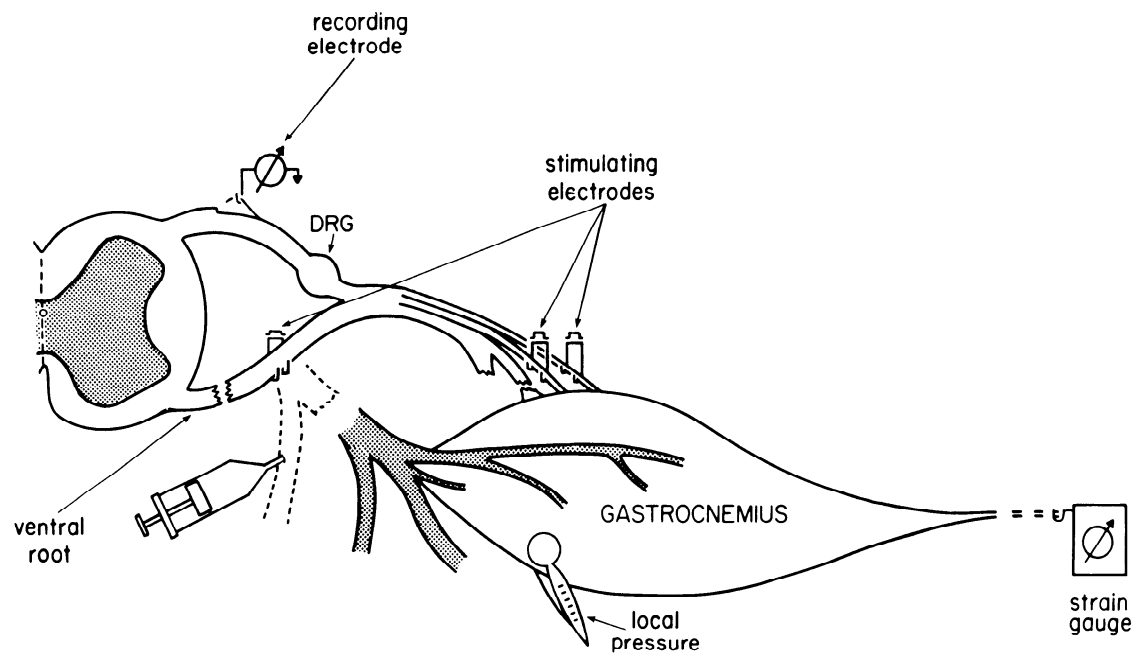

FIG. 1. Diagram of preparation. Afferent impulses arising from endings in gastrocnemius muscle were recorded from either L_7 or S_1 dorsal roots. Gastrocnemius muscle was contracted by electrically stimulating cut peripheral end of L_7 ventral root, and tension was measured with strain gauge. Receptive fields of endings were located in muscle by using forceps to apply local pressure. Bradykinin and capsaicin were injected into abdominal aorta. This figure has been modified from one by Franz and Mense (8) and by Kniffki et al. (13). DRG, dorsal root ganglion.

was dissolved in saline. Both substances were injected in 1 ml of saline and were flushed in with 1 ml of saline. The injection required 3–5 s; onset latencies were measured from the beginning of injection. None of the fibers was stimulated by the vehicles in which capsaicin and bradykinin were dissolved. The doses of bradykinin and capsaicin injected into the abdominal aorta were equal to or greater than those shown previously to stimulate groups III and IV muscle afferents in dogs (11). We did not examine the responses of groups I and II muscle afferents to static contraction, to capsaicin, or to bradykinin, because stimulation of these fibers has little or no reflex effect on the cardiovascular system (6, 14, 15, 18, 22).

Control firing rates were averaged for 30 s before either stimulating the L_7 ventral root or injecting capsaicin and bradykinin. Peak firing rates were averaged for 5 s if the fiber was stimulated by any of the maneuvers. All firing rates are expressed as impulses per second. Control heart rates were calculated by counting the arterial pressure pulses over a 30-s period, whereas peak heart rates were calculated over a 5-s period. All values are expressed as means ± SE. We used paired t tests (two-tailed) and a chi-square test to determine statistical significance.

RESULTS

Cardiovascular responses to static muscular contraction. In 19 of 23 cats, contraction of the triceps surae, evoked by stimulation of the L_7 ventral root, increased, on the average, mean arterial blood pressure from 101 ± 5 to 114 ± 6 mmHg ($P < 0.05$) and heart rate from 163 ± 8 to 167 ± 7 beats/min ($P < 0.05$). The increase in mean arterial pressure, which was observed in each of the 19 cats, started 6.4 ± 1.0 s after the onset of ventral root stimulation (range 2–16 s). The increase in heart rate, which was observed in only 7 of the 19 cats, started 8.3 ± 1.7 s after the onset of stimulation. The developed tension that caused these cardiovascular increases was 2.3 ± 0.2 kg. In the four remaining cats, contraction of the triceps surae failed to increase mean arterial pressure and heart rate, even though developed tension from this muscle group was equal to or greater than the developed tension in the 19 cats showing pressor responses to this maneuver. We chose not to record impulses from the dorsal roots of the four cats in which static contraction failed to produce a pressor response.

Effects of static contraction on the activity of group III fibers. We recorded the impulse activity of 19 group III fibers (conduction velocity 12.1 ± 1.4 m/s; range 3.2–25.0 m/s). Of the 19 group III fibers, 12 (63%) were stimulated by static contraction (mean onset latency 0.8 ± 0.5 s; Figs. 2; 3, *A* and *B*; and 4; Table 1). The most striking feature of the responses to static contraction of 10 of the group III fibers was a sudden explosive burst of impulses, starting 224 ± 84 ms (range 30–900 ms) after the onset of ventral root stimulation and decreasing to almost control levels within 20 s of the contraction period. In 5 of these group III fibers, once their initial responses to contraction were completed, no further firing was observed even though the gastrocnemius muscle continued to contract (Fig. 3*A*). In the other 5 fibers, however, once their initial responses were completed, firing increased a second time (Fig. 3*B*). The responses of the remaining 2

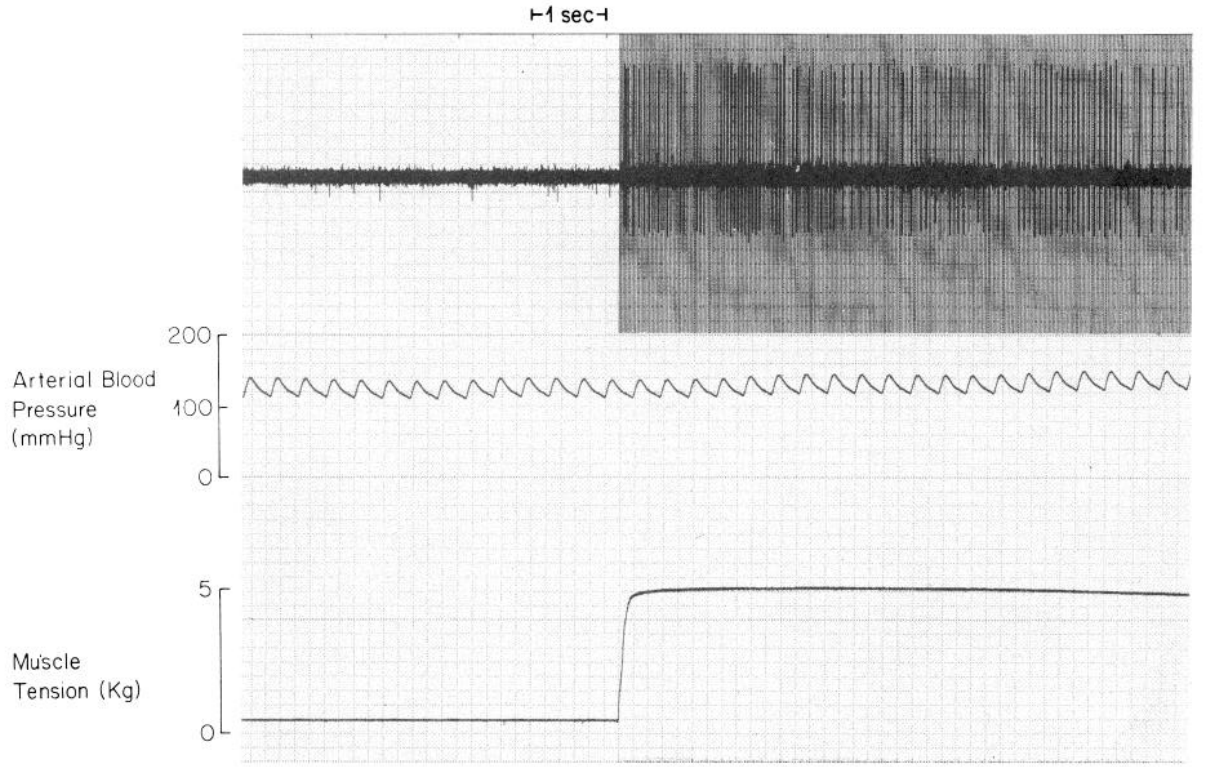

FIG. 2. Stimulation of group III muscle afferent (conduction velocity 17.3 m/s) by contraction of gastrocnemius muscle induced by ventral root stimulation. Note almost instantaneous response of group III afferent to contraction. Stimulus artifact is represented by *broad gray band* in *top trace*.

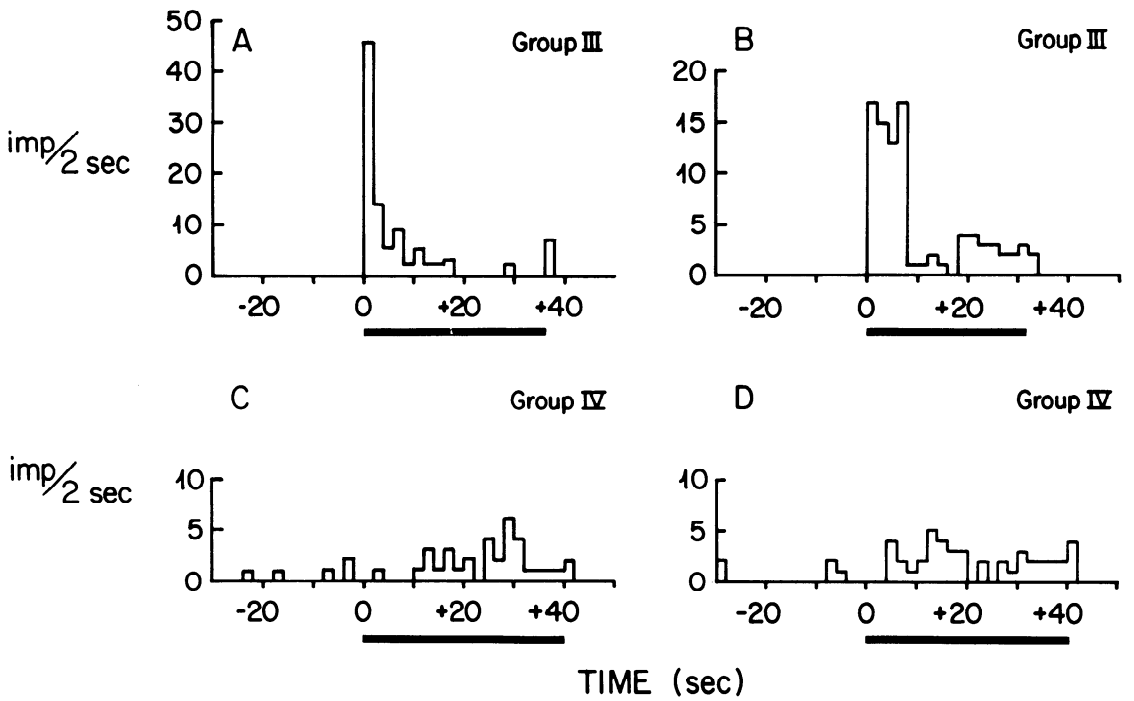

FIG. 3. Discharge patterns of 4 thin fiber muscle afferents that responded to static contraction. Contraction period depicted by *black bar*. *A*: group III fiber (conduction velocity 17.8 m/s) discharged vigorously at onset of contraction, but then its firing rate decreased even though muscle continued to contract. *B*: group III fiber (conduction velocity 9.6 m/s) discharged vigorously at start of contraction, adapted, and then fired again during contraction. *C*: group IV fiber (conduction velocity 1.3 m/s) started to fire 10 s after onset of contraction and then gradually increased its firing rate during contraction period. Note that firing of fiber slowed even though muscle continued to contract. *D*: group IV fiber (conduction velocity 1.1 m/s) fired irregularly 4 s after onset of static contraction.

TABLE 1. *Effect of static contraction on the impulse activity of 19 group III fibers and 19 group IV fibers*

	Control, impulses/s	Peak, impulses/s	Developed Tension, kg
Group III	0.1 ± 0.1	3.0 ± 0.9*	3.2 ± 0.3
Group IV	0.3 ± 0.1	1.9 ± 0.5*	3.2 ± 0.4

Values are means ± SE. * Statistical significance ($P < 0.05$) between control and peak firing rates.

group III fibers stimulated by contraction started 1 and 6 s after the onset of ventral root stimulation; their discharge patterns consisted of an irregular march of impulses throughout the contraction period. Of the 12 group III fibers stimulated by contraction, 4 fired briefly after the end of this maneuver (Fig. 3, *A* and *B*). Last, the conduction velocities of the group III fibers stimulated by contraction were not significantly different from those of the group III fibers not stimulated by contraction (13.4 ± 1.4 vs. 9.9 ± 2.5 m/s, respectively; $P > 0.05$).

We were able to examine the effects of two or three different levels of tension development by the gastrocnemius muscle on the firing of seven group III fibers stimulated by contraction. Six of the seven increased their discharge rates as the level of tension developed by the gastrocnemius increased (Fig. 4).

Eight of the 19 group III fibers were activated by gently stroking their receptive fields with a blunt glass rod. Seven of the 8 were also stimulated by stretching the calcaneal tendon; the same 7 were stimulated by static contraction, with activity in 6 starting within 1 s of the onset of ventral root stimulation. Four of these group III

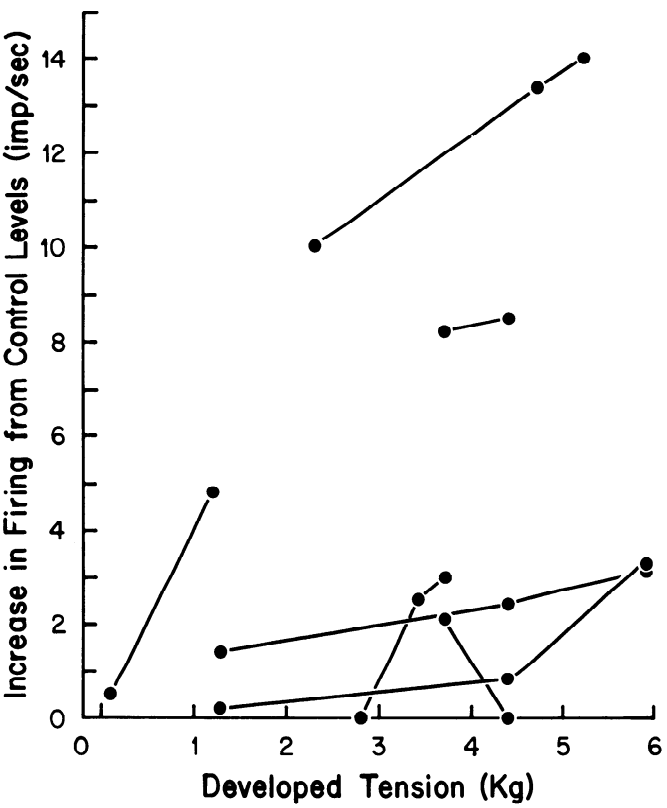

FIG. 4. Effects of different levels of tension development by triceps surae on impulse activity of 7 group III muscle afferents. *Vertical axis* represents increase in impulse activity from control levels. Note group III afferent that was so sensitive to muscular contraction that it fired a few impulses even though tension developed by muscle was only 0.1 kg. This afferent was silent for 30 s before onset of contraction.

endings were located at or near the junction of the gastrocnemius muscle and the calcaneal tendon (Fig. 2), 3 were located in the belly of the gastrocnemius, and one in the lateral head of this muscle.

Ten of the 19 group III fibers were stimulated by lightly squeezing the gastrocnemius muscle with a blunt-edged forceps. When this stimulus was applied to the forearm of the investigators it was perceived as pressure, but it was not perceived as noxious. Four of the 10 were stimulated by tendon stretch, and 5 were stimulated by static contraction. Six were located in the lateral or medial heads of the gastrocnemius, whereas 4 were located in the belly of this muscle.

The remaining group III fiber was stimulated by vigorously pinching the belly of the gastrocnemius, less forceful probing of the muscle being ineffective. When applied to the investigators, this stimulus was perceived as noxious. Neither stretching the calcaneal tendon nor static contraction stimulated this fiber.

Effects of capsaicin and bradykinin on the activity of group III fibers. Capsaicin (100–200 μg) stimulated 5 of the 19 group III fibers (Table 2). The responses of the 5 fibers stimulated by capsaicin started 7.2 ± 2.4 s after injection, activity remaining above control levels for 7.0 ± 1.8 s. Of the 12 group III fibers stimulated by static contraction, 3 were stimulated by capsaicin. Of the 14 group III fibers not stimulated by capsaicin, 9 were stimulated by static contraction.

Bradykinin (25 μg) stimulated 10 of the 19 group III fibers (Table 2). The response of the 10 fibers stimulated by bradykinin consisted of a low-frequency train of impulses beginning 19.7 ± 3.4 s after injection and remaining above control levels for 40.4 ± 4.6 s. Of the 12 group III fibers stimulated by contraction, 6 were stimulated by bradykinin. Of the 9 group III fibers not stimulated by bradykinin, 6 were stimulated by contraction.

Effect of static contraction on the activity of group IV fibers. We recorded the impulse activity of 19 group IV fibers (conduction velocity 1.1 ± 0.1 m/s; range 0.5–2.0 m/s). Thirteen of the 19 (68%) were stimulated by static contraction (Figs. 3, *C* and *D*; and 5; Table 1), with activity increasing 3.8 ± 1.1 s after the start of ventral root stimulation (range 0.3–14.0 s). In 6 of these 13 group IV fibers, the increased firing in response to static contraction reached a peak and then decreased somewhat, even though the gastrocnemius muscle continued to contract (Fig. 3*C*). In the remaining 7 group IV fibers, firing was maintained throughout the contraction. In addition, 5 of the 13 group IV fibers continued to fire for 10–12 s after the end of the contraction (Fig. 5). Also the onset latencies of the group IV fibers responding to static contraction were significantly longer than those of the group III fibers responding to this stimulus (i.e., 3.8 ± 1.1 vs. 0.8 ± 0.5 s, respectively; $P < 0.05$). We did not examine the effects of different levels of tension development by the gastrocnemius muscle on the firing of group IV fibers.

None of the 19 group IV fibers could be stimulated by gently stroking their receptive fields. Five of the 19 fibers were activated by lightly squeezing the gastrocnemius muscle with a blunt-edged forceps, a stimulus which when applied to the investigators was not perceived as

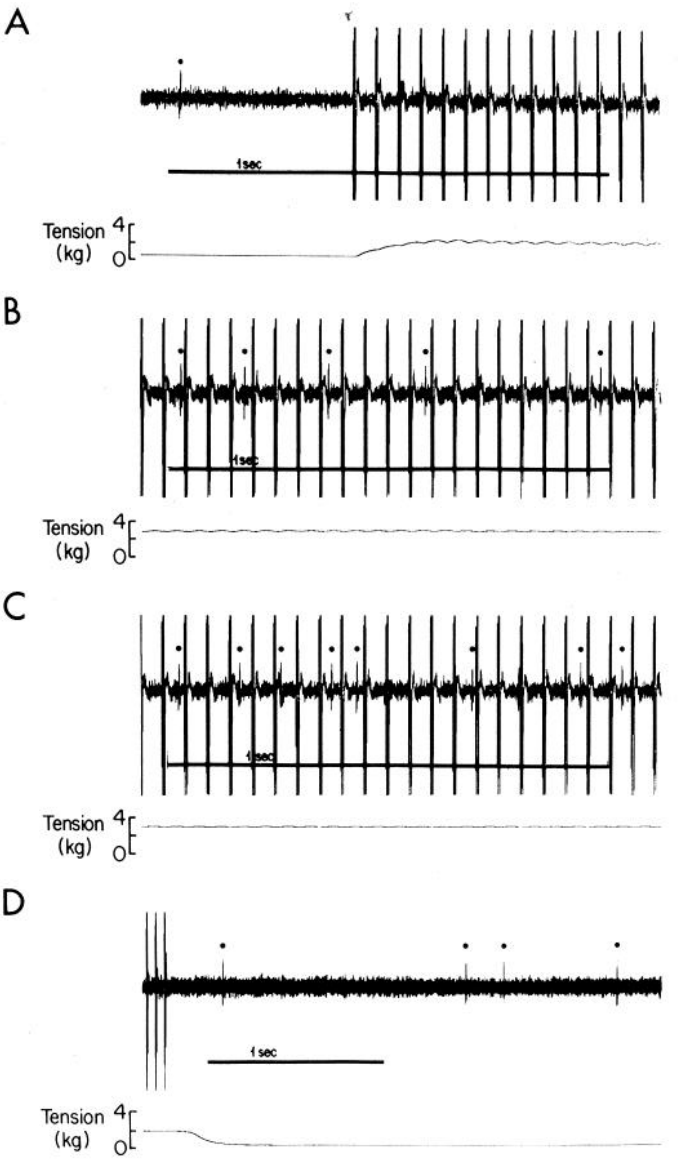

FIG. 5. Stimulation of group IV muscle afferent (conduction velocity 0.5 m/s) by contraction of gastrocnemius muscle, induced by ventral root stimulation. *Filled circle* (●) has been placed over each impulse discharged by group IV afferent. *A*: at onset of muscular contraction, group IV fiber did not fire. *B*: 8 s after end of *A*, group IV fiber has increased its firing rate over control level, which averaged 0.6 impulses/s. *C*: 21 s after end of *B*, fiber is firing at greater rate than in *B*. *D*: after the contraction period, which lasted 45 s, fiber discharged. *Horizontal bar* in *A–D* represents 1 s. Note that chart recorder speed was slower in *D* than in *A–C*.

TABLE 2. *Effect of capsaicin and bradykinin on the impulse activity of 19 group III fibers and 19 group IV fibers*

	Capsaicin		Bradykinin	
	Control, impulses/s	Peak, impulses/s	Control, impulses/s	Peak, impulses/s
Group III	0.1 ± 0.1	0.6 ± 0.3	0.1 ± 0.1	1.5 ± 0.4*
Group IV	0.3 ± 0.1	4.2 ± 1.0*	0.2 ± 0.1	1.5 ± 0.4*

Values are means ± SE. Capsaicin (100–200 μg) and bradykinin (25 μg) were injected into abdominal aorta. * Statistical significance ($P < 0.05$) between control and peak firing rates.

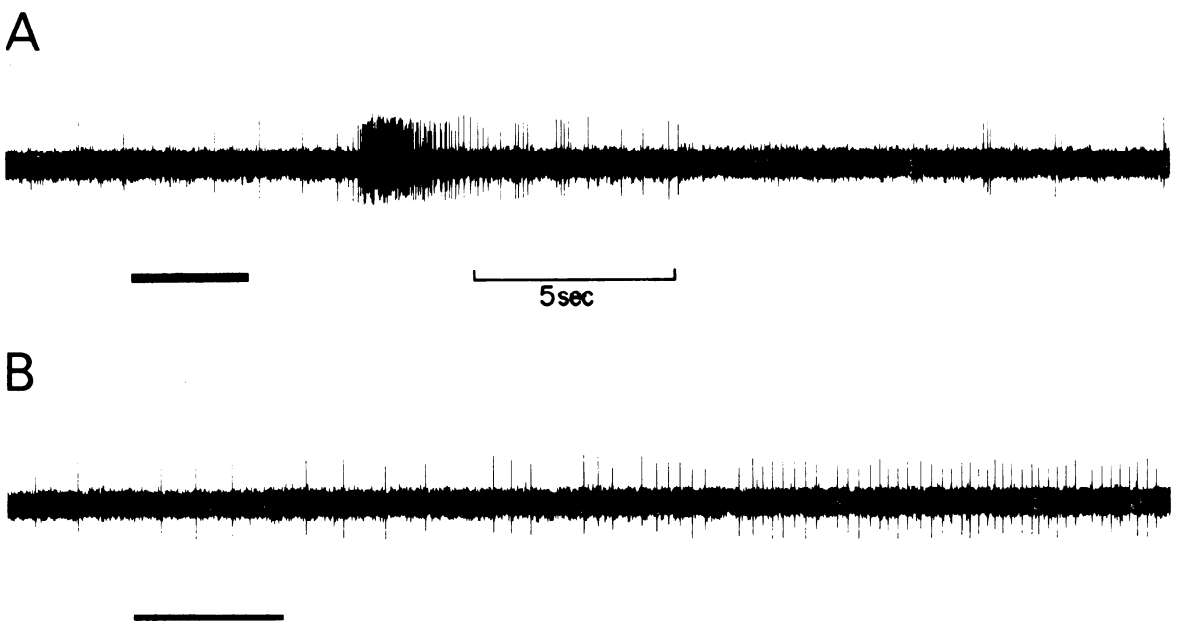

FIG. 6. Stimulation of group IV muscle afferent (conduction velocity 0.5 m/s) by capsaicin and bradykinin. Capsaicin (100 μg, *A*) and bradykinin (25 μg, *B*) were injected into abdominal aorta during *horizontal bar*. Note different discharge patterns evoked by the 2 substances.

noxious. Of these 5, 3 were stimulated by static contraction. Stretching the calcaneal tendon stimulated only 1 of the 5 fibers activated by lightly squeezing the gastrocnemius muscle. The ending of this fiber was located at the junction of the tendon and the muscle and was one of the 3 stimulated by contraction. The other 4 group IV fibers, which were activated by lightly squeezing their receptive fields, were located either in the belly ($n = 3$) or in the lateral head ($n = 1$) of the gastrocnemius muscle.

When probing the muscle, we could activate 14 of the group IV fibers only by vigorously pinching their receptive fields, a stimulus which when applied to the investigators was perceived as noxious. Of the 14, 10 were stimulated by static contraction. Stretching the calcaneal tendon fired only 1 of the 14 group IV fibers. Again, this stretch-sensitive fiber was stimulated by contraction; its ending was located in the belly of the gastrocnemius. Of the remaining 13 group IV fibers, 12 were also located in the belly of the gastrocnemius; the other ending was located in the medial head of the muscle.

Effects of capsaicin and bradykinin on the activity of group IV fibers. Capsaicin (100–200 μg) stimulated 16 of the 19 group IV fibers (Table 2). The discharge pattern of the 16 fibers stimulated by this substance consisted of a high-frequency short-lasting burst of impulses, beginning 4.9 ± 0.9 s after injection and remaining above control levels for 20.0 ± 8.6 s (Fig. 6*A*). Of the 13 group IV fibers stimulated by static contraction, 12 were stimulated by capsaicin. Of the 3 not stimulated by capsaicin, 1 was stimulated by contraction.

Bradykinin (25 μg) stimulated 12 of the 19 group IV fibers (Table 2). The discharge pattern of 10 of the fibers stimulated by this substance consisted of a low-frequency long-lasting march of impulses, beginning 18.5 ± 2.4 s after injection and remaining above control levels for 42.5 ± 9.5 s (Fig. 6*B*). The discharge pattern of the remaining 2 group IV fibers consisted of bursts of impulses beginning 4 and 11 s after injection. Of the 13 fibers stimulated by contraction, 10 were stimulated by bradykinin. Of the 7 not stimulated by bradykinin, 3 were stimulated by contraction.

Using a chi-square analysis, we found that capsaicin stimulated a greater percentage of group IV fibers (84%) than group III fibers (26%; $P < 0.05$). Bradykinin, however, stimulated approximately equal percentages of groups IV and III fibers (63 vs. 58%, respectively; $P > 0.05$).

DISCUSSION

The major aim of our study was to describe the discharge patterns of groups III and IV muscle afferents that responded to a level of static contraction that increased cardiovascular function, an effect that has been shown to be reflex in origin (5, 15). To achieve this aim, we contracted the gastrocnemius muscle by electrically stimulating the L_7 ventral root and recorded afferent impulse activity from a dorsal root innervating this muscle. We found that the majority of the groups III (i.e., 63%) and IV (i.e., 68%) afferents tested were stimulated by a level of static muscular contraction that increased mean arterial pressure.

A higher percentage of the groups III and IV afferents were stimulated by muscular contraction in our experiments than were stimulated in the experiments of Kniffki et al. (12, 13) and Mense (16). In part, this difference may have been due to the fact that we contracted the gastrocnemius continuously (i.e., statically), whereas in some of their experiments Kniffki et al. (13) and Mense (16) contracted this muscle intermittently. Hence blood flow to the contracting muscle was likely to have been less in our experiments than it was in those of Kniffki et al. (13) and Mense (16). In this regard, Iggo (10) has presented evidence that a lack of blood flow to

contracting muscle is an important determinant of the responses of group IV afferents to muscular contraction. Another possible cause for this difference is that the contracting gastrocnemius muscle may have developed more tension in our experiments than in those of Kniffki et al. (12, 13). This possibility, however, cannot be evaluated, because in the experiments of Kniffki et al. (12, 13) developed tension was not reported.

Substantial evidence has been gathered to support the concept that the exercise pressor reflex is evoked by the buildup within skeletal muscle of metabolites produced by contraction (1, 5, 15, 21, 23). These metabolites are believed to stimulate groups III and IV endings, which, in turn, are believed to signal the central nervous system that the metabolic demands of contracting skeletal muscle are not being matched by the blood supply of the muscle (17, 20). This mismatch between blood supply and demand in contracting muscle is likely to occur during static contraction, because blood flow to the muscle is mechanically obstructed by the contraction (2).

In our experiments, at least some of the group IV afferents appeared especially well suited to function as the metabolic receptors that are believed to signal a mismatch between blood supply and demand in contracting skeletal muscle. These afferents responded to static contraction with an average onset latency of 3.8 s, a period of time that was likely to allow the metabolic products of contraction to accumulate in a muscle undergoing static contraction. In addition, some of these afferents gradually increased their firing rate as the contraction period progressed, a finding consistent with the notion that they were stimulated by the buildup of metabolites in the contracting muscle. One possible explanation as to why six of the group IV afferents adapted to contraction may be that as the muscle fatigued, blood flow to it increased, an effect likely to remove the metabolites causing the afferents to fire.

The rapid stimulation of most of the group III fibers by contraction is difficult to attribute to the buildup of metabolites within the muscle. Instead, these group III fibers were likely to have functioned as mechanoreceptors, a possibility consistent with our finding that most of these afferents increased their firing when the level of tension developed by the contracting gastrocnemius muscle increased. However, the secondary increase in firing, which occurred in almost half of the group III afferents responding to contraction, raises the possibility that the endings of these afferents were sensitive both to the metabolic products of static muscular contraction and to the mechanical effects of contraction.

The rapid responses to contraction by many of the group III afferents suggest that these afferents contribute to the initiation of the exercise pressor reflex, the mean onset of which has been reported by Coote et al. (5) to be 2.5 s in cats. In addition, another reflex function of these group III afferents may be to increase heart rate at the start of exercise. For example, Hollander and Bouman (9) presented evidence that muscular contraction reflexly accelerates the heart in humans, an effect occurring 550 ms after the onset of this stimulus.

We found little correlation between the groups III and IV muscle afferents stimulated by static contraction and those stimulated by either capsaicin or bradykinin. Capsaicin has been suggested to stimulate the same afferents as those stimulated by static contraction (7, 24). However, our findings in cats gave little support to this suggestion, because capsaicin failed to stimulate most of the group III afferents that were vigorously stimulated by static contraction. This substance therefore may be useful for stimulating most of the group IV, and few of the group III, muscle afferents in both cats (present results) and dogs (11), but it appears to have little usefulness for stimulating the same muscle afferents as those stimulated by static contraction.

In our experiments, the level of tension developed by the contracting triceps surae was less than half of the maximum of this muscle group (i.e., 10–12 kg) (5, 17). Although we cannot be sure that the level and duration of the contractions used were noxious, we did find that the triceps surae, when contracted for 30–45 s, showed signs of fatigue. Human subjects sometimes report the sensation of fatigue as being uncomfortable. Therefore, because of the uncertainty regarding the noxious nature of the static contractions used in our experiments, our findings shed little definitive light on the classification by Kniffki et al. (13) of groups III and IV muscle afferents into ergoreceptive and nociceptive categories. It is interesting to speculate, however, that the group III afferents stimulated both by low levels of contraction and by nonnoxious probing of their receptive fields transduced ergoreceptive but not nociceptive information.

The onset latencies of some of the group IV muscle afferents responding to static contraction were found to be consistent with the hypothesis that these afferents were stimulated by the accumulation of metabolites in exercising skeletal muscle (1). By contrast, the onset latencies of most of the group III muscle afferents responding to contraction were much too rapid to be explained by the accumulation of metabolites and suggested that these afferents were mechanoreceptors. Moreover the rapid responses to contraction displayed by many of the group III afferents raise the possibility that they reflexly contribute to the rapid cardiovascular effects evoked by this stimulus (9).

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