J. Physiol. (1986), **370**, *pp.* 255–265
With 6 text-figures
Printed in Great Britain

BLOOD PRESSURE RESPONSE EVOKED BY VENTRAL ROOT AFFERENT FIBRES IN THE CAT

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(*Received 24 April 1985*)

SUMMARY

1. Systemic arterial blood pressure changes in response to stimulation of the distal stump of the cut spinal ventral root were investigated in anaesthetized, vagotomized, and carotid sinus-denervated cats.

2. Low intensity electrical stimulation ($<20\ T$, where T is threshold intensity) of the ventral root caused a rise in blood pressure. This elevation was abolished by paralysing the muscles with gallamine. This pressor response has been reported previously, and it is likely to be evoked by afferents excited by the contracting muscle.

3. High intensity electrical stimulation (500 T) of the ventral root caused a second and marked pressor response. This was not affected by muscular paralysis or by cutting the sciatic nerve, but it was abolished by cutting the dorsal root.

4. Threshold intensity for the second component of the pressor response was within the same range as the intensity needed for activation of C fibres in the ventral root, ranging between 200 T and 300 T. This response was graded with increasing stimulus intensity, and it showed both spatial and temporal summation.

5. From the above results, we conclude that non-myelinated fibres in feline spinal ventral root course distally to the dorsal root ganglion and then enter the spinal cord via the dorsal root. Activation of these fibres results in a marked elevation of the systemic arterial blood pressure as in other somato-sympathetic reflexes induced by peripheral C fibre activation.

INTRODUCTION

Skeletal muscle contractions induced by electrical stimulation of spinal ventral roots elicit pressor responses (Coote, Hilton & Perez-Gonzalez, 1971; McCloskey & Mitchell, 1972; Fisher & Nutter, 1974; Kaufman, Longhurst, Rybicki, Wallach & Mitchell, 1983). Activation of afferent fibres from the contracting muscle seems to cause these responses, since the responses are abolished by paralysing muscles or by sectioning the dorsal roots that innervate the contracting muscle. Because activation of peripheral non-myelinated afferent nerve fibres from skin, muscle, and viscera causes somato- and viscero-sympathetic reflexes (Hunt, 1895; Ranson, 1921; Johansson, 1962; Coote & Perez-Gonzalez, 1970; Koizumi, Collin, Kaufman & Brooks, 1970; Schmidt & Weller, 1970), stimulation of the ventral root, causing

skeletal muscle contractions, could elicit pressor responses indirectly by way of somato-sympathetic reflexes.

Another possible way to change blood pressure by ventral root stimulation would be by activating afferent fibres contained in the ventral root. Previous studies have shown that about 30% of axons in feline (Coggeshall, Coulter & Willis, 1974) and in human (Coggeshall, Applebaum, Frazen, Stubbs & Sykes, 1975) lumbosacral ventral roots are non-myelinated, and most of these seem to be sensory in function (Coggeshall *et al.* 1974; Clifton, Coggeshall, Vance & Willis, 1976; Coggeshall & Ito, 1977; Yamamoto, Takahashi, Satomi & Ise, 1977). Although we do not know the functional significance of these ventral root afferents, they are potentially important in regulating cardiovascular function because they are numerous and because they mediate a small but significant pressor response (Longhurst, Mitchell & Moore, 1980). Furthermore, experiments conducted recently in our laboratory showed that by stimulating the distal stump of the cut ventral root, we could activate many dorsal horn cells (Chung, Lee, Endo & Coggeshall, 1983; Chung, Lee, Kim & Coggeshall, 1985); this activation was mediated by fibres in continuity between the dorsal and ventral roots (Kim & Chung, 1985).

The purpose of the present study was to reveal the physiological role of ventral root afferent fibres and, particularly, their potential role for cardiovascular function. We stimulated electrically the distal stump of the cut spinal ventral root to activate non-myelinated fibres in the ventral root and observed changes in arterial blood pressure in anaesthetized cats.

METHODS

Eighteen adult cats (1·5–3·5 kg) of either sex were used. The animals were anaesthetized initially with a gaseous mixture of halothane and nitrous oxide. Intravenous injection of α-chloralose (70 mg/kg) followed. A tracheotomy was performed and the animal was ventilated with a respirator. Systemic arterial blood pressure was monitored through a cannula inserted into the carotid artery. The cannula was connected to a Statham pressure transducer. To reduce baroreceptor-mediated reflex compensation of blood pressure, we denervated the carotid sinuses bilaterally and cut the vagus nerves in the neck bilaterally. The disappearance of blood pressure responses induced by bilateral carotid artery occlusion confirmed complete denervation. Throughout the experiment, the end-tidal CO_2 level was kept between 3·5 and 4·5%; rectal temperature was maintained near 37·5 °C by an electric blanket.

The lumbosacral spinal cord was exposed by a laminectomy, and a heated mineral oil pool was formed over the exposed spinal cord. Ventral roots of the 7th lumbar and 1st sacral spinal segments were traced intradurally and cut near the spinal cord. The peripheral stump of the cut ventral root was placed on a tripolar stimulating electrode; the most distal lead (close to the dorsal root ganglion) was grounded to prevent current spread to nearby neural structures (Fig. 1*A*). In the ipsilateral hind limb, the femoral and hamstring nerves were cut so that muscle contractions induced by stimulation of the ventral root would be mainly mediated by the sciatic nerve. The sciatic nerve was placed on a pair of bipolar recording electrodes to determine the threshold intensity for α-motoneurone activation by monitoring the compound action potential evoked by ventral root stimulation (Fig. 1*A*). The threshold ranged between 10 and 20 mV using a square wave pulse with a duration of 0·5 ms. However, this approach was not useful for determining the threshold for activation of non-myelinated fibres in the ventral root because of a poor signal to noise ratio. Therefore, recordings were also made from the dorsal ramus of the spinal nerve at the S1 segment while stimulating the S1 ventral root (Fig. 1*A*). The threshold for activating non-myelinated fibres in the ventral root ranged between 3 and 5 V with 0·5 ms pulses (200–300*T*, 200–300 times that of the threshold for activation of α-motoneurone axons).

Repetitive stimuli were applied for 20–30 s at a frequency of 20 Hz. Changes in arterial blood pressure were induced by two different intensities of electrical stimulation of the ventral root. The lower intensity level of stimulation of the ventral root was suprathreshold for A fibres. The sizes of compound action potentials showed no difference when stimulus intensities were 20 *T*, 50 *T* or 500 *T*, indicating that all α-motoneurone axons were already activated at 20 *T* intensity (Fig. 1 *B*).

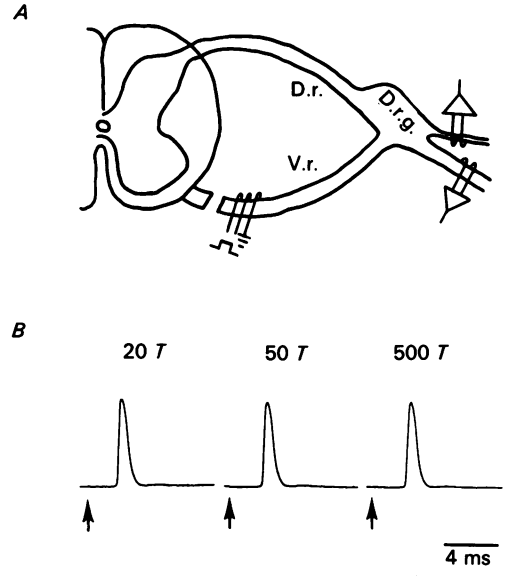

Fig. 1 *A*, schematic diagram of the experimental set-up. Electrical stimulation (usually 20 Hz for 20–30 s) was applied to the distal stump of a cut ventral root (either L7 or S1) with a tripolar electrode (the most distal lead was grounded) while the systemic arterial blood pressure was recorded. Compound action potentials were recorded from the sciatic nerve and the dorsal ramus of the spinal nerve. D.r., dorsal root; v.r., ventral root; d.r.g., dorsal root ganglion. *B*, compound action potentials recorded from the sciatic nerve show activation of α-motoneurone axons with various intensities of stimuli applied to the distal stump of the cut L7 ventral root. The sizes of the compound action potential did not change with different intensities of stimuli, indicating that the maximum number of α-motoneurone axons were already activated at an intensity of 20 *T* (20 times threshold intensity for activation of α-motoneurone axons). These recordings were obtained by averaging the tracings from 100 consecutive stimuli applied to 1 Hz. Arrows indicate times of stimuli. Conduction distance was 130 mm.

Therefore, we usually used 20 *T* to activate all of the α-motoneurone axons in the ventral root. For higher intensity stimulation, we used 500 *T* to activate non-myelinated fibres in the ventral root, since the threshold for C fibres ranged between 200 *T* and 300 *T*. Changes in blood pressure during stimulation of the ventral root were compared before and after muscular paralysis by intravenous injection (20 mg initially followed by constant infusion at a rate of 4 mg/kg.h) of gallamine triethiodide (Flaxedil).

RESULTS

Electrical stimulation of the distal stump of the ventral root with either low or high intensities produced an elevation of the blood pressure, but high intensity stimulation produced a greater elevation (Fig. 2). After paralysing the muscles by an injection

of gallamine, however, identical stimulation at low intensity no longer elicited the response whereas high intensity stimulation produced a response comparable to that seen before the injection of gallamine.

Fig. 3 summarizes the results of the experiments for all the animals. In animals that were not paralysed, blood pressure increased from $128 \pm 29/87 \pm 24$ (mean $\pm$ S.E. of mean, systolic/diastolic) at rest to $145 \pm 31/103 \pm 25$ mmHg during application of

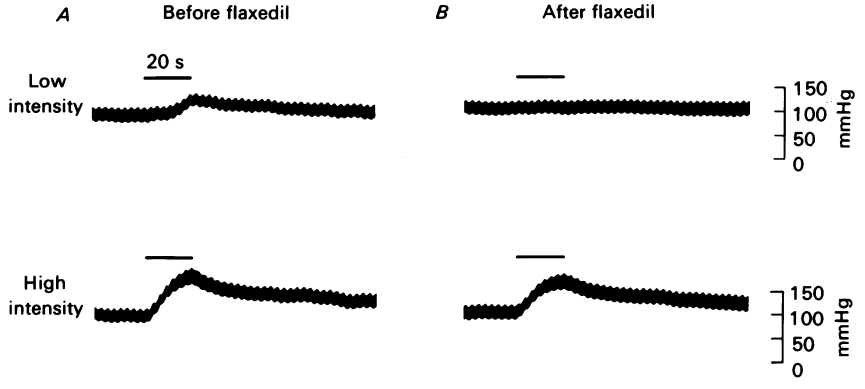

Fig. 2. Changes in systemic arterial blood pressure during stimulation of the distal stump of a cut ventral root before (*A*) and after (*B*) paralysing the animal by an intravenous injection of gallamine. The L7 ventral root was stimulated (20 Hz) with low (20 *T*) and high (1000 *T*) intensities for 20 s (indicated by bars above each blood pressure tracing). Before the gallamine injection, stimulatin of the ventral root at low intensity produced an elevation of blood pressure to a maximum of 170/130 mmHg (systolic/diastolic) from 115/90 mmHg (*A*, upper panel). This response was no longer produced after gallamine injection despite identical stimulation (*B*, upper panel). Stimulation of the ventral root at high intensity produced a marked increase in blood pressure (from 125/95 to 205/175 mmHg); this response was somewhat reduced but largely preserved (from 125/90 to 185/155 mmHg) after gallamine injection.

low intensity stimuli; it increased from $126 \pm 24/86 \pm 24$ to $184 \pm 28/143 \pm 25$ mmHg during high intensity stimulation. Paralysis of the animals with gallamine abolished blood pressure responses to low intensity stimulation ($123 \pm 20/85 \pm 22$ mmHg to $120 \pm 27/86 \pm 28$ mmHg). On the contrary, high intensity stimulation elevated the blood pressure from $126 \pm 23/88 \pm 24$ mmHg at rest to $173 \pm 32/135 \pm 28$ mmHg. These results indicate that pressor responses are elicited by stimulation of the peripheral stump of the cut spinal ventral root. Furthermore, the pressor response actually consists of two different components: (1) one is elicited by low intensity stimulation and depends on muscle contractions; (2) the other is elicited by high intensity stimulation and does not depend on muscle contractions. Activation of afferents originating from the contracting muscle probably accounts for the first component but not for the second. After these two components were clearly established, the remaining experiments concentrated on the second component since the first component, activated by afferents originating from the contracting muscle, has already been well studied (Coote *et al.* 1971; McCloskey & Mitchell, 1972; Fisher & Nutter, 1974). Therefore, the remaining experiments were performed on paralysed animals.

To determine the fibre group in the ventral root that is responsible for the second component of the pressor response, we correlated the threshold for the pressor response and that for the compound action potential recorded from the dorsal ramus of the spinal nerve during stimulation of the peripheral stump of the cut S1 ventral root. The threshold intensity for a blood pressure response was approximately 5 V

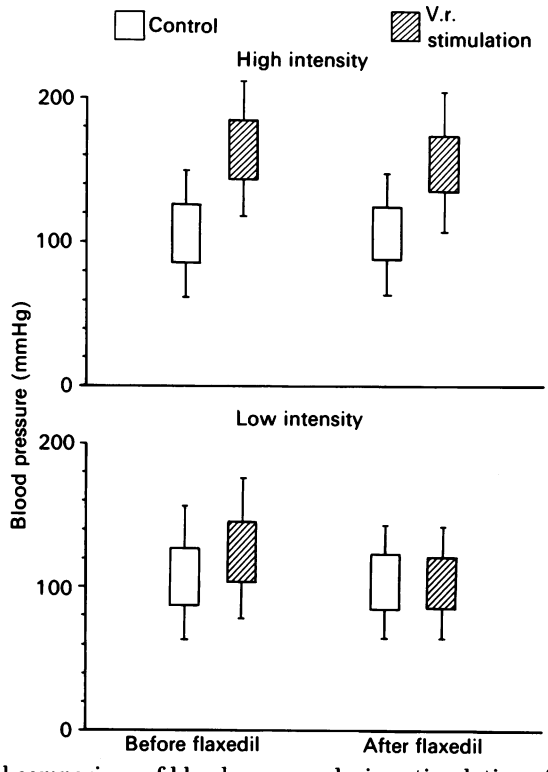

Fig. 3. Statistical comparison of blood pressure during stimulation of ventral root (L7 in all animals) with low and high intensities. Blood pressure values of both diastolic and systolic are expressed as mean ± S.E. of mean (bars) of total experiments ($n = 12$). Values of blood pressure during ventral root stimulation (V.r. stimulation) were obtained at maximum blood pressure during stimulation for 20 s period. All increases in blood pressure were statistically significant (two tailed $P < 0{\cdot}05$ by paired t test) except stimulation with low intensity after gallamine injection.

with 0·5 ms pulses and gradually reached the maximum response at about 10 V (Fig. 4*A*). Recorded changes in compound action potentials showed that the threshold intensity for C fibre activation was also 5 V with 0·5 ms pulses (Fig. 4*B*). Similar experiments on four cats showed threshold intensities after paralysis ranging between 3 and 5 V with 0·5 ms pulses. At the same range of intensities, the C fibre volley began to appear. This range of intensities was equivalent to 200–300 times that of threshold intensity for activation of α-motoneurone axons. Therefore, it is reasonable to assume that the C fibres are responsible for the pressor response elicited by stimulation of the peripheral stump of the cut ventral root in the paralysed animal.

Involvement of ventral root afferents in the production of the pressor response was demonstrated further in the experiment shown in Fig. 5. The pressor response elicited

by high intensity stimulation disappeared when the ventral root was pinched with a pair of forceps between the stimulating electrodes and the ground lead, although otherwise the same recording and stimulating conditions were maintained (Fig. 5*C*). The response reappeared when the ventral root was stimulated after moving the stimulating electrodes distal to the pinched area (Fig. 5*D*). Furthermore, pinch itself,

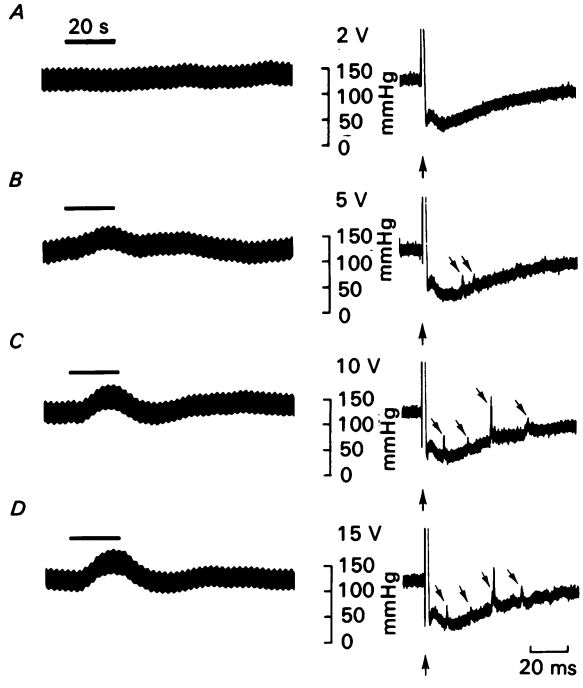

Fig. 4. Comparison of threshold intensity of stimulus for pressor response with that for evoking C fibre discharges. The distal stump of the cut S1 ventral root was stimulated for 20 s (0·5 ms pulses, 20 Hz) with varying intensities as indicated in the Figure while blood pressure responses were observed in a paralysed animal (left panel). C fibre activities (indicated by oblique arrows) recorded from the dorsal ramus of the spinal nerve during ventral root stimulation with single pulses at corresponding intensities are shown (right panel). These records were obtained after single stimulus; conduction distance was 25 mm. Upward arrows indicate times of stimuli. Note that thresholds for both the pressor response and C fibre activation were about 5 V.

as a form of mechanical stimulation of the ventral root, can affect blood pressure (Fig. 5*B*). These results indicate that the blood pressure responses to ventral root stimulation were not induced by current spreading to adjacent neural structures but by activation of fibres in the ventral roots. Repetition of this pinch experiment in four animals produced the same results.

Since the results strongly suggest that activation of non-myelinated fibres in the ventral root causes a pressor response, we next investigated the pathway of these fibres. First, we compared blood pressure responses to stimulation of the central stump with responses to stimulation of the peripheral stump after cutting the ventral root midway between the spinal cord and the dorsal root ganglion. Stimulation of

the central stump elicited no change in arterial blood pressure (Fig. 6*A*), whereas identical stimulation of the peripheral stump elicited a pressor response. These results indicate that the fibres eliciting the pressor response do not enter the spinal cord directly through the ventral root; rather, they travel distally toward the dorsal root ganglion. When these experiments were repeated in four animals, the same results were obtained.

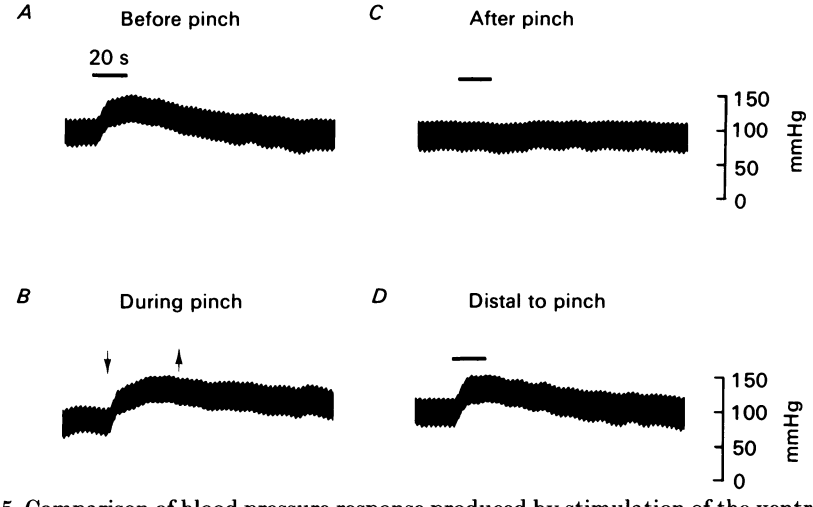

Fig. 5. Comparison of blood pressure response produced by stimulation of the ventral root before and after nerve block in a paralysed animal. *A*, as a control, the blood pressure response elicited by stimulation of the distal stump of a cut L7 ventral root (500 *T*, 50 Hz, for 20 s) is shown. *B*, a pressor response was also elicited by mechanical pinching of the ventral root with a pair of forceps between the cathode and the ground lead (two arrows indicate beginning and end of pinching). *C*, after pinching the ventral root, stimulation identical to that in *A* failed to produce a pressor response. *D*, the response reappeared when the ventral root was stimulated at a site distal to the pinched area.

Next we examined whether the ventral root fibres travelled any appreciable distance into the peripheral nerve. Sectioning the ipsilateral sciatic nerve at a level just proximal to the site where the hamstring nerve exits did not affect the blood pressure response elicited by stimulating the peripheral stump of the L7 ventral root in six animals (Fig. 6*B*). The sciatic nerve was the only important peripheral nerve left intact with access to the stimulated L7 ventral root, since all other major peripheral nerves were cut. These results further indicate that the pressor response to high intensity stimulation was not elicited by interaction between the activated motor axons and afferents originating from the muscle. Also, the ventral root fibres responsible for the pressor response did not travel into the peripheral nerve for any appreciable distance. All six experiments conducted yielded identical results.

Cutting the dorsal root of the same spinal segment, however, abolished the pressor response elicited by stimulation of the peripheral stump of the cut ventral root (Fig. 6*C*). These results lead to the conclusion that afferent information carried by ventral root fibres enters the spinal cord via the dorsal root, presumably through the dorsal root ganglion. The same results were obtained in four experiments.

The blood pressure response to stimulation of ventral roots showed spatial and temporal summations. The blood pressure increased to the same extent during stimulation of either the left or the right 7th lumbar ventral root. A significantly larger increase in blood pressure resulted when both ventral roots were stimulated simultaneously. Similar summation of responses occurred when the ventral roots of

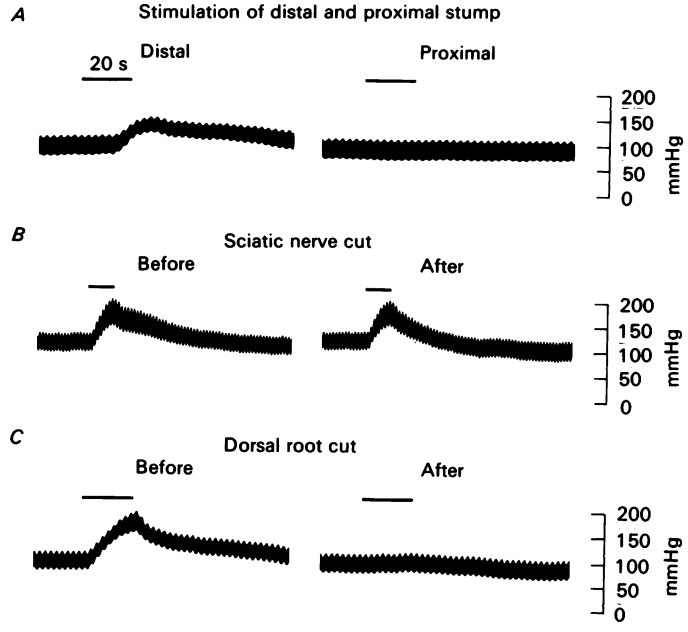

Fig. 6. Pathway of fibres in the ventral root responsible for the pressor response. *A*, *B* and *C* depict results obtained from three different experiments. *A*, the S1 ventral root was cut about midway between spinal cord and dorsal root ganglion; two sets of stimulating electrodes (including ground leads) were placed on both proximal and distal stumps (the S1 ventral root was used for this study because it is longer than the L7 ventral root). Stimulation of the distal stump produced a pressor response, whereas no response was obtained by stimulation of the proximal stump despite application of identical stimuli. *B*, cutting the sciatic nerve did not influence the pressor response elicited by stimulation of the distal stump of the cut L7 ventral root. The femoral and hamstring nerves were cut to restrict peripheral output from the L7 ventral root to the sciatic nerve. *C*, cutting the L7 dorsal root abolished the pressor response elicited by stimulation of the distal stump of the cut L7 ventral root. All time bars above blood pressure tracings indicate 20 s stimulus duration.

two different segments (L7 and S1) were stimulated on the same side. Arterial blood pressure changed little, if any, following stimulation of the ventral root with frequencies of 2 Hz or less. Stimulation at 5 Hz elicited a definite change in blood pressure; the response increased as frequency was raised to 20 Hz. In general, the arterial blood pressure response to stimulation of ventral root C fibres could be induced by a minimum frequency of 2 Hz; the frequency for a maximum response ranged from 10 to 20 Hz.

DISCUSSION

The present study has demonstrated that electrical stimulation of the peripheral stump of a cut ventral root produces an increase in systemic arterial blood pressure in the cat. Furthermore, two separate components of this pressor response were identified: (1) a smaller response that was elicited by low intensity stimulation of the ventral root and was abolished after paralysing skeletal musculature; (2) a larger response that was elicited by high intensity stimulation of the ventral root and persisted after paralysing muscle. There have been many previous reports on the increase in arterial blood pressure produced by stimulation of spinal ventral roots (Coote *et al.* 1971; McCloskey & Mitchell, 1972; Fisher & Nutter, 1974; Kaufman *et al.* 1983). The pressor response observed in these previous studies resulted from activation of afferents by the muscle contractions induced by stimulation of the ventral root. Supporting evidence includes elimination of the response by cutting the dorsal root that innervates the exercising hind limb (Coote *et al.* 1971; McCloskey & Mitchell, 1972) and disappearance of the response after muscular paralysis (Coote *et al.* 1971). Therefore, the previously reported pressor response is equivalent to the first component of the response reported in our study. The present study identified a much larger second component that only appeared when the ventral root was stimulated strongly. The stimulus intensities employed in earlier studies ranged from 5 to 30 times the thresholds of α-motoneurone axons. Although this intensity may be sufficient to induce muscle contraction fully, it is certainly not enough to elicit the second component of the pressor response, which requires a stimulus intensity of 200–300 *T*. This high stimulus intensity requirement may be the reason the second component of the response was not observed in previous studies. The first and second components differ not only in the method of elicitation but also in their underlying mechanisms. The second component is most likely mediated by direct activation of fibres in the ventral root that enter the spinal cord via the dorsal root. Several observations support this contention: (1) the response persisted even after paralysis of muscle or severance of the sciatic nerve, the only major peripheral nerve left intact to connect with the stimulated ventral root; (2) the response disappeared after the dorsal root of the same segment was sectioned; and (3) the response was no longer elicited when the ventral root was pinched distal to the stimulating site. Furthermore, the fact that the same threshold for eliciting the pressor response and for activating C fibres during stimulation of the ventral root suggests that the activated fibres that are responsible for the response are non-myelinated fibres.

Although the existence of a large number of non-myelinated afferent fibres in the ventral root is clear (see a review by Coggeshall, 1980), the physiological role of these fibres remains in question. There are only a few reports offering evidence that activation of ventral root afferent fibres may influence any part of the central nervous system. The best evidence, perhaps, may be found in the report of Longhurst *et al.* (1980), indicating that a small portion of the pressor response induced by stimulation of the sciatic nerve might be mediated by ventral root afferents, and in the study of Voorhoeve & Nauta (1983), who observed an increased firing rate of motor axons after intra-arterial injection of bradykinin in dorsal rhizotomized cats. Recently, Chung *et al.* (1983, 1985) reported the excitation of dorsal horn cells by stimulation of the ventral root.

Although the precise arrangement of ventral root fibres activated in the present study is not known, the fibres that enter the spinal cord directly through the ventral root (Maynard, Leonard, Coulter & Coggeshall, 1977; Light & Metz, 1978; Longhurst *et al.* 1980) were not involved as evidenced by the lack of pressor response when the proximal stump of the cut ventral root was stimulated. Another study from our laboratory also showed a failure to activate dorsal horn cells when the proximal stump of the cut ventral root was stimulated (Chung *et al.* 1985). The existence of fibres in the ventral root that enter the spinal cord via the dorsal root was further supported by demonstrating the presence of fibres in continuity between the dorsal and the ventral roots (Kim & Chung, 1985). It should be noted that the early morphological studies (Coggeshall *et al.* 1974, 1975) and experiments involving recordings from ventral root single fibres and their peripheral receptive fields (Clifton *et al.* 1976; Coggeshall & Ito, 1977) did not prove that these fibres entered the spinal cord directly through the ventral root. The results of these studies merely indicated the presence of fibres in the ventral root that have their cell bodies in the dorsal root ganglia and that have peripheral processes.

Activation of peripheral C fibres elicits somato-sympathetic reflexes and, consequently, induces changes in arterial blood pressure (Hunt, 1895; Ranson, 1921; Johansson, 1962; Coote & Perez-Gonzalez, 1970; Koizumi *et al.*, 1970; Schmidt & Weller, 1970; Chung & Wurster, 1976). The somato-sympathetic reflex shows both temporal (Schmidt & Weller, 1970; Chung, Webber & Wurster, 1979) and spatial summation (Chung & Wurster, 1978). In our study, the pressor response induced during high intensity stimulation of the ventral root displayed all the characteristics of the somato-sympathetic reflex. Therefore, the ventral root afferent fibres seem to have similar properties to other somatic afferent fibres in regard to eliciting sympathetic reflexes.

This work was supported by grants NS 21266, NS 18830 and NS 11255 from the National Institutes of Health. J. M. Chung is the recipient of a PHS research career development award NS 00995. J. Kim was supported in part by an alumni fund from Seoul National University, College of Medicine, Seoul, Korea. H. K. Shin was supported in part by Hanyang University, College of Medicine, Seoul, Korea.

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