Properties of the Arterial Pressor Response Induced by Stimulation of the Ventral Root Afferent Fibers in the Cat

Jun Kim, Sang Ah Seoh* and Ho Kyung Sung

*Departments of Physiology, College of Medicine, Seoul National University and Kyungsang National University**

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ABSTRACT

In an attempt to characterize the ventral root afferent fibers, arterial blood pressure responses to stimulation of the ventral root (VR) were observed in anesthetized cats. Effects of the morphine administered either intravenously or direct spinally and of the spinal lesions on the pressor responses were compared.

Followings are the results obtained.

1) Stimulation of the VR with C-strength, high frequency stimuli evoked a marked pressor response. No depressor response, which had been reported during peripheral nerve stimulation, was observed during VR stimulation with low frequency.

2) Acute cervical spinalization abolished the pressor response, indicating the involvement of supraspinal mechanism.

3) The ascending spinal pathways of the pressor response were located in the dorsolateral funiculus bilaterally.

4) Intravenously administered morphine exaggerated the pressor response to VR stimulation, while direct spinally administered morphine suppressed it.

From the above results it was concluded that the ventral root afferent fibers have more similar properties to muscular C-afferent fibers than to cutaneous C-fibers.

Key Words: Ventral root afferent, Pressor response, Morphine, Spinal lesion.

INTRODUCTION

During the past decade it has been rather well established that there are a large number of unmyelinated fibers in the spinal ventral root of mammals including human (Coggeshall et al, 1974; 1975; Coggeshall, 1980). These fibers are found to be primary afferent fibers carrying predominantly nociceptive informations and have their receptive fields in the visceral as well as somatic structures (Clifton et al, 1976; Coggeshall & Ito, 1977). Recently axons with a substance P-like immunoreactivity were observed in the lumbosacral ventral root of cat, further supporting that they are sensory in nature (Dalsgaard et al, 1982; Risling et al, 1984). If these fibers enter the spinal cord directly through the ventral root, then the so called "Law of Bell and Magendie" should be tested again. In this context a number of studies have claimed that these fibers

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enter the spinal cord through the ventral root (Maynard et al, 1977; Yamamoto et al, 1977; Light & Metz, 1978; Hosobuchi, 1980; Longhurst et al, 1980). As far as electrophysiological evidences are concerned, however, most of the ventral root afferent (VRA) fibers travel distally toward the dorsal root ganglia to enter the spinal cord through the dorsal root (Chung et al, 1983; 1985; Endo et al, 1985; Kim & Chung, 1985; Shin et al, 1985).

When the distal stump of cut ventral root was stimulated, a variety of physiological responses were produced. These include: 1. activation of many spinal neurones (Chung et al, 1983; 1985; Kim et al, 1988), 2. elevation in arterial blood pressure (Chung et al, 1986; Kim et al, 1986), 3. long postsynaptic potentials in motoneurones (Endo et al, 1985), 4. flexion reflexes (Shin et al, 1985). In spite of all these studies, however, much more investigations are needed for ascertaining the mechanism and physiological roles of the VRA systems.

The purpose of this study was to extend the previous studies for better understandings of the central actions of the VRA fibers. For this, arterial blood pressure responses to the activation of VRA were observed and effects of the morphine and naloxone were studied. In some experiments effects of acute cervical transection were observed.

METHODS

Adult cats (2.0-3.0 kg) of either sex were used. Animals were anesthetized with α-chloralose (60-80 mg/kg, i.p.). After an hour of induction period, a tracheostomy was performed. The animal was paralyzed with pancuronium bromide (Mioblock, 0.4 mg/hr) and ventilated with a ventilator. Systemic arterial blood pressure was monitored through a cannula inserted into the common carotid artery. The cannula was connected to a Statham pressure transducer. To eliminate the baroreceptor mediated reflex compensation of arterial blood pressure, the carotid sinuses were denervated bilaterally and also the vagosympathetic trunks were cut. The elevation of control blood pressure and the disappearance of blood pressure response to bilateral carotid artery occlusion confirmed the complete denervation. A cannula was inserted into the jugular vein for drug application. Throughout the experiment, rectal temperature was maintained near 37.5℃ by an electric blanket.

The lumbar spinal cord was exposed by a laminectomy. Ventral root of the L7 spinal segment was traced intradurally and cut near the spinal cord. In the ipsilateral hindlimb the common peroneal, sural and medial gastrocnemius nerves were separated for electrical stimulation. Heated mineral oil pools were made over the exposed spinal cord and hindlimb. In some experiments animals were spinalized by transection of the cervical cord.

Electrical Stimulation: The peripheral stump of cut ventral root was placed on a tripolar stimulating electrode, the most distal lead of it (the closest one to the dorsal root ganglia) was grounded to prevent the current spreading to nearby tissues. The separated peripheral nerves were placed on tripolar electrodes. In these cases the nerves were not cut but in continuity and the most central lead were grounded. The cord dorsum potential was recorded with platinum ball electrode placed on the dorsal surface of L7 spinal cord to monitor whether the current stimulating the ventral root was spread or not.

The threshold intensity for α-motoneurone activation was determined by observing the compound action potential of peripheral nerves activated by stimulation of the ventral root. It ranged between 15 ~20 and 8~12 uA using square pulses of 0.1 and 0.5 ms duration. When stimulating with supra C-strength to fully activate the unmyelinated fibers in the ventral root and peripheral nerves, 5 mA with 0.5 msec pulses were applied, since the threshold for ventral root afferent activation ranged between 200 ~300 times threshold for α-motoneurone activation

(Chung et al, 1986).

Changes in arterial blood pressure during stimulation of the ventral root and the peripheral nerves were observed and effects of the morphine, naloxone and cervical transection upon them were compared. Morphine and naloxone were administered directly to the spinal dorsal surface or intravenously injected. Some spinal lesions were made on the dorsal sulcus area and the dorsal part of lateral funiculus to identify the ascending spinal pathways of the ventral root afferent information eliciting changes in the arterial blood pressure. At the end of the experiment the spinal cord was removed and prepared for histological examination to confirm the spinal lesions.

RESULTS

The first series of the experiments was directed towards eliciting the blood pressure responses to electrical stimulation of the distal stump of cut ventral root with varying frequencies (0.5～50 Hz). As already mentioned, supra C-strength intensities of stimulus were required to evoke any change in arterial blood pressure. Fig. 1 shows a typical example of the responses. When an L7 ventral root was stimulated with 5 mA, 0.5 msec square pulses of 1～2 Hz for 20 secs, there was no change in arterial blood pressure, while 10～50 Hz stimulation evoked apparent pressor responses. Peak pressor response was about 35 mmHg at 20 Hz stimulation. The mean value of peak responses in the first series of 9 cats was 20.8±6.2 (s.e.) mmHg. The lower left trace in Fig. 1 shows comparable result obtained during the stimulation of common peroneal nerve with 20 Hz, supra C-intensity.

The frequency response curves of the L7 ventral root, medial gastrocnemius (MGS) and sural nerve to the electrical stimulation with C-strength were constructed on the Fig. 2. The values plotted on the

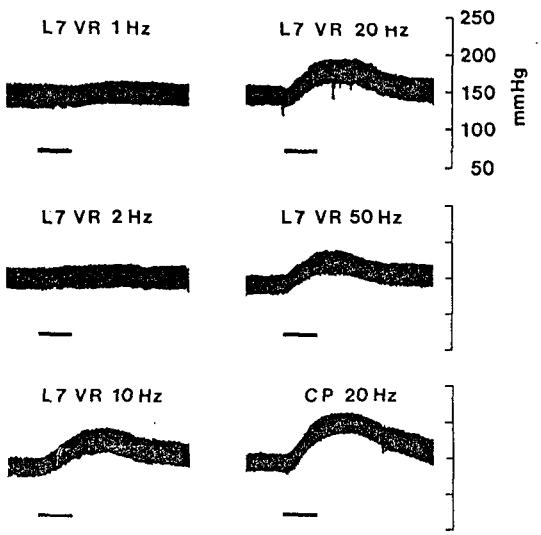

Fig. 1. Arterial blood pressure response to ventral root stimulation with varying frequencies. Ventral root was stimulated with C-strength, 0.5 ms. Square pulses were applied for 20 secs. For comparison the response with common peroneal nerve stimulation is shown. Horizontal bars indicate 20 sec time scale.

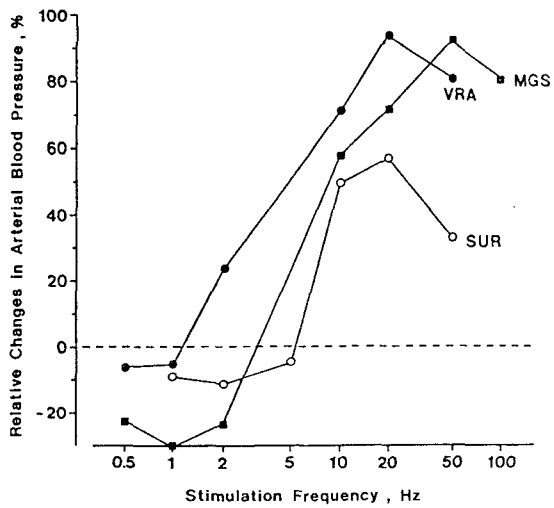

Fig. 2. Frequency responses of the arterial blood pressure to stimulation of ventral root (VRA), medial gastrocnemius nerve (MGS) and sural nerve (SUR). Changes in arterial blood pressure is expressed as percentile to maximum pressor responses to each nerve stimulation. Abscissa was expressed with logarithmic scale.

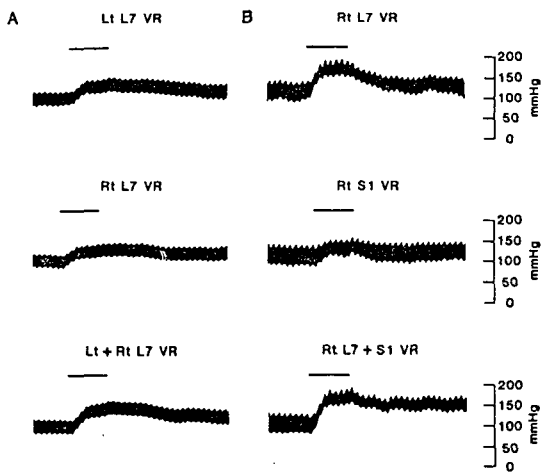

Fig. 3. Spatial summation of pressor responses elicited by stimulation of the ventral roots. A. Blood pressure responses during stimulation of left, right and both left and right L7 ventral root. B. In another experiment, pressor responses during stimulation of L7, S1 and both L7 and S1 ventral roots. Note that stimulation of two ventral roots produced a larger response in both A and B.

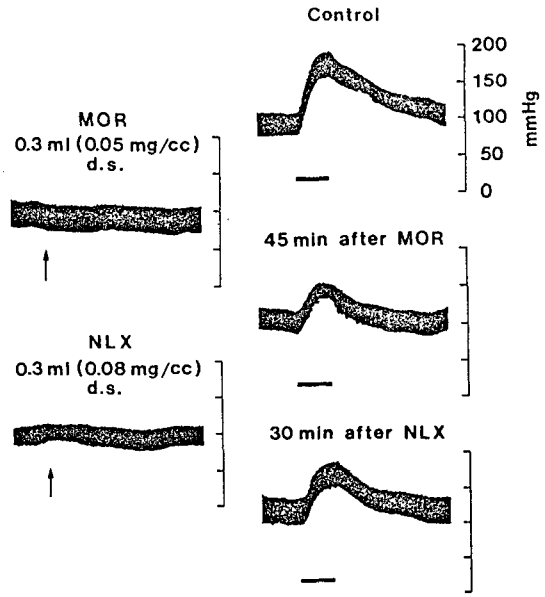

Fig. 4. Effects of morphine and naloxone administered direct spinally on the arterial blood pressure responses to stimulation of the ventral root. The pressor response decreased markedly by direct spinal morphine and naloxone partially reversed the morphine effect. Horizontal bars indicate time intervals during which the ventral root was stimulated. d.s.: direct spinal administration, MOR: morphine, NLX: naloxone.

graph are the relative amplitude (%) to peak pressor response to each nerve. As can be seen in the figure the peak response to ventral root afferent activation was at 20 Hz, while that to MGS was at 50 Hz. The pressor response to sural nerve stimulation did not show prominent peak to any particular stimulation frequency as others did, but responded to rather broad frequency range (10～50 Hz). The stimulation of peripheral nerves, especially in the case of MGS, with lower frequencies resulted in depressor responses. On the contrary, stimulation of the ventral root afferent fiber did not evoke any significant depressor response.

The pressor response to stimulation of the ventral root showed spatial summation (Fig. 3). Blood pressure increased to the same extent during stimulation of either the left or the right L7 ventral root (Fig. 3A). A significantly larger increase in blood pressure was resulted in when both ventral roots were stimulated simultaneously. Similar summation occurred when the ventral roots of two different segments (L7 and S1) were stimulated on the same side (Fig. 3B). Usually the increase in blood pressure evoked by the S1 VRA activation was smaller than that evoked by the L7 VRA activation, probably due to the marked difference in the size of these two roots.

In Fig. 4 & 5, the effects of morphine on the pressor response to activation of the VRA fibers were represented. Since morphine is known to exert its action on the spinal cord as well as supraspinal structures (Barton et al, 1980), morphine was administered either directly to the dorsal surface of lumbosacral spinal cord (0.1-0.3 ml, 0.2 mg/ml) or intravenously (2 mg/kg). Morphine direct to the spinal cord itself did not change the resting arterial blood pressure, but decreased the pressor response

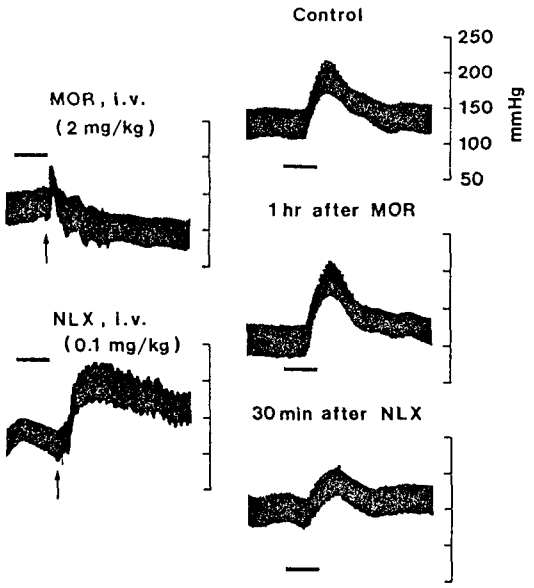

Fig. 5. Effects of morphine and naloxone administered intravenously on the arterial blood pressure responses to stimulation of the ventral root. The pressor response was exaggerated by intravenous morphine. Note that intravenous morphine depressed the arterial blood pressure and naloxone elevated the depressed arterial blood pressure even over the control level. Horizontal bars indicate 20 sec time scale. MOR: morphine, NLX: naloxone.

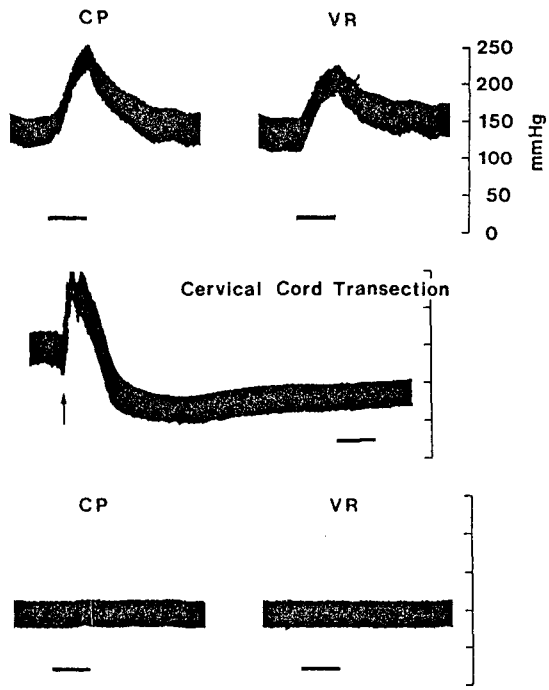

Fig. 6. Effect of acute cervical spinalization on the arterial pressor response elicited by stimulation of the common peroneal nerve and ventral root. Spinal cord was transected at C4 level. Arterial blood pressure decreased immediately and not recovered upto an hour. After transection the arterial pressor responses were abolished. Horizontal bars indicate 20 sec time scale. CP: common peroneal, VR: ventral root.

to VR stimulation progressively, down to a half value of control 45 minutes after administration (Fig. 4). Naloxone (0.3 ml, 0.08 mg / ml), the morphine antagonist, reversed partially the effect of direct morphine administration. Intravenous morphine, in contrast to direct spinal administration, depressed the systemic arterial blood pressure immediately and persistently (Fig. 5), and pressor response to the VR stimulation was exaggerated by intravenous morphine. Naloxone elevated the depressed systemic arterial blood pressure even over the control level and the exaggerated response to the VR stimulation was partially recovered.

The next step of the experiment was the determination of ascending pathways of these VRA informations to the higher centers. At first several animals (n=4) were spinalized by cervical cord transections. Fig. 6 represents one such experiment. After arterial pressor responses to stimulation of the L7 ventral root as well as the left common peroneal nerve were confirmed, cervical spinal cord at the level of C3-C5 was ligated with a cotton thread for 20~30 sec. By this the spinal cord was functionally transected while the blood vessels were not cut. Spinalization produced severe depression in the arterial blood pressure immediately and remained at low blood pressure. The lower traces in Fig. 6 were taken 1 hr after cervical transection. Stimulation of

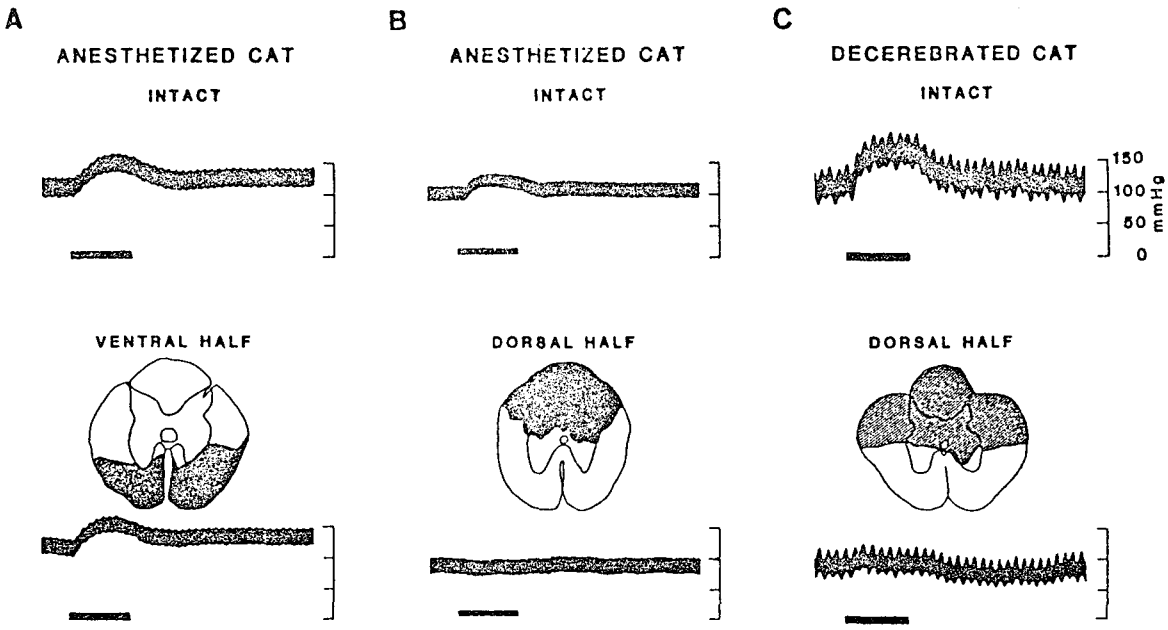

Fig. 7. Effects of lesions in the ventral and dorsal halves of the spinal cord on the arterial pressor response to ventral root stimulation. While ventral half lesion did not affect the pressor response (A), dorsal half lesion completely abolished the pressor response in both anesthetized (B) and decerebrated (C) cats. The shaded area in cross sections of spinal cord indicates the damaged region. Horizontal bars indicate 20 sec time scale.

the ventral root and common peroneal nerve did not evoke any change in arterial blood pressure, indicating that these pressor responses involve supraspinal structures.

In the next series of experiment, the ascending pathways for the pressor response evoked by the VRA activation were located by making lesions in the spinal cord. Fig. 7 shows the typical results in anesthetized and decerebrated animals. When the distal stump of the cut L7 ventral root was stimulated before making any spinal lesion, the arterial blood pressure was elevated by 40 mmHg. A lesion in the ventral half of the spinal cord at the L1-L2 level did not change the pressor response significantly (Fig. 7A). However, an additional lesion in the dorsal half of the spinal cord completely abolished the pressor response. When a lesion was made in the dorsal half of the spinal cord in another animal, the pressor response disappeared as shown in Fig. 7B. In this case a mechanical manipulation of the remaining ventral spinal cord resulted in a pressor response. Similar results were obtained in decerebrated cats, although the baseline fluctuations in the arterial blood pressure was more promiment in this preparation (Fig. 7C).

The ascending spinal pathways of VRA information in the dorsal spinal cord were further localized by small lesions on the various part of the dorsal half of the spinal cord at the L1-L2 level. As shown in Fig. 8 intact animal produced a pressor response of 35 mmHg to the VRA stimulation. When a lesion was made on the dorsal part of the lateral funiculus (DLF) of the ipsilateral side, the activation of the VRA resulted in a 20 mmHg pressor response. An additional lesion on the contralateral DLF resulted in further decrease in pressor response by 10 mmHg. When the entire dorsal half was damaged, the pressor response to VRA stimulation was abolished. A dorsal column lesion alone, however, did not result in significant changes in pressor response.

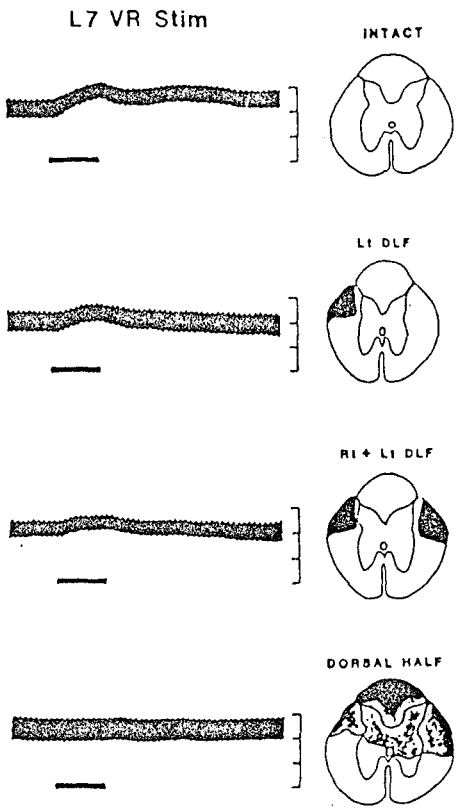

Fig. 8. Effect of lesions in the dorsolateral funiculus on the pressor response to ventral root stimulation. By left DLF lesion, pressor response decreased to almost half of intact response and additional right DLF lesion abolished remaining response. A subsequent lesion in the dorsal half of the spinal cord abolished the pressor response completely. Horizontal bars indicate 20 sec time interval during which ventral root was stimulated. DLF: dorsolateral funiculus.

These results suggested that the ascending pathways carrying informations from the VRA system were in the dorsolateral funiculus, bilaterally.

DISCUSSION

It is no longer doubtful that there are a significant number of unmyelinated, primary afferent fibers in the spinal ventral root of mammals including human (see Coggeshall's review, 1980). This fact brought about serious questions against the so called "Law of Bell & Magendie", which stands for the fact that sensory axons enter the spinal cord through the dorsal root and motor axons leave through the ventral root. Horseradish peroxidase (HRP) staining experiments (Maynard et al, 1977; Yamamoto et al, 1977; Light & Metz, 1978) demonstrated that some primary afferent fibers enter the spinal cord through the ventral root. Other clinical (Hosobuchi, 1980) as well as physiological studies (Longhurst et al, 1980; Voorhoeve & Nauta, 1983) supported this contention.

However, the majority of the VRA fibers seem to enter the spinal cord through the dorsal root. Magendie, himself, observed that stimulation of the ventral root could elicit pain reactions, which were abolished by dorsal root section (Magendie, 1822). This phenomenon is usually called "recurrent sensibility" and was also demonstrated in human (Frykholm et at, 1953). Furthermore, stimulation of the distal stump of the cut ventral root in a series of physiological studies resulted in: 1. activation of dorsal horn cell receiving convergent inputs from periphery (Chung et al, 1983; 1985), 2. elevation of systemic arterial blood pressure (Chung et al, 1986; Kim et al, 1986), 3. production of long-latency postsynaptic potentials in motoneurons (Endo et al, 1985) and spinal reflex (Shin et al, 1985). In addition some morphological evidences demonstrated that no unmyelinated axons were found to enter the spinal cord through the ventral root, but many of the unmyelinated axons in the ventral root made U-turns or entered the spinal pia mater (Risling et al, 1984). Our present results also supported the above.

The mechanism of pressor response to the activation of VRA is not known. The VRA informations might act either on the preganglionic sympathetic neurons in the thoracic spinal cord or on the supraspinal structures especially in medulla oblongata. Although there are some evidences that afferent

inputs from the spinal level to the sympathetic preganglionic neurons may exist (Corbett et al, 1971; Malliani et al, 1969; 1973), it is not known to what extent these spinal afferent projections are significant physiologically. As shown in Fig. 3, there was no change in arterial blood pressure response to stimulation of either VRA or common peroneal nerve after acute cervical transections. This result suggests that the VRA informations should be transmitted to the supraspinal structures to elicit any pressor response at least in acute experiments.

It is well known as a somatosympathetic reflex that the arterial blood pressure responds to the activation of peripheral nerve (see Sato & Schmidt's review, 1973). A somatosympathetic reflex would be elicited exclusively by strong, noxious stimuli and it shows both temporal (Schmidt & Weller, 1970; Chung et al, 1979) and spatial summation (Chung & Wurster, 1978). The activation of peripheral nociceptive nerves results in either a pressor or a depressor response according to the stimulation intensity and frequency. Whether the peripheral nerves activated are muscle afferents or cutaneous afferents is also an important factor. The pressor response is said to be mediated through the ascending spinal pathways in the dorsolateral sulcus area and the depressor response, in the dorsolateral funiculus (Chung & Wurster, 1976; Chung et al, 1979).

The pressor response to VR stimulation in present study, could be compared with other author's results of arterial blood pressure response to the activation of nociceptive peripheral nerves: 1. Stimulation of the ventral root with low frequency stimuli in present study did not evoke any depressor response. Others reported that cutaneous nerve stimulation with A δ-intensity, low frequency (Chung & Wurster, 1976; Chung et al, 1979; Iwamoto et al, 1983) and muscle afferent with C-strength, low fequency (Lim et al, 1987; Kim et al, 1988) could evoke depressor responses. 2. The ascending pressor pathways were in the DLF bilaterally, which confirmed the previous report (Kim et al, 1986). It is usually known that cutaneous pressor inputs are transmitted through the dorsolateral sulcus area (DLS) and depressor input, through the DLF region (see Mitchell's review, 1983). Some ascending pressor pathways are also found in lateral funiculus (Kozelka et al, 1981; Kozelka & Wurster, 1985) and in the ventral half of the spinal cord (Iwamoto et al, 1984). 3. VRA inputs were summated spatially which were comparable to the result of Chung & Wurster's study (1978). 4. Direct spinally administered morphine suppressed the pressor response while intravenous morphine exaggerated it. This result is similar to those observed during activation of nociceptive cutaneous and muscle afferent nerves (Kim et al, 1988: Kwon, 1988). So far we do not know the separative effects of morphine on the somatosympathetic reflexes. Suppression of the pressor response by the morphine administered direct spinally indicates that morphine certainly has direct inhibitory action on the spinal processing of nociceptive informations. Supraspinal action of the morphine depends upon choice of species, site of injection, state of arousal, respiration, type of opioids and dose of opioids (Faden & McIntosh, 1986). Among the above the state of arousal seems to be the most important one. Since the nociceptive inputs are the most powerful stimuli for the arousal, it is possible that the intravenous morphine, although it depressed the resting arterial blood pressure, could not affect the somatosympathetic reflex evoked by noxious peripheral nerve stimulation.

From the above discussions it seems that the ventral root afferent fibers producing pressor response might have similar properties to the muscle C afferent fibers have, but not similar to the cutaneous C afferent fibers.

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═ 국문초록 ═

고양이 척수 전근내 감각신경 자극으로 유발된 승압반응의 생리학적 특성

서울대학교 및 경상대학교* 의과대학 생리학교실

김 전·서 상 아*·성 호 경

척수 전근내 구심신경의 특성을 알아보기 위하여 고양이에서 L7 척수 전근을 전기자극하여 유발되는 동맥혈압의 변동을 관찰하였다. morphine을 정맥내 혹은 직접 척수에 투여하여 그 효과를 관찰하였으며 척수에 부분적인 손상을 가하여 척수 전근의 구심정보가 척수의 어느 부위를 통하여 중추로 올라가 승압반응을 유발하는지를 결정하여 다음과 같은 결과를 얻었다.

1. 척수전근을 C-강도, 높은 빈도로 자극하면 현저한 승압반응을 유발하였으며 낮은 빈도로 자극할 때에는 피부 혹은 근육감각신경을 자극할 때 보이는 감압반응이 유발되지 않았다.

2. 경수부위를 절단하였을 경우 승압반응이 소실되어 승압반응이 전적으로 척수 상부구조를 통하여 일어남을 알 수 있었다.

3. 승압 반응의 척수내 상행경로는 척수의 dorsolateral funiculus에 양측성으로 존재하였다.

4. 정맥내로 주사한 morphine은 척수 전근내 구심섬유가 자극되어 유발되는승압반응을 강화시켰으나 척수에 직접 투여한 morphine은 승압반응을 억제하였다.

이상의 결과로부터 척수 전근내에 존재하는 구심성 섬유들은 기능적으로 근육 감각신경 중에 승압반응을 유발하는 C-섬유와 유사한 성질을 갖는다고 사료된다.