A Role for the Dorsal Column in Nociceptive Visceral Input Into the Thalamus of Primates

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Al-Chaer, Elie D., Yi Feng, and William D. Willis. A role for the dorsal column in nociceptive visceral input into the thalamus of primates. J. Neurophysiol. 79: 3143–3150, 1998. A possible role of the dorsal column (DC) in the processing of visceral pain has gained attention after studies in the rat have revealed that the DC transmits a major part of the pelvic visceral nociceptive input from the colon into the thalamus. Furthermore, clinical interventions aimed at interrupting ascending DC axons near the midline were successful in relieving the pain suffered by patients with cancer of the pelvic organs. The purpose of this study was to check whether a DC lesion in monkeys would reduce the responses of thalamic neurons to graded colorectal distension (CRD) as in rats. Experiments were done on anesthetized male monkeys (Macaca fascicularis). Extracellular single cell recordings were made in the ventrolateral complex of the thalamus, mainly the ventral posterolateral (VPL) nucleus, in response to visceral and cutaneous stimulation. Of 80 VPL cells isolated, CRD activated 25, inhibited 25, and had no effect on 30 neurons. The responses of six visceral sensory VPL neurons were recorded before and after a lesion of the DC at or above the T10 spinal segment. Lesions of other spinal tracts were made after the DC lesion. The results show that the DC lesion significantly reduced the responses of the thalamic neurons tested with CRD by >50%. Lesions of other tracts did not have a consistent effect. These results corroborate findings in the rat and support the proposal that the DC plays an important role in transmitting nociceptive visceral input into the thalamus and subsequently in visceral pain.

INTRODUCTION

The dorsal column-medial lemniscus (DC-ML) system traditionally has been viewed as a pathway responsible for the discriminative aspects of tactile sensations and for kinesthesia. This concept was adopted at the turn of this century (Brown-Séquard 1868; Head and Thompson 1906; Stanley 1840) and was based on the pathological alterations seen in certain disease states associated with DC lesions. However, a strong body of evidence now suggests that the DC plays an important role in viscerosensory transmission and in visceral pain.

Based on clinical observations (Hirshberg et al. 1996) and earlier experimental studies (Amassian 1951; Berkley and Hubscher 1995; Rigamonti and Hancock 1974, 1978; Sarnoff et al. 1948), our group conducted a number of experiments in rats that established that the DC is the major spinal pathway for nociceptive visceral input into the ventral posterolateral (VPL) nucleus of the thalamus (Al-Chaer et al. 1996a–c, 1997b–d). In a study that assessed the effect of spinal cord lesions on visceral input into the VPL nucleus, the role of the DC substantially outweighed that of the ventro-lateral columns (VLC) (Al-Chaer et al. 1996a). The visceral information was relayed in the dorsal column nuclei (DCN) because lesions of the nucleus gracilis (NG) dramatically reduced colorectal input onto neurons of the contralateral VPL nucleus (Al-Chaer et al. 1997b). Single cell recordings in the NG have shown that ~50% of the NG neurons can be activated by stimulation of one or the other of the pelvic visceral organs (Berkley and Hubscher 1995). Al-Chaer et al. (1996b) found that colorectal input into the NG is mediated largely by postsynaptic dorsal column (PSDC) neurons the cell bodies of which are found in and around lamina X. In fact, single cell recordings from PSDC neurons located near the central canal have revealed that the responses of these neurons to colorectal distension (CRD) and to colon inflammation resemble those of the NG and VPL neurons to the same stimuli (Al-Chaer et al. 1996a, b). Furthermore, colon inflammation similarly potentiates the responses of PSDC, NG, and VPL neurons to CRD, suggesting that these neurons belong to the same viscerosensory channel ascending from the level of the spinal cord to the brain stem and the thalamus (Al-Chaer et al. 1996c, 1997c,d).

The purpose of this study was to examine the role of the DC in transmitting nociceptive visceral input into the thalamus in monkeys. Single cell recordings were made from viscerosensitive VPL units before and after a lesion of the DC at the T10 spinal segmental level. The responses of these cells to CRD and to somatic stimulation were recorded before and after spinal lesions. The hypothesis tested was that the DC of the monkey plays an important role in the processing and transmission of viscerosensory information to the thalamus. The results obtained indicate that the DC does indeed play a major role in visceral input into the thalamus and that this role is more substantial than that of ventrolateral or dorsolateral spinal tracts. Preliminary results of this study have been reported in abstract form (Al-Chaer et al. 1997a).

METHODS

Experiments were done on eight adult male monkeys (Macaca fascicularis) weighing between 2 and 2.5 kg. The monkeys were sedated with ketamine (10 mg/kg im) and then anesthetized with 4% halothane in a mixture of oxygen and nitrous oxide (30:70). Tracheal and intravenous cannulae were inserted, and anesthesia was maintained initially by an infusion of α-chloralose (60 mg/kg iv) followed by a continuous infusion of pentobarbital sodium (5 mg·kg⁻¹·h⁻¹). The animals were paralyzed with pancuronium (0.2–0.25 mg·kg⁻¹·h⁻¹) and artificially respirated. A laminectomy
was performed to expose the spinal cord at the level of T10. Animals then were transferred to a stereotaxic frame in a shielded recording room, the skin and muscles of the back were retracted and the dura was cut and reflected to expose the cord. The exposed spinal cord then was covered with warm mineral oil. End tidal CO₂ was monitored and maintained at 4 ± 0.5%, and body temperature was kept near 37°C by a thermostatically controlled heating blanket. The arterial blood oxygen saturation level was monitored with a laser oxygen rectal probe and was kept at 98 ± 2%. The adequacy of the depth of anesthesia was evaluated by the examination of the pupillary reflexes, monitoring of the electrocardiogram and assessing the stability of the expired CO₂. A pneumothorax was performed to minimize respiratory movements during recording. Craniotomies exposed the cortical surface above both thalami.

**Stimulation**

The visceral stimulus used was CRD. It was adapted from the model used in rats (Gebhart and Sengupta 1996; see also Al-Chaer et al. 1996a,b). The stimulus was applied using an inflatable balloon inserted rectally. The balloon was constructed from a latex glove finger attached to a length of tygon tubing (10 cm). The tubing was connected, via a T connector, to a manual pump and to a pressure transducer to monitor stimulus intensity. The balloon was inflated and left overnight to overcome the tension in its walls. CRD consisted of consecutive inflations of the balloon to pressures ranging between 20 and 80 mmHg, applied in increments of 20 mmHg for 20 s every 4 min. CRD stimuli having an intensity of ≥40 mmHg are considered noxious (Ness and Gebhart 1988; Ness et al. 1990).

The cutaneous stimuli employed were brushing (BR) of the receptive field using a camel hair brush, an innocuous stimulus; pressure (PR), using a large arterial clip applied to a fold of skin, a stimulus that causes a sense of pressure if applied to human skin; and pinch (PI) using a small arterial clip that exerts a force of 550 g/mm², a distinctly painful stimulus if applied to human skin.

**Single cell recording**

Recordings from individual thalamic neurons were made using tungsten microelectrodes (125 μm shank; 12 MΩ). The electrode was inserted stereotaxically into the brain aiming at the VPL nucleus. It was lowered slowly while brief taps were applied to the contralateral hindlimb or the perineal area. When multiunit activity became distinctly audible on the audio monitor, the site coordinates were recorded and the electrode was moved in small increments until a VPL unit was well isolated. In two preparations, the electrode was inserted stereotaxically into the thalamus at the rostral-lateral end of a planned grid (AP: 9; L: 10) and was lowered gradually until a single cell was isolated. The electrode later was moved caudally or medially to explore a square grid (1 × 1 mm²). Once a thalamic unit was isolated, it was examined for cutaneous input, using low- and high-intensity cutaneous stimuli, and for visceral input using graded CRD stimuli. The cutaneous receptive field was mapped, and the unit’s response to CRD was determined. Units with visceral and cutaneous inputs usually had a cutaneous receptive field in and around the perineal area. Occasionally we recorded from a cell in the hindlimb area of the VPL nucleus that responded to CRD. The extracellular action potentials recorded were fed into a window discriminator and displayed on an oscilloscope screen. The output of the window discriminator was led into a data collection system (CED 1401+) and a personal computer to compile rate histograms or wavemark files using the Spike 2 software program. Responses of a VPL cell to consecutive applications of cutaneous stimuli (BR, PR, and PI) were recorded. The response to each intensity of CRD, on the other hand, was stored separately. Twenty seconds of baseline activity preceded the application of a distension stimulus. Each stimulus lasted 20 s. Four minutes were allowed to elapse between two consecutive stimuli. The responses were calculated as the difference between the rate of firing during the response and that during the baseline recording.

**Spinal cord lesions**

After isolating a single VPL unit that responded to both cutaneous and visceral stimuli and recording its responses, a lesion of the DC at the level of T10 was made, after which the responses of the cell to the same stimuli were tested again. The DC lesion was initially shallow and restricted to the midline. It was done using a sharp (B-D 22 G 1/2) needle. The needle tip was inserted into the midline of the fasciculus gracilis and moved 2–3 mm rostrocaudally under view through a surgical microscope. A more extensive DC lesion (wider and deeper) then was made at the same level, and the cell was tested again. For the second DC lesion, the needle was inserted into the dorsolateral sulcus to make it easy to crush the DC with fine forceps. The DC then was squeezed with the fine forceps. Twenty to 30 min later, a lesion of the VLC, ipsilateral to the recording site, was made at the same level as the DC lesion, and the cell was tested again. Subsequent lesions of the contralateral VLC and of the dorsolateral funiculi (DLF) were later made separately until the response of the VPL unit to CRD was totally abolished. For the VLC lesions, the cord was lifted using a fire polished glass rod and a needle or the fine forceps was inserted into the ventrolateral quadrant. DLF lesions were made using the forceps.

**Histology**

At the end of each experiment, a continuous current (1 mA for 20 s) was passed to mark the recording site. The spinal cord at the level of the lesions, and the brain were removed and put into a formalin solution (10%). The tissue was kept in 20% sucrose before frozen sectioning at 50 μm. The recording sites were identified, and the extents of the spinal lesions were determined by drawing enlarged projections of consecutive cord sections using a camera lucida.

**RESULTS**

A total of 80 neurons in the lateral thalami were isolated and tested for input from CRD and for somatic receptive fields. Twenty-five of these 80 cells (31%) were excited by CRD, and 25 were inhibited (31%). Twenty-six viscerosensitive cells (33%) could not be activated by somatic stimulation. On the other hand, 30 somatically activated cells did not respond to CRD (38%). Seventy-five percent of the viscerosensitive neurons were located in the caudal part of the VPL nucleus (VPLc) and 25% were located in the oral part of the VPL nucleus (VPLo). Figure I shows the recovered recording sites for eight VPL cells isolated in eight different monkeys (1 cell/monkey). Because most neurons were located in the area of representation of the lower body, most of them responded to cutaneous stimulation in the perineal region, the inner thigh, the caudal aspect of the hindlimb, or the tail. Three cells activated by CRD responded also to cutaneous stimulation on the forelimb. The cutaneous input onto these cells was largely innocuous. Only one VPL cell that was excited by CRD had a convergent high-threshold cutaneous input. Six viscerosensitive cells responded to deep tissue stimulation applied to the muscles of the leg or arm or to joint movement.
Neuronal response characteristics

For this study, a neuronal response to a specific stimulus was defined as an average change in the firing rate from baseline of at least three spikes/s during the application of the stimulus. For instance, a VPL neuron was considered to be activated by CRD, if during CRD of any intensity the average firing rate of the neuron increased by at least three spikes/s. On the other hand, a VPL neuron was judged to be inhibited by CRD if its firing rate dropped by at least three spikes/s below baseline. Neurons activated by CRD exhibited an increase in their responsiveness that correlated with the increase in stimulus intensity. The population stimulus response curve was approximated by a linear regression for all the neurons in each category. These regression lines show that the rate of change (slope) was similar across response types (excitatory and inhibitory; Fig. 2). This graded response was observed regardless of the threshold of activation of the VPL unit by CRD. The threshold of activation was defined as the lowest intensity of CRD that evoked a neuronal response and was determined with increasing steps of 20 mmHg of intracolonic pressure for the cells tested. The approximate accuracy is estimated to be 10 mmHg across the pressure spectrum (20–80 mmHg). Of the 25 VPL neurons excited by CRD, 13 had a threshold of 20 mmHg, 7 neurons were activated at 40 mmHg, 2 neurons were activated at 60 mmHg, and 3 neurons required 80 mmHg CRD. Of the 25 VPL neurons inhibited by CRD, 13 neurons responded at 20 mmHg, 5 neurons responded at 40 mmHg, 4 neurons at 60 mmHg, and 2 neurons at 80 mmHg. One VPL neuron showed a poststimulus inhibition that could be evoked with a CRD as low as 20 mmHg. The mean thresholds for VPL units with CRD input were 36 ± 4.2 mmHg (n = 25) for excitatory responses and 35.8 ± 4.2 mmHg for inhibitory responses. Vertical box plot displays the median, 10th, 25th, 75th, and 90th percentiles as the boxes with error bars and the 5th percentile as small circles (the percentile increases downward).
(mean ± SE; range 20–80 mmHg; n = 25) for inhibitory responses (Fig. 3). There was no obvious correlation between the threshold of activation by CRD and that by cutaneous stimuli, although most viscerosomatic neurons were low-threshold somatic cells.

Effects of spinal lesions

In six different preparations, spinal cord lesions were made after recording the responses of a VPL neuron to CRD and somatic stimuli. A lesion of the DC was made first, followed by a lesion of the ipsilateral VLC (iVLC) and, when appropriate, the contralateral VLC (cVLC) and the DLFs. Successive lesions were made until the neuronal response to CRD was abolished. Six viscerosensitive VPL units were isolated in six different monkeys and tested for cutaneous stimuli and CRD before and after a lesion of the fasciculus gracilis at the level of T10. Of the six viscerosensitive VPL cells, five were excited by CRD and one was inhibited. The cell inhibited by CRD had no somatic receptive field. Two of five cells activated by CRD responded to innocuous cutaneous stimuli applied to the anal and perianal area for one cell and to the inner aspect of the arm for the second cell. One cell responded to CRD and to high-intensity noxious stimulation applied to the scrotum and inner thigh junction. Another cell responded to CRD and to the application of shearing force to the rectal wall. The fifth cell was activated by flexion of the hind paw. The DC lesion decreased dramatically the responses of these cells to visceral stimulation. The responses of a viscerosensitive VPL neuron activated by CRD and cutaneous stimulation are illustrated in Fig. 4. The figure shows the thalamic location (Fig. 4A), reconstruction of the spinal cord lesions (Fig. 4B), spike shape (Fig. 4E), and responses of a VPL unit to graded colorectal distension (Fig. 4C) and to graded mechanical cutaneous stimuli (Fig. 4D) before and after the DC lesion and after an iVLC lesion. The responses were reduced dramatically by a DC lesion and totally abolished by a lesion of the iVLC. In two cases, a lesion of the DC greatly reduced the responses of viscerosensitive thalamic neurons to CRD and cutaneous stimulation. However, subsequent bilateral VLC lesions did not have a significant effect on the remaining components of the responses to CRD. Bilateral DLF lesions, made after the VLC lesions, abolished the

![Diagram](http://jn.physiology.org/content/118/6/3146/F4)

**Fig. 4.** Responses of a VPL neuron with dorsal column (DC) and VLC inputs. *A:* drawing of a cross-section through the posterior thalamus illustrating the recording site in the VPL nucleus. *B:* drawing of a cross-section through the spinal cord at the T10 segment illustrating the sites of a DC lesion and of a VLC ipsilateral to the recording side (iVLC) lesion. *C:* rate histograms of the responses of a thalamic neuron to graded CRD. Responses were recorded before and after consecutive spinal lesions (T10 spinal segment). *Bottom:* waveform traces of the CRD stimuli (20, 40, 60, and 80 mmHg). *D:* rate histograms of the responses to cutaneous stimulation (BR: brush; PR: press; PI: pinch). Stimuli were applied for 10 s each. Responses were recorded before and after consecutive spinal lesions made at T10 spinal segment. *E:* traces of spikes indicate no change in the spike shape before and after the lesions.
neuronal responses to CRD and cutaneous stimulation. One of the cases is illustrated in Fig. 5.

One cell that responded to cutaneous stimuli applied to the arm and to CRD was tested before and after the DC lesion, after the lesion of the iVLC and after the lesion of the cVLC. Its responses to CRD nearly were abolished by the DC lesion. When the iVLC lesion was made after the DC lesion, the remaining responses to visceral stimulation were abolished except for a small component of the response to 80 mmHg. However, the cell’s responses to cutaneous stimuli were not affected by any of the lesions.

In two cases, the responses of two viscerosensitive VPL neurons to CRD were considerably affected by a lesion of the iVLC made after the DC lesion. For instance, the DC lesion reduced the response to 80 mmHg in one case to ~75% of its initial rate and the following iVLC lesion eliminated the remaining component.

The effects of a lesion of the DC on the responses of a
Bruèggemann et al. 1994; Chandler et al. 1992) have shown that viscero-somatic neuronal responses to colorectal distension (CRD) were graded with stimulus intensity. A lesion of the DC, made at the T10 spinal segment, reduced the responses of five VPL neurons that were excited by graded colorectal distension. The DC lesion also dramatically attenuated the mean responses to CRD more dramatically than did the iVLC lesion. The DC lesion attenuated the responses to innocuous cutaneous stimuli applied to the lower areas of the body. The iVLC lesion abolished the response to noxious cutaneous stimulation applied to the inner thigh in one case.

**DISCUSSION**

In this study, approximately half of the viscerosensitive VPL neurons sampled were excited by CRD and half were inhibited. Responses to visceral distension were graded with stimulus intensity. A lesion of the DC, made at the T10 spinal segment, reduced the responses of five VPL neurons excited by CRD by >50% and completely abolished the response of 1 neuron that was inhibited by CRD. On the other hand, lesions of other spinal tracts did not have a consistent effect on the responses to CRD.

**Characteristics of VPL neuronal responses**

The VPL nucleus is now considered an important site for processing visceral information and relaying it to higher cortical centers (Al-Chaer et al. 1996a; Berkley et al. 1993a; Brüggemann et al. 1994; Chandler et al. 1992). Especially interesting for the primate VPL nucleus in pelvic visceral pain processing. Our results show that the population of viscerosensitive VPL neurons sampled was divided equally between excited and inhibited cells. The excitatory responses to CRD were graded with the intracolonic pressure, as reflected by an increase in firing rate in proportion to the increase in stimulus intensity. The somatic receptive fields of these cells were unilateral and mostly located on the hind parts of the body although a few receptive fields were on the forelimb. These observations are similar to those reported earlier by Brüggemann et al. (1994) in the monkey. Several studies have addressed the role of the VPL nucleus in pain in general and in visceral pain in particular. Chandler et al. (1992) did a pioneering study of the visceral representation in the macaque lateral thalamus using bladder distension as a visceral stimulus. Their sample of viscero-somatic neurons consisted mostly of cells excited by noxious cutaneous stimuli and thus apparently activated by spinothalamic tract input. Brüggemann et al. (1994) had a different result concerning the responses of VPL neurons to visceral stimulation. They reported that most of their sample of VPL viscerosensory cells were low-threshold somatic neurons. Therefore, they came to the conclusion that the DC mediates visceral input onto these cells.

The viscerosensitive neurons isolated in the present study were almost all low-threshold somatic neurons, but they responded to high-intensity, presumably noxious, CRD (see Brüggemann et al. 1994). The assumption that high-intensity CRD is a noxious stimulus in monkeys follows behavioral studies in man that have shown that CRDs of intensities ≥40 mmHg is painful (Ness et al. 1990). Ness and Gebhart also reported that CRD is a noxious visceral stimulus in rats when intensities of CRD reach or exceed 40 mmHg. They described pseudoaffective reflexes exhibited by rats when tested with CRD (Ness and Gebhart 1988). Occasionally, the VPL neuronal firing rate in response to maximal CRD did not exceed the response to gentle brushing of the skin or to innocuous pressure, raising a question about the nociceptive quality of the visceral response. These observations correlate with earlier ones made in the rat (Al-Chaer et al. 1996a,b). One may argue that the responses of somatic low-threshold cells in the CNS to noxious intensities of CRD are themselves low-threshold responses and therefore do not relate to visceral nociceptive processing and subsequently cannot contribute to the perception of visceral pain. This interpretation is based on the intuitive supposition that neurons putatively involved in a sensory discriminative nociceptive system should exhibit, in nonpathological conditions, their highest frequencies of firing when noxious stimuli are applied to their receptive fields. Such a property most often is verified for convergent neurons tested with a single modality of stimulation such as graded temperatures or increasing intensities of mechanical stimulation under standardized temporal conditions (Kenshalo et al. 1979; Mendell and Wall 1965; Price and Browe 1975). However, Al-Chaer et al. (1996c, 1997c,d) have shown that visceral sensory neurons in the VPL nucleus, the NG, and the PSDC system exhibit different properties vis-a-vis different modalities of input. For example, whereas colon inflammation with mustard oil (MO) sensitized the responses of neurons to CRD, inflammation had an opposite effect on the responses to cutaneous stimuli. These observations suggest that modality-specific neuronal channels may possibly be involved in separately relaying visceral and somatic inputs. Le Bars and Chitour (1983) have reported in the rat that the responses of dorsal horn cells to noxious heat stimuli are smaller in...
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PSDC system, previously unnoticed and characterized by a sensory system correlates with the effect of these lesions on Chaer et al. 1996b, 1997c). Our group has shown that these reduced the thalamic responses to visceral stimulation. The presumably is mediated by second-order neurons located rates.

Viscerosensory neurons in the somatosensory cortex (Bérugemann et al. 1997), in the thalamus (Al-Chaer et al. 1996a; Berkley et al. 1993a; Brittgemann et al. 1994; Chandler et al. 1992) or in the spinal cord (Al-Chaer et al. 1996b; Berkley et al. 1993b; Chandler et al. 1996; Milne et al. 1982; Ness and Gebhart 1987; Zhang et al. 1996) respond to visceral stimulation with a phasic-tonic profile. These neurons were shown consistently to exhibit a graded increase in firing rate directly related to the stimulus intensity and time bound by the stimulus duration. This leaves the door open to speculations about the role of spike timing versus that of firing rate in coding for visceral events, especially nociceptive ones.

Role of the DC

Several opinions have been presented about the role of the DC in the processing and relaying of visceral nociceptive signals to higher brain centers (Apkarian et al. 1995; Berkley 1997; Gybels 1997; Hirshberg et al. 1996; Nauta et al. 1997). Our group has reported previously that lesions of the DC in the rat can dramatically lessen the responses of VPL neurons to graded CRD, indicating that the DC has an important role in mediating noxious viscerosensory input into thalamic and presumably other brain centers (Al-Chaer et al. 1996a). Localized lesions of the NG later corroborated this initial observation; although less potent in their effect, tiny NG lesions induced significant reductions in the responses of VPL neurons to CRD (Al-Chaer et al. 1997b). Recordings made in the NG have shown that a large percentage of NG neurons receive visceral input from one or more visceral organs (Al-Chaer et al. 1996b, 1997d; Berkley and Hubscher 1995; Rigamonti and Hancock 1978). This input presumably is mediated by second-order neurons located near the central canal with axons projecting in the DC (Al-Chaer et al. 1996b, 1997c). Our group has shown that these second-order neurons constitute a separate component of the PSDC system, previously unnoticed and characterized by a predominant visceral input.

A striking difference between the observations made in the rat and those made in the monkey lies in the extent of an effective DC lesion. Whereas in the rat a superficial midline lesion of the fasciculus gracilis almost totally abolished the VPL neuronal response to CRD, a similar lesion in the monkey was not as effective. To observe a dramatic decrease in the VPL neuronal responses to CRD, the DC lesion in the monkey had to be made deeper and wider than the ones usually made in the rat. However, the lesion did not extend ventrally or laterally beyond the DC, sparing therefore the dorsal commissure and the DLF. The difference between the monkey and the rat may be the presence of the corticospinal tract underneath the DC in rats, presumably causing a different distribution of the positions of ascending axons in rats and monkeys. Another difference noted between the monkey and the rat is the apparent lack of a substantial effect of VLC lesions in monkeys. In the rat, Al-Chaer et al. (1996a) have reported that a lesion of the VLC ipsilateral to the recording site causes a significant reduction in VPL neuronal responses to CRD. However, in the monkey, the effect of VLC lesions was not substantial within the limited number of observations made, although in two individual cases, the VLC lesion did have a potent effect. Furthermore, in the rat, Al-Chaer et al. (1996a) have observed that a DC lesion followed by a VLC lesion ipsilateral to the recording site eliminated the responses of VPL neurons to CRD. This was not the case in the monkey. Although the predominant visceral input onto the VPL cells examined was via the DC, the contribution of other spinal pathways, in particular the iVLC, to this input was not consistent across the cells sampled. Some cells appeared to receive an additional component of the visceral input via the cVLC and some others via the DLFs. The role of the DLF in visceral input onto VPL neurons in the monkey is not surprising for it is possible that the axons of some postsynaptic dorsal column neurons have branches that travel in both the dorsal and the dorsolateral funiculi (Rustioni et al. 1979; see also Willis and Cogshall 1991). These axons project onto neurons in the DCN. Axons of DCN neurons, afterward, relay this information to the VPL nucleus. The postsynaptic dorsal column system receives a major input from the colon of the monkey (unpublished data) and the rat (Al-Chaer et al. 1996b). However, in the rat, the axons of the PSDC project almost exclusively in the DC (Giesler et al. 1984). This morphological difference presumably underlies the lack of an effect of a DLC lesion on the responses of VPL neurons to CRD. On the other hand, a role of the DLC in pelvic visceral input onto NG neurons has been reported in rats (Berkley and Hubscher 1995). Nevertheless, whereas the DC lesion consistently produced a large decrease in the VPL neuronal responses to CRD, VLC, or DLC lesions did not have a consistent effect in monkeys. Because this study was aimed at examining the effect of DC lesions on the responses of VPL neurons to CRD, the observations were limited to the first significant effect of a spinal lesion on the neuronal firing rates.

In this study, we have shown that a lesion of the DC reduced the thalamic responses to visceral stimulation. The effect of DC lesions on evoked potentials within the somatosensory system correlates with the effect of these lesions on behavioral tasks that require processing of sensory information (Makous et al. 1996). Therefore it is tempting to specu-
late that a lesion of the DC would invariably alter the sensation of visceral pain regardless of whether this pain is coded for by activity patterns or by firing rates. Consequently, we conclude that the DC pathway plays an important role in the processing and relaying of visceral nociception to higher brain centers.

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