Epigastric Antinociception by Cervical Dorsal Column Lesions in Rats

Yi Feng, M.D.,* Minglei Cui, Ph.D.,* Elie D. Al-Chaer, Ph.D.,† William D. Willis, M.D., Ph.D.‡

Background: Previous clinical evidence and electrophysiological studies in the authors’ laboratory have implicated the dorsal column (DC) as an important pathway for the transmission of visceral colorectal pain. This study examined, behaviorally and electrophysiologically, the role of the DC in mediating epigastric nociception using a visceral pain model involving duodenal distension in rats.

Methods: For behavioral testing, the writhing-like responses produced in awake rats by graded intraduodenal balloon distension (0.1 to 0.7 ml) were tested. A DC mechanical lesion at the C2 level or a sham operation (SH, same spinal cord segment exposed but no DC lesion) was performed. The writhing-like responses to duodenal distension were tested again and the rats were compared with other rats with no lesions and with SH rats. For electrophysiologic testing, the extracellular activity of single neurons was recorded in the ventrobasal nucleus of the thalamus in anesthetized rats. The ventrobasal cells that responded to duodenal distension were tested further with this visceral stimulus before and after a lesion of the DC.

Results: The mechanical DC lesion significantly reduced the intensity of the writhing-like responses and increased the threshold volume that would elicit writhing-like responses compared with rats with no lesions and SH rats without any observable neurologic deficit. A lesion of the DC also significantly reduced the responses of ventrobasal cells to duodenal distension.

Conclusions: The DC plays an important role in signaling epigastric nociception in this experimental model. A mechanical DC lesion can produce significant visceral antinociception in rats. (Key words: Dorsal column; rat; visceral nociception.)

VISCERAL pain is a common clinical manifestation caused by inflammation, cancer, ulcer, and other chronic diseases. Treatment is usually frustrating but is critical to the daily functioning and quality of life of patients. Several ascending pathways carry viscero-sensory information from peripheral receptors.¹ These are the spinothalamic and spinoreticular tracts and a pathway in the dorsal column (DC).

Recent neuroanatomic and neuropysiologic findings from our laboratory in animal experiments have shown that the postsynaptic DC pathway plays a more important role than that of the spinothalamic tract in visceral nociception.² A midline lesion of the DC can abolish thalamic neuronal and DC nuclei (nucleus gracilis) neuronal responses to pelvic visceral stimulation.²,³ Evidence from clinical case studies also indicated that a restricted lesion in the midline of the posterior columns at T10 can eliminate pelvic cancer pain that is insufficiently controlled by morphine.⁴,⁵ The results remained excellent even in patients in whom somatic structures of the pelvic body wall were involved. Neurologic testing reveals no additional neurological deficit and no analgesia to pinprick stimuli applied to the body surface, despite relief of the visceral pain. Because of neuroanatomic differences in the innervation of pelvic and epigastric viscera,⁷ it is unclear if the DC pathway also plays an important role in nociceptive transmission from epigastric organs. Further, it is unknown if a mechanical lesion of the cervical cord would have as dramatic an effect in relieving epigastric pain as a lesion in thoracic cord does in relieving pelvic pain. We hypothesized that the DC has a similar role in processing epigastric and pelvic nociception.

The writhing-like movement represents an unconditioned response by the animals to noxious visceral stimulation and is characterized by repeated contractions of the abdominal muscles accompanied by extension of the hind limbs.⁸ Colburn et al.⁹ created a mechanical

* Postdoctoral Fellow
† Assistant Professor, Departments of Internal Medicine and Anato
my and Neurosciences;
‡ Professor and Chairman, Department of Anatomy and Neurosci
ences; Director, Marine Biomedical Institute.

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Address reprint requests to Dr. Willis, Department of Anatomy and Neurosciences, MBII, 501 University Boulevard, The University of Texas Medical Branch, Galveston, Texas 77555-1069. Address electronic mail: wdwillis@utmb.edu

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visceral pain model in which writhing-like behavioral responses can be reproducibly induced by duodenal distension. In the present study, we tested the behavioral improvement in the writhing-like response in awake rats produced by duodenal distension after a DC lesion at the C2 level. Correspondingly, we tested extracellular activity of single neurons to duodenal distension in the ventrobasal nuclei (including ventral posterolateral [VPL] and ventral posteromedial [VPM]) of the thalamus before and after a DC lesion in anesthetized rats. The ventrobasal nucleus is considered one of the major supraspinal relays of nociceptive inputs in rats. Some of the results have been reported in abstract form.10

Materials and Methods

Animals
Male Sprague Dawley rats (300 - 350 g) were used for the experiments. The animals were housed individually in a temperature-controlled (21 ± 1°C) room with a 12-h light:12-h dark cycle (lights on from 9:00 A.M. - 9:00 P.M.) and were allowed free access to food and water. Surgical and experimental protocols used in this study were approved by the Animal Care and Use Committee at the University of Texas Medical Branch, Galveston.

Surgical Preparation
Intruduodenal Balloon Catheter Implantation. Using a modification of the method described by Colburn et al.,9 chronic intruduodenal balloon catheters were implanted in the rats. Briefly, under inhalation anesthesia (2% to 2.5% halothane), a 1.5-cm laparotomy was made and the gastroduodenal junction was exposed. A latex rubber balloon catheter (15-cm-long PE50 catheter with an attached 8-mm-long balloon distensible to a 2 ml fluid volume) was advanced through the pylorus into the first portion of the duodenum after gastrotomy. The distal end of the catheter was tunneled subcutaneously to the base of the skull, externalized, and anchored to the dermis with a silicone sleeve and super glue. The animals were allowed 7 days to recover before the first set of behavioral tests.

The intruduodenal balloon catheter implantation and cervical DC surgery did not affect the rats' normal diet and behavior. The weight of the rats decreased 5-20 g during the initial 3-5 days after implantation surgery and then gradually increased. Two samples of duodenum from two rats were examined 7 and 12 days after balloon catheter implantation, respectively, and showed that there was no severe mucosal injury, only slight acute inflammation.

Behavioral Tests
Each animal was initially allowed 30 min to get used to its surroundings. The rat was placed in a 30 × 50 × 30 cm polypropylene box. At least two rats were tested simultaneously. The behavioral investigator was blinded as to whether there was a DC lesion.

The intruduodenal balloon was distended for 1 min at 15-min intervals by 0.1-ml stepwise increments of saline using a 1-ml syringe (from 0.1 ml to 0.7 ml). The tail arterial blood pressure was monitored during testing using a cannula connected to a pressure transducer that fed into a CED 1401+ (Cambridge Electronic Design Limited, Cambridge, UK) data analysis instrument. The intraballoons pressure was obtained through a barostatic device and led to a data collection system (n = 6). The pressure changes in the balloon were recorded graphically and reflected the contractile activity in the duodenal wall during the inflation. The intraluminal pressure produced by the duodenal wall against the balloon could be determined from the balloon pressure inside the duodenum minus the balloon pressure in the atmosphere.

The writhing-like response behavior before and after a DC lesion was evaluated using the scale (0-4) proposed by DeLeo et al.11 as follows: 0, normal body position and exploratory behavior; 1, halt in activity, “wet dog” shaking, excessive facial grooming, and teeth chattering; 2, hunching, abdominal nipping, hind paw biting, and immobility of hind limbs; 3, stretching of the hind limbs, arching, and dorsoflexion of the hind paws; and 4, stretching of the body, extension of the hind limbs.

Dorsal Column Lesion
Mechanical Dorsal Column Lesion. A DC lesion was made 7 days after balloon implantation. Under halothane anesthesia, the rat’s head was fixed in a stereotaxic frame. The head was pushed forward and down to extend the cervical 1 - 2 intervertebral space. An incision was made in the midline near the base of the skull, and the muscles were retracted. An intervertebral fasciotomy was done to reveal the spinal cord. A 27-gauge needle tip was inserted into the midline or 0.5 mm lateral to the midline of the DC on both sides and moved 1 - 2 mm rostrally under view through a surgical microscope. In a preliminary study of four rats, we
found that if hind limb extension and body stretching could still be induced after a DC lesion, we considered it an ineffective lesion. By comparing the behavior with histologic data, we found that all ineffective lesions were located in the fasciculus gracilis (see fig. 1A). When the lesion area was extended more laterally on both sides of the DC, no rats reached a score of 3 or above (75% were at a scale of 1 or less, and 25% were at a scale of 2). The effective lesions in the rest of the experiments were either large midline or bilateral lesions as described before. Similar observations were also made in the electrophysiology experiments.

**Mechanical Dorsal Column Sham Lesion.** The same surgery was done to expose the cervical spinal cord, but no DC lesion was made in 12 rats. Animals were allowed 5 days to recover from cervical surgery before the second set of behavioral tests. Each incision was locally anesthetized with 1% lidocaine (with adrenaline 1:10,000). The surgical procedures were done under sterile conditions.

**Electrophysiologic Experiments**

Anesthesia was induced with 4% halothane, followed by 2% to 2.5% halothane during the surgery. Body temperature was monitored using a rectal probe and maintained at about 36-37°C with a regulated heating blanket placed under the animal. Tail venous and arterial cannulas, a tracheal cannula, and the intraduodenal balloon catheter were inserted. The rats were paralyzed with pancuronium bromide infused intravenously at 0.2 mg·kg⁻¹·h⁻¹. Arterial blood pressure was maintained at approximately 100-120 mmHg by adjusting the halothane (range, 1% to 1.2%). After paralysis, all rats were artificially ventilated with oxygen and nitrogen (1:1), and the end-tidal carbon dioxide level was kept between 3.5% and 4.5%.

A partial craniotomy and cervical laminectomy were performed to expose the dura above the thalamus and spinal cord to record neuronal responses and to create a DC lesion, respectively.

Single-cell activity of individual neurons of the VPL or VPM nuclei was recorded using tungsten microelectrodes (125-µm shank; 12 MΩ). The electrode was inserted stereotaxically into the brain and aimed to search the VPL or VPM or both areas. The electrode was slowly lowered until a single unit was well isolated. The cutaneous receptive field was mapped by brushing the skin, and the unit’s responses to graded duodenal distension (0.2 ml, 0.3 ml, 0.4 ml, and 0.5 ml) were determined. The recorded extracellular action potentials 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. Each duodenal distension stimulus lasted 20 s and was followed by an 8-min interval before the next step stimulation. A DC lesion was made at the C2 level (as in the behavioral group). The unit’s responses to duodenal distension and cutaneous stimuli were tested again 30 min after the lesion.

**Histologic Confirmation**

At the end of each electrophysiologic experiment, the last recording site was marked by passing a continuous current (150 μA for 30 s). The spinal cord at the level of the lesion and the brain were removed and fixed in 4% paraformaldehyde before being frozen sectioned at 60 μm. The ventrobasal recording sites were identified, and the extent of each spinal cord lesion was confirmed.

**Statistical Analysis**

For behavioral tests, Friedman’s test was applied to the sham (SH) and lesion groups, separately for prelesion and postlesion data, to determine if the scales tended to differ across volumes. A repeated measures analysis of variance was applied to the SH and lesion data (fig. 2) to measure the extent to which changes from before versus after the lesion in the lesion group differed from those in the SH group. To accomplish this, the difference between prelesion and postlesion scores was calculated at each volume. The difference scores were then analyzed for the effects of volume, group, and their interaction. Although the data are not strictly continuous, the procedure is considered appropriate because of the robustness of the method. Pairwise comparisons were made with Wilcoxon rank sum tests or Wilcoxon signed tests because the data are not continuous. We terminated testing when the intensity of duodenal distension reached the maximum twice (scale 4), because we assume that it would be more painful and that there would have been more risk of balloon rupture with the next higher volume stimulation. As a result, we used the missing-data imputation technique of carrying the last value forward. Mean threshold distension volume to elicit a writhing-like response was compared before and after a DC lesion using paired t tests. The significance of differences in mean blood pressure responses to duodenal distension with or without a DC lesion was determined using unpaired Student’s t tests. The mean intraluminal pressure and

**DISTENTION VOLUME (ml)**

Fig. 2. Median intensities of writhing-like response (WR) to duodenal distension comparing (A) pre-sham lesion (pre-SH) with sham lesion (SH; *P* = 0.71 using the Wilcoxon signed rank test), (B) pre-lesion and after lesion (**P* < 0.01 by the Wilcoxon signed rank test) and (C) genuine lesion with SH lesion (**P* < 0.05, ***P* < 0.01 by Wilcoxon sum rank tests).

frequency of duodenal contraction before and after DC lesion were compared using paired Student’s t tests.

For electrophysiologic experiments, distension-evoked ventrobasal responses were expressed as percentages of the baseline. The differences between responses before lesions and after lesions were tested using paired t tests. All data are expressed as mean ± SD. A *P* value <0.05 was considered significant.
Results

Behavioral Tests

To examine the effects of a DC lesion on epigastric nociceptive behaviors, 30 rats were divided into two groups: a lesion group (n = 18) and a sham (SH) group (n = 12). Six rats (three in the lesion group and three in the SH group) were excluded from analysis because the balloon was broken before the second set of tests. Histologic sections from three rats in the lesion group showed that the cervical spinal cords remained intact despite attempts to create a lesion. The dura was punctured but there was no lesion of the cord. The writhing-like response to duodenal distension showed no change compared with prelesion responses. Thus data from these three rats were combined with the SH group.

Drawings of the ineffective lesions, the extent of effective lesions of the cervical spinal cord from all behavioral and electrophysiologic experiments, and the histologic data of lesions from two representative rats used for electrophysiologic experiments are shown in figure 1. In the case of the electrophysiologic experiments, there was both an ineffective and effective lesion separated by 1 mm.

The median intensities of writhing-like responses for duodenal distension in the two groups are shown in figure 2. The lesion group includes only rats with effective lesions. Graded intraduodenal balloon distension produced graded writhing-like responses. The writhing-like response varied at low inflation volume (≤0.3 ml) and consisted of shaking like a wet dog, cessation of activity, teeth chattering, and hind paw biting. Stretching of the body with hind limb extension (the classic writhing response) was progressively more frequent at greater inflation volumes (≥0.4 ml) and was induced in all rats. The cervical DC lesion can be seen to decrease significantly the intensity of the writhing-like response compared with prelesion and SH surgery (figs. 2B, 2C). The median scores for all four repeated measures groups, before and after surgery for the SH and lesion groups, were significantly different across volumes according to Friedman’s test (all P < 0.001). Our interpretation is that the difference scores are higher in the SH group compared with the lesion group, but the difference is not consistent across volumes. No differences were found at volumes of 0.1 and 0.2 ml, but significant differences were found at 0.3, 0.4, 0.5, 0.6, and 0.7 ml.

The threshold inflation volume to produce writhing-like responses is shown in figure 3. Before the DC lesion, the mean threshold volume to elicit writhing-like responses was 0.23 ± 0.06 ml. A DC lesion increased the threshold volume significantly to 0.37 ± 0.18 ml (P < 0.05 by paired t test, n = 10); the analysis was truncated by not including the data from two rats in which no writhing-like responses were elicited by graded duodenal distension after the DC lesion.

Duodenal contractions did not appear to change after the DC lesion (24.8 ± 7.4 contractions/min vs. 23.8 ± 10.1 contractions/min; P > 0.05), nor did intraluminal pressure produced by the duodenal wall against the balloon (86.0 ± 45.4 mmHg vs. 72.5 ± 23.7 mmHg at 0.4 ml inflation; P > 0.05).

The blood pressure response to duodenal distension was tested in 15 rats (8 in the SH group and 7 in the lesion group). Responses were quantified as the change in mean arterial pressure (ΔMBP expressed in mmHg). The ΔMBP increased in a graded manner after graded duodenal distension in all rats. The resting blood pressure showed no significant difference between the DC lesion group and the SH group (144 ± 26.8 mmHg vs. 137.7 ± 18.8 mmHg; P > 0.05). The pressor response (ΔMBP) was decreased after the DC lesion, but this was not significant when compared with the SH lesion group (17.4 ± 8.2 vs. 25 ± 8.1 mmHg; P > 0.05), whereas the writhing-like response was reduced significantly at 0.6 ml inflation (scale 4 vs. scale 1; fig. 4).

Electrophysiologic Experiments

A total of 80 thalamic neurons in the ventrobasal nuclei of 30 rats were isolated. Forty-nine were somatic, 16 were somatovisceral, and 15 were viscerceptive.
neurons. Of the 16 somatovisceral neurons, 92% of those tested responded only to innocuous stimulation of the skin (brush). Figure 5 shows 16 recovered recording sites of these cells in the VPL and VPM nuclei of the thalamus. All of the cells, including excited (n = 13) or inhibited (n = 3) cells, were located on the edge of the VPL or VPM nuclei. However, in this study we report only the results from excited somatovisceral and visceroreceptive cells tested before and after a DC lesion (n = 9) in nine animals. Of the nine characterized thalamic neurons, three had cutaneous receptive fields in and around the neck and shoulder; three had their re-

ceptive fields in the face and nose area; one had its receptive field around the epigastrum and mid-back area; one had a receptive field on the paws; and one had no cutaneous receptive field.

Ventral posterolateral unit responses to duodenal distension were increased after inflation in a graded manner by 0.2, 0.3, and 0.4 ml and became saturated at 0.5 ml distension (see fig. 6, CONTROL). A limited midline lesion did not effectively attenuate the visceroreceptive responses to duodenal distension (see fig. 6, DC1 LESION; fig. 1D); a wider bilateral lesion (1 mm apart from DC1 LESION) abolished the visceroreceptive responses to duodenal distension (see fig. 6, DC2 LESION; fig. 1E).

Figure 7 shows the effects of DC lesion on the average responses of nine excited units to graded duodenal distension. The mean responses before the lesion increased over baseline by 68 ± 35.6%, 78 ± 20.9%, 145.9 ± 40.9%, and 121.1 ± 27.2% after distension of 0.2, 0.3, and 0.5 ml, respectively. A limited midline DC lesion (DC1) resulted in no significant change in the responses when compared with before the lesion (P > 0.05); a wide DC lesion (DC2) at the C2 level dramatically decreased the responses to 5 ± 10.1%, 0.9 ±

Fig. 4. Blood pressure tracings of one rat (top), median writhing-like response (middle), and the mean pressor response (bottom) to 0.6 ml duodenal distension. Left-hand plots indicate sham lesion group measures and right-hand plots indicate lesion group measures (*P < 0.05).

Fig. 5. The location of 16 viscerosensitive cells in the ventrobasal nucleus of thalamus. Solid circles indicate excited cells, and open circles indicate inhibited cells.
CERVICAL DORSAL COLUMN LESION

Fig. 6. Responses of a viscerosensitive cell in the VPL nucleus to graded duodenal distention (0.2, 0.3, 0.4, and 0.5 ml). (A) Neuronal responses to graded duodenal distention before (CONTROL) and after a limited midline dorsal column lesion (DC1 LESION) and after a wider bilateral dorsal column lesion (DC2 LESION) at the C2 level. The histologic data for this animal are shown in figure 1D and 1E. (B) Spike shape throughout the procedures. (C) The dorsal column lesion area at the C2 level; the hatched area indicates the first midline lesion, and the dark area indicates the second wide bilateral lesion. (D) The dark area delineates the cutaneous receptive field on the shoulder. (E) The location of the neuron in the ventral posterolateral nucleus.

10.6%, 1.14 ± 7%, and 2.1 ± 10.0%, respectively (P < 0.05 by paired t test).

Discussion

The goal of this study was to verify behaviorally and electrophysiologically the role of the DC in mediating epigastric nociceptive processing. Our findings clearly indicate that the DC plays an important role in epigastric nociceptive transmission. A mechanical lesion of the DC at the C2 level can significantly reduce the writhing-like responses induced by duodenal distension, without producing any observable deficiency in the spontaneous activity of the rats. The DC lesion can also abolish the responses of ventrobasal thalamic neurons to noxious duodenal distension.

Many clinical studies in patients and healthy volunteers have consistently documented that human experimental pain evoked by distension of the gastrointes-
tinal tract is similar to the sensation of pathologic pain. Inflation of a balloon in the duodenum produces an intense pain, and sites of pain referral are predominantly epigastric and periumbilical.

In the current investigation, we used writhing-like responses induced by graded intraduodenal distension to mimic clinical epigastric pain. A DC lesion not only significantly reduced the intensity of the writhing-like responses but also increased the threshold to elicit writhing-like responses, whereas there was no effect on intraluminal pressure or duodenal wall motility. The weight of the rats remained unchanged or there was a slight increase by several days after the DC lesion. No rat showed any observable neurological deficit. The results of behavioral tests imply that a DC lesion can indeed produce epigastric antinociception in rats. Interestingly, we found that the pressure response to duodenal distension was not eliminated by a DC lesion, even though the writhing-like responses were reduced significantly. It can be inferred that the cardiovascular response to duodenal distension is mediated through a pathway other than the DC and that the mechanism of the changes produced by a mechanical DC lesion is different than that of intraluminal morphine or clonidine treatment, which do affect neurological responses.

Nociceptive responses of neurons in the VPL and VPM nuclei of the thalamus have been recorded by many investigators in monkeys, cats, and rats. The properties of the nociceptive neurons in the VPL nucleus are appropriate for a role of these neurons in the sensory-discriminative aspects of pain. Lenz et al. recorded nociceptive neurons in the human ventral posterior thalamus, presumably including VPL, VPM, and VPI stimulation at the same time often evoked pain. In one patient with angina pectoris, stimulation in the VPL nucleus caused anginal pain. Another clinical report from Davis et al. showed that microstimulation in the area ventral and posterior to the ventrocaudal thalamus in the human brain evoked visceral pain sensation. These observations strongly suggest that the ventral posterior nuclei are involved in processing visceral information.

The DCs or a pathway in the DC has not been considered to play a role in mediating visceral nociception until a recent series of studies, although this conjecture was suggested as early as 1945 by White. Recently, Al-Chaer et al. in our group compared the roles of the DC and the spinothalamic tract in the processing of visceral nociceptive information transmitted to the VPL nucleus of the thalamus in rats and primates before and after sequential lesions of the DC and of the ventrolateral column at a thoracic level. A lesion of the DC dramatically reduced the responses of VPL cells to colorectal distension while the effect of a ventrolateral column lesion was minor compared with that of a DC lesion. We concluded that the DC contains a pathway more important for transmitting visceral sensation than the spinothalamic tract. A similar conclusion was also reached by Apkarian's group, based on a mathematical analysis of thalamic neuronal responses. Further observations indicated that visceral nociceptive information reaches the DC nuclei through the postsynaptic DC path.

In the current report, viscerceptive neurons were observed in the VPL and VPM nuclei of the thalamus in rats. All neurons in the VPM nucleus with responses to colorectal distension had receptive fields on the face. Similar viscerceptive neurons in the VPL nucleus had receptive fields on the upper body. The DC lesion reduced dramatically the behavioral and ventrobasal neuronal responses to colorectal distension. This observation is consistent with a role of the DC-medial lemniscus system in visceral nociception. Furthermore, we also found that a lesion of the fasciculus gracilis could not eliminate effectively the writhing-like responses, nor the responses of ventrobasal neurons to noxious duodenal distension, as seen after colorectal distension. However, when the lesion was extended bilaterally to the dorsal intermediate septum and fasciculus cuneatus at the C2 level, the writhing-like responses to duodenal distension were greatly diminished or abolished, as were the responses of ventrobasal neurons to noxious duodenal distension. This observation supports the theory that the axons of the DC pathway emanating from postsynaptic DC cells in the caudal segments of the spinal cord terminate in the nucleus gracilis, whereas projection cells in the more rostral segments of the spinal cord terminate in the nucleus cuneatus. Anatomically, duodenal afferents reach the spinal cord through dorsal roots T4-L1 in humans. These fibers presumably converge on neurons at segmental levels where ascending projections travel in the funiculus cuneatus rather than in the funiculus gracilis. Supportive evidence comes from a study by Wang et al., in which Phaseolus vulgaris-leucoagglutinin (2.5%) was microinjected iontophoretically into lamina X at the T7 or S1 levels of the spinal cord, respectively. They found that the ascending postsynaptic DC projection from the sacral spinal cord was localized in the midline; in contrast, the postsynaptic DC projection from the mid-tho

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racic spinal cord was localized in the dorsal intermediate septum. This difference might explain why nociceptive signals from upper abdominal viscera were not affected after limited midline cervical myelotomy but were nearly abolished after a wide DC lesion that included the dorsal intermediate septum bilaterally. The DC is also responsible clinically for discriminative touch, vibratory sensibility, and position sense. The discriminative functions include two-point discrimination, stereognosis, and graphesthesia. Loss of DC function may result in loss of these sensations and a reduced capability of recognizing gradations of tactile and pressure stimulation, and it may cause positional hallucinations. There has been no clinical evidence that clearly shows any special gastrointestinal functions for the DC in humans. Partial DC lesions generally cannot eliminate tactile sensations and discriminative functions. Hirshberg et al. reported eight clinical cases of pelvic visceral cancer pain successfully treated by posterior midline myelotomy. They did not detect any signs of postoperative neurologic deficits, and no patients reported any disorder of gastrointestinal function after midline myelotomy. Nauta et al. reported a case in which a punctate midline myelotomy performed in a patient effectively eliminated intractable pelvic pain caused by colon inflammation after radiation. Before the surgery, the patient had already suffered a slight proprioceptive neurologic deficit due to diabetes mellitus. After posterior midline myelotomy, the severe pain was markedly reduced without any further neurologic deficit for at least 10 months. The patient had a normal appetite without nausea and weight increased.

The evidence from our experiment on epigastric nociception shows that cervical DC lesions do not change duodenal wall motility significantly. The weight of the rats stayed unchanged or was slightly increased and they had normal spontaneous activity without any signs of neurologic deficit after a DC lesion.

In conclusion, our promising experimental results in rats imply that the DC plays an important role in epigastric nociceptive processing and that a DC lesion at the C2 level should provide effective pain relief for epigastric pain. However, it is unclear if a DC lesion in humans comparable to that required in rats to relieve epigastric nociceptive responses can be done without causing some neurological deficit.

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