Spinal regions involved in baroreflex control of renal sympathetic nerve activity in the rat

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Zahner MR, Schramm LP. Spinal regions involved in baroreflex control of renal sympathetic nerve activity in the rat. Am J Physiol Regul Integr Comp Physiol 300: R910–R916, 2011. First published January 12, 2011; doi:10.1152/ajpregu.00646.2010.—Spinal cord injury causes debilitating cardiovascular disturbances. The etiology of these disturbances remains obscure, partly because the locations of spinal cord pathways important for sympathetic control of cardiovascular function have not been thoroughly studied. To elucidate these pathways, we examined regions of the thoracic spinal cord important for reflex sympathetic control of arterial pressure (AP). In anesthetized rats, baroreceptor relationships between pharmacologically induced changes in AP and changes in left renal sympathetic nerve activity (RSNA) were generated in spinal intact rats and after acute surgical hemisection of either the dorsal, left, or right T8 spinal cord. None of these individual spinal lesions prevented the baroreceptor-mediated increases in RSNA caused by decreases in AP. Thus, baroreceptor-mediated increases in RSNA in rats are mediated by relatively diffuse, bilateral, descending, excitatory projections. The ability to reduce RSNA at increased AP was impaired after both dorsal and left hemisections, and baroreceptor gain was significantly decreased. Baroreceptor-induced maximum decreases in RSNA were not affected by right hemisections. However, baroreflex gain was impaired. Because both dorsal and left hemisections, but not right hemisections, attenuated the decrease in RSNA at elevated AP, we conclude that pathways involved in the tonic inhibition of spinal sources of sympathetic activity descend ipsilaterally in the dorsal spinal cord. Our results show that many lesions that do not fully transect the spinal cord spare portions of both descending excitatory pathways that may prevent orthostatic hypotension and descending inhibitory pathways that reduce the incidence of autonomic dysreflexia.

sympathetic preganglionic neurons; rostral ventrolateral medulla; cardiovascular regulation; spinal cord injury; descending spinal pathways


In patients, SCI rarely transects the spinal cord completely. Therefore, SCI patients may be left with varying degrees and combinations of inadequate descending sympathetic drive and disinhibited spinal sympathetic reflexes. Because increasing evidence suggests that some recovery of somatic (4, 5) and cardiovascular (22–25, 27) function can occur after SCI, and these studies have been conducted in the rat, it is important to identify the spinal pathways in that animal that mediate the sympathetic limb of baroreceptor regulation and, perhaps, the recovery of cardiovascular regulation.

Damage to descending sympathoexcitatory pathways could degrade the ability of baroreceptor regulation to increase sympathetic activity during orthostatic hypotension. In spinal intact mammals, baroreceptor input regulates sympathetic activity at its principal, although not exclusive, source in the brainstem. Experiments in cats and rats have described multiple locations of descending sympathoexcitatory pathways (12, 15, 17–19, 35, 36, 38). Therefore, we asked whether descending baroreceptor-regulated pathways were widespread, as suggested by previous studies, or were spatially restricted.

Damage to descending pathways that normally inhibit tonic, spinally generated sympathetic activity could degrade the ability of baroreceptor regulation to control sympathetic activity during the hypertensive crises common during autonomic dysreflexia. We have previously shown that ongoing RSNA increases after dorsal hemisection in rats (35, 37) due to reduced descending inhibition of spinal sympathetic systems. Therefore, we asked whether spinal lesions might degrade baroreceptor efficacy by reducing the ability of a baroreceptor to decrease RSNA at elevated AP. To answer these questions, we created baroreceptor response relationships between AP and left RSNA after either dorsal, left (ipsilateral to the recorded renal nerve), or right (contralateral to the recorded renal nerve) surgical hemisection at the T8 spinal level. Our observations suggest that the pathways responsible for the excitation of RSNA at decreased AP are spatially diffuse and widespread. Additionally, the pathways involved in the descending tonic inhibition of spinal activity, and thus the degree of inhibition of RSNA at elevated AP, are largely unilateral and located in the dorsal spinal cord.

MATERIALS AND METHODS

Thirty-nine male Sprague-Dawley rats (Charles River, Raleigh, NC) weighing between 275 and 350 g were surgically prepared in accordance with the Guide for the Care and Use of Laboratory Animals (National Research Council, 1996) by using procedures approved by the Johns Hopkins University Committee on Animal Care and Use.

Surgical preparations and renal nerve recordings. We anesthetized rats in a plastic chamber and then with a nose cone using 2% isoflurane in O2. We discontinued isoflurane after administration of α-chloralose (100 mg/kg iv; Sigma) via a left jugular cannula. We maintained anesthesia at a surgical plane by supplemental doses of α-chloralose (25 mg/kg) and determined the depth of anesthesia either by corneal reflexes during the recovery from paralysis (see below) or
by the variability of RSNA and AP when rats were paralyzed. We
maintained body temperature between 35 and 37°C with a heating pad
and lamp and monitored it with a rectal probe. The trachea was
 cannulated for mechanical ventilation using a rodent ventilator (CWE,
 Ardmore, PA). The left carotid artery was cannulated for measure-
 ment of AP, which was recorded simultaneously with Cambridge
 Electronic Design Micro1401 hardware and Spike 2 software. The left
 and right femoral veins were cannulated for the separate administra-
 tion of depressor and pressor drugs. Rats were then secured in a
 stereotaxic apparatus and paralyzed with gallamine triethiodide (40
 mg/kg) via the jugular vein cannula. Supplemental doses (10 mg/kg)
 were delivered as required. We exposed the thoracic spinal cord via a
dorsal laminectomy from T6 to T8 and removed the underlying dura.
We administered 1 ml of physiological saline subcutaneously prior to
surgery, and blood loss was monitored and replaced with 25% human
albumin (CSL Behring, Bern, Switzerland) intravenously as needed.

RSNA recording. We have described the preparation for RSNA
recording in detail elsewhere (6). We approached the left kidney via
a left flank laparotomy and retracted it. We deflected the adrenal
gland, the fat covering the psoas muscle, and the paraspinal muscles
from the renal nerve, which typically was located at the junction of the
aorta and the renal artery or was found traversing the aorta. We
dissected the renal nerve from the renal vasculature and surrounding
tissue with the aid of an operating microscope. We immersed the
nerve in mineral oil, and mounted it on a bipolar hook electrode. We
cut the distal end of the renal nerve to prevent recording afferent
activity. RSNA was amplified by a differential amplifier with a
bandpass of 300–3,000 Hz. Sympathetic activity was further pro-
cessed by rectification and low-pass filtering at a time constant of 0.5
s and recorded with the raw AP. To match the filtered RSNA to AP
for baroreceptor curves, AP was also filtered at a time constant of 0.5
s. After experiments, we cut the proximal end of the renal nerve, and
the remaining noise was subtracted from all previous values of RSNA.

Baroreflex. We obtained baroreflex function curves by plotting the
reflex response of RSNA to decreases and increases in AP caused by
the vasodilator sodium nitroprusside (SNP; 50 μg/ml) and the α-ad-
renergic agonist phenylephrine (125 μg/ml), respectively, in succes-
sive ramped infusions. We administered SNP first, beginning at a rate
of 150 μg/h and increased by 150 μg/h approximately every 15 s until
an AP of 60 mmHg below baseline, or a maximum rate of 750 μg/h
was reached. Immediately following SNP administration, we admin-
istered phenylephrine, beginning at a rate of 375 μg/h and increasing
by 125 μg/h every 15 s. These infusions produced an approximately
linear increase in AP from 60 mmHg below baseline AP to 60 mmHg
above baseline AP at a rate of ~1.5 mmHg/s. We quantified RSNA
during baseline recording prior to SNP and phenylephrine delivery,
and between the SNP-induced nadir in AP and the phenylephrine-
induced peak AP. We fit baroreflex curves to a sigmoidal function
when the data justified that relationship (31) or to a linear function
when the data were clearly not sigmoidal. We took the gain of the
baroreceptor function curves as the maximum slope of the sigmoidal
curves and the gain of the linear curves as the slope of the computed
line. In this study, baroreceptor tests consisted of a continuous
phenylephrine-induced increase from an SNP-induced nadir in AP.

Spinal cord lesions. To determine the spinal cord regions involved
in regulating RSNA, we generated baroreceptor curves before and
after surgical lesion of the spinal cord with a 0.6-mm surgical blade.
Spinal lesions consisted of either a thoracic (T8), bilateral dorsal
hemisection, a left hemisection (ipsilateral to the left renal nerve), or
a right hemisection (contralateral to the left renal nerve), in separate
groups of rats (Fig. 1). We performed a baroreflex test prior to spinal
lesions (control) and after cardiovascular and neural signals returned
to steady state after spinal cord lesions (10–15 min).

Histology. At the end of experiments, we perfused rats transcardi-
ally with buffered saline followed by 4% buffered paraformaldehyde
(pH 7.4). Spinal cords were removed and postfixed in paraformalde-
hyde solution overnight. After cryoprotection in 30% sucrose for 48 h,
we cut 50-μm serial parasagittal sections for dorsal hemisections and
horizontal sections for left and right hemisections on a sliding mi-
crotome, mounted them on gelatin-coated glass slides, and air-dried
them. We microscopically reconstructed lesions from each rat in
transverse planes under darkfield illumination for verification of
lesion location and extent (Fig. 1).

Data analysis. We fit RSNA responses to changes in AP to a
sigmoidal function or a linear function, as appropriate, using Prism
software (version 4.0, GraphPad). The sigmoidal function was de-
scribed by the following equation: \[ y = A_1/[1 + \exp[A_2(x - A_3)]] + A_4, \]
where \( y \) was the RSNA, \( x \) was AP, \( A_1 \) was the range of RSNA, \( A_2 \)
was the gain coefficient, and \( A_3 \) was the minimum RSNA of the reflex
curve (31). Maximum gain and change in RSNA were calculated
according to each fitted curve. In four rats the baroreceptor curve was
fit to a linear as opposed to sigmoidal function. Of these rats, two had
dorsal hemisections and two had left hemisections. All baroreceptor
curves from rats with a right hemisection were appropriately fit by
sigmoidal functions.

To account for small differences in baseline pressures between rats,
we grouped the AP measurements for each baroreflex curve into 10
mmHg bins and expressed them as the change from the AP before
delivery of SNP and phenylephrine (denoted as baseline AP). To
compare the effect of spinal hemisection alone on sympathetic activ-
ity, we expressed ongoing RSNA after hemisection as a percentage of
the RSNA prior to the lesion. For quantification of RSNA during
baroreceptor testing, we also normalized RSNA to the values recorded
prior to hemisection.

Thus, we recorded RSNA prior to the control baroreceptor tests,
followed by hemisection and an appropriate recovery period (~10–15
min). After stabilization, we recorded RSNA for an additional period
to obtain a postlesion baseline. Finally, we recorded RSNA during AP
changes produced by SNP and phenylephrine. During the barorece-
ptor responses as AP increased from the SNP-induced nadir and
reached baseline level, RSNA was slightly smaller than that measured
during the baseline period. This was most likely due to the viscoelastic
properties of the carotid sinus (8).
We expressed percent changes in RSNA and the maximum gain of the baroreflex curves as means ± SE. Statistical analyses employed either a paired t-test or one-way ANOVA with repeated measures for comparison between stimuli within groups, one-way ANOVA (with Tukey’s posttests), or two-way ANOVA for comparison between groups, as appropriate. We considered values of $P < 0.05$ significant.

**RESULTS**

We performed baroreflex tests in 39 rats. Ten rats were dismissed from analysis due to inaccurate lesions or spinal hemorrhages during surgery, resulting in successful experiments in 11, 10, and 8 rats after dorsal, left, and right hemisections, respectively.

**Effect of dorsal hemisection on the baroreflex.** Dorsal hemisection did not significantly affect baseline AP or heart rate (HR). In control recordings, baseline AP was 116 ± 5.4 mmHg and HR was 447.3 ± 5.4 beats/min. After dorsal hemisection, AP was 114 ± 4.3 mmHg and HR was 452.1 ± 9.0 beats/min. Figure 2A shows a representative tracing of the effect of dorsal hemisection on baseline AP, ongoing RSNA, and representative baroreflex responses. Dorsal hemisection elicited a significant increase in the baseline RSNA (lesion baseline; 126 ± 13% $P < 0.05$).

Baroreceptor-induced increases in RSNA upon decreasing AP were not affected significantly by dorsal hemisection (Fig. 2B). In the control baroreceptor responses prior to lesions, a 60-mmHg decrease in AP increased RSNA to a maximum plateau of 157 ± 8% of baseline RSNA. After dorsal hemisection, the 60 mmHg decrease in AP increased RSNA to a maximum plateau of 181 ± 15% relative to control baseline RSNA.

Although dorsal hemisection did not significantly affect the increase in RSNA upon decreasing AP, these lesions attenuated the reduction in RSNA at all AP elevations above baseline ($P < 0.05$). In control baroreceptor responses, a 60-mmHg increase in AP decreased RSNA to a minimum of 23 ± 6% relative to baseline RSNA. After dorsal hemisection, a 60-mmHg increase in AP decreased RSNA to a minimum of 80 ± 15% relative to control baseline RSNA.

Dorsal hemisection also significantly decreased the maximum gain of the baroreflex. Prior to lesions, the maximum baroreflex gain was $-2.97 ± 0.25 \Delta%\text{RSNA}/\Delta\text{AP}$. Dorsal hemisection reduced the maximum gain to $-1.97 ± 0.39 \Delta%\text{RSNA}/\Delta\text{AP}$ ($P < 0.05$ Fig. 2C).

**Effect of left hemisection on the baroreflex.** To determine the importance of ipsilateral pathways for baroreflex regulation of RSNA, we conducted baroreflex experiments in rats before and after left hemisection. In control recordings, mean baseline AP was 116.4 ± 5.6 mmHg and HR was 457.0 ± 16.4 beats/min. Figure 3A shows a representative tracing of the effect of left hemisection on the ongoing AP and baseline RSNA as well as representative baroreflex responses. Left hemisection did not significantly affect baseline AP (106.9 ± 4.4 mmHg), HR (457.0 ± 9.1), nor ongoing RSNA (110% ± 12% relative to control). Baroreceptor-induced increases in RSNA upon decreasing AP were not affected by left hemisection (Fig. 3B). In the control baroreceptor responses, prior to lesions, a 60-
mmHg decrease in AP increased RSNA to a maximum plateau of 154 ± 8% of baseline RSNA. After left hemisection, a 60-mmHg decrease in AP increased RSNA to a maximum plateau of 173 ± 17% relative to control baseline RSNA.

Although changes in RSNA after baroreceptor unloading were not significantly affected after left hemisections, these lesions attenuated the reductions in RSNA at all elevations of AP > 10 mmHg above baseline (P < 0.05). In control baroreceptor responses, a 60-mmHg increase in AP decreased RSNA to a minimum of 22 ± 4% relative to baseline RSNA. After left hemisection, a 60-mmHg increase in AP decreased RSNA to a minimum of 71 ± 9% relative to control baseline RSNA.

Left hemisection also significantly attenuated the maximum gain of the baroreflex. The maximum gain was −2.83 ± 0.25 Δ%RSNA/ΔAP prior to lesions. Left hemisection reduced the maximum gain to −1.76 ± 0.26 Δ%RSNA/ΔAP (P < 0.05 Fig. 3C).

**Effect of right hemisection on the baroreflex.** To determine the contribution of contralateral pathways in baroreflex regulation of RSNA, we conducted baroreflex experiments before and after right thoracic hemisection. In control recordings, mean baseline AP was 113.6 ± 3.6 mmHg and HR was 458.9 ± 16.1 beats/min. Figure 4A shows a representative tracing of the effect of right hemisection on the ongoing AP and baseline RSNA, as well as representative baroreflex responses. Right hemisection did not significantly affect baseline AP (109.8 ± 4.0 mmHg), HR (458.9 ± 16.1), nor ongoing RSNA (88.9% ± 11% relative to control). Baroreceptor-induced increases in RSNA upon decreases in AP were also unaffected (Fig. 4B). In the control baroreceptor responses prior to lesions, a 60-mmHg decrease in AP increased RSNA to a maximum plateau of 147 ± 7% of baseline. After right hemisection, a 60-mmHg decrease in AP increased RSNA to a maximum plateau of 152 ± 18% relative to control baseline RSNA. Furthermore, these lesions did not significantly affect reductions in RSNA at increased AP. In control baroreceptor responses, a 60-mmHg increase in AP decreased RSNA to a minimum of 18 ± 5% relative to baseline RSNA. After right hemisection, a 60-mmHg increase in AP decreased RSNA to a minimum of 28 ± 5% relative to control baseline RSNA.

Although the right hemisection had no effect on the maximum or minimum level of RSNA upon changes in AP, it significantly decreased the maximum gain of the baroreflex. The maximum gain was −3.37 ± 0.15 Δ%RSNA/ΔAP prior to lesions. Right hemisection reduced the maximum gain to −1.86 ± 0.18 Δ%RSNA/ΔAP (P < 0.05 Fig. 4C).

**DISCUSSION**

This study provides important new anatomical and physiological information on the pathways necessary to retain adequate baroreceptor control of AP after SCI. We investigated the thoracic regions of the rat spinal cord that are important for baroreceptor regulation of RSNA after spinal lesions. Our results suggest that the pathways responsible for baroreceptor-induced increases in RSNA at reduced AP descend diffusely and bilaterally in the spinal cord. We also show that the
pathways responsible for baroreceptor-independent, tonic inhibition of ongoing, spinaly generated, sympathetic activity are localized dorsally and ipsilaterally. Although the regulation of the descending inhibitory pathways is independent of the baroreceptors, the tonic inhibition that they mediate, coupled with baroreceptor-induced withdrawal of sympathetic activation (discussed below), determines the minimum level of RSNA at elevated AP.

The spinal pathways mediating ongoing sympathetic activity in the rat (15, 35, 36, 38) cat (12, 17–19), and human (13) have been well described. Although some evidence from postmortem studies in humans indicates that spinal sympathetic pathways are relatively diffuse (34), others have concluded that these pathways are more concentrated in the dorsolateral white matter (13). The data from humans agree with electrophysiological data from cats, which suggest that a sympathoinhibitory pathway descends in the ventral spinal cord and a sympathoexcitatory system descends in the dorsolateral funiculus (12, 17). Although the locations of descending spinal pathways in human and cat appear similar, many species-related differences exist. For example, the major portion of the crossed corticospinal tract in rats is located in the dorsal columns, whereas in cats, as in most other mammals, it is located in the dorsolateral funiculus.

Few experiments have been conducted on pathways explicitly involved in the baroreflex regulation of AP. In particular, prior to this study, the regions of the spinal cord in the rat necessary to retain baroreceptor-mediated regulation of RSNA after SCI have not been investigated. Although we have previously studied the baroreceptor regulation of RSNA in rats after contusion lesions of the spinal cord (33), in the present experiments surgical lesions provided better localization of spinal pathways involved in baroreceptor regulation after SCI.

Baroreceptors are most commonly thought to regulate sympathetic activity by inhibiting the ongoing activity of brainstem neurons which, in turn, excite spinal sympathetic preganglionic neurons. A decrease in AP decreases the discharge rate of baroreceptors, which, in turn, decreases tonic inhibition of bulbospinal sympathoexcitatory neurons, resulting in increased sympathetic activity. Conversely, an increase in AP increases the discharge rate of baroreceptors, increasing the inhibition of bulbospinal sympathoexcitatory neurons, resulting in decreased sympathetic activity (2, 16). According to this view, baroreceptors modulate sympathetic activity exclusively by regulating descending excitation of spinal sympathetic preganglionic neurons. Therefore, spinal cord lesions would be expected to affect baroreceptor regulation of sympathetic activity exclusively by destroying descending excitatory drive to sympathetic preganglionic neurons. However, spinal sources of sympathetic drive also exist (6, 32, 35, 37, 38). In animals with intact spinal cords, these sources are tonically inhibited by descending pathways that do not appear to be under baroreceptor regulation. Thus, in addition to reducing baroreceptor-mediated sympathoexcitatory drive at low AP, damage to descending spinal pathways releases a spinal source of sympathetic activity. Destruction of the pathways responsible for the tonic inhibition of the spinal activity compromises the ability of the baroreceptor system to reduce sympathetic activity at elevated
AP. Although some evidence suggests that baroreceptors also modulate sympathetic activity via bulbospinal sympathoinhibitory pathways, this modulation appears to be relatively modest, and its existence does not significantly alter the interpretation of the present experiments (9–11, 26, 30). Thus, although we cannot rule out the effect of a spinal component of the baroreflex in our experiments, unless the spinal control is near or above the lesion site its effects would appear to be substantially smaller than those mediated supraspinally.

The analysis of our data has assumed that all of the effects of spinal lesions on baroreceptor regulation resulted from damage to descending spinal pathways. We have considered whether some of the observed effects were caused by concomitant damage to ascending or propriospinal pathways. Although we cannot rule out effects caused by damage to nondescending pathways, we know of no reports of spinal baroceptor afferents. Furthermore, spinal circuits are not responsive to non-ischemic levels of AP (1).

The ability of the baroreceptor system to prevent orthostatic hypotension by increasing sympathetic nerve activity at reduced AP is manifested by the height of the plateau of RSNA on the left side of the baroreceptor function curve. This parameter was not significantly affected by hemisections of either the left or right spinal cord nor by bilateral lesions of the dorsal spinal cord. Because the ability of the baroreceptor system to increase RSNA was not compromised after such widespread lesions, we conclude that the descending pathways that mediate this increase are diffuse and bilateral. The clinical significance of this diffuse projection is that some degree of baroceptor-mediated sympathoexcitation may be spared by even large spinal cord lesions.

The effect on baroreceptor regulation of releasing the activity of spinal sympathoexcitatory systems by destroying descending inhibitory pathways is more complicated. In earlier studies from this laboratory, acute lesions that interrupted ipsilateral, but not contralateral, dorsal descending pathways significantly increased ongoing RSNA (32, 35, 37). We have discussed extensively the origins of ongoing RSNA activity after spinal cord lesions in previous publications (6, 32, 35–38). Evidence from those studies supports the hypothesis that this activity is produced by spinal sympathoexcitatory interneurons, which in spinal intact rats are underdescending tonic inhibition from brain stem systems. Once released from descending inhibition by spinal transection or lesions, activity in these interneurons (and thus in their postsynaptic, preganglionic neurons) is generated either by the endogenous activity of spinal networks or by excitation of spinal networks by primary afferents. Thus, in our experiments, rats with acutely lesioned dorsal spinal cords still had some degree of baroceptor-controlled sympathetic activity. However, dorsal lesions also destroyed baroceptor-independent pathways responsible for the inhibition of spinal activity. Therefore, during baroceptor responses there was a reduced inhibition of sympathetic activity at increased AP.

Our observations support the prediction of diminished baroceptor efficacy at elevated AP after dorsal spinal lesions. Acute left hemisections and dorsal hemisections dramatically reduced the ability of the baroceptor system to inhibit RSNA at elevated pressures. Because the decreases in RNSA at elevated AP were attenuated only after left and dorsal hemisections (but not right hemisections), we conclude that the descending inhibitory pathways are largely ipsilateral. Dorsal and left hemisections did not improve the ability of the baroceptor system to increase RSNA at reduced AP as might have been expected because these lesions destroyed not only the more localized, descending, inhibitory systems but some diffuse, descending, excitatory systems as well.

Studies from other laboratories have described a baroceptor-mediated inhibition of sympathetic activity at spinal levels (9–11, 26, 30). These workers suggested that at increased AP, in addition to baroceptor-mediated inhibition of descending excitatory pathways, baroceptor-mediated activation of descending sympathoinhibitory pathways contributes to the decrease in sympathetic nerve activity. In those experiments, conducted in rats in which neurons in the RVLM had been previously inhibited by medullary injections of glycine, large increases in AP modestly reduced the magnitude of a spinal sympathetic reflex (not ongoing sympathetic activity). Although we tested the effects of much smaller increases in AP, the inhibitions of ongoing RSNA observed in both the present and earlier studies (11–13, 30) were much larger than those attributed to spinal sympathoinhibition. Thus, although we cannot rule out the effect of a spinal component of the baroreflex in our experiments, its effects would appear to be substantially smaller than those mediated supraspinally.

Perspectives and Significance

Collectively, these data suggest that baroceptor-mediated excitation of RSNA is mediated by diffuse bilateral pathways in rats. If descending sympathoexcitatory pathways are similarly distributed in humans, their partial sparing after SCI could reduce the probability of orthostatic hypotension upon assuming an upright posture. Additionally, our data suggest that the ipsilateral, dorsal, descending pathways mediate tonic inhibition of spinally generated sympathetic activity. Although baroceptor regulation is not directly mediated by this descending inhibition, the presence of tonic inhibition is necessary to achieve minimum levels of sympathetic activity during baroceptor activation at elevated AP. If descending inhibition of spinal sources of tonic sympathetic activity is similarly localized in humans, sparing of the dorsolateral portions of the spinal cord could reduce the probability of autonomic dysreflexia and hypertensive crises after spinal injury. The potential danger that dorsal spinal lesions might exacerbate autonomic dysreflexia is mitigated by the fact that dorsal spinal lesions would destroy not only descending inhibitory pathways but descending excitatory pathways as well. The distribution of descending sympathetic regulatory pathways we describe is encouraging in that many incomplete spinal lesions may retain a spinal substrate for spontaneous or rehabilitation-induced recovery of cardiovascular regulation after SCI.

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DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the author(s).

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