Introduction
Lumbar intervertebral discs are recognized to be one of the major sources of low back pain. Recently, it has been demonstrated that lower lumbar intervertebral discs and adjacent tissues in the rat are innervated from L1 and L2 dorsal root ganglia (DRG) neurons through the paravertebral sympathetic trunk. 1,2,3 Although this sensory pathway has been investigated histologically and anatomically, mechanisms of discogenic low back pain are still unknown. The purposes of this study, using neurophysiological techniques in a rat model, were to investigate if sympathetic afferent discharges originating from lower lumbar intervertebral discs and adjacent tissue pass through the L2 dorsal root and to characterize these afferent units.

Materials & Methods
Thirty-one adult male Lewis rats were sedated and anesthetized by an intramuscular injection of ketamine hydrochloride (43 mg/kg), xylazine (7 mg/kg) and torbutrol (0.1 mg/kg). Supplemental half doses were used as required to maintain anesthesia throughout the experiment. Activity from peripheral receptive fields from L2 dorsal and ventral rami was abolished by cutting L2 dorsal and ventral rami, respectively. At this point, the L2 root was connected with only rami communicante to the lumbar sympathetic chain. A laminectomy from L1 to L6 was performed to expose L2 dorsal nerve root and the dorsal aspect of L5/6 intervertebral disc. After cutting the dura, a pool was formed from skin flaps, and the spinal cord and nerve roots were immersed in warm (37°C) mineral oil to prevent nerve roots from drying. The left L2 dorsal root was detached from the spinal cord and draped over one or two bipolar platinum recording electrodes to examine afferent units. The L2 root was then split to record from a smaller number of units. The impulses were amplified, monitored on an oscilloscope and an audio monitor, digitized and analyzed using PC-based spike discrimination and frequency analysis software (R.C. Electronics, Goleta, CA). For later detailed analysis, the data were also simultaneously recorded on an analog tape recorder (MR-30; TEAC, Montebello, CA).

Mechanical Stimulation:
Lumbar structural elements including the dorsal aspects of L5/6 discs and adjacent tissues were mechanically probed with blunt glass rods and a 25 ga needle. Once the receptive fields were identified, mechanical threshold of the afferent units were characterized using calibrated nylon filaments. Separately, the test probing on bone lamina was performed to insure no artifact responded to lumbar spinal motion.

Electrical and Chemical Stimulation:
If the disc and adjacent tissues did not respond to any mechanical stimulation, electrical stimulation with a bipolar electrical stimulator (1 to 20 V) was applied to the dorsal aspects of L5/6 disc and adjacent tissues to evoke action potentials and obtain latency. Later, based on the latencies and the distance measured, nerve conduction velocity (CV) was calculated. Finally, the chemical application with 2% carrageenan (0.1 ml) to L5/6 disc was performed to produce inflammation. The change of discharge rate and the response of L5/6 disc to mechanical probing were observed and recorded.

Results
Receptive fields: Psoas muscle units were observed in about 40% (12/31) of the experiments. No systematic attempt was made to evaluate quantitatively the mechanical thresholds because of the variable location of the receptive fields. Only three units were identified in the DRG (Figure 1). Meanwhile, we could not get any units in the L5/6 discs.

Spontaneous discharge: Spontaneous irregular bursting discharges, not responsive to any mechanical stimulation, were sometimes observed. These may be visceral sympathetic afferents (Figure 2).

Conduction velocities: We identified 42 units with CVs, 7.86±4.9 m/s (2.56 to 21.60). Most of these CVs belonged to A-delta fibers.

Inflammation: Inflammation with carrageenan was tested in 19 experiments. In only 5 cases, response to mechanical probing of the L5/6 disc was produced after exposure to carrageenan.

Discussion
Our studies appear to be the first to functionally demonstrate that afferent units from the dorsal aspect of the lower lumbar spine pass through L2 dorsal roots via lumbar sympathetic trunk. Other studies have recorded sensory spinal activity from the ventral side of the spine through the sympathetic chain 4 and the rami communicantes.5 Our study also demonstrated that L5/6 intervertebral discs units were not responsive to mechanical stimulation under normal conditions, while after the inflammation, some of these mechanically insensitive afferents responded to mechanical stimulation. These “silent nociceptors” have been reported in the digestive system related to the autonomic nerve fibers partially passing through sympathetic chain. The afferent fibers innervating the colon became sensitized to mechanical stimuli during inflammation.6 In one clinical series, approximately one-third of locally anesthetized back surgery patients had no pain although the affected annulus fibrosus was mechanically stimulated.7 Similarly, it has been reported that in lumbar discography to a group of asymptomatic patients, only 10% was painful.8 Our results may be a reasonable explanation for the clinical phenomena that mechanical stimulation to lumbar disc may not always produce pain. Inflammatory changes in the tissue may cause silent nociceptors to become responsive to mechanical stimuli.

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Reference: