

## Minireview

# The Spinal Ganglion —An Ignored Nucleus?

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## Abstract

Somatic primary afferent neurons located in dorsal root ganglia and their cranial nerve equivalents have been recognized as not indispensable in sensory transmission for a long period of time. Active spontaneous activities, extra- and postspike spikes, four response patterns, unusual action potential waveforms, as well as somatovisceral and bilateral activation were shown in the dorsal root ganglion neurons. These findings suggest that synaptical/ junctional activities, dichotomized peripheral processes, somatovisceral and bilateral cross innervation exist in the ganglion. It would thus be tempting to reevaluate its structure and function and restore the nuclear essence of dorsal root ganglion to its original place.

**Key Words:** dorsal root ganglion, spontaneous activity, extra/postspike spike, small depolarization, atypical action potential, excitatory postsynaptic potential, synaptic/junctional contact, dual innervation, cross-innervation

## Introduction

The dorsal root ganglion (DRG) is distinguished by its accessibility, simplicity of connections, and unique T-shaped neurons. It is not surprising that the DRG has been considered as a rewarding model for the study of basic principles of sensory processing and that its neurons have been the subject of intensive study for over a hundred years (16).

To date much is known about the various aspects of DRG structure and function. Questions, however, remain regarding the involvement of DRG neurons in the generation of spontaneous activity, the functional significance of action potential propagation into their somata, and the possibility that these cell bodies have integrative roles. In order to examine these issues we undertook a systematic study of the DRG in the cat, rat, and toad models.

Experiments were conducted first on adult cats anesthetized with pentobarbital. Microelectrode recordings were made mainly from the S1 and L7 DRG neurons. Primary afferent A fibers of the related dorsal roots and the sciatic nerve were electrically stimulated while they were kept intact, locally anesthetized, or locally anesthetized and sectioned. In the latter two cases, the relevant ventral roots and

the gluteal nerve were also routinely cut. Most experiments were done under the latter two conditions. However, no differences were observed in the results among these three designs.

## DRG Study in Cats

### *High- and Low-Frequency Spontaneous Activity of Cat DRG Neurons In Vivo (9)*

Spontaneous and extra discharges were observed in 50 cells. Fifteen neurons showed background activity under conditions with no extra-ganglionic origin and without any intentional stimulation. The rate of firing varied from 12.5 to 100 c/sec (Fig. 1). The action potentials recorded from the neurons at high frequency often showed a distinct prepotential preceding their spike potentials; in contrast, the action potentials of neurons firing at low frequency often displayed a distinction potential after hyperpolarization. These activities are basically rhythmic. However, their rhythmicities are irregular observed by the histogram taken over a long period of time. These rhythmicities and prepotentials are reminiscent of those of pacemaker cells.

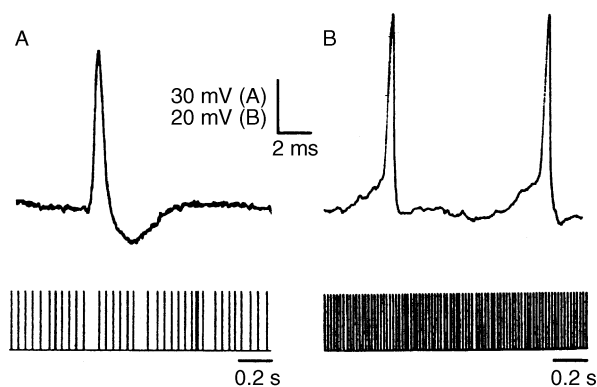


Fig. 1. (A) Representative records of low-frequency spontaneous discharges (ca., 18Hz). Top panel: an intracellularly recorded action potential. Note the distinct afterhyperpolarization. Bottom panel: rate-meter records of window-discriminated spikes. Each bar represents a single action potential. Note that a somewhat irregular firing rate is evident only over a longer time period. (B) Representative records of high-frequency spontaneous activity (ca., 60Hz). Top panel: intracellularly recorded action potentials. Note the presence of distinct prepotentials during the rising phase. Bottom panel: rate-meter records as in A. [Reproduced with permission from G. W. Lu et al.: Brain Res. Bull. 31: 523-530, 1993(9). ©Elsevier.]

#### *Extra- and Postspike Spikes of Cat DRG Neurons In Vivo* (9)

When the neurons were electrically stimulated at low frequency, the expected evoked action potentials might be followed by one or more extra action potentials (Fig. 2). These extra spikes occurred 5 msec or more after their preceding spikes. They were variable in latency and easily missed even at very low frequency stimulation.

Postspike spikes (Fig. 3) were shown during high frequency stimulation. These postspikes events occurred immediately following their preceding evoked potentials and lasted at even higher frequencies of stimulation.

Both the background and stimulus-triggered extra and postspike activities interacted with the evoked discharges. The interaction between the spontaneous and evoked activities was related to their relative frequency. The lower frequencies of discharges were always collided by higher ones despite they were spontaneous or evoked activities. If these normally occurred spontaneous and extra activities could distort the evoked activity or sensory information, the intentionally applied stimulus-evoked activity might exert its role in the reverse direction: distortion and/or block of arriving information signals.

#### *Response Pattern of Evoked Potentials of Cat DRG Neurons In Vivo* (6)

Sixty seven neurons showed response to the

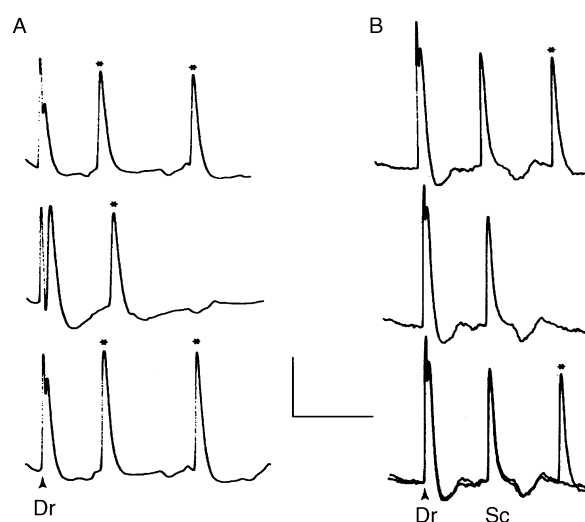


Fig. 2. Examples of stimulus-related discharges occurring in the form of extra spikes (asterisks) and seen during low-frequency repetitive stimulation at 2 Hz. (A) Three consecutive stimuli delivered to the Dr produced one or two extra spikes in addition to the expected evoked spikes. Note the latency change in both the extra spikes and the evoked spikes. Note also that one of the extra spikes missed (middle trace) during repetitive stimulation. (B) In another DRG neuron an extra spike follows the evoked spikes induced by stimulation of both Dr and Sc. The bottom trace was obtained by superimposing the top and middle trace. Note again that the extra spike missed. Arrowheads mark stimulus artifacts. Dr = dorsal root, Sc = sciatic nerve. Calibration for both A and B = 8 ms, 15 mV. [Reproduced with permission from G. W. Lu et al.: Brain Res. Bull. 31: 523-530, 1993(9). ©Elsevier.]

stimulation of the dorsal roots (Dr), the sciatic nerve (Sc), and both Dr and Sc. The average threshold for Dr stimulation (3.92V) was 1.6 times higher than the Sc stimulation (2.41V). The conduction velocity from the Sc averaged 71 m/sec in average, with a range of 36 to 110 m/sec, suggesting the cells are type A cells in the ganglion. Both conduction velocity and threshold values indicate that the Dr branch or the central process of DRG neurons is smaller than their Sc branch. Their peripheral fibers are thus in the range of A $\alpha$ , A $\beta$ , and probably A $\delta$  but not C fibers.

The general pattern of responses of these type A ganglion neurons to repetitive stimulation of their peripheral (Sc) and central (Dr) processes included primarily 4 changes: including latency (jitter), amplitude (reduction), configuration (decomposition), and number (missing spikes) (Figs. 4 and 5).

Interestingly, some neurons responded to stimulation of their Dr and Sc in different ways. The same neuron showed jitter at low frequency stimulation when Dr was stimulated while no change in latency was shown during Sc stimulation. At higher frequency of stimulation, the Dr stimulation induced responses started to miss while the Sc evoked ones began to change their shape. At even higher frequency of stimulation, the Dr evoked responses started to change

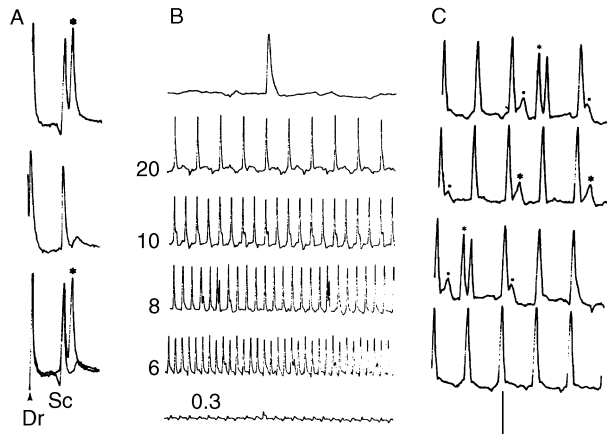


Fig. 3. Stimulus-related discharges of three different DRG neurons occurring in the form of postspike events (asterisks) at low (A) and high (B,C) frequency of stimulation. (A) Recorded during stimulation at 2 Hz. The top and middle tracings are superimposed on the bottom tracing. Note the latency change in the evoked spikes, and the missing S-like spike in the middle trace. (B) Recorded at different frequencies of stimulation (numbers next to the tracings represent interpulse intervals, or IPI, in ms). Note a series of postspike events (humps M-like, IS-like) which all disappear at a high stimulation frequency (IPI of 0.3 ms or 3000 Hz). (C) Recorded during stimulation at 125 Hz (8 ms IPI). Note the intermittent postspike events (asterisks). Calibration: A and C = 8 ms, 30 mV; B = 8 ms, 50 mV for the uppermost trace; 40 ms, 30 mV for the other trace. [Reproduced with permission from G. W. Lu et al.: *Brain Res. Bull.* 31: 523-530, 1993(9). ©Elsevier.]

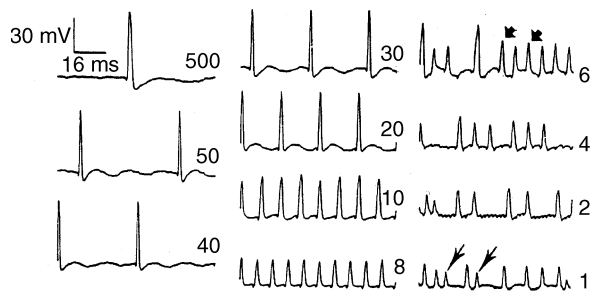


Fig. 4. Examples of the changes in spike number, amplitude and configuration seen in a cat spinal ganglion neuron following repetitive stimulation of the sciatic nerve. Numbers to the right of the trace indicate stimulus inter-pulse intervals (IPIs) in ms. Note that the full spike decreases in amplitude at a frequency of stimulation greater than 20 ms IPI (50 Hz), and begins to decompose into non-myelinated (thick arrows) and myelinated (thin arrows) components. Complete spike failure is shown at 6 ms IPI (167 Hz). [Reproduced with permission from G. W. Lu et al.: *Neuroscience* 39: 259-270, 1990(6). ©Elsevier.]

their shape but it was still different from the response induced by Sc stimulation.

The majority of cells were missing at high frequency of stimulation. The full spikes (S) of these cells were unable to follow 200 Hz and began to degrade into non-myelinated (NM) and myelinated

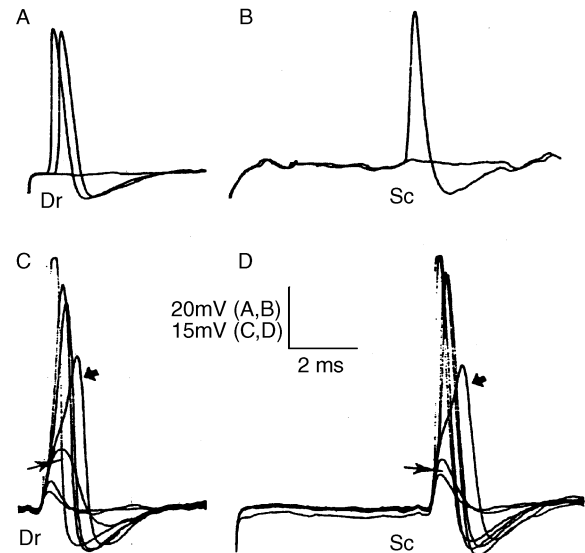


Fig. 5. Faster sweep examples of the changes in latency, number, amplitude, and configuration of the evoked action potentials recorded from different spinal ganglion neurons following repetitive stimulation of the dorsal root (Dr) or sciatic nerve (Sc). (A) Three sweeps taken at different rates of stimulation are superimposed to illustrate the jitter in onset latency (at 25 Hz), and the eventual inability of 20 Hz stimulation to evoke a response in a 1:1 manner. (B) Two superimposed sweeps illustrate full spike failure in another neuron occurring at 20 Hz. (C and D) About seven superimposed sweeps obtained in two different neurons illustrate the reduction in action potential amplitude, and decomposition of the full spike into non-myelinated (thick arrows) and myelinated (thin arrows) components, during progressive increases in stimulation frequency from 10 to 167 Hz. [Reproduced with permission from G. W. Lu et al.: *Neuroscience* 39: 259-270, 1990(6). ©Elsevier.]

(M) spikes. The term full spike is composed mainly of the S, NM and M spikes. The term NM or initial segment (IS) spike covers M spike. All these terms are analogous to those used for the spinal motoneurons.

If we count the number of full spikes, NM spikes, and M spikes separately at different frequencies of stimulation, then three types of change could be categorized. In the first group there was no decomposition until the full spike missed suddenly. In the second group the full spike decomposed only into NM spikes. In the third group the full spike decomposed into NM and M spike alternately, and the ability of frequencies following was thus in the order of  $M > NM > S$ .

When the stimulus frequency at which the full spike starts to jitter or miss as a standard of frequency following ability, these type A spinal ganglion neurons showed a quite wide spectrum of frequency following, ranging from 2 Hz to 500 Hz. The ability of frequency following of the DRG neurons appeared to have no significant correlation with the recording position, refractory period, and latency. There seems to be a

**Table 1. Parameters of typical and atypical spike potentials.**

	RT (ms)	FT (ms)	AMP (mV)	DUR (ms)	Area ( $\mu$ Vs)
Typical (n=49)					
Mean $\pm$ SE	0.29 $\pm$ 0.01	0.70 $\pm$ 0.04	91.04 $\pm$ 2.51	1.75 $\pm$ 0.10	165.38 $\pm$ 24.11
Range	0.16-0.49	0.13-1.52	50-110	0.95-4.03	18-585.66
Atypical (n=8)					
Mean $\pm$ SE	1.13 $\pm$ 0.15*	0.61 $\pm$ 0.09	75.00 $\pm$ 4.91 <sup>#</sup>	2.99 $\pm$ 0.44*	370.50 $\pm$ 99.09 <sup>Δ</sup>
Range	0.67-1.80	0.35-1.13	60-90	1.10-4.66	53.13-1000
Pooled sample (n=57)					
Mean $\pm$ SE	0.41 $\pm$ 0.04	0.69 $\pm$ 0.03	88.76 $\pm$ 2.32	1.93 $\pm$ 0.12	194.12 $\pm$ 22.26
Range	0.16-1.80	0.35-1.52	50-110	0.95-4.66	18-1000
Ratio (atypical / typical)	3.89	0.87	0.82	1.71	2.24

Significantly different from the typical parameter at the \* $P < 0.0001$ , <sup>#</sup>0.05, and <sup>Δ</sup>0.001 levels. [Reproduced with permission from G. W. Lu et al.: Brain Res. Bull. 31: 531-538, 1993(21). ©Elsevier.]

tendency of correlation between the ability of frequency following and the conduction velocity.

#### *Unusual Waveform of Action Potentials of Cat DRG Neurons In Vivo (21)*

In addition to the typical configuration of action potentials, 23% of the sample of 60 DRG neurons displayed a distinct prepotential and slow rise time in the rising phase (also known as atypical action potential). Interestingly, the two kinds of action potentials could even be seen in some single neurons, responding to Dr and Sc stimulation (Fig. 6).

As shown in the Table 1, the rise time (RT), duration(DUR), and area of these atypical action potentials were almost 4.0, 1.7, and 2.2 times larger than those of the typical action potentials. The fall time of typical spikes is 2.4 times longer than their rise time, while the rise time of the spike preceded by prepotentials is 1.9 times longer than the fall time (Table 1).

Some neurons illustrated delayed or distorted depolarization that progressively developed into a postspike hump, M-like and NM-like component. Some neurons exhibited a small and late depolarization when the neurons were stimulated with straddling threshold (0.9 threshold) intensity (Fig.7). These minidepolarizations were relatively constant in their size and occurred in an "all or none" manner.

#### **DRG Study in Toads**

##### *Responses of Toad DRG Neurons In Vitro (1, 2, 7, 8, 10, 12, 13)*

Similar to the cat DRG neurons *in vivo*, the

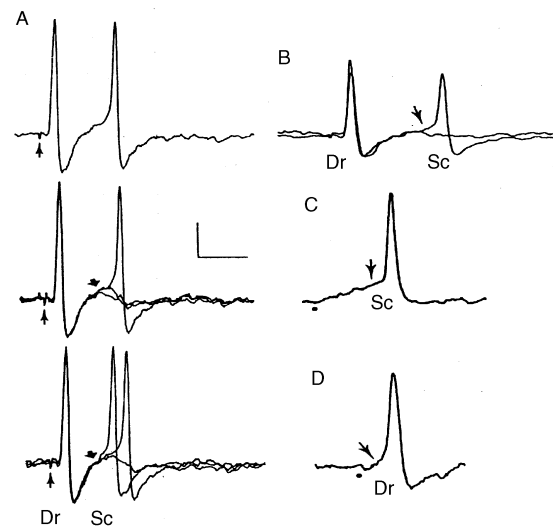


Fig. 6. Intracellularly recorded action potentials illustrating the presence of prepotentials recorded from four of our spinal ganglion neurons. (A) (Top trace) Single sweep tracing. Note the difference in action potentials recorded from the same neuron, and the clearly observable prepotential preceding the sciatic-evoked spike (Middle and bottom traces). Superimposed records of three consecutive sweeps in the same neuron (bottom trace). Note the failure of the prepotential to trigger a spike, and the jitter in onset latency of the spike potentials triggered by prepotentials (thick arrows). Stimulation frequency was 1 Hz for the top trace, and 2 Hz for the middle and bottom traces. The dorsal root (Dr) and sciatic nerve (Sc) stimulus pulses were separated by 5 ms. Thin arrow, stimulus artifact. (B) Two superimposed traces illustrating the prepotential (thin arrow) in another neuron. Full spikes were recorded at 2 Hz, and the failing sciatic spike was observed at 10Hz. (C and D) Prepotentials (thin arrows) were also recorded (at 2 Hz) in two additional spinal ganglion neurons activated by sciatic (C) or dorsal root (D) stimulation only (dots indicate stimulus onset). Calibrations: A= 20 mV, 8 ms; B= 30 mV, 4 ms; C and D = 25 mV, 4 ms. [Reproduced with permission from G. W. Lu et al.: Neuroscience 39: 259-270, 1990 (6). ©Elsevier.]

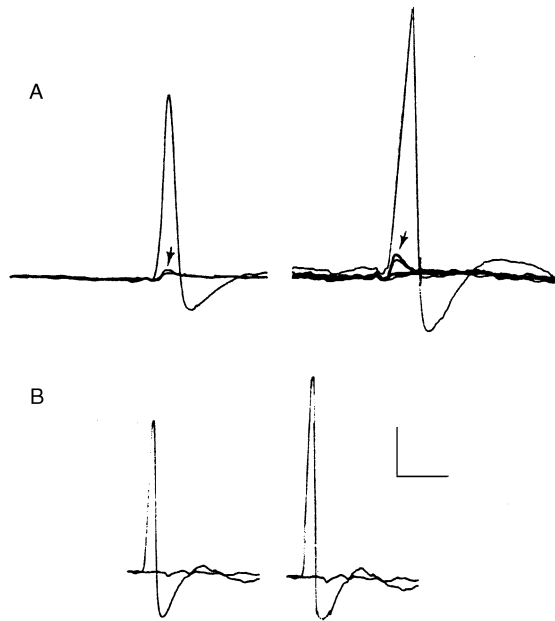


Fig. 7. Intracellularly recorded action potentials from two DRG neurons exhibiting the presence (A) and absence (B) of small depolarizations (arrow) (A) Responses to subthreshold stimulation (0.9T) of the Dr. The left-hand trace shows three, and right-hand trace about 10 superimposed sweeps in the same neuron. Note that the small depolarizations in A appear intermittently, and occur with a different onset latency from the action potential. (B) Two superimposed sweeps following similar stimulation of the Dr in another DRG neuron. The right-hand trace is an expanded view of the left-hand trace. Calibration: A= 4 ms, 25 mV (left trace), 15 mV (right trace); (B)= 4 ms, 25 mV (left trace), 20 mV (right trace). [Reproduced with permission from G. W. Lu et al.: Brain Res. Bull. 31: 531-538, 1993(21). ©Elsevier.]

above mentioned changes were also seen in toad DRG neurons in vitro as stimulus frequency progressively increased. Latency drift or delay, amplitude reduction, decreased and enlarged after hyperpolarization as well as decomposition of full spike into wave NM and M through missing were also seen. Nonsynchronous responses occurred in 70% toad DRG neurons when the related Dr and Sc were stimulated. The ability of related Dr and Sc to follow high frequency stimulation differed greatly, averaging 126 and 323 Hz. Following ability to high frequency stimulation was significantly decreased when the DRG was perfused with a solution containing high magnesium and low calcium or GABA (Fig. 8).

#### *Synaptic/Junctional Contact (1, 2, 6-10, 12, 13, 17, 19, 21)*

To discuss and understand the mechanisms underlying the facts mentioned above, many questions could be raised. Why are the DRG neurons so active even in the absence of any extraganglionic stimulation? How to explain the occurrence of extra- and

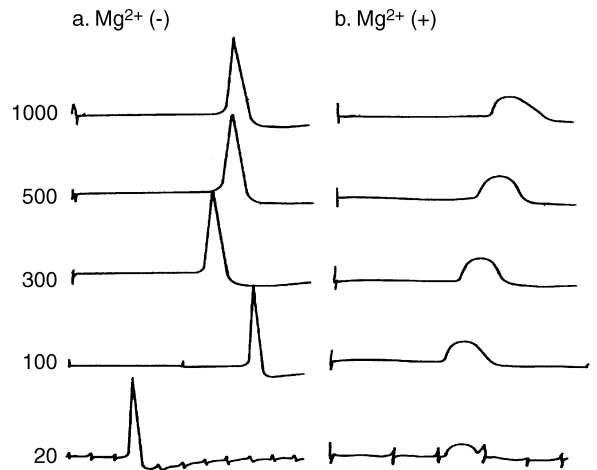


Fig. 8. Action potentials (a) and dome-like depolarization (b) during perfusion of  $Mg^{2+}$  free (a) and high  $Mg^{2+}$  (b) solution. Note the missing of action potentials and dome-like depolarizations as well as the jitter in their latencies. Calibration: 20 ms, 40 mV, except 100 and 20 ms IPI in (a), which are 40 ms, 40 mV. [Reproduced with permission from G. W. Lu et al.: Chin. Sci. Bull. 41: 1735-1740, 1996(13).]

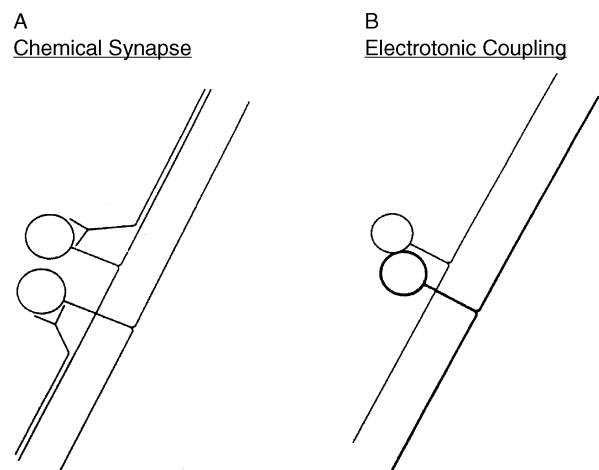


Fig. 9. Diagram illustrating the proposed synaptic (A) and electrotonic coupling (B) contact in DRG. A: A chemical synapse between a DRG neuron and a presynaptic terminal coming via Dr and Sc in two neurons. B: An electrotonic coupling contact between two cell bodies of two DRG neurons by a juxtaposition. Explanation: see text. [Reproduced with permission from G. W. Lu et al.: Brain Res. Bull. 31: 531-538, 1993(21). ©Elsevier.]

postspike spikes or events triggered by electrical stimulation of Dr and Sc? Why is the frequency following spectrum of the DRG neurons so wide? How to interpret the poor ability of DRG neurons to follow high frequency stimulation? Why single DRG neurons respond to stimulation of their peripheral and central process so different? And what are the nature or mechanisms underlying the prepotential, the small

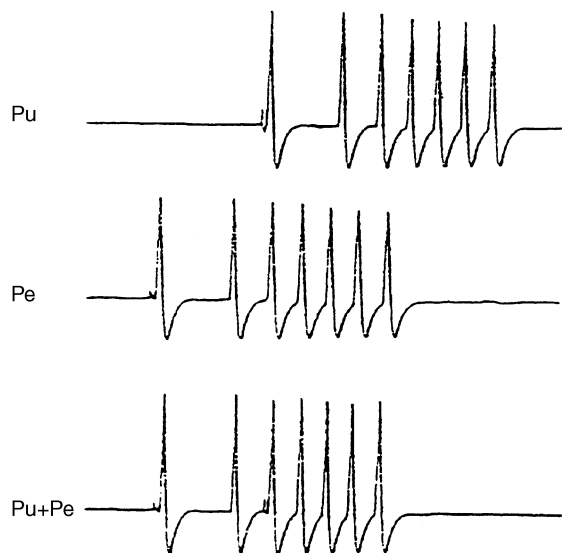


Fig. 10. Multiple responses of single DRG neurons to single pulse stimulation of Pu and Pe. Note the longer latency and slower rising phase shown in the extra spikes. Calibration: 10 ms, 20 mV. [Reproduced with permission from G. W. Lu et al.: Chin. J. Neurosci. 3: 59-63, 1996 (15).]

depolarization and the distorted repolarization?

One of the answers might be the existence of a second independent pathway in the DRG. The pathway might exert its role by way of chemically mediated synapse and/or electrically coupled junction (Fig. 9). The variable onset latency of the prepotentials, the prepotential triggered spikes, and their inability to follow repetitive stimulation are very reminiscent of responses judged to be synaptically mediated. This might explain the origin of the slowly-rising prepotentials that preceded these atypical action potentials.

The generation of the small depolarization that occurred during subthreshold stimulation might be more appropriately explained by an electrotonic coupling junction. Specialized membrane juxtaposition between two DRG neurons may allow the action potentials to spread exponentially from one stimulated neuron to its neighbor through a low resistance path. When the stimulus intensity is subthreshold for the recorded neighbor neuron but superthreshold for the stimulated one, the former's action potential would be observed as a small, all-or-none depolarizing potential with fixed latency and size.

### DRG Study in Rats

*Dual Innervation on Both Somatic and Visceral Tissues of Single Rat DRG Neurons In Vivo (5, 11, 14, 15, 18, 20)*

The DRG neurons, as somatic sensory neurons,

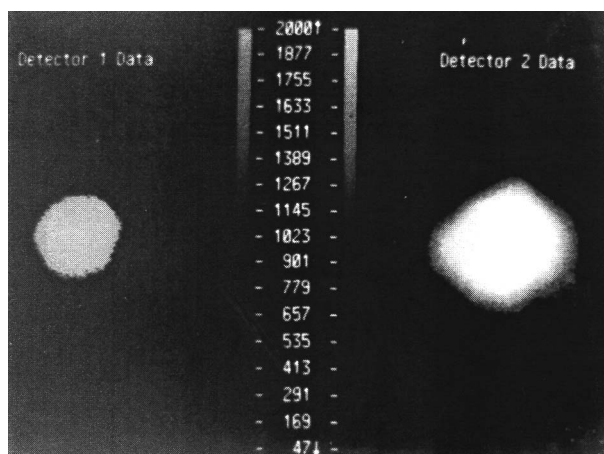


Fig. 11. Confocal microphotograph of a double labeled cell in S1 spinal ganglion. Confocal microphotograph of the cell taken by two-detector system. Left, NY labeled nucleus. Right, FB labeled cytoplasm. [Reproduced with permission from G. W. Lu et al.: Chin. Sci. Bull. 43: 137-139, 1998(20).]

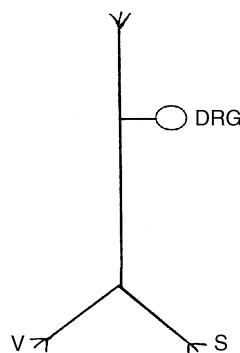


Fig. 12. Diagram illustrating a DRG neuron and its proposed dichotomized primary afferents. V: Visceral tissue, S: Somatic tissue. [Our unpublished results]

responded to somatic stimulation. However, some DRG neurons responded to receptive filed stimulation of both somatic and visceral areas, while other neurons responded to both pudendal and pelvic nerve stimulation (Fig. 10). The neurons responded with multiple discharges when only one stimulus pulse was delivered to pudendal or pelvic nerve. The pudendal nerve stimulation induced responses and the pelvic nerve stimulation induced response could be collided with each other. The positive result of the collision indicates that the stimulated is the two-branches of the parent primary afferent rather than it's the proximal and distal points.

After injection of fast blue (FB) was injected into the subcutaneous tissue of the perineum and nuclear yellow (NY) into the subserous lamina of the bladder wall in the rat, single labeled FB, NY, and double labeled FB+NY cells were found in the DRG

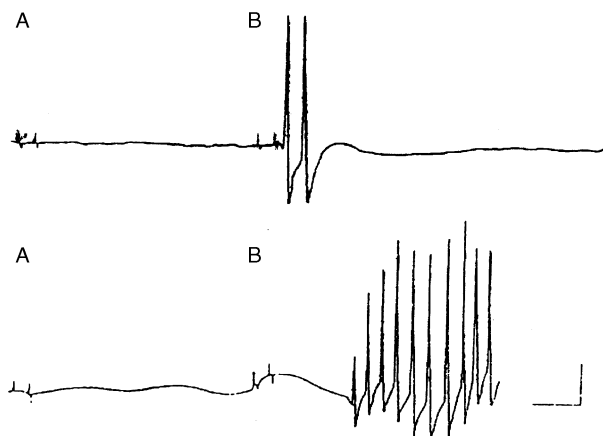


Fig. 13. Short (top) and long (bottom) latency responses of DRG neurons to contralateral Dr stimulation (B) Note no responses were shown when ipsilateral Dr was stimulated (A). Two small vibrations on the trace: stimulus artifacts. Calibration: 20 mV, 10 ms. [Reproduced with permission from G. W. Lu et al.: *Chin. J. Neuroanat.* 13: 327-330, 1997(3).]

of L6, S1 and S2 segment (Fig. 11). The amount of FB, NY and FB+NY labeled neurons were 146, 186 and 81 out of a total of 463 labeled cells and their proportion was 40%, 51% and 9%, respectively. The results indicate that the DRG neurons dually innervate both the somatic and visceral tissues and the convergence of somatic-visceral sensory information from the related tissues via a dichotomized primary afferent (Fig. 12).

#### *Cross Innervation between Bilateral DRG Neurons in Rats In Vivo (3)*

A total of 68 units were intracellularly recorded from L6-S1 DRG in the rat. The discharges were evoked by stimulation of Sc and Dr (Sc/Dr), pelvic (Pe) and Dr (Pe/Dr), Sc, Pe and Dr (Sc/Pe/Dr) and contralateral Dr. The number of units responded to Sc/Dr, Pe/Dr, Sc/Pe/Dr and contralateral Dr were 33, 11, 16 and 8, respectively. The conduction velocity of Sc, Pe and Dr was  $36.8 \pm 17.9$ ,  $36.8 \pm 18$ , and  $32.1 \pm 16.4$  m/s, respectively. The responses evoked by stimulation of contralateral Dr were divided into three types: short latency, long latency and both (Fig. 13). The results suggest that DRG neurons have branched central and peripheral processes and receive input from crossed fibers of opposite DRG (Fig. 14).

### Conclusion

It has been generally recognized for more than 100 years that the DRG is only a simple assembly of afferent pathways and that the cell body of DRG

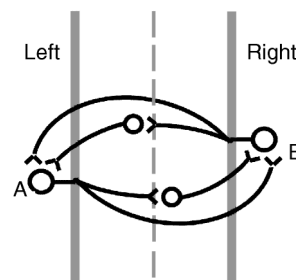


Fig. 14. Diagram showing the proposed cross innervation between bilateral DRG neurons. Note the neurons close to midline are interneurons. [Our unpublished results]

neurons serves solely on a nutritive depot for its processes (4). This classical concept on DRG and its neurons should be challenged by above mentioned facts. It thus should be acceptable that the DRG is a “laterally displayed portion of the spinal cord” (22) and that the nuclear essence of DRG should be restored to its original place.

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