

The Vascular Pattern of the Human Dorsal Root Ganglion and Its Probable Bearing on a Compartment Syndrome

Wesley Wilkin Parke, PhD,* and Joseph Leo Whalen, MD, PhD†

Study Design. A descriptive anatomic investigation of the vasculature of the dorsal root ganglions.

Objectives. To determine whether the blood supply of the various spinal ganglions is sufficiently consistent to derive a "generic" description and illustration that would be applicable to all spinal levels, and to ascertain whether this vascular pattern is inherently predisposed to the development of a closed compartment syndrome.

Summary of Background Data. The few previous descriptions of spinal ganglionic vasculature do not include photographic evidence showing uniformity in the arterial distribution plan at all ganglionic levels. The venous drainage, although verbally reconstructed from microscopic sections, lacks any indication of its probable role in the etiology of a compartment syndrome.

Methods. Three perinatal cadavers received latex/India ink injections, and their removed radiculomedullary systems were cleared, transilluminated, and macroscopically photographed. Paravertebral sections were grossly removed from the spines of two adult anatomic cadavers and received retrograde venous injections of a fine suspension of barium sulfate. The intervertebral foraminal tissues were then dissected from the bone, and radiographs of them were made. For comparative reference, a nerve root/ganglion complex of a rabbit was arterially injected with a more dilute preparation of the latex/India ink suspension.

Results. Macroscopic photographs of perinatal dorsal root ganglions showed that the pattern of the intraganglionic arterial distribution was sufficiently consistent to allow a graphic rendering and labeling of a "generic" ganglion. The series of incomplete retrograde venous injections adequately indicated the pressure labile location of a periganglionic venous plexus.

Conclusions. The common development, structure, and function of the human dorsal root ganglions have resulted in the evolution of a uniform nutritional vascular pattern that can be conceptualized in a single visual image. Its plan of a primarily internal arterialization with a superficial venous drainage renders it vulnerable to the ischemic conditions consequent on external pressures and/or internal edematous swelling. This vascular arrangement may contribute to a propensity for the ganglion to develop a compartment syndrome when subjected to compression by periforaminal degenerative or neoplastic space-occupying lesions. [Key words: spinal ganglion, neural ischemia, radiculopathy, mechanosensitivity] *Spine* 2002;27:347-352

Relatively contemporary and reasonably comprehensive accounts of the radiculomedullary circulation provide little or no mention of the nutrient vascular distribution to the dorsal root ganglion (DRG).^{1,3,4,7,17} The scarcity of citations specifically referable to these blood vessels may give the impression that such information is generally unknown. However, the vascular patterns unique to the DRG received a well-detailed treatment by Bergmann and Alexander² several decades before most investigations of the intrinsic radicular circulation were conducted. Although some basic elements of their report have been briefly substantiated by two subsequent articles,^{5,21} it has not been superseded in its wealth of observation, description, and illustration. The lack of a general awareness of this work may be attributed to the fact that its publication preceded the development of sufficient interest and background information necessary to emphasize its clinical significance.

Unfortunately, Bergmann and Alexander relied only on pen-and-ink graphics to corroborate their verbal descriptions of the larger vessels, although they provided an exceptional series of photomicrographs to illustrate the microscopic cross-sections of their injections of the finer intraganglionic vasculature. Subsequently, Day,⁵ in 1964, in a study of the arterial circulation of the lumbosacral plexus, published photographs of darkly injected DRGs, but they were confined to a few examples of the lower lumbosacral ganglions. Somogyi et al,²¹ in 1973, provided fairly clear photographic depictions of the T6 and L4 ganglions that substantiated the drawings of Bergmann and Alexander, but a comprehensive photographic display of a series of injected DRGs derived from the four major regions of the spine was still lacking.

More recent studies^{7,11,12,18,19} have concentrated on the functional aspects of DRG circulation with a greater regard for the measurable physiologic reactions than for the morphologic intricacies of the vascular pattern. Although many of these physiologic responses are widely understood to be caused by alterations in the nutritive system, interest in the structural arrangements of the vessels has not received equivalent consideration. This apparent neglect of the probable roles of the vascular peculiarities of the DRG may originate somewhat in the recognition that much of the vessel architectonics of the entire human radiculomedullary circulation is unique to the species, and the functional generalizations derived from various experimental animals are demonstrably more reliable than the morphologic ones.¹⁶ Therefore, the major intentions of this study were to provide a pho-

From *The Robert E. Van Demark Institute of Anatomical Research, Division of Basic Medical Sciences, University of South Dakota School of Medicine, Vermillion, South Dakota, and the †University of Southern Illinois School of Medicine and The Orthopedic Institute of Doctors Hospital, Springfield, Illinois.

Acknowledgment date: March 13, 2001.

First revision date: June 28, 2001.

Acceptance date: July 27, 2001.

Device status category: 1.

Conflict of interest category: 12.

tographic review of the human DRG vasculature relative to diverse spinal areas to possibly formulate a graphic generalization that could conceptually represent a pattern applicable to the “generic” DRG, and to determine whether this pattern might be a significant factor in the production of a DRG compartment syndrome.

Materials and Methods

The observations described here were derived from the arterial injections of three human perinatal cadavers, one adult rabbit, and venous injections of vertebral segmental tissues of two adult human anatomic cadavers.

The perinatal series consisted of tissues taken from a stillborn of 35 weeks gestation that had been injected as part of a previous investigation, and the spines from a week-old neonate and a 29-week-old spontaneously aborted fetus acquired specifically for this investigation. All the specimens received a perfusion of a 2:1:1 ratio of India ink, neoprene latex, and water in a modification of the Spalteholz technique, which has previously given excellent results with small-bore vessels.¹⁴ The preterm fetuses were injected *via* one of the exposed umbilical arteries, and the neonate was perfused through a cutdown of a femoral artery.

The perinatal specimens having been stored by freezing were allowed to reach ambient room temperatures (approximately 20 C) before they were injected with the perfusate of the same temperature. Injection pressure was supplied by a hand-held syringe, and the extent of the vascular bed perfusion was monitored by noting the degree of penetration evident in the conjunctival and gingival membranes.

After the arterial perfusion, the intravascular pressures were allowed to equalize before the specimens were preserved by body-cavity and fascial plane injections with an aqueous solution of 2.0% formalin and 0.5% phenol.

Three days after embalming, the entire spines were removed and bleached in a 1.5% solution of hydrogen peroxide, dehydrated in 100% ethanol, and cleared in methyl salicylate to permit transillumination for photography. The spinal cord, the dural sac, and all the spinal ganglions were dissected *en bloc*, and the lateral parts of the dura were permitted to remain intact to splint the roots and keep them in their proper relation during hardening. The adult Belgian female rabbit was injected *via* a femoral artery with a medium of the same constituents but of a more dilute nature to allow deeper capillary penetration to demonstrate arteriovenous anastomoses and consequent periradicular venous drainage.

Vertebral segmental areas of adult anatomic cadavers received a venous injection of a fine suspension of barium sulfate (Micropaque, Demancy & Co., Ltd., Birmingham, UK) *via* the approaches described by Bergmann and Alexander for their ink-injected gross arterial studies.² The paraspinal tissues of lumbosacral regions of conventionally prepared anatomic cadavers were removed *en bloc* from two male bodies aged 65 and 72 years. Exposed intercostal and/or lumbar veins were identified, cannulated, and injected with the Micropaque suspension. Because the removal of the paravertebral sections cut numerous branches of the paraspinal and vertebral epidural venous plexuses, maintaining any consistent degree of intravascular pressure was virtually impossible, and the individual preparations provided only partial injections. The subarticular intervertebral tissues related to the DRGs were then dissected

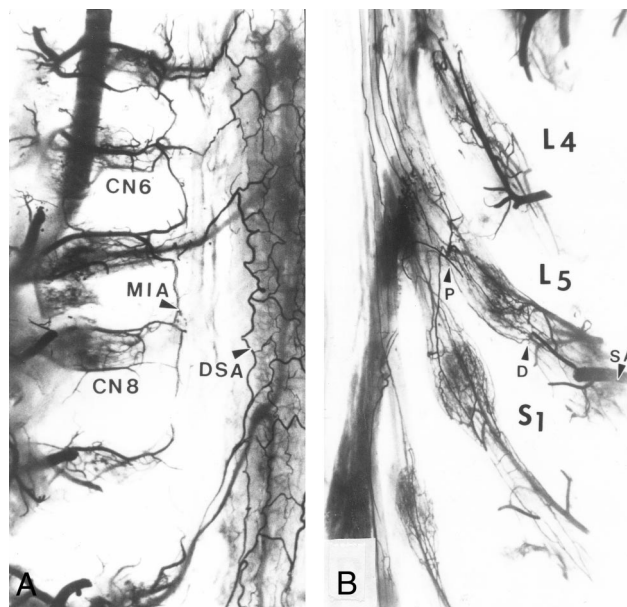


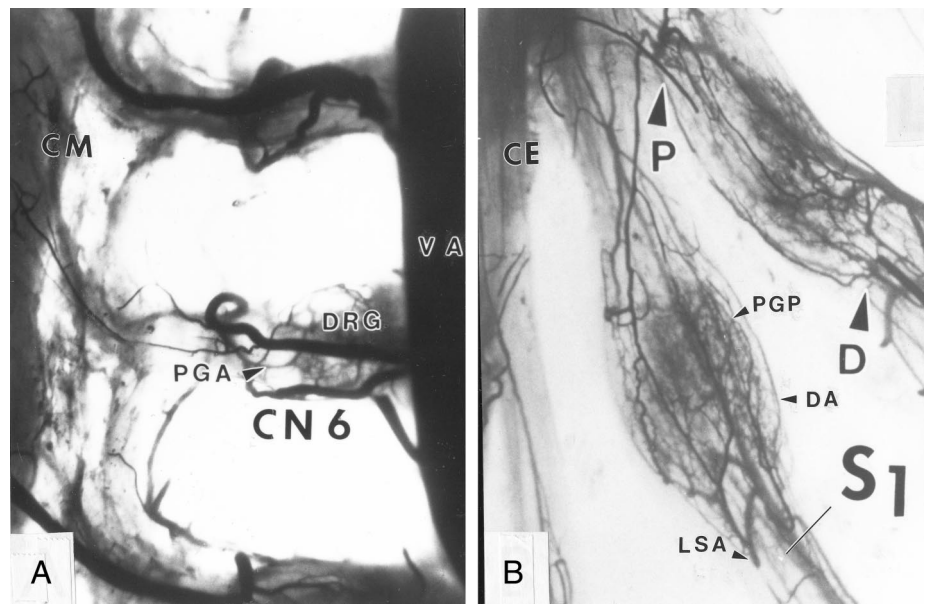
Figure 1. **A**, Dorsal macrophotograph of an injected, cleared and transilluminated example of the lower cervical and upper thoracic radicular/dorsal root ganglion (DRG) complexes taken from a 35-week stillborn perinate. Notice the consistent general pattern of ganglionic arterial vasculature. A meningeal intersegmental artery (MIA) runs on the external surface of the dura. DSA = dorsolateral spinal artery. **B**, Cleared, transilluminated macrophotograph of the lumbosacral radicular/DRG complexes taken from a week-old neonatal cadaver. Notice the consistency of the DRG vascular pattern in both **A** and **B**. P = proximal polar ganglionic arteries; D = distal polar ganglionic arteries, SA = lumbar segmental artery; CN6 = cervical nerve six; CN8 = cervical nerve eight.

from their foramina and placed on Kodak mammogram cassettes for radiography.

Results

Transilluminated macrophotographs of the vasculature of the human perinatal DRGs provided an unprecedented series of finely injected arterial specimens representing all regions of the vertebral segmentation. The salient features of the DRG arterial vascular pattern as described at specific levels by Bergmann and Alexander,² Day,⁵ and Somogyi et al²¹ were shown to be characteristic of the ganglions throughout their entire distribution. In essence, it was shown that the nuclear parenchyma is highly vascularized primarily from vessels entering the proximal and distal poles of each ganglion. These, through predominantly longitudinal derivatives that course parallel to the axis of the ganglion, supply the cordlike arrangements of the neuronal nuclei with a dense capillary bed. A secondary, much finer, network of arteries that is also mostly derived from the proximal and distal polar sources forms a fine reticular system over the surface of the ganglion. This periganglionic plexus communicates with the deeper vessels by a system of anastomotic, centripetally coursing, fine channels. The distal and proximal polar arteries are derived from the epidural branches of the intersegmental vertebral arteries. As can be seen in the macrophotographs, this basic DRG vascu-

Figure 2. **A**, Ventral view of the sixth cervical (DRG), showing detailed view of recurrent course of proximal ganglionic arteries. CM = cervical medulla; VA = right vertebral artery. **B**, Enlarged detail of first sacral DRG illustrating the intrinsic arterial supply to the capillary beds of the cordlike arrangements of the neuronal cell bodies. P and D indicate the proximal and distal polar arteries of the L5 ganglion. This specimen provided an exceptional example of the fine periganglionic arterial plexus on the surface of the DRG and a fine external dural artery with the subdural space separating the two. LSA = branch of lateral sacral artery; PGA = proximal ganglionic artery; CE = cauda equina; PGP = periganglionic plexus; DA = dura artery.



lar pattern exists from the cervical to the sacral regions (Figures 1 and 2) and can be generalized as in Figure 3.

As the radicular branches of the intervertebral segmental arteries penetrate the dura they give off cranial and caudal branches that course along the dural outer surface and, joining their counterparts from the adjacent segments, form a relatively prominent previously undescribed longitudinal channel that is here named the meningeal intersegmental artery. It is most consistently derived from the arteries of the dorsal radicular vessels and thus constitutes an additional longitudinal pathway collateral to the dorsolateral spinal arteries as shown in Figure 1A and graphically depicted in Figure 3.

The rabbit injection provided a photographic illustration of the diversity of radicular vascular patterns in other species. The shorter spinal nerve roots of nonhuman mammals allows a much finer system of arteries to support the capillary bed, although the functional relation of a proximal and distal source to the radicular vessels was still apparent. However, the more dilute injection medium passed through the radicular arteriovenous anastomoses and outlined part of the periganglionic plexus of veins that drains to the epidural and paravertebral venous complexes (Figure 4).

The injection of the cadaver segmental veins was a limited success. Isolation of lower intercostal and lumbar veins permitted cannulation, but the numerous open channels of the paravertebral and epidural venous plexuses failed to provide the closed system necessary for the development of adequate pressure. Figure 5 is an example of one of the better injection results and illustrates how the clotted blood components apparently resisted the retrograde venous penetration. Regardless of its incomplete nature, this result indicates the presence of a superficial periganglionic venous plexus that apparently receives the major efferent blood flow from the interior of the DRG parenchyma, and supports the observations

that Bergmann and Alexander described from their histologic sections.

■ Discussion

Following the 1934 classic report of Mixter and Barr,¹⁰ a consensus prevailed that acute spatial encroachments on the lumbosacral nerve roots were responsible for most back pain and sciatica. As early as 1948, Lindblom and Rexed⁸ postulated that compression of the DRG by dorsolateral disc protrusions and/or hypertrophies of facet joints might also be implicated. Yet, it was not until 1977 that Howe et al⁶ published a physiologically based approach showing that although chronically injured roots are quite sensitive to compression, acute pressure on normal dorsal roots failed to elicit the expected nerve excitations. By contrast, prolonged periods of repetitive nerve firings followed a minimal acute compression of the normal DRG.

An excellent article by Yoshizawa et al²⁴ emphasizes the spatial relations of the nerve root and its associated DRG to their nutritional sources. Their experiments indicated that the distal extension of the intradural space envelops the DRG with cerebrospinal fluid (CSF) and challenged some prevailing conceptions that had placed the subarachnoid angle of reflection, and thus the distal limit of the CSF, proximal to the ganglion. The work of Yoshizawa et al also provided evidence that the blood flow volume in the dorsal root of dogs is less than that of the gray matter of the cord and the peripheral nerves, but greater than that of the cord white matter, whereas the DRG blood flow volume is approximately twice that of the nerve root and similar to the gray matter blood flow in the spinal cord. By using the hydrogen clearance method, they were able to show that the DRG blood flow was reduced 40 to 45% by a compression of 60 g on the distal side of the ganglion, and 10 to 15% by equivalent pressure on its proximal side. However, they noted that

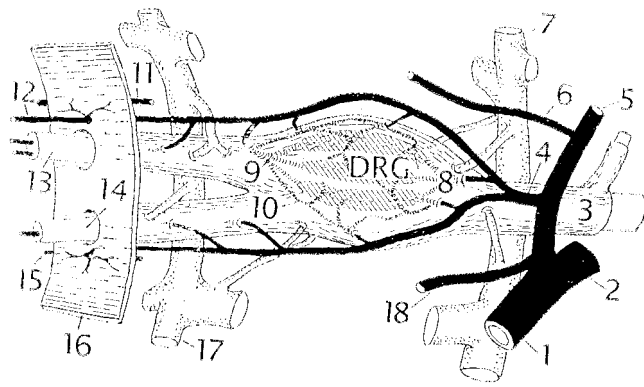


Figure 3. Labeled schema of a "generic" dorsal root ganglion (DRG) with its regional vascular and dural relations. 1 = segmental artery; 2 = lumbar intercostal or cervical somatic artery; 3 = spinal nerve; 4 = radiculomedullary branch of segmental artery; 5 = dorsal branch of segmental artery; 6 = internal artery to laminae; 7 = paravertebral venous plexus; 8 = distal polar DRG arteries; 9 = proximal polar DRG arteries; 10 = periganglionic arterial plexus; 11 = meningeal intersegmental artery; 12 = medullary artery to dorsolateral spinal artery; 13 = dorsal nerve root; 14 = ventral nerve root; 15 = smaller inconsistent meningeal intersegmental artery; 16 = spinal dura; 17 = epidural venous plexus; 18 = dorsal artery to vertebral bodies and their ligaments.

compression on the proximal side had a more prolonged effect, which might have resulted from the fact that the constricting block produced an additional lack of the highly oxygenated CSF that normally bathes the more distal structures and has been shown to be an ancillary nonvascular source of root nutrition by Rydevik et al.²⁰

Bergmann and Alexander speculated that the arterial supply of the spinal ganglions may be rather vulnerable.² They based this belief on the assumption that certain distribution characteristics rendered the DRG blood flow "inefficient" because the source arteries did not branch from their origins in a manner that is hemodynamically optimal with respect to the direction of the main intervertebral artery blood flow. Instead, the vessels branched off at right angles or actually recurred counter to the prevailing vascular current.

In 1989, Rydevik et al¹⁹ published a very significant work that can be related to both the vascular and the surrounding anatomic peculiarities of the DRG. They recorded the endoneurial pressures within the normal rat dorsal nerve root and its DRG and observed it to be higher than the internal fluid pressure within this animal's sciatic nerve, with a reading of 3.7 cm of water. They then noted that it rose to as high as 9.6 cm of water subsequent to a mechanical deformation. Because miniature compartment syndromes have been shown to exist after compression of a peripheral nerve,⁹ it was concluded that such a high pressure elevation in the DRG would likely affect the nutrition of the neuron cells, and a long-standing edema could result in DRG endoneurial fibrosis. Although Rydevik et al¹⁹ recognized that the pressure increase within the closed fibrous dural compartment of the DRG would impede normal vascular flow, they did not relate it to any predisposing char-

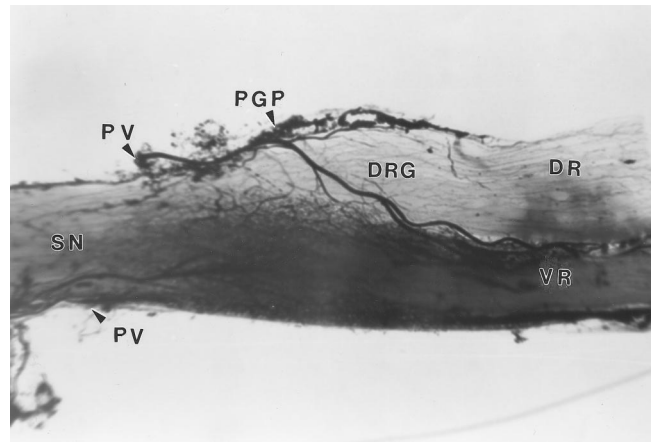


Figure 4. Macrophotograph of an injected and cleared radicular/dorsal root ganglion complex from L4 in an adult rabbit. The dilute latex/India ink suspension traversed numerous arteriovenous anastomoses and partially filled the periganglionic venous plexus shown draining to the paravertebral veins and epidural veins (removed). Notice that the shorter lumbosacral nerve roots of nonhuman specimens show a very fine arterial supply intrinsic to the roots and do not require the larger, longer radicular arteries characteristic of the long roots of the human cauda equina. DR = dorsal root; VR = ventral root; SN = spinal nerve; DRG = dorsal root ganglion; PV = proximal vein; PGP = periganglionic plexus.

acteristics of the vascular pattern. The computer-generated model of peripheral nerve compartment syndrome proposed by Myers et al¹¹ placed the primary locus of venous vulnerability at the point where the vein obliquely exits through the perineural sheath. However, the more spheroidal nature of the DRG may indicate that an additional factor of compressive lability is offered by the subcapsular arrangement of the periganglionic venous plexus (Figure 6).

Bergmann and Alexander claimed that the vascular pattern in the human DRG "is fundamentally different from the pattern seen in the rhesus monkey,"² and to this it may be here added that it is different from any of the nonhuman species that the present authors have examined. This human uniqueness is also evident in comparisons of the radicular circulation as well. As has been cautioned by Parke,¹⁶ the physiologic analogies between various aspects of spinal organization may more readily yield valid cross-specific generalizations than the particular structural details, especially in relation to the vascular patterns of the radiculomedullary circulation. This is well illustrated by the injection of the rabbit root and DRG complex shown in Figure 4, where one can note that the shorter nonhuman nerve roots do not require the longer longitudinal radicular arteries needed to reach the greater distances to the capillary beds that occur only in the much longer roots of the human cauda equina. Nevertheless, this figure shows the general interspecific functional relations between the DRG arterial supply and its peripheral venous drainage.

The demonstrable fact that when a mechanical pressure is applied to an endoneurial vascular system, a venostasis is achieved at a significantly lower pressure than that required to stop the arterial flow¹³ accounts for the

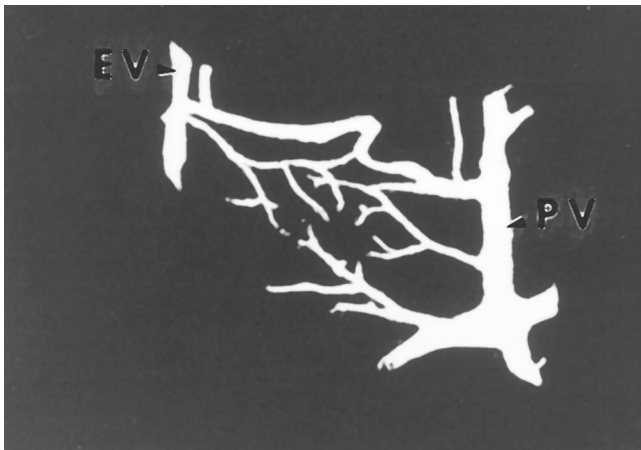


Figure 5. Radiograph of an incomplete retrograde injection of Micropaque into a paravertebral vein draining the intervertebral foramina of L1 in an adult human anatomic cadaver. Despite inadequate pressure and blockage by postmortem blood clots, a “ghost” of the L1 dorsal root ganglion is outlined by a partly filled periganglionic venous plexus. The postinjection formalin-fixed intervertebral foramina were dissected from bone and underwent radiography on a mammography cassette. PV = paravertebral veins; EV = epidural veins.

observations of Watanabe and Parke,²² which showed that the nerve root constrictions of a lumbar stenosis may produce a point of complete blockage of the radicular veins while sparing their confluent radicular arteries. Thus, as indicated in a publication by Parke,¹⁵ this greater lability of the efferent side of the radiculomedullary circulation may be more responsible for ischemic neuropathies than was previously supposed.

With regard to the intrinsic vascularity of the human DRG as described and illustrated here, it is obvious that the structural arrangement of the vessels presents an inherent vulnerability to compressive factors. Weinstein²³ made a functional allusion that the DRG could be regarded as the “brain” of the motion segment unit. This metaphor may be taken one step further with respect to its circulation. Like the cerebral vascularity, the main arterial supply of the DRG cellular masses tends to immediately run deep and central to their position. The efferent veins then, as in the cerebral cortical tissues, generally run a separate centrifugal course and rise to the surface to collect in a periganglionic plexus that is distributed counter-current to the much finer, but equivalently located, periganglionic arterial plexus. Although Bergmann and Alexander provided no illustration depicting the pattern of this plexus, it may be mentally reconstructed from their successively presented microscopic sections of injected specimens.² They also verbally described the general course of these veins and their manner of forming the superficial plexus and its drainage to the regional tributaries of the intervertebral veins. In addition, the series of partly successful injections of intervertebral venous channels described here (Figure 5) gave fragmentary but ancillary support to the existence of a venous plexus lying on the surface of the DRG.

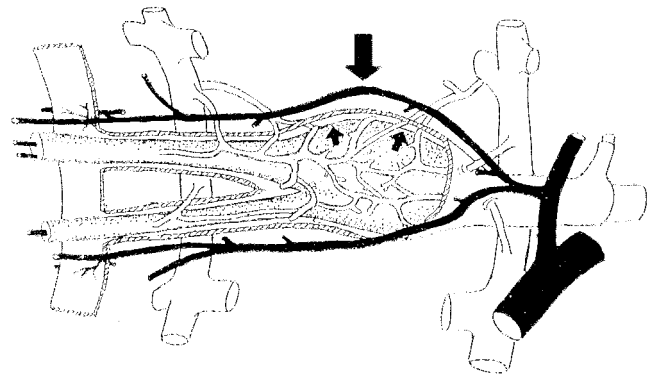


Figure 6. Schema duplicating the anatomic relations labeled in Figure 3, but here the periganglionic venous plexus has been reconstructed to show its vulnerable position adjacent to the fibrous dura, where external pressures (large arrow) or edematous internal pressures (small arrows) could readily create a venostasis leading to a compartment syndrome.

An analysis of the unique structural arrangement of the vascularity of the human DRG in relation to its relatively unyielding fibrous adnexa, as graphically rendered in Figure 6, shows that by the position and arrangement of the major venous drainage, the human DRG is remarkably predisposed to the development of a classic compartment syndrome. The total effect of an external mechanical compression may be complex. In an acute phase, the compression would force the dural capsule to directly impede the immediately underlying efferent flow in the periganglionic plexus and thus resist the normal afferent nutrition to the deeper parenchymal constituents. By subsequent alterations in the vascular permeability, a resulting endoneurial edema would then reflexly push the periganglionic venous plexus against its unyielding fibrous container and create a more chronic venostasis. Prolonged maintenance of this condition could lead to endoneurial fibrosis, increased sensitivity, and repetitive ectopic firing of the contained neural elements.

Because of the salient role of the DRG in the causes of back pain and sciatica, a primary value of this report may depend on the creation of a general awareness and visual image of its vascular vulnerability. This should influence not only the judicious consideration of the surgical relief of spatial encroachments but also a reluctance to subject them to unnecessary manipulation.

■ Key Points

- A basic pattern of vascularization is common to all dorsal root ganglia.
- Pressure on or within the ganglion may produce a venostasis in the periganglionic venous plexus that may lead to a compartment syndrome.
- Because of the possible vascular reaction, pressure on the dorsal root ganglion should be relieved and/or avoided.

References

1. Amundson G, Garfin SR, Parke WW. Vascular anatomy of the spine. In: Levine AM, Eismont FJ, Garfin SR, et al, eds. *Spine Trauma*. Philadelphia: WB Saunders, 1998:61-74.
2. Bergmann L, Alexander L. Vascular supply of the spinal ganglion. *Arch Neurol Psychiatry* 1941;46:761-82.
3. Corbin JL. *Anatomie et Pathologie Arteriellles de la Moelle*. Paris: Masson et Cie, 1961.
4. Crock HV, Yoshizawa H. *The Blood Supply of the Vertebral Column and Spinal Cord in Man*. New York: Springer-Verlag, 1977.
5. Day MH. The blood supply of the lumbar and sacral plexus in the human foetus. *J Anat (Lond)* 1964;98:105-16.
6. Howe JF, Loeser JD, Calvin WH. Mechanosensitivity of dorsal root ganglia and chronically injured axons: A physiological basis for the radicular pain of nerve root compression. *Pain* 1977;3:25-41.
7. Lazorthes G, Gouaze A, Zadeh Jo, et al. Arterial vascularization of the spinal cord. *J Neurosurg* 1971;35:253-62.
8. Lindblom K, Rexed B. Spinal nerve injury in dorsolateral protrusions of lumbar disks. *J Neurosurg* 1948;5:413-32.
9. Lundborg GL, Myers R, Powell H. Nerve compression injury and increased endoneurial fluid pressure: A miniature compartment syndrome. *J Neurol Neurosurg Psychiatry* 1983;46:1119-24.
10. Mixer WJ, Barr JS. Rupture of intervertebral disc with involvement of the spinal canal. *N Engl J Med* 1934;211:210-5.
11. Myers RR, Murakami H, Powell HC. Reduced nerve blood flow in edematous neuropathies: A biomechanical mechanism. *Microvasc Res* 1986;32:145-51.
12. Nakamura S-I, Myers RR. Injury to dorsal root ganglia alters innervation of spinal cord dorsal horn lamina involved in nociception. *Spine* 2000;25:537-92.
13. Olmarker K, Rydevik B, Holm S, et al. Effects of experimental graded compression on blood flow in spinal nerve roots: A vital microscopic study on the porcine cauda equina. *J Orthop Res* 1989;7:817-23.
14. Parke WW, Rothman RH, Gammell K. Arterial vascularization of the cauda equina. *J Bone Joint Surg* 1981;63(A):53-62.
15. Parke WW. The significance of venous return impairment in ischemic radiculopathy and myelopathy. *Orthop Clin North Am* 1991;22:213-21.
16. Parke WW. Point of view (a cautionary discussion on the necessity, advantages and pitfalls of animal models in research). *Spine* 1995;20:765.
17. Parke WW. Applied anatomy of the spine. In: Herkowitz HN, Garfin SR, Balderston RA, et al, eds. *Rothman-Simeone: The Spine*, 4th ed. Vol I. Philadelphia: WB Saunders, 1999:29-73.
18. Pedowitz RA, Rydevik BL, Hargrens AR, et al. Neurophysiologic and histologic changes induced by acute graded compression of the pig cauda equina. Presented at the International Society for the Study of the Lumbar Spine, Miami, Florida, 1988.
19. Rydevik BL, Myers RR, Powell HC. Pressure increase on the dorsal root ganglion following mechanical compression: Closed compartment syndrome in nerve roots. *Spine* 1989;14:574-6.
20. Rydevik B, Holm S, Brown MD, et al. Diffusion from the cerebrospinal fluid as a nutritional pathway for spinal nerve roots. *Acta Physiol Scand* 1990;138:247-8.
21. Somogyi B, Undi F, Kausz M. Blood supply of the spinal ganglion. *Morphol I Orv Sz* 1973;13:191-5.
22. Watanabe R, Parke WW. Vascular and neural pathology of lumbosacral spinal roots in spinal stenosis. *J Neurosurg* 1986;64:64-70.
23. Weinstein JN. In: Frymoyer JW, Gordon SL, eds. *New Perspectives in Low Back Pain*. Park Ridge, IL: American Academy of Orthopedic Surgeons, 1989:97.
24. Yoshizawa H, Kobayashi S, Hachiya Y. Blood supply of nerve roots and dorsal root ganglia. *Orthop Clin North Am* 1991;22:195-211.

Address reprint requests to

Wesley W. Parke, PhD
 Division of Basic Medical Sciences
 USD School of Medicine
 Vermillion, SD 57069
 E-mail: wparke@usd.edu