Spinal Root Disease

Radiculopathy and Arachnoiditis

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INTRODUCTION

RADICULAR LOW BACK pain is widely considered to be the most common neuropathic syndrome.1 Despite their minute anatomical extent and seemingly secure location within the spinal canal, the anterior (ventral) and posterior (dorsal) nerve roots are subject to mechanical, inflammatory, infectious, and even vascular insults. Injury to the posterior roots arising from degenerative conditions of spinal structures such as the lumbar discs is by far the most prevalent cause of painful radicular syndromes.^{1,2} The degree to which chronic low back pain syndromes involve the nerve roots and may therefore be characterized as neuropathic, or "a direct consequence of a lesion or disease affecting the somatosensory system," remains a matter of vigorous debate. Estimates range widely from a high of 4.5% of all adults over age 30 in one sample to fewer than 5% of patients experiencing low back pain.^{3,4} There is

no definitive clinical tool to localize neuropathic pain to the nerve root level; however, there is an enduring, broad consensus that radicular syndromes constitute a major health and socioeconomic problem worldwide.

The archetypal clinical presentation of radicular pain, commonly referred to as sciatica in lay terms, is distinctive and the pain may localize to either the dorsal root or its ganglion. Animal models have linked radicular pain to ectopic discharge and neuronal hyperexcitability in the dorsal root or its ganglion.⁵ Introduction of the proinflammatory nucleus propulsus into the epidural space, even in the absence of nerve root compression, is a well-established cause of this pain pattern.⁶ In contrast, radiculopathy is defined by conduction block in the distribution of a spinal nerve or its roots.7 Radiculopathy may result in weakness when motor fibers are blocked or numbness when sensory fibers are involved. The resulting myotomal or dermatomal