Benign Nerve Tumor and Posttraumatic Nerve Palsy: A Common Thread?

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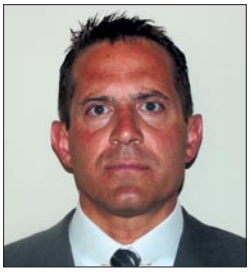
Although the 4 articles in the March E-Focus of *The American Journal of Orthopedics* address seemingly disparate topics—intraneural ganglion of the peroneal nerve, recurrent schwannoma in the finger, peroneal palsy associated with distal femoral physeal fracture, and the epidemiology of benign nerve tumors in the hand from a single practice—there is, arguably, a *common thread*. Indeed, the anatomic spectrum of potential injury to nerve, and the functional implications thereof, are the common thread—which lends itself nicely to a discussion of nerve dysfunction, variable pathogenesis, and appropriate treatment.



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Firstly, nerve dysfunction, whether secondary to tumor, traction injury, or inflammatory neuritis, and the prognosis following treatment depend, in large part, on whether nerve sheath is injured alone or in combination with axonal disruption. We know from our understanding of compression neuropathy that the spectrum of pathoanatomic change begins with segmental demyelination and progresses to intrinsic fibrosis, axonal dropout, and muscle denervation. Indeed, neurophysiological testing shows conduction delay when nerve sheath alone is injured (demyelination). Decreased amplitude and, ultimately, muscle fibrillation occur only when progressive axonal dropout occurs.

The papers regarding schwannoma by Lincoski and colleagues and Wilson and colleagues, and, for that matter, the one by Aprin and coauthors, each show that nerve dysfunction may be minimal and transient when nerve fascicles are preserved. There may be little sensorimotor abnormality on physical exam in cases of encapsulated schwannoma and early presentation of nerve sheath ganglion cysts—and, absent entertaining the preoperative diagnosis of tumor or studying a magnetic resonance image, the correct diagnosis may be elusive altogether. Successful treatment for each requires excision prior to irreversible axonal disruption.

The paper by Sloboda and coauthors identifies the rarity of concomitant peroneal nerve palsy and distal femoral physeal fracture. Most peroneal nerve injuries associated with knee dislocation are neuropraxias and can be treated nonoperatively. Rarely, more serious injury, neurotmesis, may occur following this injury or distal femoral physeal fracture, necessitating a more aggressive surgical approach. Thus, if some modicum of clinical recovery is not present by 3 to 6 months after injury, neurolysis and possible nerve grafting may be advisable to ensure that motor reinnervation, in accordance with Seddon's law of nerve regeneration at a rate of 3 cm per month, is restored by 12 months—to avoid irreversible fatty infiltration and atrophy.

In conclusion, the functional implication of nerve pathology is inextricably linked with the anatomic basis for the dysfunction; so, too, is treatment predicated on the pathoanatomy. ■

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