Magnificent MRI and fascinating selective nerve fascicle damage

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A century ago less 2 years, the distinguished French neurologist Joseph Dejerine and his colleagues¹ meticulously studied the clinical sequelae of nerve lesions in World War I soldiers. They showed clearly that partial lesions in a major peripheral nerve can produce very selective sensory and motor deficits that could mimic the clinical picture of damage to just a distal branch of that nerve. Thus a proximal nerve lesion can masquerade as a more distal one, befuddling the clinician and causing important errors in localization. Unfortunately, these important observations have been lost in the mists of time.

The anterior interosseous nerve (AIN) may seem like an inconsequential branch of the median nerve, but a lesion here impairs the all-important action of pinching between the thumb and forefinger. The cause and management of spontaneous, subacute lesions of the AIN have long been poorly understood. One view has been that these anterior interosseous neuropathies are due to entrapment at the elbow and require surgical decompression. The other has been that these are focal inflammatory lesions (likely a restricted form of acute brachial plexus neuritis [neuralgic amyotrophy, Parsonage Turner syndrome]) that may best be treated with immunosuppressant medications.²

In this issue of *Neurology*[®], Pham et al.³ not only resolve this controversy but also add 2 more important messages. This is a 3-for-the-price-of-1 article.

The first message is about technology. MRI is, of course, excellent at showing lesions that compress nerves, and nerve tumors. These researchers now show that, using 3T high-resolution MRI, the age of magnetic resonance neurography has arrived. Nerves can be imaged with clarity and small intraneural lesions can be identified.

The second message addresses a controversy that has long raged, in spite of the prescient work of Dejerine et al. Peripheral nerves are made up of discrete bundles of nerves—the fascicles. There are 2 views of their organization. The first, championed by Australian anatomist Sunderland⁴ in work dating back to the 1940s, is that the fascicles within a nerve pursue a chaotic course with many branchings and joinings along the course of the nerve (a plexiform structure). Many others have argued persuasively, on the basis of clinical observations and human and animal experiments, that the opposite is the case: fascicles are arranged somatopically in an orderly cable-like arrangement.5 Does this matter? Indeed it does. If there is a cable structure, a proximal partial nerve lesion could selectively damage those fascicles going to form a distal branch. Such a lesion would be mislocalized as damage to the branch. What Pham et al.³ have shown, in 20 patients with subacute anterior interosseous neuropathies, is that the lesions are not in the AIN itself but within the main median nerve trunk in the upper arm, involving just those fascicles destined to form the AIN more distally. These lesions are about 15 cm proximal to the origin of the AIN in the forearm. This is the most compelling evidence to date to illustrate the clinical implications in localizing partial nerve lesions involving selective nerve fascicles.

The third message is the obvious corollary to the second. Subacute anterior interosseous neuropathies are not a surgical condition.

This study of MRI of peripheral nerves not only demonstrates the diagnostic power of the technique but also reveals the unexpected location of the nerve lesion causing subacute anterior interosseous neuropathies, and thus shows that surgical release of the nerve is not required. Most importantly, however, it proves the long-predicted phenomenon that partial lesions of a major nerve trunk involve specific fascicles and thus can produce the clinical picture of a lesion of a distal nerve branch. Clinical and electrodiagnostic studies have strongly suggested that this occurs in partial lesions of many peripheral nerves.^{1,5} Now we have proof. Awareness of this "fascicular phenomenon" is crucial for neurologists seeing patients with focal nerve lesions, be they compressive, traumatic, inflammatory, or otherwise.

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DISCLOSURE

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