**Fascicular anatomy, nervi nervorum, and paresthesia**

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The letter to the editor by Moon[1] addresses the interesting issue of the poor correlation between paresthesia and motor response. He attributes the poor sensory success of median and radial nerve block to variable fascicular anatomy. This could also be explained by the common mistake of equating dermatomal distribution to osteotomal distribution. For instance, the osteotome on the radial side of palm has contributions from the ulnar nerve as well the median nerve. In general, a muscle's bony attachment is supplied by the same nerve that supplies the corresponding muscle. As for the radial nerve, Moon's approach “4 finger breadths above the lateral epicondyle” could leave the posterior antebrachial cutaneous nerve unblocked because it branches off more proximally to supply the skin on the back of the forearm.

Moon also proposes that the variable fascicular anatomy along the peripheral nerve explains “excellent motor response without paresthesia.” This finding is contradictory to paresthesia without motor response in 23% of patients reported by Choyce et al.[2] The concept of fascicular anatomy is commonly explained by the assumption that the axons supplying proximal structures are more superficial in the nerve fiber than the ones that supply distal structures. Because the median and ulnar nerves supply muscular structures followed by sensory axons to the hand, then one would expect sensory fibers to be more centrally located until the nerve reaches the wrist. Conversely, the radial nerve has multiple sensory branches (posterior brachial cutaneous, posterior antebrachial cutaneous) as it traverses the arm along with motor axons. If one accepts this rationale, then the phenomenon of “paresthesia, but no motor response” is likely with the radial nerve because of its peripherally located sensory axons, but less likely with the median and ulnar nerve. However, Choyce et al[2] found no difference among nerves in paresthesia and motor response. Because the brachial plexus provides little cutaneous sensory innervation above the clavicle, one would expect the fascicular anatomy at the level of the trunks to have centrally located sensory axons, with consequent difficulty obtaining a paresthesia. But on the contrary, Urmey et al[3] found a 75% incidence of paresthesia with no motor response at 1 mA during interscalene block. Moon's explanation predicts that a paresthesia technique for interscalene block would have a poor success rate, which is not the case.

I believe it is incorrect to assume that stimulation of sensory axons is responsible for the sensation of paresthesia. Neurophysiologists widely believe that axons are only sensitive to mechanical stimulation at the distal terminal receptors. This insensitivity is altered by nerve injury and is a source of neuropathic pain. Insensitivity to nociceptive mechanical stimulation (needle) of midaxons favors another pathway for eliciting the sensation of paresthesia: the nervi nervorum, which have a role in supplying autonomic innervation to vessels in the perineurum and have nociceptors as well.[4] The absence of nervi nervorum in the central nervous system permits stereotactic surgery on awake patients and extralemniscal myelotomy with no pain or paresthesia. This brings up an interesting question: is it possible to elicit paresthesia in a purely peripheral motor nerve? Where do the nervi nervorum impulses travel? Do they eventually travel to the sensory axons of corresponding nerve or do they have a different sensory pathway?

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