To the Editor:


I have read with interest the paper by Berry and coworkers entitled Isolated Suprascapular Nerve Palsy: A Review of Nine Cases.1 I am concerned that they have dismissed too hastily the possibility that neuralgic amyotrophy (NA) – referred to in their paper as "brachial plexus neuritis" – may be responsible for isolated suprascapular neuropathies (ISNs). The authors report that they had encountered no instances of ISN among the 18 cases of NA they had assessed. Moreover, in the discussion of differential diagnosis, they imply that NA generally affects multiple nerves about the shoulder girdle, i.e., in the example they give, there would have to be lesions of at least the musculocutaneous nerve and either the posterior cord, or the axillary plus radial nerves, to cause the muscle involvement described.

In fact, NA frequently involves just one nerve, particularly the long thoracic, suprascapular, axillary, or anterior interosseous. This was demonstrated quite well by Parsonage and Turner, in their two joint papers on the subject, concerned with 136 patients and 82 patients: single nerve lesions were present in 33% and 61%, respectively.2,3 They reported, however, in both series that ISNs constituted only 3-4% of their cases. In contrast, we have assessed approximately 125 patients with NA and have found that ISN probably represents the single most common mononeuropathy presentation.

A few other points are worth noting. First, we have observed, as have others, that whenever NA affects the suprascapular nerve it often involves the fibers innervating the infraspinatus muscle much more severely than those supplying the supraspinatus or, less often, vice versa.4 Thus, in distribution at least, the ISN caused by NA can mimic the ISN caused by entrapments at both the suprascapular notch and the spinoglenoid notch. Second, in our experience, one of the many antecedent factors with NA, in addition to infectious diseases, operations, childbirth, etc., is non-specific mild trauma, such as is often sustained during sporting events. Third, NA can affect young athletes, just as it can affect any young adult.5

I believe that two of the most helpful historical markers in the recognition of NA concern the pain that heralds the formal onset of the disorder: 1) it frequently begins abruptly at night, awakening the patient from sleep, and is quite severe – a very atypical presentation for an entrapment lesion, 2) it is always preceded by a "latent period" of variable duration (several hours to 2-3 weeks), separating it from the antecedent event. Thus, the high school athlete who plays football or wrestles Friday evening, and is then asymptomatic until early Sunday morning, when he is awakened from sleep by severe unilateral shoulder pain that is followed a few days later by weakness and wasting of one or more of the forequarter muscles, has experienced a bout of NA, and not a sport-related nerve injury. Unfortunately, many physicians are unaware of these aspects of NA and, as a result, misdiagnoses occur, sometimes culminating in unnecessary surgical decompression of nerves. Several probable instances of errors of this nature can be found in the literature, particularly the Sports Medicine literature.5

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Reply:

We would agree with Dr. Wilbourn’s statement that the differential diagnosis of isolated suprascapular nerve palsy should include neuralgic amyotrophy and there is general agreement as to the clinical features of this condition, namely, the sudden onset with severe, often nocturnal pain and the subsequent development of wasting and weakness about the shoulder and upper limb. In our experience with approximately 85 patients with brachial plexus neuritis, the commonest site of dense involvement has been within the territory of the circumflex nerve (axillary). Careful examination has almost invariably revealed some involvement of adjacent nerve territories. We did find involvement of the suprascapular nerve in 18 patients along with some involvement of adjacent nerves but not on an isolated basis. It is of interest, therefore, that Dr. Wilbourn has found isolated suprascapular involvement to be the commonest form of involvement in this condition.

The statement that mild trauma in a sporting event is an antecedent factor in neuralgic amyotrophy does raise the interesting and fundamental question as to the process by which some entities are defined. The diagnosis of neuralgic amyotrophy is based upon a configuration of historical and clinical evidence, at times supplemented by EMG findings of denervation, and there is no single piece of evidence which is specific for the diagnosis or confirmatory of it. Isolated nerve palsies are at times relatable to musculoskeletal strains and vigorous activity of an athletic or other type and, in our experience, these can involve the lateral popliteal nerve, the femoral, suprascapular and the long thoracic nerve. Under such circumstances, therefore, it is a matter of a subjective and perhaps arbitrary interpretation as to whether such an isolated nerve palsy is attributed to neuralgic amyotrophy, a condition whose causal mechanism remains unknown or, more directly, to athletic or minor trauma. Such an individual choice in interpretation as to the cause of the palsy will necessarily shape the personal experience of individual examiners and their perception of the incidence of traumatic versus brachial plexus neuritis in their personal series.

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