Entrapment Neuropathies of the Upper Extremity

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Introduction

The incidence of upper extremity entrapment neuropathies in athletics is not known. The neurologic symptoms are often intermittent, appearing only during or after a specific exercise activity. As a result, the physical examination findings in the office setting are normal. In many instances, abnormal physical examination findings are present only when the athlete is evaluated immediately after exercise.

There are numerous causes associated with nerve entrapment syndrome in the athlete. In general, these may be divided into acute traumatic injuries and repetitive microtraumatic (overuse) injuries associated with certain sports and athletic activities. Entrapment neuropathies appear to be more common in the upper extremity. In a recent review of 216 sports-related nerve injuries, 86% were in the upper extremity, and 40% were the so-called "burner" or "stinger" injuries [1]. Peters [2•] reported on 83 consecutive sport climbers (68 men, 15 women, average age 32 years) evaluated at a sports medicine center; 62 (75%) of the climbers reported orthopedic problems, 21 of which were compressive neuropathies, with 14 occurring in the upper extremity and seven occurring in the lower extremity.

In theory, any nerve can be affected at any level. The manifestations of the entrapment depend on many factors, such as the type of nerve affected (sensory, motor, mixed), the specific site of compression, the etiology (intrinsic vs extrinsic), the duration (acute vs chronic), and other factors. The presentation may also be affected by any known risk factors for overuse (such as repetitive activities involved in a certain sport). Diabetes, tobacco use, connective tissue disorders, hypothyroidism, alcoholism, malnutrition, renal insufficiency, and genetic factors may contribute to the development of a neuropathy.

This article reviews the general history and physical examination, radiographic and laboratory evaluation, and treatment of upper extremity entrapment neuropathies in the athlete. Cervical disorders and acute traumatic nerve injuries, including burners, are not reviewed; for these conditions, the reader is referred to other sources [3•,4•,5].

History and Examination

Symptoms of nerve compression depend on many factors; the clinical presentation of the athlete with an entrapment syndrome depends on the nerve affected, and whether any other structures (artery, nerve) are involved. Pain is common; it is often described as a dull ache that is worse during or after activity, and it may be sharp and radiating. The athlete may also complain of numbness, paresthesias, sensory loss, exertional fatigue, weakness, and muscle atrophy. Concomitant venous obstruction may lead to edema, cyanosis, and venous collateralization, whereas arterial obstruction may produce coolness, numbness, activity-related pain, and exertional fatigue.

Due to the fact that physical examination findings are often minimal in the athlete with an entrapment neuropathy, a search of historic features is extremely helpful. Symptoms are usually insidious in onset, but may be precipitated by acute trauma, including fracture and dislocation. Overuse risk factors should be reviewed. Change in exercise activity, frequency, intensity, and duration, such as is seen when changing sports season, level of competition, or position should be documented. The use of new exercise training, techniques, and equipment may contribute to these conditions. The history of previous orthopedic injuries and treatment, and of symptoms consistent with underlying toxic-metabolic and connective tissue diseases should also be reviewed.
For most upper extremity entrapment neuropathies, the entire extremity, including the spine, should be examined. Physical examination may reveal swelling, discoloration or temperature change, tenderness to palpation, pain with resisted movements, sensory loss that may or may not involve the entire nerve distribution, and decreased reflexes. Muscle testing will occasionally reveal atrophy, weakness, and possibly muscle fasciculation. Palpation and percussion at the site of nerve compression may reproduce pain and paresthesias; tenderness over a localized segment of a peripheral nerve usually indicates its site of compression. A positive Tinel's sign (reproduction of symptoms with tapping of a nerve) may be present. Other findings include congenital anomalies, anatomic malalignments, functional deformities, and a change in biomechanics.

If the history is suggestive of an upper extremity entrapment neuropathy but the athlete has no obvious physical examination findings, it can be very helpful to examine the athlete immediately after engaging in the activity that causes symptoms. Soft tissue swelling, tenderness, weakness, incoordination, or sensory deficits may be noted. I have found post-exercise examination extremely helpful in the identification of entrapment neuropathies in many athletes, including baseball and softball pitchers, weight lifters, volleyball players, golfers, tennis players, and motocross racers.

Evaluation
Numerous studies may be helpful in determining the cause of an entrapment neuropathy. Screening blood work may assist in confirming suspicions of a toxic-metabolic or connective tissue disease. Routine radiographs should be obtained to rule out bony abnormalities such as osteoarthritis, fractures, exuberant callus formation or deformity, traction spurs, tumors, and anatomic variants. Plain radiographic tomography, arthrography, bone scan, computerized tomography scan, magnetic resonance imaging (MRI), invasive and noninvasive vascular studies, and other radiographic investigations may be required to confirm the diagnosis and to determine the etiology of the entrapment neuropathy. Electromyography (EMG) and nerve conduction studies (NCS) may be useful in demonstrating the presence, location, pathophysiology, and severity of a nerve lesion; these findings can assist in providing a prognosis [3••,6•]. Unfortunately, due to the fact that many upper extremity entrapment neuropathies are intermittent in nature, the EMG and NCS at rest are often normal. Also, the EMG examination is not standardized from one laboratory to another, and thus results may be inconsistent. The study is a two part process that includes NCS and the needle electrode examination. Three clinically concrete, distinct conditions may result from an EMG study: 1) the specific suspected neurogenic lesion is present and valuable information about the lesion may be obtained; 2) a neurogenic lesion is present; however, it is different than the one suspected clinically, and this may or may not be proved to be related to sports participation; and 3) no neurogenic lesion is present (eg, disuse atrophy causing muscle wasting will produce a normal EMG study). Despite some limitations, the EMG may be of assistance in the evaluation nerve entrapment syndromes and other sports injuries to the peripheral nervous system. Further discussion of the EMG is beyond the scope of this article and readers are encouraged to review other sources [4••,6•].

Treatment
Treatment of compressive neuropathies depends on the etiology. Any risk factor that may be contributing to the disorder should be identified and corrected if possible. Often, correcting these errors results in improvement or resolution of symptoms. Rehabilitation exercises are the cornerstone of treatment. Improvement in posture, muscle balance, joint flexibility, and muscle endurance, strength, and power should be attainable through a structured rehabilitation exercise program under the guidance of a qualified physical therapist or certified athletic trainer. The use of relative rest, nonsteroidal anti-inflammatory drugs (NSAIDs), oral or injectable corticosteroids, and padding, splinting, or bracing should also improve symptoms. Referring the athlete for professional sports evaluation by a well-trained coach can be very helpful in the identification of flaws in technique or equipment that may be contributing to the neurologic disorder. Golf and tennis professionals, swimming and weight lifting instructors, batting and pitching coaches, and others may add their expertise to the treatment armamentarium. At times, surgical intervention is required if the athlete does not respond to conservative measures. Nerve decompression, neurolysis, neuroma excision, nerve resection, repair of a traumatically injured nerve, or nerve or muscle transfer may be required.

Disorders
Thoracic outlet syndrome
Thoracic outlet syndrome (TOS) is found most commonly in athletes engaged in weight lifting and repetitive throwing motion, such as pitching and swimming, although it has been reported in runners as well [7]. Because the compression is usually dynamic, symptoms are intermittent and reflect the location and degree to which the brachial plexus, subclavian artery, or subclavian vein are compressed, thus making the diagnosis difficult. Symptoms may include pain, paresthesias, weakness, fatigability, swelling, or discoloration. Compression may occur at three levels: the interscalene triangle, costoclavicular space, and pectoralis minor insertion on the coracoid process. There are three classic tests for TOS that may reproduce symptoms [4••]. In Adson's test, the athlete's neck is extended and head rotated toward the affected limb while taking and holding a deep breath. In Wright's test, the head is turned toward the unaffected limb and the affected arm is...