Peripheral Nerve Injuries in Sport

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KEYWORDS

• Peripheral nerve • Nerve injury • Sports related

KEY POINTS

- Sport-related peripheral nerve injuries (SNRIs) can be acute or chronic and often related to the specific mechanics of individual sports.
- The diagnosis of SNRIs includes identifying its pathophysiology and severity in order to initiate appropriate treatment.
- The diagnosis of SRNIs requires a detailed clinical history and examination, the use of imaging and electrodiagnostic techniques, and awareness of other conditions that affect peripheral nerves.
- Treatment includes medications, rest, physical and occupational therapy, injection therapy, and modification (if possible) of sporting mechanics. Surgical treatment is indicated when these measures fail but can be successful when candidates are appropriately selected.

INTRODUCTION

Sport-related peripheral nerve injury (SRNI) can affect any level of the peripheral axis, including the nerve root, plexus, and peripheral nerves. Nerve injuries that occur during a specific sport account for less than 0.5% of all traumatic peripheral nerve injuries, but recent studies suggest a higher rate in the United States.[1–5] The risk and type of SRNIs varies by sport and can be generally classified by their onset, that is, acute or chronic, or whether they occur in full-contact or noncontact sports. Most acute SRNIs occur in full-contact sports whereby high-velocity impacts cause acute nerve traction or compression, for example, stingers with football tackles. In contrast, chronic SRNIs tend to occur in the setting of high-frequency repetitive movements, for example, ulnar neuropathy in baseball pitchers and suprascapular neuropathy in swimmers and volleyball players.[6]

The diagnosis of SRNI requires a detailed clinical history, examination, and the appropriate use of diagnostic modalities (eg, high-frequency ultrasound, MRI,

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computed tomography [CT], and electrodiagnostic [EDX] studies). Moreover, SRNIs often occur in the setting of musculoskeletal symptoms. This can make a clinical diagnosis more challenging, especially when symptoms are nonspecific in nature and distribution, that is, vague shoulder pain, scattered numbness or paresthesias not confined to a single dermatome, or intermittent pain without weakness.[7] This article reviews the diagnosis and treatment of SRNIs and further addresses some of the more common and complicated SRNIs seen in competitive and noncompetitive sports.

FACTORS

Factors that predispose athletes to injury include improper technique, age, overtraining, the number of repetitive stresses, and protective equipment.[8,9] Any disruption along the nerve pathway, such as a ganglionic cyst, increases the risk for nerve injury.

MECHANISM AND PATHOPHYSIOLOGY

There are many mechanisms whereby peripheral nerves can be injured during sports participation, including compression, stretch, traction, laceration, and crush.[7] The severity and duration of these different mechanisms result in varying degrees of axon loss, demyelination, or a combination of both. Axon loss is the most common form of pathophysiology with SRNIs followed by demyelination. Prognosis with axon loss lesions varies depending on the severity and location of injury, whereas the prognosis with focal demyelinating lesions is good assuming that further compression is avoided. Confirming pathophysiology often requires use of the EDX examination that includes nerve conduction studies and needle electromyography in addition serial clinical evaluations.

The decision to return to play after sustaining a potential SRNI is less distinct than the evolving guidelines for field concussion assessments. Defining the mechanism and severity of an SRNI requires a detailed description of how the injury occurred and an examination that incorporates both musculoskeletal and neurological components. The use of a grading scale for peripheral nerve injuries, that is, the Sunderland grades I to V, ranging from I or neurapraxia/focal demyelination to V or complete nerve transection or loss of function, allows for more accurate prognosis but can at times only be determined by serial examinations and imaging and electrophysiological tests[7] (**Table 1**).

Table 1
Sunderland classification and expected recovery

Injured Structure	Seddon	Sunderland	Spontaneous Recovery
Myelin	Neurapraxia	Grade 1	Excellent
Myelin, axon	Axonotmesis	Grade 2	Good
Myelin, axon, endoneurium		Grade 3	Variable
Myelin, axon, endoneurium, perineurium		Grade 4	Poor
Myelin, axon, endoneurium, perineurium, epineurium	Neurotmesis	Grade 5	None

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