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REVIEW

A narrative review of nerve compression syndromes in overhead throwing athletes

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Abstract

Purpose of Review The purpose of this narrative review is to highlight upper extremity nerve compression syndromes and peripheral neuropathies reported in throwing and overhead athletes.

Recent Findings The overhead-throwing athlete may experience unique patterns of injuries and pathology related to the biomechanics and demands of the throwing motion, a demanding manoeuvre that places a significant amount of stress across the upper limb. Nerve injuries that may appear in high-level throwers include suprascapular and long thoracic neuropathy, quadrilateral space syndrome, and thoracic outlet syndrome. Nerve compression syndromes around the shoulder may appear with pain, paresthesia, and upper limb weakness. Overlapping features may be common among these compression neuropathies or mimic other common shoulder pathologies.

Summary Prompt differential diagnosis and successful treatment should be based on knowledge of key anatomical features, pathophysiology, clinical examination, and appropriate paraclinical studies.

Keywords Peripheral nerve injury · Peripheral neuropathy · Shoulder pain · Overhead athlete · Throwing athlete · Thrower · Sports

Introduction

Shoulder pain and dysfunction of the overhead and throwing athlete may be caused by variable conditions, including glenohumeral pathology (capsulolabral or cartilage injuries), rotator cuff pathology, scapular dyskinesis, or pathology of neurovascular structures. Prompt differential diagnosis is imperative for successful treatment. Nerve compressions and nerve-related conditions in the overhead throwing athlete may result from thoracic outlet syndrome, axillary-, suprascapular,- and long thoracic compression neuropathies, which, although rare, may cause profound morbidity. Symptoms may include fatigue, vague shoulder pain, a sense of heaviness or cramping in the upper limb, decreased overhead or throwing motion performance, weakness of grip, and dysaesthesia. The throwing motion is a fast and demanding manoeuvre and requires synchronization of the kinetic chain. The coordinated function of the kinetic chain effectively reduces the need to generate large forces at the shoulder. Besides, the shoulder structures maintain a delicate equilibrium ("throwers paradox") between sufficient laxity and stability, thus maintaining and maximizing the force that can be generated. Finally, the shoulder's muscle-tendon units and capsule-labral structures effectively absorb this force during the throwing motion, especially after the release phase and arm deceleration [1–5]. This review study discusses the leading causes of neurological pain in the shoulder of the throwing athlete.

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Quadrilateral space syndrome

Anatomy

Quadrilateral space syndrome (QSS) results from compression of the axillary nerve or the posterior circumflex humeral artery (PCHA) within the quadrilateral space (QS) and was originally reported in 1983 [6]. The axillary nerve and PCHA transverse the QS bounded by the teres minor superiorly, the teres major inferiorly, the long head of triceps medially, and the surgical neck of the humerus laterally [5]. The axillary nerve innervates the deltoid and teres minor muscles, which are responsible for the abduction and external rotation (at 90 abduction) of the arm, respectively. The PCHA provides the main perfusion of the humeral head. (Fig. 1)

Aetiology

The QSS is most commonly attributed to repeated overhead activity in throwers and overhead athletes, where fibrous bands or muscle hypertrophy is usually the specific pathoanatomy relating to compression. Rarer causes include tumours and labral cysts [1, 8]. The Mayo group differentiated QSS cases to neurogenic QSS (nQSS) presenting with non-dermatomal paraesthesias and QS point tenderness and a vascular type of QSS leading to PCHA thrombosis and/or aneurysm with distal embolization and digital ischaemia [8].

Diagnosis

The correct diagnosis requires a high suspicion index due to the condition's relatively low frequency, combined with the nonspecific clinical presentation [8, 9]. Initial symptoms usually result from compression of the axillary nerve. The overhead athlete with QSS may complain of intermittent, poorly localized shoulder pain, most often posteriorly located (over QS), weakness, and distally radiating non-dermatomal paraesthesias, aggravated by forward flexion or abduction and external rotation of the arm [1, 6, 8]. Upon inspection, atrophy of the deltoid and teres minor can be noticed due to denervation. The diagnosis is usually made based on the presence of Hagert's clinical triad of nerve

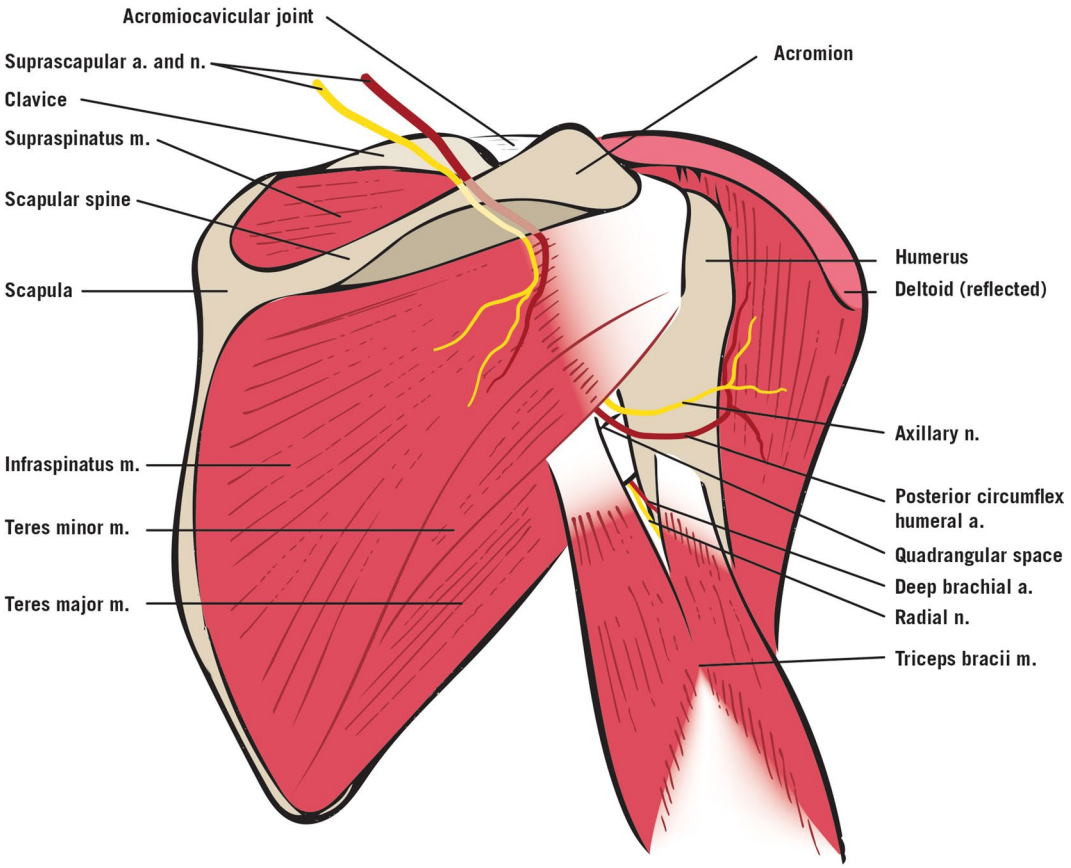

Fig. 1 The quandrangular space (QS) is located at the posterior shoulder. It is bounded from teres minor at the superior aspect, the surgical neck of the humerus laterally, the teres maor inferiorly, and the long head of triceps medially. The axillary nerve and the posterior circumflex humeral artery cross the QS. Figure used with permission from [6]

compression diagnosis: (1) tenderness over the quadrilateral space (usually discrete point tenderness), (2) possible paraesthesia over the lateral shoulder and upper posterior arm with positive scratch-collapse test at the QSS, and (3) deltoid weakness with decreased strength in shoulder abduction [1, 5, 6, 10, 11]. Differential diagnoses include other sources of shoulder pain in the thrower, such as rotator cuff and labral pathology, C5-C6 radiculopathy, thoracic outlet syndrome, brachial plexus neuritis, and suprascapular nerve injury. Also, other conditions with axillary nerve injury, such as blunt trauma or anterior shoulder dislocation, and an uncommon compression site of the axillary nerve between the proximal humerus and the latissimus dorsi tendon should be excluded [12, 13]. Although no "gold standard" diagnostic test for QSS exists, magnetic resonance imaging (MRI) is usually the first choice. MRI often demonstrates focal fatty infiltration and atrophy of the teres minor Muscle and can exclude other causes of shoulder pain [12]. Dynamic PCHA occlusion during abduction-external rotation manoeuvres can be shown with angiography [8]. However, the accuracy of vascular studies alone for confirming the diagnosis of nQSS is unclear. Electrodiagnostic studies can be performed after vascular imaging studies suggestive of nQSS; however, there is low EMG sensitivity due to technical difficulties in targeting the teres minor, which might be improved by ultrasound guidance.

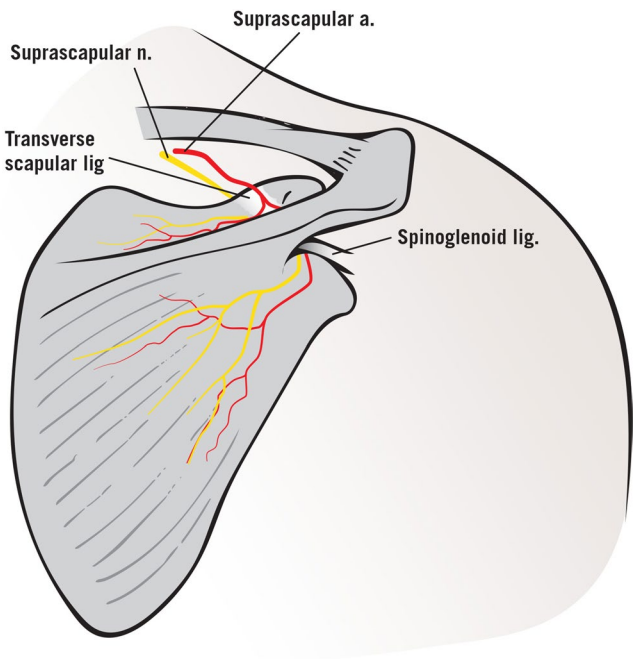

Fig. 2 The suprascapular nerve courses through the suprascapular nothch of the scapula covered by the transverse scapular ligament and then through the spinoglenoid notch covered most of the times by a spinoglenoid ligament. Figure used with permission from [6]

Treatment

The first line of treatment is usually conservative, with oral anti-inflammatory medications,

physical therapy, and limitation of activity. Physical therapy includes internal rotation stretching with posterior rotator cuff and periscapular muscle strengthening and stretching. Almost 30% of throwers will not respond to conservative measures and require surgical intervention. Surgical decompression involves the removal of fibrous bands or other space-occupying lesions and neurolysis. Although more demanding, the anterior (deltopectoral) approach reduces postoperative morbidity. The posterior approach has been found to be the easier and quicker method to identify the quadrangular space and secure its contents [8–10, 12–14].

Suprascapular nerve compression

Although an uncommon injury, the suprascapular nerve palsy is the most frequently injured peripheral branch of the brachial plexus in throwing and overhead athletes [15, 16].

Anatomy

The suprascapular nerve is a mixed motor and sensory nerve that originates from the C5 and C6 roots (occasionally including C4) and arises from the upper trunk of the brachial plexus at Erb's point. The suprascapular nerve crosses the posterior triangle of the neck and passes beneath the transverse scapular ligament through the suprascapular notch to reach the supraspinatus fossa. The suprascapular nerve then branches into 1–2 small and short motor branches to the supraspinatus muscle and contributes terminal sensory nerve endings to the glenohumeral joint, the acromioclavicular joint, the coracohumeral ligament and in about 15%, provides cutaneous sensory fibres to the upper lateral arm (deltoid patch). The suprascapular nerve then courses obliquely along the floor of the supraspinatus fossa and continues around the lateral margin of the base of the scapular spine, also known as the spinoglenoid notch, to enter the infraspinatus fossa. It provides 3–4 longer and larger motor branches to the infraspinatus muscle. (Fig. 2)

Pathophysiology

In the overhead athlete, the cause of suprascapular nerve injury is usually repetitive microtrauma to the nerve through several mechanisms, including compression, traction, and friction. Compression of the nerve can occur from space-occupying masses, in the notches and overlying ligaments,

and from rotator cuff tendons due to its relatively fixed position under the cuff tendons and within the suprascapular and spinoglenoid notches. Ganglion cysts that compress the nerve are usually found with capsulolabral or SLAP tears and are created from a one way valve mechanism. Overlying hypertrophic or calcified transverse scapular ligament and the shape of the suprascapular notch are possible causes for compression or injury of the nerve [17–19]. Compression at the spinoglenoid notch occurs from the spinoglenoid ligament tightening during cross-body adduction and internal rotation during the follow-through phase of throwing or serving [20]. During extreme abduction and full external rotation, the medial tendinous portions of the infraspinatus and supraspinatus muscles impinge against the lateral edge of the scapular spine, compressing the infraspinatus branch of the suprascapular nerve [21]. All these mechanisms may also create traction, mechanical stretching or friction as the suprascapular nerve displays several turns and sharp changes of direction around critical osseous points during its course while it is concomitantly fixated in some other points. This can be exacerbated by repetitive overhead activities, characterized by scapular motion, simultaneous contraction of the infraspinatus muscle and the extreme torque and angular velocity placed on the shoulder [22]. Nerve stretching during forceful external rotation has been shown to be the cause of a decrease of shoulder external rotation power up to 30% in volleyball players [22, 23] and a slowing of nerve conduction velocities in baseball pitchers as the season progressed [24]. In contrast, some throwers may show infraspinatus atrophy and compensation with the teres minor [22, 25].

Diagnosis

The symptoms depend on the chronicity and tend to be less severe when the injury is localized to the spinoglenoid notch. The athlete may have poorly localized lateral and posterior shoulder pain, usually exacerbated by activity. The clinical examination reveals tenderness at the location of nerve compression, while tenderness over the acromioclavicular joint may also be present due to the sensory fibres. Atrophy of the infraspinatus and supraspinatus or the infraspinatus alone will be seen in later stages.

Muscle testing will reveal a weakness in shoulder external rotation and a positive scratch collapse test over the level of nerve compression. The reliability of electrodiagnostic studies for suprascapular nerve pathology is low. An ultrasound-guided lidocaine injection in the suprascapular notch can help in diagnosis if pain is relieved; however, if not, it cannot preclude the diagnosis. An MRI can help identify soft tissue masses compressing the nerve and quantifying rotator cuff muscle atrophy and fatty infiltration. Other diagnoses of shoulder-specific pathology can be ruled out.

Treatment

If there is no evidence of a space-occupying well-defined lesion, the initial treatment is nonsurgical management with activity modification, NSAIDs, and analgesic medications, combined with strengthening exercises for the rotator cuff, deltoid, and periscapular muscles [26]. Surgical intervention is warranted in cases without improvement after six months of nonsurgical treatment or with a compressive lesion. The superior approach decompresses the nerve at the suprascapular notch, using either a split or elevation of the trapezius. A posterior approach is used to decompress the nerve at the spinoglenoid notch. This approach spares the deltoid and requires subperiosteal elevation of the infraspinatus to access the spinoglenoid notch. An arthroscopic approach is used in cases with soft tissue mass like paralabral cysts related to posterior labrum or SLAP tears [1, 23].

Thoracic outlet syndrome

Thoracic outlet syndrome (TOS) includes a range of upper limb neurovascular compressive pathology affecting the brachial plexus (neurogenic TOS, NTOS) and the subclavian vessels (vascular TOS) as they course through the cervicoaxillary canal. Although rare, TOS is often overlooked as the cause for the limitation of high-level athletic performance in overhead athletes. NTOS is more common in females than males by a ratio of 3.5:1.2.

Neurogenic TOS

Anatomy

The C5 to T1 roots form the brachial plexus, which course between the anterior and middle scalene muscles to leave the neck. NTOS is caused by dynamic compression of the brachial plexus nerves at the supraclavicular scalene triangle level, between the clavicle and first rib, or in the infraclavicular level at the subcoracoid space posteriorly to the pectoralis minor (Fig. 3).

Pathophysiology

The aetiology of NTOS in throwers and overhead athletes is usually the combined result of predisposing anatomic variations (musculotendinous abnormalities, fascial bands, or cervical ribs) with local responses to acute or repetitive injury leading to fibrosis and hypertrophy of the scalene or

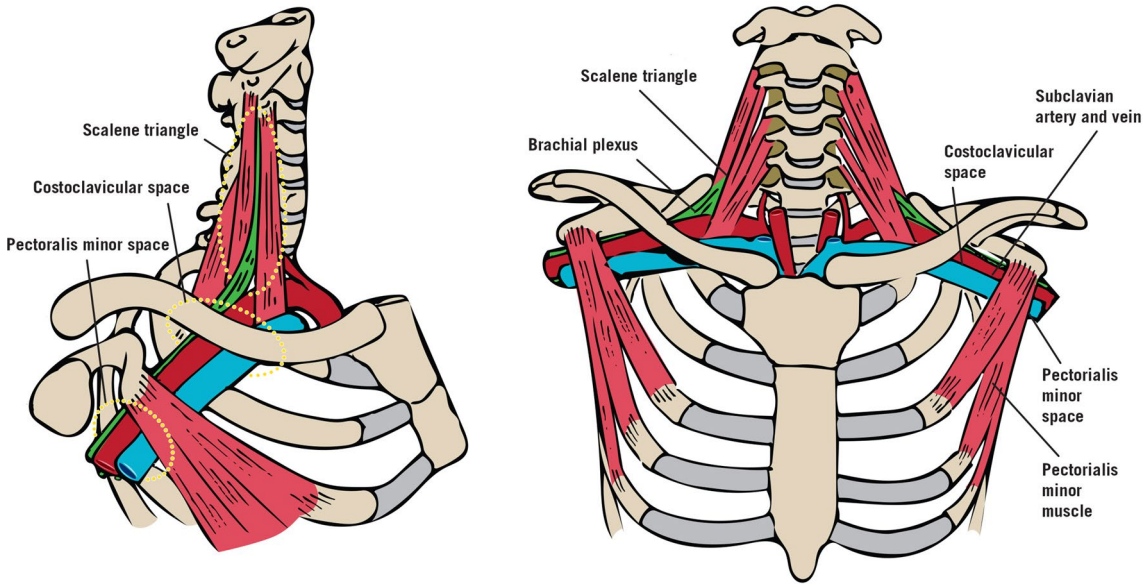

Fig. 3 Neurogenic thoracic outlet syndrome is caused by dynamic compression of the brachial plexus as it courses through the cervicoaxillary canal. At the supraclavicular level it can be compressed between anterior and middle scalene muscles at the scalene triangle, at the costoclavicular space it is compressed between first rib and clavicle, and at the infraclavicular level it may be compressed posteriorly to the pectoralis minor muscle. Figure used with permission from [6]

pectoralis minor muscles. NTOS in throwers and overhead athletes may also occur due to excessive scapular depression of the dominant arm, inadequate scapular muscle stabilization, or scalene, trapezius, and pectoral minor muscle hypertrophy [1, 27, 28].

Diagnosis

Athletes with NTOS may experience pain, numbness, or paresthesia/dysaesthesia that may involve the entire upper limb without following a single peripheral nerve or nerve root distribution and are usually exacerbated with overhead activities. However, in many cases, the lower trunk or ulnar nerve distribution (C8 to T1) is primarily affected. Other complaints include arm weakness or fatigue, heaviness, and difficulty gripping the racket, bat or throwing object because of intrinsic muscle weakness [2–32]. Clinically, muscle weakness, especially of hand intrinsics, may be found. Symptoms may be elicited with overhead arm position and by palpation over the brachial plexus at the supraclavicular or infraclavicular level. Provocative tests include the Adson manoeuvre, costoclavicular manoeuvre, and the Wright and Roos stress tests [1, 23, 28]. Standardized clinical diagnostic criteria for NTOS based on the symptoms, history, and physical examination have been proposed [30]. Imaging and EDX studies are usually negative or exclude other diagnoses.

Treatment

Non-operative management is the first line of treatment for NTOS, and the main goal of physical therapy is to treat muscle imbalances in the neck, upper back, shoulder, and scapular regions. Usually, it takes four to six months of physiotherapy to achieve significant improvement, and evidence shows that most patients (50 to more than 80%) with NTOS do not require further operative intervention [1, 30, 33–35]. Surgical treatment is warranted for athletes with refractory symptoms and insufficient improvement with non-operative measures. Surgical decompression for NTOS consists of anterior and middle scalene muscle resection, at times removal of the first rib, and brachial plexus neurolysis. Depending on the surgeon's preference supraclavicular or transaxillary approach can be used. Additionally, the pectoralis minor tendon can be released through an infraclavicular incision [27]. After surgical decompression, full rehabilitation and return to high-level sports participation may take nine to 12 months. Postsurgical outcomes show almost 85–90% of the high-level overhead athlete population reporting significant symptom improvement [31, 36].

Long thoracic nerve palsy

Anatomy

The long thoracic nerve originates from the ventral rami of C5-C7 and innervates the serratus anterior muscle. It passes through the middle scalene muscle and penetrates

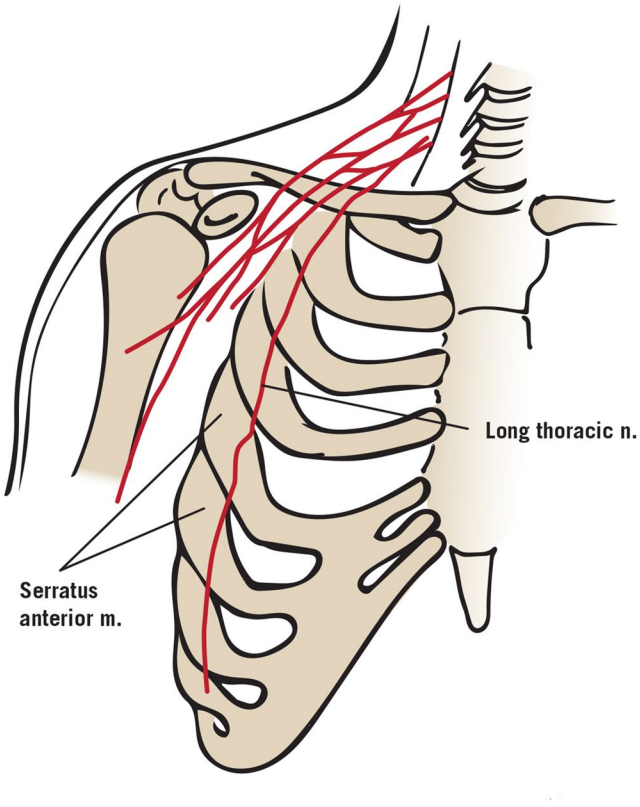

Fig. 4 The long thoracic nerve innervates the serratus anterior muscle after penetrating its proximal part. Figure used with permission from [6]

the proximal serratus anterior muscle. These two muscles are the points the nerve is relatively tethered to and may be compressed. The serratus anterior keeps the scapula close to the thorax and stabilizes it during arm motion (Fig. 4).

Pathophysiology

Long thoracic nerve palsy can occur by repetitive microtrauma and traction during throwing motion while the arm is in the overhead position and the head is tilted in the contralateral direction. Another mechanism is during maximal scapular protraction [1, 28, 37].

Diagnosis

The athlete may feel neck, shoulder, and scapular discomfort or pain, reduced velocity/performance and weakness during throwing motion. Clinically, there is usually decreased forward flexion range of motion and strength, as well as scapular dyskinesis during arm forward flexion and wall push-ups. MRI may reveal fatty infiltration or atrophy of the serratus anterior. Electrodiagnostic studies are often inconclusive.

Treatment

Non-operative management is the initial treatment strategy for athletes with long thoracic nerve palsy. Physiotherapy focuses on the range of motion exercises and strengthening of periscapular and rotator cuff muscles. Gradual improvement can be noted, requiring up to two years for maximum recovery in some cases. To a small extent, scapular dyskinesis may persist [37, 38]. Surgical decompression may be required if no improvement is noted in the early stages. Nerve transfer of thoracodorsal to long thoracic nerve has shown good functional recovery [39]. In later stages, muscle transfers of the sternal head of pectoralis major or, rarely, scapulothoracic fusion may be necessary [40, 41].

Conclusion

Treatment of the throwing athlete with shoulder pain remains among the more challenging aspects of orthopaedic sports medicine. Nerve compressions and pain syndromes should be included in the differential diagnosis of these athletes. Early detection of symptoms, careful clinical examination, and exclusion of other usual structural pathologies around the shoulder may raise suspicion of possible nerve compression syndrome. A coordinated approach among trainers, therapists, and physicians is required to comprehensively treat nerve compression symptoms and related shoulder pain in the throwing athlete.

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Data availability No datasets were generated or analysed during the current study.

Declarations

Ethics statement (Human ethics and consent to participate) Not applicable.

Competing interests The authors declare no competing interests.

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