Review

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Stingers—A Review of Current Understanding and Management

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Review

Stingers—A Review of Current Understanding and Management.

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Abstract: Stingers are transient sensory and motor loss of one upper extremity due to stretch or compression injury to the brachial plexus or the exiting cervical nerve roots. All nerve roots that make the brachial plexus are prone to injury, but the upper roots of the brachial plexus (C5 and C6) are most frequently affected. Although the true incidence of stingers is hard to know, American Football and Rugby are the two sports with the highest incidence. Mostly, stingers have a promising prognosis. However, with advancing sports medicine and rehabilitation, so did the general understanding and management of stingers among the medical professionals. This review is a summary of the current understanding of the pathology, clinical features, investigative modalities, novel radiologic measurements, management, rehabilitation and return-to-play decision protocol for stingers.

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1. Introduction and Definition

Stingers, also known as "burners", are defined as transient sensory and motor loss of one upper extremity due to stretch or compression injury to the brachial plexus or the exiting cervical nerve roots (Figure 1)[1-4]. Stingers most commonly occur during contact sports like American Football and rugby, but other sports like wrestling, boxing, cycling, and falls from horseback have been implicated as causes.[2, 5-7]. The symptoms can last for few seconds resolving at the field, to hours, mostly resolving within a day, or, in some severe instances, taking up to 2-6 weeks to completely resolve[3, 6, 8, 9]. The athlete typically complains of sharp pain in the supraclavicular area that is followed by unilateral arm pain that may or may not follow a dermatomal distribution and reaches the fingertips [4, 9, 10]. Associated paresthesia, tingling, numbness, extremity weakness ranging from mild to complete loss of strength with a sensation of "dead-arm" and, decreased cervical range of motion are all features that can be present[6, 8, 9]. All nerve roots that make the brachial plexus are prone to injury but, owing to the hyperextension and direct compression mechanism of injury of stingers and the underlying anatomic features, the upper roots of the brachial plexus (C5 and C6) are most frequently affected[8] (Figure 2). Since stingers are a common occurrence in contact sports and the treating physician must make sensitive decisions that impact the athlete's career, understanding of stingers and comprehensive knowledge about the types of injury, anatomy, mechanism of injury, treatment, and prevention is of the utmost importance to render the best care. Here we review the current understanding of stingers and their management with the intention to give insight to health professionals.

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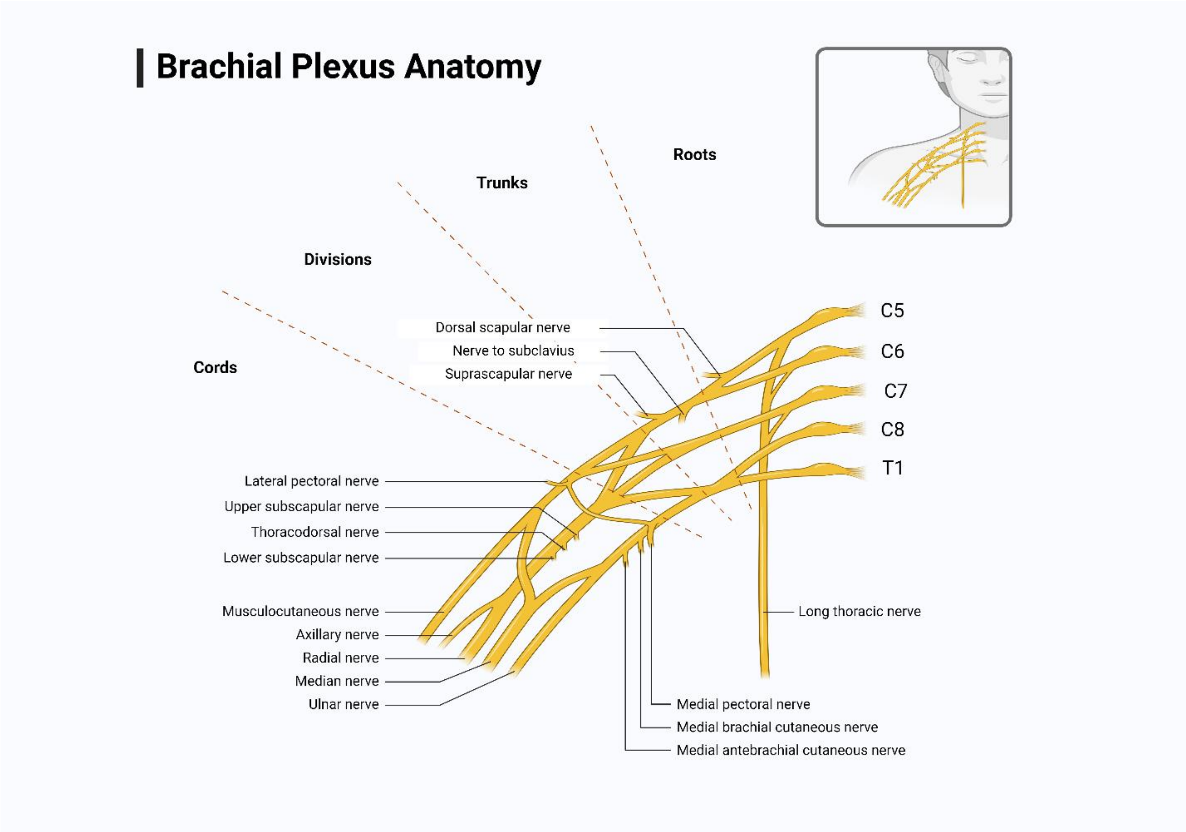

Figure 1. Anatomic illustration of Brachial Plexus. Created in BioRender. Gebeyehu, T. (2024) https://BioRender.com/g33z849.

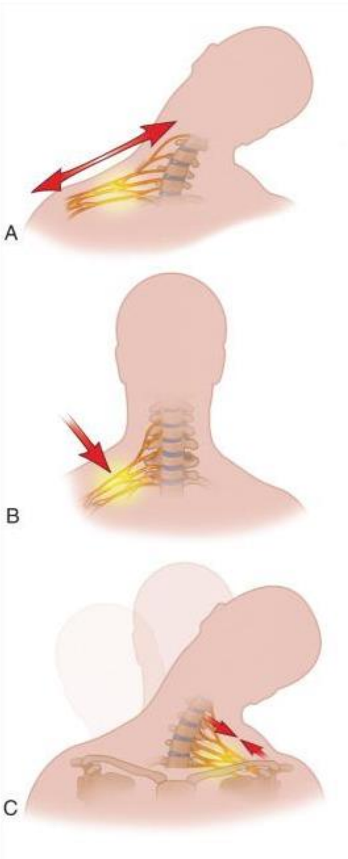

Figure 2. Various mechanisms of injury causing Stingers. **A**. Stretch injury with depressed shoulder and laterally hyper-extended neck. **B**. Direct injury at Erb's point. **C**. lateral flexion and hyperextension of the neck

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compressing the brachial plexus. Used with permission from Clinical Tree Publications, DeLee, Drez, & Miller's Orthopaedic Sports Medicine, Orthopedics, Stingers, Online publication, January 27, 2024. Stingers - Clinical Tree.

2. Epidemiology

The true incidence of stingers is hard to know due to underreporting by athletes. This underreporting is either intentional for fear of negative implications in the career of a professional athlete or from overlooking symptoms because of their transient and mild nature[2, 9]. However, considering the anatomy of the nerves involved, athletes affected most are those participating in activities that can potentially involve a violent contralateral neck flexion and ipsilateral shoulder depression or direct compression of the lateral neck and shoulder area[11, 12]. The sports reported to be associated with stingers include but are not limited to American football, rugby, wrestling, hockey, gymnastics, and boxing.[12-15]. The highest incidence was noted in American football[3, 12-15], especially in players in the defensive ends (25.8%) when athletes engaged in tackling (36.7%)[2]. Offensive linemen had a 23.6% stinger rate[2]. The National Collegiate Athletic Association (NCAA) injury surveillance program reported a 2.04 per 10,000 athlete exposure injury incidences between 2009-2015[2]. A study by Beaulieu-Jones et al. showed stingers accounted for 62.2% of neck injuries in athletes playing for the National Football League [16]. Starr et al., using a survey, identified that 50.3% of high school, college, and professional football players had a lifetime occurrence of stingers, with the highest incidence in running backs (69%), defensive linemen (60%), linebackers (55%), and defensive secondaries (54%)[17]. Another study in Japan by Kawasaki et al. showed that 33.9% of high school and university rugby players had stingers with a recurrence rate of 37.3% [5].

3. Mechanism of Injury

The two main mechanisms implicated in stingers are stretching and compression of the nerves of the brachial plexus[3, 9, 18], (Figure 2). Compression of the nerves can happen either at the nerve root area by bony elements of a narrowed neural foramina during axial loading or hyperextension and lateral flexion of the neck. Alternatively, compression may occur by direct impact at Erb's point. [9, 18]. Compression of the nerve roots is seen in Chronic Stingers where there has been repetitive trauma to the neck resulting in anatomical changes to the cervical vertebra and surrounding structures. One of the anatomic changes that takes place is cervical canal and neural foraminal stenosis due to changes in the sub-axial space over time[19]. Injury by compression at the neural roots happens mostly during tackling in football or rugby in athletes that had these anatomic changes[20, 21]. Compression at Erb's point affects nerves distal to the root area. Erb's point is located where the C5 and C6 join to form the superior trunk bundle right above the clavicle, and it is where the nerves are most superficial[11]. Markley et al. studied tackle athletes and showed that protective shoulder pads can cause brachial plexus injury by compressing nerves at Erb's point against the medial scapula when an athlete is tackling[22]. Stretch injury, on the other hand, is usually due to ipsilateral shoulder depression and contralateral neck flexion, which is common in specific tackling techniques that use the top of the head or helmet as the point of contact and are now banned, namely clothesline and spear tackling [23].

All contact athletes are at risk of injury from any of the mechanisms of injury. However, age and length of involvement in contact sports play a crucial role in determining which type of injury an athlete most experiences. Older and ''veteran' athletes that experienced multiple impacts usually have anatomical changes in the cervical spine[8, 19, 24, 25]. The changes include scalene muscle hypertrophy, cervical canal and foramen stenosis, degenerative changes like osteophyte formation and, denticulate ligament stiffening that expose the nerves to repeated trauma leading to loss of the neural protective tissue, which in turn makes the nerves less compliant and prone to trauma [7, 26]. Therefore, the changes within the cervical spinal column and the resulting neuroforaminal narrowing in 'veteran' athletes cause them to have injuries closer to the spinal cord area where the nerves originate. This is because there is high tension and less compliance of the nerves during an impact that would hyperextend the neck in the opposite direction to the course the nerves follow[7, 26]. Younger athletes, however, experience more distal nerve damage due to absence of changes within the cervical column that would spare the nerves from chronic damage and a preserved neural compliance at the root area[8, 9].

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4. Grading[8]

Grading for stingers is based on the severity of neuronal injury (Table 1). Neurapraxia or Grade I stinger refers to a mild injury where the axon of the nerves is not severed but will have a temporary interruption of conduction because of neuronal stretch. This type of injury is usually accompanied by normal neuronal and surrounding tissue (Schwann cells, endoneurium, epineurium, and perineurium) and lasts for a very short time, with complete neurologic recovery expected. Axonotmesis, or Grade II Stinger, refers to a more extensive injury where the axon of the nerve is severed, and Wallerian degeneration of the severed axon takes place. This type of injury can have a longer-lasting effect on neurologic deficit, usually taking up to 2 weeks to resolve and a maximum of a year and a half to fully recover. Grade III stinger refers to a more severe case and is *rare in athletics*. It can be grade IIIA, which is also known as neurotmesis, where complete irreversible neural damage has taken place, or in the worst form, there can be a nerve root avulsion (Grade IIIB Stinger) where the damage is pre-ganglionic[8]. Grade III stingers are irrecoverable through the body's innate healing and require surgical intervention to facilitate functional recovery. However, complete recovery is rare and affected individuals are usually left with permanent deficits. [8, 27-31].

Table 1. Grades of Stinger injury and the mode of treatment. *[27-30].

Grade	Nerve Injury	Mode of Treatment
I	Neurapraxia	Usually transient with no need for any intervention. Pain control, rest, and physiotherapy as needed if symptoms persist. Consider MRI and other imaging with serial examinations if symptoms persist for >5 minutes.
II	Axonotmesis	Pain control, rest and physiotherapy, serial monitoring with Electrodiagnostic Studies, Imaging (MRI, radiographs).
IIIA	Neurotmesis	Imaging, Electrodiagnostic Studies, Nerve Repair ± Nerve Transfer*, Pain control and physiotherapy.
IIIB	Nerve root avulsion	Imaging, Electrodiagnostic studies, Nerve Transfer*, pain control and physiotherapy

5. Clinical Presentation

Athletes typically present with unilateral upper limb weakness and a dermatomal or non-dermatomal burning or shock-like sensation (hence the terms "burner" and "stinger") starting at the supraclavicular area and radiating down the upper limb after forceful contact between the shoulder and neck[9, 32-34]. Other sensory symptoms like numbness and sensation of "dead arm" are also common[34]. The symptoms widely vary, from being only sensory abnormality in a dermatomal fashion to complete loss of strength and sensation in the affected limb [35]. A Canadian study of football players by Charbonneau et al. reported tingling in 77%, numbness in 61%, weakness in 44%, and neck pain in 17% of athletes.[36] Depending on the severity of the injury, symptom duration can be seconds to days[3]. Knowing the mechanism of injury is crucial in evaluating an athlete with stingers since it can inform other possible injuries. The mechanism of injury is usually witnessed by the sideline medical staff or from video recordings of the incident. Other clinical symptoms and findings like persistent neck pain with limited range of motion, lower extremity involvement, bilateral upper extremity involvement, headache, dizziness, nausea, vomiting, changes in mentation, speech, and vision, changes in vital signs like blood pressure, brachial-brachial index, heart rate, and temperature may indicate an alternative diagnosis of vertebral fracture, spinal cord, brain, or vascular injury. The presence of these symptoms warrants further evaluation[3, 37-40].

6. On Field Evaluation and Management

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The evaluation of injured athletes starts within the field or on the sidelines. Similar to the evaluation of other traumatic injuries, suspected stingers should begin with an advanced trauma life support (ATLS) structured assessment to rule out other serious and life-threatening injuries involving the airway, breathing and circulation[34, 41]. All athletes with neck injuries should be considered to have sustained spinal cord injury until proven otherwise [40]. Therefore, spinal cord and other life-threatening injuries should be thoroughly evaluated and ruled out before other lower priority examinations can be performed [7, 41]. If such an injury cannot be ruled out from the assessment, head and neck stabilization should be considered early, and the athlete should be transferred to an appropriate trauma facility. Any manipulation of the area, as in the removal of the helmets and shoulder pads, should be done with the utmost care, at the right site with the necessary equipment and assistance from other care givers under the supervision of practitioners familiar with the acute management of spinal cord injury to minimize the risk of neurologic worsening and adverse outcomes.[7, 19, 41]. It is also noted in some studies that helmets and shoulder pads contribute to spine alignment, further supporting the notion of not rushing to remove them[42-44]. The exceptions to immediate removal of helmets or shoulder pads according to these literature would be difficulty in assessing/managing airway because of the design of the helmet, facemask or chinstrap; during cardio-pulmonary resuscitation; need for chest access for external defibrillation; and, the presence of scalp injury requiring visual access[43, 44]. Suspicion of spinal cord or other life-threatening injury warrants immediate imaging to assess the extent of the injury. Once the possibility of a serious injury is ruled out, the athlete should be evaluated with the appropriate physical examination, focusing on inspecting for asymmetry indicating dislocation or fracture, persistent cervical or neck tenderness, range of motion for neck and shoulder, and a thorough evaluation of limb strength[8, 9, 33, 34]. It is important that muscles innervated by the brachial plexus should be evaluated in detail beyond cursory grip strength testing only. Weinstein [34], in model management of stingers explains at least a minimum testing of the spinatii, deltoid, biceps, brachioradialis, triceps, serratus anterior, wrist flexors, and extensors, as well as grip strength, is mandatory with the unaffected side serving as a control. Pain due to nerve injury within the spinal foramina can be reproduced with extension and lateral flexion (Spurling's test), while brachial plexus injury lacks this feature [9].

Other suspected injuries of vascular origin should be evaluated in a hospital setting to confirm their presence and characterize them for the appropriate management. CT Angiography, MR Angiography supplemented with digital subtraction angiography, and ultrasonography are all used in confirming and planning interventions for vascular injuries[8, 41, 45].

7. Imaging

The use of imaging can reveal important findings in athletes with stingers and is utilized for diagnosis and screening[10, 46, 47]. However, indications for imaging are a controversial subject due to absence of definitive recommendations or guidelines [48]. A study by Standaert and Herring on expert opinion and controversies mentions the findings of imaging are not always clear [18]. A more recent modified Delphi consensus study with the cervical spine research society by Schroder et al shows a super majority (84.5%) of spine surgeons agree that athletes who experience first-time stingers and symptoms lasting from seconds to less than 5 minutes need no imaging studies[47]. Again, in contrast to the old consensus, Schroder et al. shows if the symptoms persist for more than 5 minutes or if athletes have more than one episode of stingers, regardless of the duration of symptoms, a super majority (84.5%) of surgeons agree further consideration including imaging is indicated[47]. The basic imaging to be considered are cervical radiographs with anterior, posterior, lateral flexion/extension and odontoid views, and MRI[8, 9, 18]. CT scan is an alternative for those who cannot do MRI[8, 9, 18] Single-photon emission computed tomography (SPECT) can facilitate the diagnosis of an occult cervical vertebra fracture[4].

Chronic stingers are believed to be a result of degenerative anatomic changes in the cervical spine[8, 19, 24, 25]. The use of imaging, hence, can possibly identify those at risk of sustaining stingers based on changes observed in the imaging. Torg et al. described the Torg-Pavlov ratio to confirm and screen for stingers (Figure 3a)[3]. The Torg-Pavlov ratio is determined by dividing the distance from the midpoint of the posterior aspect of the vertebral body to the nearest point on the corresponding

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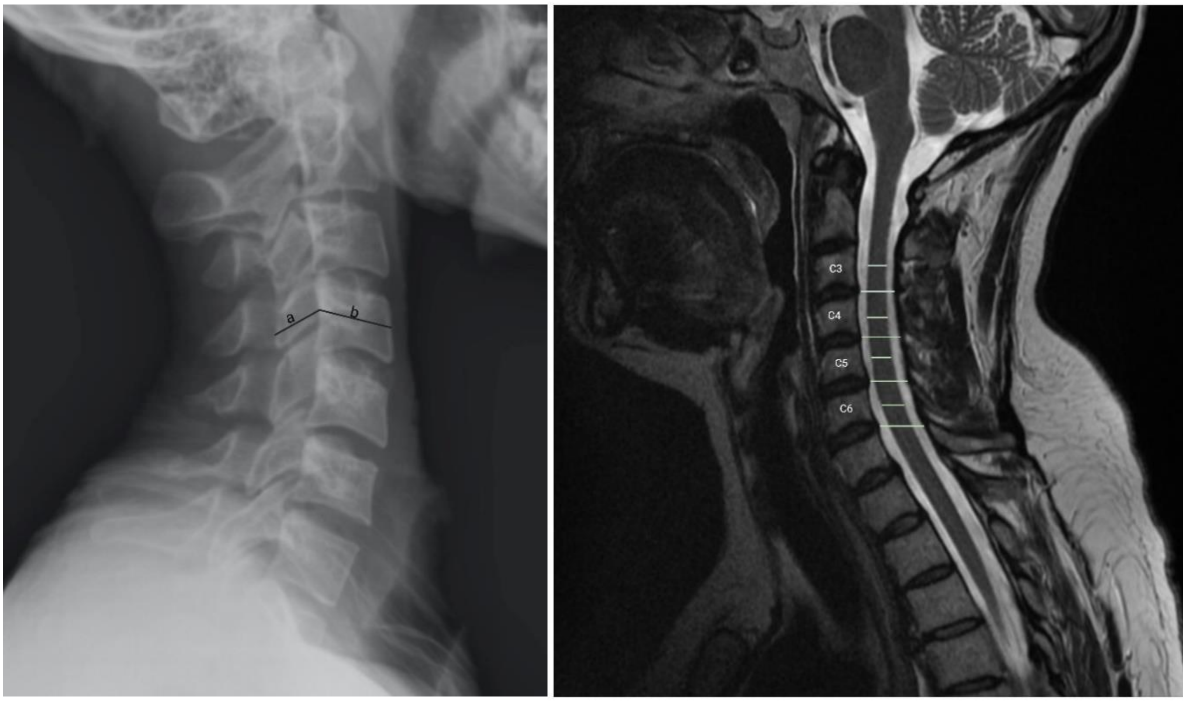
spino-laminar line, by the anteroposterior width of the vertebral body on a plain radiograph[3]. According to Torg et al, 95% of all reported cases had a Torg-Pavlov ratio of <0.8[3]. However, a very low positive predictive value for asymptomatic athletes% makes this measurement not useful for screening[49]. The role of MRI in screening is also a recent novel development. Presciutti et al.[19], while studying the changes in the sub-axial space for chronic stingers, determined the mean sub-axial cervical space available for the cord (MSCSAC). MSCSAC is the average of the values determined by subtracting the sagittal cord diameter from the disc-level spinal canal diameter between C3 and C6[19] (Figure 3b) and is a better alternative than the classic Torg-Pavlov ratio[19]. This novel measurement has a sensitivity of 80% and a negative predictive value of 0.23 for stingers when 5.0mm is used as a cut-off. When a cut-off value of 4.3 mm is used, MSCSAC has a 96% specificity with a 13.25 positive likelihood ratio. Presciutti et al. also found that these measurements are 20% more accurate than the classic Torg ratio[19]. Other novel measures used to objectively identify cervical spinal canal stenosis are cord-to-canal area ratio (Figure 4a), which is the ratio of transverse cord area to transverse canal area, and cord compression ratio (Figure 4b), which is calculated by dividing the transverse cord diameter by sagittal cord diameter (a/b) [50]. These measures are highly predictive of individuals at risk of cervical spine injury after minor trauma and can indirectly predict those at risk for chronic stingers.



A. **B.**

Figure 3. A. Torg-Pavlov Ratio, TPR= a/b. **B.** Mean Sub-axial cervical space available for the cord is the mean of the values for cord diameter deducted from, disk level canal diameter at each level between C3 and C6.

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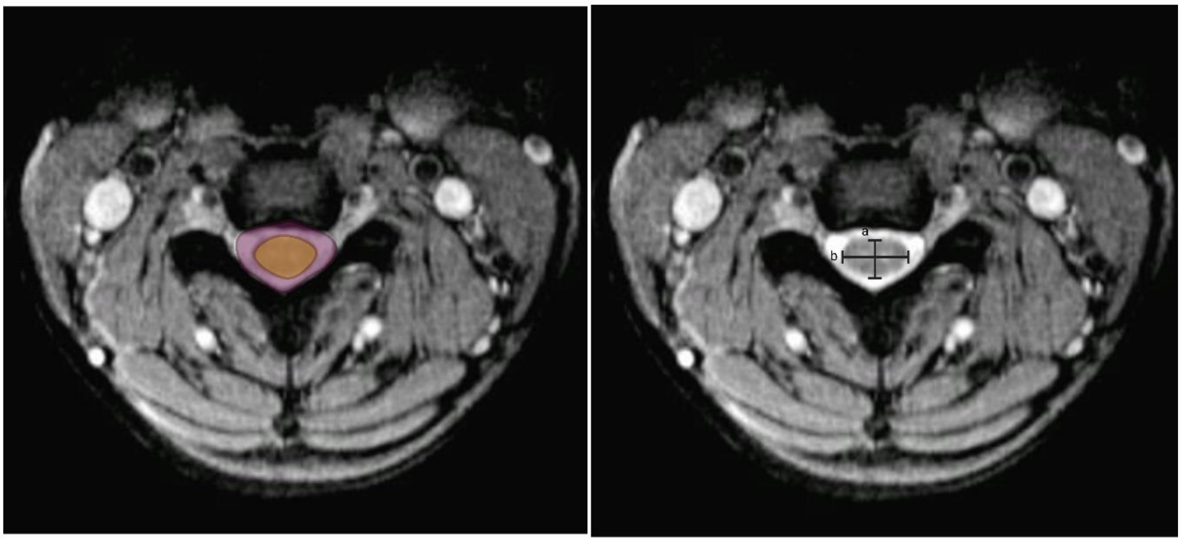

A. B.

Figure 4. A. Illustration of Cord to Canal Area Measurement, the ratio of area under inner circle (orange, [*cord*]) to area under outer circle (area under pink and orange combined, [*canal*]). **B.** Cord compression ratio =a/b, ratio of sagittal[a] to axial [b] cord diameter.

8. Electrodiagnostic Studies

Nerve conduction tests and electromyography are utilized as additional tools to clinical findings and imaging and are used to monitor progress[8, 50]. These can be used to inform the clinician about the site, severity, and regeneration of nerves after injury, based on signals generated from normal and involved muscles or nerves[8, 50]. For instance, limb muscle involvement can imply plexus or root injury, while paraspinal muscle involvement implies root injury which is because of the anatomical orientation of the posterior rami nerves supplying the paraspinal muscles[50]. Sensory amplitudes can also be used to localize injury sites. A normal peripheral sensation signal conduction velocity, and amplitude is seen in preganglionic injury. However, normal amplitude does not rule out post-ganglionic injury[50]. The severity of injury also correlates with the presence and magnitude of abnormal spontaneous electrical activities that manifest as positive sharp waves and fibrillation. The absence of positive sharp waves and fibrillations, however, does not rule out nerve root injury or rule in brachial plexus injury[50]. Abnormal electrical activity takes 2 weeks to develop and 3-5 weeks to maximize[50]. Complete nerve transection is known for absence of muscle unit recruitment and has the worst prognosis. On the other hand, discrete motor unit recruitment that occurs despite an attempt to maximally contract muscle is an indication of a conduction block, which can be a result of severe stretch injury (neurapraxia) or axonal degeneration. However, after the first few weeks, when neurapraxia is expected to subside, it is a sign of axonal degeneration[50]. Long polyphasic potentials and nascent potentials (axonal regeneration potentials, ARPs) develop over time and are seen as signs of reinnervation and regeneration of axons, respectively[50]. Terminal collateral sprouts polyphasic potentials identify chronic and recurrent injury[50]. For the various reasons mentioned, even though it is possible to use EMG as early as 7 days, it is more specific after 4-6 weeks of the injury and afterward, and, is best utilized in the follow-up period rather than in the acute phase[3, 8, 9, 50].

9. Return to Play

Most stingers are mild, lasting a few seconds to minutes, and do not interfere with participating in the next game or practice[3, 4, 35, 36]. The decision to clear athletes to return to play is a controversial issue that is reliant on expert opinions as there are no available definitive studies[1, 3, 7-10, 12, 32, 47]. The current expert consensus takes the presence and duration of symptoms, number of episodes of stingers, and imaging findings to either give a temporary or permanent withdrawal from contact sports[10, 47]. If an athlete experiences the first episode of a stinger and the symptoms fully resolve in less than five minutes (no strength, sensory, and range of motion limitation is seen), the athlete can return to play the same day[47]. Athletes with more than one episode of stingers or symptoms lasting more than 5 minutes have a relative contraindication to immediately return to play as they need to have imaging and serial examinations to rule out underlying cervical abnormality[10, 47]. The most important parameter to consider is pain and weakness-free full range of motion of the neck and shoulder joints [3, 7-10, 47]. If the athlete sustains a grade IIIA or IIIB injury, it is an absolute contraindication to return to play, and further treatment is indicated (Figure 5).

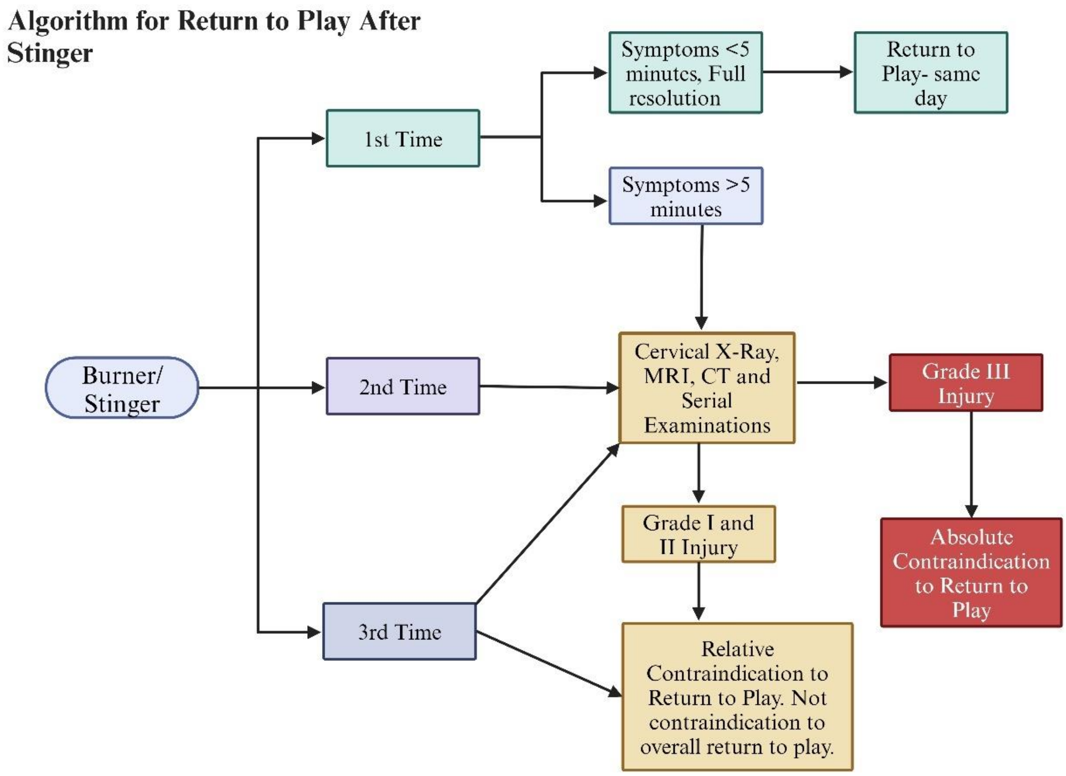

Figure 5. Algorithm for return to play after a stinger injury. Created in BioRender. Gebeyehu, T. (2024) https://BioRender.com/y07q789.

10. Treatment

Management of stingers is dependent on the severity of nerve injury or grade of injury[8, 9, 34]. For athletes who sustained Grade I and II injuries, pain control, reduction of inflammation, physical rehabilitation, and prevention of recurrence are the mainstay of treatment [8, 9, 34]. For athletes who sustained Grade III injury with nerve root avulsion or neurotmesis, timely surgical reconstruction of the severed nerve with the addition of the non-operative treatment protocols is the mainstay of treatment[8, 9, 27, 51].

Pain is controlled with rest, analgesics, and a cervical collar[50]. Cervical region epidural injections can also be used; however, utmost caution should be exercised because of the risks of traumatizing the cord directly during the procedure or indirectly with the high pressure from the administered medication within a narrowed and compromised canal[52].

The main target of physical therapy is correcting postural abnormalities, flexibility, and strength[50]. A prominent postural abnormality noticed after such injury is the reduction or absence of normal cervical lordosis[50], which needs to be corrected. Associated with this postural imbalance is usually associated thoracic kyphosis, scapulae protraction, glenohumeral joint internal rotation and hyperflexion of the lower cervical and upper thoracic vertebral segments, mid-cervical hypermobility, and, weakening along with shortening of various neck, shoulder and back muscles[50]. These abnormalities are all targeted with the appropriate physiotherapy muscle strengthening and stretching to return balance[50].

Prevention focuses on addressing the biomechanical factors that led to the injury, which includes proper tackling technique for the athlete, use of protective equipment like a cowboy collar and shoulder pads, restoration of neck area motion, and strengthening[3, 50, 53].

Stingers are usually caused by nerve stretch injuries, and neurotmesis and nerve root avulsion are uncommon possibilities for athletes with contact sports injuries. Surgical treatment is therefore rarely needed. In the rare instance where surgical treatment is needed, it is a salvage procedure for individuals with loss of function due to severe nerve damage (Grades IIIA and IIIB) with no return of function in sight[27, 51]. The surgery encompasses exploring the brachial plexus to identify the injury of the brachial plexus anatomy and strategize repair options. Surgical repairs options include direct anastomosis, interposition grafting and nerve transfer with associated partial or complete sacrifice of a less needed healthy nerve. Shoulder abduction and elbow flexion are the two important functions that are the focus of surgical repair. Garg et al. and Yang et al. performed a meta-analysis and systematic review independently, trying to compare the surgical options and determine the superiority of nerve transfer alone or a combination of nerve transfer with nerve repair. Both concluded from their synthesized data that nerve transfer is a better option in terms of motor outcome for shoulder abduction and elbow flexion[27, 51]. However, the systematic review by Yang et al. showed that for shoulder abduction, neither of the surgeries was superior to the other due to a post hoc statistical analysis that entailed a large number of statistical manipulations to demonstrate a significant benefit from nerve transfer [27]. Therefore, the issue of what type of surgery should be preferred is not yet settled and is an open area for future research.

The timing of surgery is also another controversial issue. The current suggestion is waiting for 3 to 6 months to allow re-innervation to take place and settling of concomitant injuries that don't make surgery feasible[30]. This approach was advocated for cases of nerve root avulsion or damage where regeneration is not expected[27]. However, Kato et.al documented that outcomes are dependent on the timing of surgery, and early surgery within 1 month of injury showed great outcomes in terms of functional recovery[31]. This better outcome is also sensible considering a motor endplate that becomes atrophic and de-nucleated, dimming the hope of recovery with time-lapse (especially after 1 year of injury), especially considering the long physiologic period it takes for a nerve to regenerate, reach, and re-innervate a muscle [28-30]. In conditions where a motor end plate has been rendered dysfunctional, microvascular transfer of normal muscle with a motor endplate, in addition to nerve grafting, has been suggested[29].

11. Conclusion

Stingers are injury to the nerve roots forming the brachial plexus, most commonly to C5 and C6. They have common occurrence in contact sports like American football, rugby, wrestling, hockey, gymnastics and boxing. The mechanism of injury can be stretching or compression of the nerve roots. Grading of stingers is based on the severity of the nerve damage which can be neural stretching, severed axon, damaged neuron or nerve root avulsion. The manifestations can be sensory or motor deficit after injury to the neck region. Stingers are mostly managed conservatively with rest, pain control, physiotherapy and rehabilitation. However, in certain rare and unlikely instances for athletes, surgical repair of the nerve roots is needed. The prognosis is mostly promising except where there is complete nerve damage or nerve root avulsion.

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