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Congenital Cervical Stenosis: a Review of the Current Literature

Alyssa M. Goodwin[1,2] · **Wellington K. Hsu**[1,2]

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Abstract

Purpose of Review Congenital cervical stenosis (CCS) is a phenomenon in which an individual has a narrow canal due to abnormal anatomy which can present with earlier degenerative symptoms due to a reduced sagittal diameter. The diagnosis of CCS is important to individual treatment and preventative measures. Often, athletes are warned against sport participation that may cause damage to the cervical spine. There may be a predisposition in certain populations, but lack of data limits conclusions. The current review investigates recent literature on the definition, pathoanatomy, clinical presentation, and management of CCS. It specifically interrogates potential populations predisposed to this condition.

Recent Findings The current literature reveals a potential predisposition for CCS in the black population when compared to the white population; however, many studies do not report race when discussing CCS patients. The lack of data limits a consensus on specific populations with a congenitally narrow canal.

Summary CCS may be more prevalent in specific populations. With knowledge of populations more at risk for this condition, physicians and teams can be alert when evaluating players and young adults. Furthermore, this may provide insight into risk for symptoms with degenerative disease. These findings introduce an avenue for further research into CCS.

Keywords Congenital cervical stenosis · Genetic syndromes · Adjacent segment degeneration · Transient quadriplegia

Introduction

Abnormal narrowing of the spinal canal commonly occurs through degenerative changes and/or developmental defects. In patients with developmental narrowing, clinical presentation of myelopathy can occur earlier with more severe symptoms than those with only degenerative disease [1•]. Congenital cervical stenosis (CCS), also known as developmental cervical spinal stenosis, is a phenomenon in which an individual has a narrow canal due to abnormal anatomy [2–4] which can present with earlier symptoms due to a reduced sagittal diameter [5, 6].

CCS was first considered by Payne and Spillane in 1957 in their discussion of cervical spondylosis and the influence on symptom presentation with a smaller size of the spinal canal [7]. Following this, many researchers published case reports, primarily in young patients, presenting with symptoms of bilateral upper-extremity dysesthesias, referred to as "burning hand syndrome" after minor trauma [8–10]. It was postulated that this symptom presentation was due to a pre-existing narrowed cervical spinal canal with limited ability to adapt sufficiently [11], resulting in more significant clinical symptoms in the setting of minimal degenerative changes, trauma, and other causes of compression [12].

The diagnosis of CCS is important to individual treatment and preventative measures. Often, athletes are warned against sport participation that may cause damage to the cervical spine [13]. Increased awareness of this condition may prevent catastrophic injuries from otherwise avoidable accidents and guide physician recommendation on appropriate precautions. This review will reveal current knowledge regarding the definition, pathophysiology, mode of diagnosis, clinical presentation of CCS, and associated spinal canal pathologies.

Diagnosis

Historically, the Torg-Pavlov ratio (TPR) was described to identify the presence of CCS which relies on plain radiographs to measure the relative ratio of the sagittal

✉ Alyssa M. Goodwin
Alyssa.goodwin16@gmail.com

[1] Department of Orthopaedic Surgery, Northwestern University, 303 E Superior, Chicago, IL 60611, USA

[2] Simpson Querrey Institute (SQI), Northwestern University, 303 E Superior, Chicago, IL 60611, USA

diameter of the spinal canal to the sagittal diameter of the vertebral body (CCS < 0.82) (Fig. 1) [14]. This comparison was limited, however, because it did not consider the structures that occupy space in the spinal canal such as the spinal cord and the posterior longitudinal ligament (PLL) [15]. Additionally, it excluded athletes from participating in sports due to a larger vertebral body resulting in a false diagnosis of CCS [13]. Currently, the TPR has mostly been abandoned due to low specificity, poor correlation with canal size, and variability among ethnicities [16].

While plain radiographs are limited to the evaluation of osseus structures, magnetic resonance imaging (MRI) provides the ability to evaluate soft tissue abnormalities that may contribute to cervical spondylosis and cervical spinal canal stenosis [18]. Additionally, MRI is the primary modality to define spinal cord occupation ratio (SCOR) which is defined as the ratio between the area of the cord and the dural sac (Fig. 2) [15]. A SCOR ≥ 75% is diagnostic of CCS, whereas a normal SCOR value is 58.3% and does not indicate CCS [4]. Other studies define a SCOR ≥ 70% as the threshold for CCS [15]. In patients with degenerative cervical myelopathy (DCM), measurements were taken above and below the pathology at the vertebral body level, as to not bias the results [15].

Kinetic MRIs (kMRI) are a unique diagnostic tool that allow for patients to be examined in various positions that may reveal a pathology not seen on a static MRI [19]. In a study evaluating the effect of flexion and extension on the cervical canal size with a kMRI, Sayit et al. found a dramatic decrease in canal distance at the C4–C5 level upon flexion of the cervical spine. In a patient that already has structures compromising this space, detrimental symptoms may occur, as seen in athletes with extreme flexion [20]. This is a

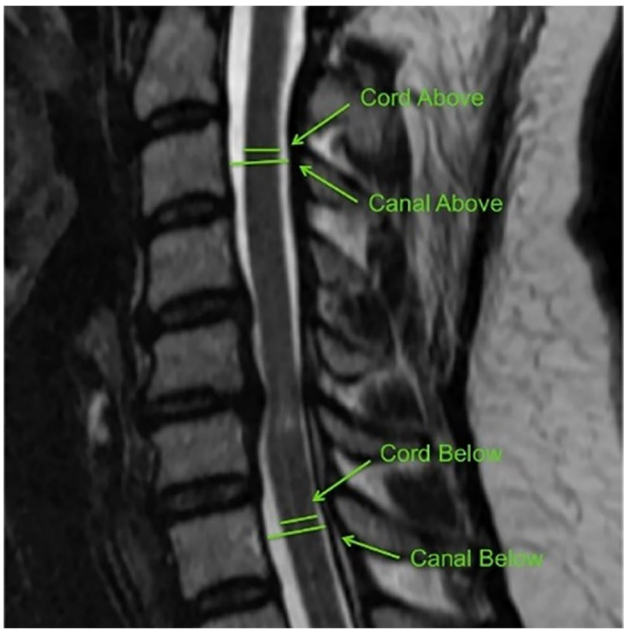

Fig. 2 Spinal cord occupation ratio (SCOR) measurements on MRI. SCOR takes the ratio of diameter of cord above and cord below divided by canal above and canal below. SCOR = $\frac{cord\ above + cord\ below}{canal\ above + canal\ below} \times 100$ [17]. Courtesy of Nouri et al. labeled for reuse according to and published with permission from Elsevier

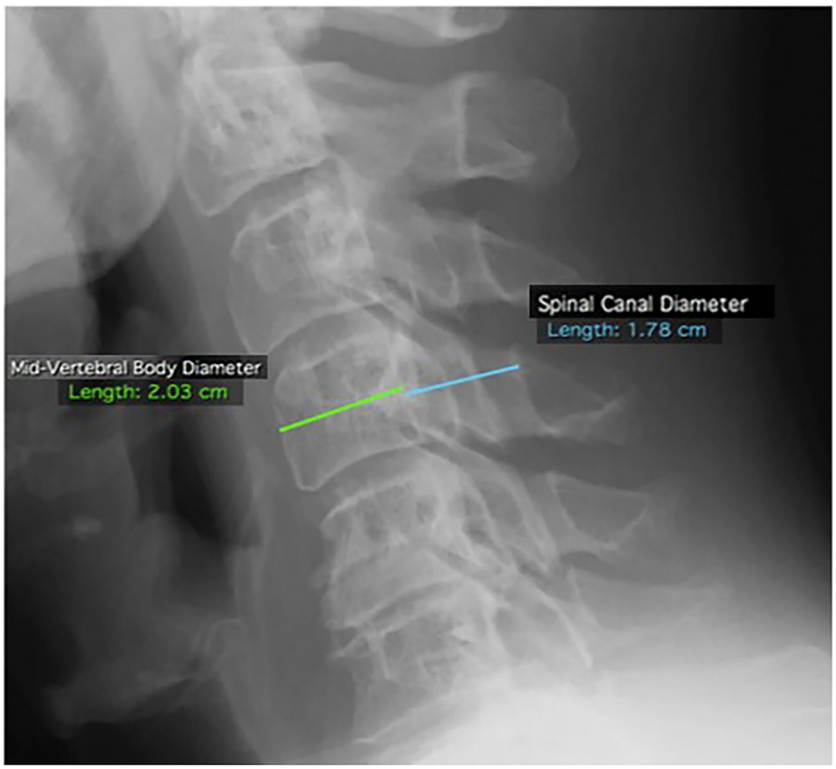

Fig. 1 A plain lateral radiograph illustrating the vertebral body diameter and spinal canal diameter measurements that comprise the Torg-Pavlov ratio (canal-body ratio) [17]. Courtesy of Nouri et al. Labeled for reuse and published with permission from Elsevier

valuable tool to evaluate symptomatic patients where a static MRI does not reveal stenosis.

Epidemiology

While there is no consensus on the definition of CCS in the evidence-based literature, historically, this has been defined as an anteroposterior (AP) canal diameter that measures < 12–13 mm on a plain lateral radiograph of the cervical spine [15]. Since this value was initially utilized in the rheumatoid arthritis population, more recent studies use an AP value of < 10 mm on MRI which has allowed for more specific evaluation of patients [2••]. Kasai et al. measured the AP diameter in male cadavers who died in northeastern Thailand, ranging in age from 22 to 93 years old with digital calipers. They defined CCS in cadavers with an AP diameter of < 12 mm at C4, 5 and 6 and concluded that CCS has a prevalence of 20.1% [21]. In a separate study with similar protocol, Lee et al. measured 3000 cadavers and identified the prevalence of CCS in the general population to be 5% [22].

There are few studies examining population-specific characteristics in relation to CCS. Spinal cord area has been negatively correlated with age, positively correlated with height, and with no relationship to body weight [23]. In a study evaluating patients with DCM by region, Nouri et al. found pre-existing congenital stenosis in populations from Latin America (9.8%), Asia–Pacific (11.6%), and North America (8.3%). Notably, the incidence rate in the European population was 2.3% [24]. Lee et al. measured cadaveric specimen sagittal canal diameter and found a significant difference between women and men at all levels (C3–C7) at an average of 13.7 mm and 14.4 mm, respectively ($p < 0.001$), but no significant difference between the black (14.0 mm) and white (14.2 mm) populations [22]. In contrast, other studies have found significant differences in measurements between African and American populations. Ndubuisi et al. examined space available for the spinal cord (SAC) at C4 (SAC4), C5 (SAC5), and C6 (SAC6) in adult Nigerians in the cervical spine. At each subaxial cervical spine level, the mean values in the Nigerian population (SAC4 4.5, SAC5 4.6, SAC6 4.9) were much smaller than those in Americans (SAC4 6.5, SAC5 6.7, SAC6 6.8) and Eastern Europeans (SAC4 6.9, SAC5 7.0, SAC6 7.7); however, their findings were similar to measurements in the Japanese population (SAC4 4.2, SAC5 4.5, SAC 4.8) [14, 25–27]. The authors suggested that the prevalence of narrow SAC measurements is most significant at C4 leading to the overall findings of the study; however, they did not statistically compare the SAC measurements between the different populations [28]. Similarly, a study examining incidence of cervical stenosis by Murone et al. concluded that Asians had smaller canal diameters than whites [5].

In a cadaver study comparing the South African white and black populations, Taitz measured mid-sagittal diameters of foramina at vertebral segments C3, C4, and C6. They found that the black males (C3: 13.5, C4: 13.4, C6: 13.7) had significantly narrower foramina than the white males (C3: 13.9, C4: 14.2, C6: 14.4) ($p < 0.05$) [29]. Ezra et al. demonstrated, utilizing a Torg ratio < 0.80, that CCS was significantly higher in the African American (AA) population at levels C3 (15.1%), C4 (18.5%), and C5 (19.2%) compared to European Americans at levels C3 (5.3%), C4 (9.9%), and C5 (6.1%) ($p = 0.008$, $p = 0.043$, $p = 0.001$, respectively) [30]. The data in these studies suggest that further research on the relationship between ethnicity and CCS is warranted.

Pathoanatomy

In a retrospective cross-sectional study in a Japanese population with and without CCS, Miyazaki et al. measured the osseous spinal canal area (OSCA), the dural sac area (DSA), and the spinal cord area (SCA). They found OSCA and DSA to be significantly smaller from C3 to C5 in patients with CCS compared to those without ($p < 0.05$). However, the spinal cord area was not significantly different. Furthermore, they concluded that the CCS group had significantly shorter pedicle axis length and lateral masses when compared to the non-CCS group ($p < 0.05$). OSCA was not related to the size of the spinal cord, further emphasizing the need to measure SCA and OSCA when evaluating for CCS [31].

Jenkins et al. defined CCS as < 10-mm mid-sagittal canal diameter at levels C3–C7 on cervical MRI and sought to evaluate anatomical abnormalities that might contribute to CCS. The authors concluded that by defining the spinal canal with a "triangle model" that includes the lamina-disk angle (LDA), the lamina-pedicle angle (LPA), and the spinal canal distance (SCD) (Fig. 3), differences between CCS and control patients could be established. patients with CCS had a significantly shorter lateral mass at all cervical levels, and at C4–C6, the posterior canal distance (PCD) was smaller than controls. Additionally, in the CCS group, the LDA was significantly more obtuse while the LPA was significantly more acute. With a more acute LPA angle and shorter laminar length, there is a higher chance of spinal canal diameter (SCD) narrowing. The authors concluded that the pathoanatomy contributing to CCS involve abnormal development of the posterior elements. Specifically, with a decrease in the LPA and increase in the LDA, there is a shortened SCD that may eventually lead to CCS [2••].

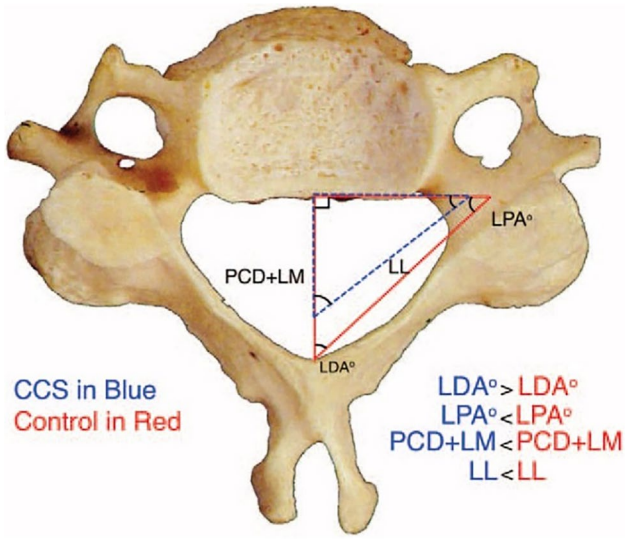

Fig. 3 Triangle model comparing measurements between a CCS and control patient. The posterior canal distance (PCD) with AP lateral mass (LM) composes the spinal canal diameter (SCD). Lamina-pedicle angle (LPA) is formed by the intersection of the and the laminar length (LL) (hypotenuse). Lamina-disk angle (LDA) is formed by the intersection of the SCD and the LL [2]. Courtesy of Jenkins et al. published with permission from Wolters Kluver

Although CCS most commonly occurs at lower cervical levels, atlas hypoplasia is a rare cause [32]. These patients often have compression of the spinal cord at the C1 level and carry a high risk of severe spinal cord injury, even with minimal trauma.

Congenital malformation of the atlas may involve various pathologies. Most commonly, the posterior arch is involved. In these patients, the most common pathology was a type A defect where there is a failure of the posterior midline fusion of 2 hemiarches, causing a midline cleft to form in the posterior arch. Symptoms often present in adulthood with cervical myelopathy due to aging processes or trauma [33].

Congenital narrowing of the cervical spine is an important risk factor for development of degenerative disease and traumatic injuries due to limited space in the canal. In a patient with CCS, an otherwise inconsequential disk herniation can have a greater impact on clinical symptoms. Morishita et al. demonstrated that patients with a developmentally narrow canal had significantly greater pathological changes in all cervical disk segments than subjects with a wide canal [34]. There is a lower threshold of degenerative changes to result in spinal cord compression and less cerebrospinal fluid (CSF) surrounding the spinal cord leads to increased risk of injury [3, 35].

In patients with DCM, the C5 region is most frequently affected. Previously, this region has been established as the narrowest cervical spinal canal region [4].

Nouri et al., using SCOR, found that CCS patients with DCM were on average 5.5 years younger and presented with worse neurological and functional impairment than patients without CCS. Additionally, the prevalence of DCM patients with CCS was 8.4% in this study [15]. Other studies examining surgical patients with DCM found a prevalence of CCS of 21.7% [6].

Spondylotic changes in the cervical spine most often occur at C5–C6, less commonly at C6–7 and C4–5, and are a site for CSM [36]. In a study by Ohwada et al., they described 12 patients with DCM found to have CCS. The average age was 30 years old [37]. In a retrospective review of plain radiographs, Edwards and LaRocca identified that myelopathy was present in patients with a sagittal cervical spinal canal < 10 mm and premyelopathic changes were present in patients with a diameter ranging from 10 to 13 mm. Symptomatic spondylosis occurred in patients with a diameter of 13–17 mm. Notably, patients with a diameter > 17 mm were not prone to cervical spondylosis [38].

Associated Syndromes

There are rare genetic syndromes that are associated with CCS. These include achondroplasia, mucopolysaccharidoses (MPS), Klippel-Feil syndrome (KFS), down syndrome, and Jeune syndrome (Table 1) [39–41]. Generally, the short stature and hindered pedicle growth decreases the space in the spinal canal in these individuals [42]. In achondroplasia, endochondral ossification is impaired and results in short, thickened laminae and pedicles. Additionally, the vertebral bodies are often concave posteriorly and protrude into the canal, further reducing the canal space [43]. While congenital thoracolumbar stenosis is often seen, cervical stenosis in achondroplasia is rare and presents with symptoms of cervical myelopathy or radiculopathy in early adult life. Achondroplasia has an incidence rate of 1 in 25,000 live births and reduced life expectancy of approximately 10 years [44]. Similarly, MPS has an incidence rate of approximately 1 in 22,000 to 25,000 live births. In these patients, cervical myelopathy is the most common presenting symptom due to musculoskeletal involvement at the cranio-cervical junction. Additionally, there are many types of MPS which affect difference cervical levels [44]. Constanzo et al. discuss patients with MPS type VI (36% incidence) had CM due to atlanto-axial instability, whereas those with MPS type VI (54% incidence) had cervical stenosis contributing to CM [45]. KFS is a condition where there is abnormal fusion of C2 and C3 which can predispose them CCS. Incidence rates are 1 in 40,000 to 42,000 live births with a slight preference for females [46]. Down syndrome, with an incidence of 1 in 660 live births, displays cervical instability which may lead

Table 1 Genetic syndromes associated with congenital cervical stenoies

Genetic syndrome	Incidence	Pathology	Symptoms
Achondroplasia	1 in 25,000 live births	Short, thickened laminae and pedicles	Cervical myelopathy or radiculopathy
Mucopolyssacharidosis	1 in 22,000–25,000 live births	GAG accumulation and MSK disruption at cranio-cervical junction	Cervical myelopathy
Klippel-Feil syndrome	1 in 40,000–42,000 live births	Fusion of C2 and C3	Cervical myelopathy or radiculopathy
Down syndrome	1 in 660 live births	Atlanto-axial hypermobility	New hyperreflexia, gait disturbance, loss of bladder control, decreased exercise tolerance
Jeune syndrome	1 in 100,000 to 300,000 live births	Hypoplastic posterior arch of C1	Quadriparesis, absent diaphragm function

to cervical stenosis. This manifests in symptoms such as new hyperreflexia, gait disturbance, loss of bladder control, and decreased exercise tolerance [42]. Lastly, Jeune syndrome has an incidence of 1 in 100,000 to 300,000 live births and may manifest as quadriparesis and absent diaphragm function due to a hypoplastic posterior arch of C1 causing cervical stenosis [47].

Clinical Presentation

Patients often present after motor vehicle accidents, falls from heights, on-the-job injuries, hyperextension, or insidious onset with primary symptoms consisting of neck pain and a combination of motor and sensory findings in both the upper and lower extremities [42]. Early cases described "burning hand syndrome" where the injury, in combination with the narrow cervical spine, caused a central cord syndrome [9]. Primarily, the various presenting symptoms are consistent with cervical myelopathy (CM). A congenitally narrow spinal canal is a major risk factor for the development of CM [48] and acute spinal cord injury (SCI). Aebli et al. determined a cutoff value of 8-mm canal diameter to be best predictive of minor trauma resulting in SCI due to decreased space available [49].

Transient quadriplegia (TQ), a rare phenomenon involving a wide range of sensory and motor weakness of both the upper and lower extremities, has been well reported in the literature [3, 13, 50, 51]. Torg et al. determined the TQ incidence to be 1.3 cases per 10,000 athletes [52]. Often, the symptoms resolve in ten to fifteen minutes, but some may last 24 to 36 hours. Of the patients who present to the ED with TQ, about 74% will have resolution of symptoms within 15 min, while 11% will have symptoms persisting for more than 24 hours, with 80% of patients experiencing neural deficits in all four extremities [53]. Most commonly, it occurs with high trauma in athletes with undiagnosed CCS [54]. Importantly, transient compression occurs where there is extreme flexion or extension of the spine. Torg et al. identified no correlation between CCS and irreversible SCI after TQ [55]. Castro et al. postulated that the relative risk of a sports-related TQ is between 645 and 3225 times greater in individuals with a history of previous TQ [56]. Casey et al. discussed return to play (RTP) parameters and determined contraindications for RTP included > 2 episodes of TQ, cervical myelopathy, reduced ROM, neurological deficit, or cervical discomfort [57•].

Management and Outcomes

In patients with mild myelopathy and radiculopathy caused by a herniated disk, conservative treatment is indicated due to the high potential for the herniated disk to regress [58].

Operative management of CCS involves surgical decompression of the spinal cord at the affected levels which may occur via anterior cervical discectomy and fusion (ACDF) or posterior cervical decompression and fusion.

Anterior

Yu et al. discussed anterior decompression as an effective treatment for CCS in patients with CSM due to potential to increase CSF, improve blood supply, and create stability that acts as a protective factor [18]. However, some authors have suggested that anterior decompression may incite postoperative instability and/or bone spurs at adjacent surgical levels in patients with a narrowed spinal canal [6, 59, 60]. Additionally, Zhang et al. examined CSM patients treated with ACDF and found that those with CCS had a significantly higher incidence of adjacent segment degeneration (ASD) after anterior fusion [61]. Dohler et al. [62] reported that 67% of their patients who underwent anterior interbody fusion experienced instability of adjacent segments and Ross et al. [63] reported adjacent segment disk herniation in 29% of their patients. In a meta-analysis, Kwok et al. reported an increased incidence of clinical ASD in patients with CCS requiring reoperation within 4 years after ACDF with plating [64]. Similarly, another meta-analysis found a significant association between CCS and ASD in patients undergoing ACDF [65].

Posterior

Hirabayashi initiated the use of posterior cervical expansive open-door laminoplasty for the treatment of CSM in patients with CCS [66]. It was found to successfully relieve spinal cord compression, improve blood supply, and resolve pre-operative symptoms, However, there was also reported a high recurrence rate of spinal cord compression [67] and poor post-operative sagittal alignment [68]. Iwasaki et al. determined expansive laminoplasty is preferred in a patient with CCS who presents with myelopathy to avoid future degenerative changes at adjacent levels [69]. Similarly, Yoshida et al. found no complications in their laminoplasty group at 3.5-year follow-up [58]. Additionally, Wang et al. examined posterior single-door laminoplasty in patients with CCS compared to those without and found comparable JOA scores and improvement rates in neurological function, with slightly longer post-operative recovery time [70]. In our practice, we generally favor a posterior decompressive approach in the setting of multilevel congenital cervical stenosis causing myelopathy to address the posterior-based anomaly that has been established for this condition.

Generally, surgical outcomes in CCS patients parallel those in non-CCS patients. Although severity of symptoms often correlates with worse surgical outcomes for DCM, patients with CCS will likely have similar outcomes to patients without CCS despite the present of neurological symptoms and myelopathy [15].

Conclusion

While there are more established definitions of CCS and more effective imaging for diagnosis in recent years, there are still key factors missing from the literature. The studies reporting on athletes' symptoms after inconsequential trauma provide important insight as these individuals are in a place where their chosen activity exposes an otherwise undetected pathology. Due to this, it is important for race to be reported and analyzed, to search for any discrepancies in the various populations as previously found in post-mortem studies. This further knowledge will guide treatment of individuals, especially high trauma athletes, to optimize their participation in something they love while maintaining their quality of life.

Author Contribution Alyssa Goodwin – idea generation, manuscript research, writing, and preparation

Dr. Wellington Hsu – idea generation, manuscript editing.

Data Availability Not applicable.

Code Availability Not applicable.

Declarations

Conflict of Interest Alyssa Goodwin – No conflicts; Dr. Wellington Hsu – Advisory board member of Stryker, Medtronic, Promimic, Surgalign.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

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- •• Of major importance

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