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Spinal Cord Injuries and Sports

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Abstract

Sports are not an insignificant cause of SCI (mainly by contact and collision sports) and they may play a crucial role in the process of SCI patients adaptation. Sports practice by SCI patients must be balanced with the wide array of complications stemming from SCI.

Several complications can arise in a patient with SCI that may potentially interfere with sports practice, such as authonomic dysreflexia, urinary tract infection, pressure ulcers, bone loss and fractures, entrapment nerve syndromes and shoulder overuse As long as a suitable exercise protocol is applied with proper medical surveillance, exercise is safe and recomended in SCI patients. This review aims to discuss the particularities of sports as mechanism of injury and as a way of dealing with the injury after SCI.

Keywords: Spinal cord injury; Tetraplegia; Paraplegia

Introduction

The spinal cord encompasses a wide array of complex associations between descendant and ascendant tracts, with upper and lower motor neurons that work together in strict cooperation. Resultant motor and sensory pathways, as well as autonomic targets, are crucial to maintaining organic integrity [1].

As such, the injury of the spinal cord, not only affects the conduction of sensory and motor signals but also the autonomic nervous response. "Tetraplegia" and "paraplegia" refer to distinct levels of motor and/or sensory function impairment. Tetraplegia implies loss or impairment of motor and/or sensory function in cervical segments, in opposition to paraplegia where these are spared. Paraplegia refers to loss or impairment of motor and/or sensory function below cervical segments (thoracic, lumbar or sacral).

Additionally, it is also important to classify the Spinal Cord Injury (SCI) as complete or incomplete. The lesion is considered incomplete if any sensory or motor function in the lowest sacral segments (S4-S5) is preserved (sacral sparing). A complete injury implies the loss of those functions in the same segments (no sacral sparing) [2].

The diagnosis of SCI can be made through a complete neurologic examination. The American Spinal Cord Injury Association (ASIA) has published a frequently cited neurological classification that standardizes the evaluation and classification of these patients. The 2011 revised classification aims to provide consistent terminology in order to describe SCI as good as possible [2].

The pathophysiology and epidemiology of SCI consequences, especially the long-term ones, cannot be fully ascertained unless a tremendous bias is accepted. This difficulty is partially related to the lack of adherence of these patients to long-term studies. Thus, the design of appropriate targeted therapies will not be possible as long as this deficit is not corrected [3,4].

This review aims to describe and discuss the particularities of SCI patients as well as the main precautions that both healthcare professionals and patients should take to address potential complications. Such actions are of utmost importance, particularly in extreme conditions such as sports, where physiological limits are frequently attained.

Complications of SCI

An exhaustive list of consequences of SCI includes autonomic dysreflexia (AD),

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thermoregulatory abnormal responses, urinary tract infection (UTI), pressure ulcers, entrapment nerve syndromes and shoulder overuse. Subsequently, some of these consequences will be discussed, focusing on some particularities of athlete's population. The main goal is the early identification of potential complications, as well as the implementation of preventive measures. Treatment strategies should be implemented when those are not effective. A large part of the consequences of SCI is directly related to forced inactivity while others result from the nervous lesion itself. The importance of prevention and treatment of complications amongst this population is even greater since athletes with SCI potentially achieve physiologic thresholds during training and competition [5-13].

Autonomic dysreflexia

The AD is a life-threatening syndrome that occurs in SCI individuals with lesion at T6 level or above. The first indexed article in PUBMED/MEDLINE database about AD in Spinal Cord injury patients dates from 1974 [14]. Since then, the number of studies that have tried to fully understand AD has steadily increased, is 2016 the year with most related publications.

AD can be caused by a noxious stimulus below the injury level, culminating in a massive sympathetic discharge which elicits, in turn, reflex sympathetic activity. Patients with complete tetraplegia show the greatest risk of developing AD (91%) in opposition with those who suffer from incomplete tetraplegia (27%) [15]. The most frequent noxious stimulus are: urological (e.g. bladder distention, urinary tract infection, bladder or kidney stones, catheterization), intestinal (e.g. constipation, impaction) and skin lesions (e.g. ingrown toenail and pressure area). Other stimulus such as fractures, menstruation, sexual intercourse or burns can equally induce AD [16-18].

Due to the vasoconstriction below the level of injury, systolic blood pressure can acutely increase up to 300mmHg and diastolic blood pressure up to 200mmHg in severe cases. A rise in systolic blood pressure higher than 20mmHg is considered consistent with AD. Since resting blood pressure in individuals with high cervical injury is frequently 20mmHg lower than non-injured individuals, a sudden rise compatible with this life-threatening condition can easily be missed by the clinician [19-23].

Besides hypertension, AD can be characterized by the presence of headaches, profuse sweating and flushing above the level of the lesion, bradycardia, apprehension, anxiety and even cognitive impairment. Surprisingly, AD may be silent or have minimal symptoms such as sweating. Additional symptoms, like nasal congestion and signs such as visual field defects and penile erection or cold peripheries, may also be present [24,25].

Interestingly, such nervous deregulation constitutes a double-edged sword in terms of athletic performance. It can have ergogenic effects with the potential death risk to the athlete [26].

Despite being potentially dangerous, many researchers believe that AD is frequently the result of an intentional induction by elite athletes with SCI to enhance their performances, a technique known as "boosting". Evidence is available for the fact that boosted athletes show better performances (6.9-9.7% of improvement) than non-boosted athletes [26].

AD generally induces bradycardia at rest, being tachycardia also considered a frequent finding in this situation. Under exercise conditions, tachycardia is the most frequent response, probably due to vagal withdrawal and unopposed sympathetic stimulation. This new "autonomic doping" can be induced in athletes through bladder distention, prolonged sitting, a broken toe or even sitting on the scrotum. Questionnaires have concluded that AD is considered by athletes to be more useful in long-distance events [26].

The International Paralympic Committee (IPC) banned the practice of "boosting" in 1994. Until recently, the rule was to submit the athletes to a systolic pressure measurement, with a threshold of 180mmHg just prior to the competition, and another measurement minutes apart confirming the high value [26-28]. None athlete has been tested positive to our best knowledge so far. In May 2016 IPC has tightened the rules imposing a lower threshold of 160mmHg to decrease the probability of existing athletes competing with AD [28].

The management of AD relies on the identification of its cause with immediate withdrawal of trigger factors. Time is precious during detection of primary etiology. If the trigger factor is not detected, the pharmacological treatment plays a major role. Valuable pharmacological options include: nifedipine, nitrates - contra-indicated in patients on 5-phosphodiesterase inhibitors (PDE5) inhibitors, phenoxybenzamine, captopril and magnesium sulfate [16,24,29,30]. Dysreflexic episodes are generally stopped with the simple removal of stimulus without the need of medication [26]. If untreated, AD may have catastrophic consequences: intracranial and retinal hemorrhage, seizures, cardiac ischemia, arrhythmias, and ultimately death [24, 26].

Thermoregulatory abnormal responses

SCI patients have to deal with thermoregulatory dysfunctional responses. Undeniable evidence indicates that sweating and vasomotor responses below the level of injury are disrupted. As expected, the higher the level of injury, the lower the area of skin with functional evacuating heat properties. Therefore, tetraplegic individuals with complete lesions are much more affected in both cool and warm environments during exercise protocols [31]. In the literature, there is unequivocal evidence that SCIs predispose to hyperthermia and even hypothermia, being the latter much less studied as a clinical finding [32,33].

Urinary tract infection

UTI represents the most common infection among SCI patients with a higher prevalence in comparison to non-injured individuals. The prevalence of asymptomatic bacteriuria is over 50% in these patients and approaches 100% when indwelling catheters are used. The increased risk is thought to be inextricably intertwined with impaired storage and voiding function [34,35]. In patients with SCI, UTI is defined by the presence of both physical symptoms and high amounts of bacteriuria [36]. The most common isolated bacteria is *E. coli* [37].

Symptoms related to an UTI in an athlete with SCI consist of acute or worsening fever, rigors, altered mental state, lethargy, acute haematuria, dysuria, flank or pelvic discomfort, development of or increase of urinary incontinence, increased frequency of voiding, increased spasticity, AD and sense of unease [38].

A recent Australian consensus about UTI in athletes with SCI, states that routine dipstick testing should not be used and asymptomatic bacteriuria should not be treated. When athletes present symptoms compatible with ITU, urine should be analyzed by microscopy, and submitted to culture and sensitivity assays.

Indwelling catheters should be changed prior to the collection of urine and empirical therapy with antibiotics can be initiated [38,39]. The recommended duration of antibiotic treatment in patients with SCI has not been established [36].

The use of antibiotic prophylaxis is not recommended, except for the cases of recurrent UTIs and always with medical supervision [34,35]. Educational programs can help mitigate the risk of UTI. Different sources of information can be delivered to injured individuals such as written material and knowledge tests with the collaboration of nurses and physicians [40].

A tendency towards overtreatment with antibiotics is an undeniable fact. The indiscriminate use of these therapeutic agents can lead to multidrug-resistant bacteria. In this context, previous antibiotic use and hospitalization were found to be risk factors for the development of multi-resistant organisms [35,41,42].

Pressure ulcers

Pressure ulcers are one of the most frequent problems faced by SCI Patients. The lifetime prevalence of this disabling problem in these individuals is near 85%, mostly in ischial and sacral regions. These represent strong limitations to daily activities and are responsible for an important reduction in self-reported quality of life. They also increase health care visits with all the costs implied to the health care system [43]. A cross-sectional observational study demonstrated that factors as male gender, a long time passed since SCI, a lower level of education, absence of an intimate partner, tetraplegia and older age were positively associated with the risk of development of pressure ulcers, in patients with more than 11 years of age [44]. Patients with metabolic syndrome, smoking habits and excessive drinking are also at higher risk [45].

Preventive measures to avoid the development of pressure ulcers are, thus, essential. The first step is to assess risk factors. In the past, devices to modify interface pressure distribution made their way as the main agents of prevention, such as mattresses and pressure-relieving cushions. Further insights on the etiology of pressure ulcers as well as new technological achievements will expectably reduce the incidence of this frequent and recurrent complication [46].

Bone loss

Another complication of SCI is marked bone loss which evidences a quick onset after acute injury. The most affected bones are the metaphyses of proximal tibia and distal femur [3]. The mean post-injury time till the first fracture is 9-years [4]. Thus, athletes with SCI might have an increased risk of fracture even with minor trauma [47].

A 12-month follow-up study concluded that bone loss can be predicted shortly after acute injury, through the evaluation of mineral bone density at lumbar spine and femur [48]. The impact of sympathetic denervation in bone tissues was already demonstrated in animal models. Altogether, these evidences support that three pathways responsible for conducting to bone mineral loss might be the consequence of sympathetic dysfunction: one that leads to reduced bone deposition and mineralization, other triggering increased bone resorption, and the last one impairing bone metabolism through vascular deregulation [3]. Another additional mechanism accounting for the loss of normal bone structure consists in the loss of mechanical loading forced by paralysis. The increased expression of sclerostin in osteocytes might be responsible for this, being actually considered a key mediator in the pathogenesis of osteoporosis in SCI patients [4].

Vitamin D insufficiency is related to increased bone turnover and bone loss as well as other non-skeletal diseases. The serum concentration of 25-hydroxyvitamin D (25(OH)D) constitutes a robust and reliable biomarker, considered to be the best determinant of vitamin D status [49]. Mineralization defects start to emerge in patients with 25(OH)D serum levels below 75nmol/L [50].

In addition, high prevalence of insufficiency and deficiency of vitamin D amongst patients with SCI has been encountered. Several factors as limited sun exposure, low diet vitamin D intake and the use of some medications, especially those metabolized by the P450 cytochrome CYP3A4, may contribute to vitamin D insufficiency. Although there are still no specific recommendations available for SCI patients regarding this concern, SCI individuals should have routine vitamin D screening tests to check their vitamin D status. Supplementation with daily or weekly regimens is recommended in vitamin D deficient individuals, and dose adjustments may be required [51].

Entrapment nerve syndromes

Wheelchair athletes are prone to nerve entrapment syndromes. Even non-athletes that rely on wheelchair show upper limb nerve injuries in as high as 73% of the cases. The two most common syndromes concerning wheelchair athletes are the entrapment of median nerve at the wrist and ulnar nerve entrapment at the wrist and elbow. It is hypothesized that these prevalent entrapments result from repetitive trauma and overpressure. Interestingly, training athletes do not evidence higher prevalence of median neuropathy. In this particular population, such prevalence might even be lower, despite the hours of training pushing miles. In one study, the duration of disability was significantly related to the dysfunction of median nerve [52,53].

Shoulder overuse

The overreliance on the upper limbs of wheelchair users justifies the higher prevalence of shoulder pain and injuries. The movements and forces applied on the shoulder, required to propel the wheelchair, culminate in the lesion of tendons with disabling pain [54-56]. A study involving 52 wheelchair users, half of them athletes, concluded that the most prevalent pathologies are bicipital tendinitis and rotator cuff impingement. The same authors demonstrated that participation in sports activities neither reduces nor increases the risk of shoulder pain [57].

Sports as a Key Partner

A suitable exercise protocol can be the key issue, allowing disabled individuals to reintegrate the society, lessening their feeling of dependence. It is crucial to understand that SCI individuals are usually physically deconditioned and acute exercise responses vary with the level as well as completeness of the lesion. For example, individuals with complete lesions above the fourth thoracic level bear a severely impaired cardiac acceleration with maximum rates below 130beats/min. Furthermore, the decreased venous return might result in cardiac muscle atrophy in tetraplegic individuals. It was demonstrated that an exaggerated heart rate response appears with exercise in paraplegics, as well as higher levels of VO_2 requirements, in comparison with non injured patients. Cardiovascular diseases can easily emerge in low conditioned persons. In fact, obesity and diabetes are four times more common in SCI patients comparing to the normal population. Bone density, joint contractures and thermal dysregulation are important limitations as well. The importance of

medical advice before engaging into an exercise program cannot be overemphasized [1,58,59]. Provision of written information to any injured patient willing to engage in regular exercise activities is just as important. The SCI Action Canada website puts together a substantial amount of relevant knowledge from the literature and offers simple and reliable information in 13 different languages, including activity guidelines in a comprehensible appealing manner for patients, constituting an important key source of information [60].

Exercise Management and Prescription in Patients with SCI

Despite the fact that SCI patients are visibly limited in the choice of a sport modality, some options are still available. To improve cardiovascular health, the following activities are recommended: wheeling, arm cycle, recumbent stepper, aquatics, cycling and circuit training. A minimum of 2 days per week of 20-30 minutes of moderate to vigorous intensity are strongly recommended. As far as muscle strength and endurance are concerned, the recommendations do not vary very considerably from those targeted to the population in general. Muscle strength and endurance can be achieved through free weights, elastic resistance bands and weight machines [1,58]. A review of 13 studies evaluating cardiorespiratory endurance training concluded that SCI patients may increase their VO_2 max and physical work capacity in 20% and 40% respectively, after 4 to 20 weeks of training [61]. Few is known about resistance but it seems logical and justified to incorporate resistance training in SCI patients. In addition, flexibility and range of motion should not be underestimated and must be well addressed too. Functional electrical stimulation is another strategy to force muscle contractions that can be employed without pain. It can be used to increase muscle mass and prevent contractures. Financing issues, expenditure of time and complexity of the equipment have prevented the progressive employment of this approach [58,61,62].

The practice of exercise by SCI patients does not occur without risks. The above-mentioned AD is probably one of the most threatening ones. Every exercise must be immediately interrupted in such a context. The deficit of cutaneous sensation may be amenable to skin breakdown without symptoms. Blunted heart rate response is another problem that may impair vigorous activities. Therefore, being able to maintain a light conversation ("talk test") while exercising might be a good indicator of exercise intensity. Decreased or absent muscular contraction on the lower limbs can be responsible for hypotension after exercising. Therefore, vascular flow optimization strategies such as warm up and cooldown should be encouraged [58]. To counteract this and to improve athletic performances, some wheelchair athletes frequently strap their legs, in an attempt to increase lower body positive pressure. Such practice is supposed to help in the redistribution of blood flow during exercise. There is not enough evidence to support this, but it may be beneficial by a facilitation of venous return to the heart [63].

The difficulties faced by disabled athletes cannot be overemphasized. However, a remarkable increase in the number of athletes taking part in Paralympic Games has been reported. In the Rome 1960 Paralympic Games, 400 athletes competed in eight different sports, being all of them suitable for SCI athletes. The recently Rio 2016 Paralympic Games recorded a participation of 4316 athletes, divided in 22 different modalities [28].

Conclusions

Great efforts have been made with great success in the last decades to protect athletes from SCI. However, sport-related activities still provoke too many injuries. Further measures can still potentially reduce the number of injured individuals, namely through regulation changes and educational strategies.

SCI patients have specific particularities that must be known and considered by all involved healthcare-professionals. The patients themselves must also be informed about the complications stemming from a SCI. AD is a potential life-threatening situation, at times intentionally induced by athletes due to its potential ergogenic effects. Such practice, considered as doping for more than two decades now, must be strongly discouraged as it can culminate in death.

Sports are beneficial for a stronger personal fulfillment, wellbeing and the integration of patients with SCI. They are considered safe in people with SCI, provided that there is proper clinic monitoring and surveillance. Furthermore, the improvement of physical fitness reduces dependency and costs to the healthcare system.

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