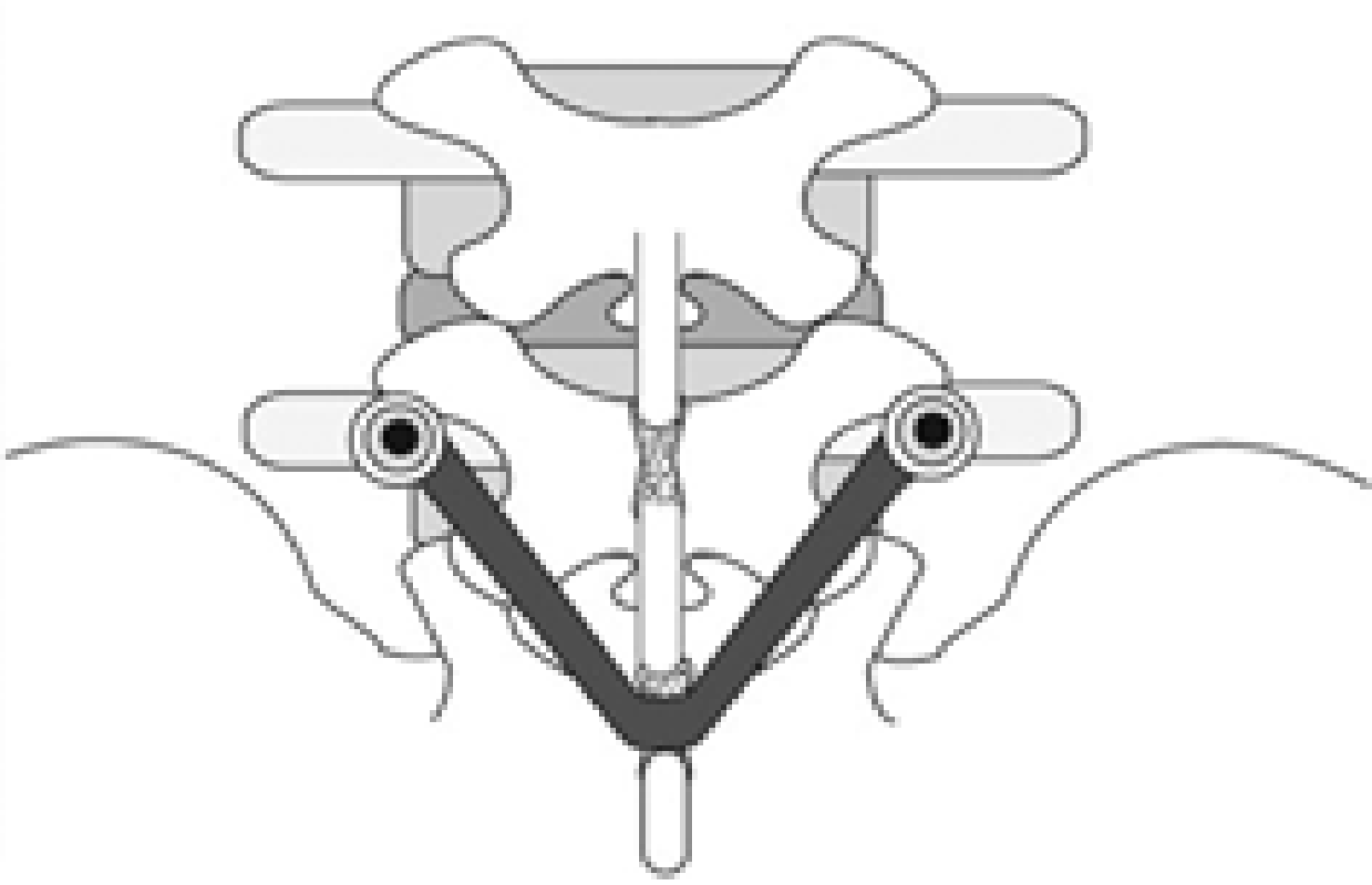

Introduction

Sports-related neurosurgical injuries

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Neurosurgeons have long had a vested interest in traumatic injuries to the brain and spine, and have more recently taken a leadership role in the prevention of mild and severe traumatic injuries. Trauma remains the leading cause of death in children in the US, and severe traumatic injuries remain a leading cause of morbidity, lost productivity, and death among otherwise healthy adults. Many of these injuries are suffered during sports and other recreational activities.

During the past few years, sports-related head and spine traumas have received significantly more attention in the lay press, especially the impact of concussions and chronic traumatic encephalopathy from repetitive brain injury. As neurosurgeons are among the few specialists who care for patients with the full spectrum of nervous system injuries, from mild to severe, it is essential that we continue to shape the field in research, clinical practice, and public policy discussions. Several excellent articles in this volume address these timely topics, including the range of injuries in soccer, impact of chronic repetitive head injury, return to play after concussion, decompressive surgery for severe head injury, all-terrain vehicles, and sports-related spine and peripheral nerve injuries. We hope you will find this issue of *Neurosurgical Focus* enlightening and enjoyable. *(DOI: 10.3171/2011.9.FOCUS11258)*

Disclosure

Dr. Maroon owns stock in ImPACT Applications, Inc.

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Mechanisms and consequences of head injuries in soccer: a study of 451 patients

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Object. The goal of this study was to evaluate the incidence and mechanisms of head injury during soccer games and to describe the results after spontaneous resolution of symptoms or after treatment.

Methods. In a retrospective study from 2005, records on 451 players from the German Soccer Association who had suffered various injuries were collected. The study used a questionnaire in which the player described the accident and the playing situation as well as the clinical course after trauma. This questionnaire also included information about the physical symptoms of the players and the length of their rehabilitation. Two groups were formed: one with head injuries (case group), and the other with injuries of other body parts (control group).

Results. Of the injuries reported, 108 (23.9%) were related to the head, 114 (25.3%) to the knee, 58 (13%) to the ankle, 56 (12%) to the calf, and 30 (7%) to the shoulder. The areas of the head most frequently involved were the facial and occipital regions. In the head injury group, the head duel was the most common playing action to lead to trauma. In those cases, the body part that hit the injured player was the elbow, arm, or head of the opponent. The most common playing situation was combat in the penalty area. The median hospitalization time after the trauma was 2 days for the case group and 5 days for the control group. The rehabilitation time for the case group was also shorter (median 6.5 days) than for the control group (median 30 days).

Conclusions. Trivial head injuries in soccer can have a long and complicated course. Nevertheless, the temporary disability is shorter in most cases than for players with injuries to other parts of the body. Modifying the rules of play would be necessary to reduce the incidence of head trauma. *(DOI: 10.3171/2011.10.FOCUS11184)*

Key Words • **head injury** • **trauma** • **soccer** • **mechanism of injury** • **outcome** • **disability**

Soccer is the most popular sport in the world, with approximately 200,000 professional and 240 million amateur players.[23] In the last 16 years, a variety of studies have reported on injuries to the head and other parts of the body. Head injuries can be the result of the contact of one head with another head, with the upper extremity of the opponent (usually the elbow), with the ground, with the ball, with the foot or knee, and (rarely) with goalposts.[13] Head injuries were reported to amount to 4%–22% of all injuries in soccer, although the severity of all the injuries and the mechanisms involved have not been well documented.[24,37,39]

In 1991, Tysvaer and Løchen[38] postulated that "heading" the ball could lead to chronic brain injuries such as those noticed in boxers.[19] Since the publication of these results, several cross-sectional studies[4,21,27–30,36] have indicated that soccer can cause measurable sustained brain impairment, and this has raised significant concerns about the effects of repetitive heading in soccer.

The frequency of soccer injuries overall is estimated to be approximately 10–35 injuries per 1000 playing hours.[14] The majority of injuries occurred at the lower extremities, mainly in the knees and ankles, although, as Dvorak and Junge[14] mentioned, the number of head injuries was probably underestimated. This is due to the fact that the player is afraid to mention his or her symptoms to a trainer for fear of being prevented from returning to play.[18] In an anonymous study done by Delaney et al.,[11] more than 46% of university soccer players experienced a concussion in just one fall season, and almost two-thirds of the same group experienced a concussion over the 12-month period while playing soccer.[12] Serious head injuries are well documented, whereas mild head injuries are often not examined. Usually only players who had concussions with severe symptoms presented for evaluation and diagnosis in the emergency department.

Most of the head injuries are skull fractures or an internal head injury like intracranial hemorrhage, epidural or subdural hematoma, or cerebral contusion.[11] Delaney et al.[13] reported that the side of the head seems to be the most vulnerable area for concussions, because most concussive blows occur to the side or temporal part of the head. These results differed from our study.

Abbreviation used in this paper: ARAG = Allgemeine Rechtschutz Versicherungsgesellschaft.

The acute injuries occur in a split second, and it is difficult for medical staff to provide exact information about the injury mechanisms to avoid their consequences. Therefore, based on a series of cross-sectional studies in active and older retired soccer players of the Hessian soccer association (Hessischer Fußball Verband), and using a standardized questionnaire, we tried to describe the most common mechanisms and consequences of head injuries in soccer in a study of 451 amateur players.

Methods

Patient Population

As a source for our retrospective study, we used the claim register of the ARAG, the sport insurance provider for all athletes of the Hessian sports association (Landessportbund Hessen). The claim register of the ARAG includes all registered injuries that have been treated by medical staff. We included all injuries from the previous year, and enrolled 841 people in the study. After the data interpretation and statistical evaluation, we ended up including 451 players in the study.

Our study was retrospective and assisted by a standardized questionnaire. We used coded numbers and anonymous data. We excluded 390 players who, although they matched the case and control groups, did not provide complete information; we included only players who returned completely filled and well-documented questionnaires. We then designated 2 groups. The case group included all athletes who got a head injury during a match or while training (108 players). The control group included all athletes who got injured on other parts of the body during a match or while training (343 players). We did not document overlaps between injury groups.

Questionnaire Used

The questionnaire included 2 parts. In the first part, we collected all the pieces of information on the mechanisms and consequences of the injuries as well as the matching of data and the participant's capacity to remember the injuries. This part of the questionnaire was based on a validated questionnaire that had been established by Bochum University for the study of injuries in soccer, and we modified it with information on training characteristics.[16]

The second part of the questionnaire was for information on the symptoms and type of head injury. Importantly, this part also included the specification of time of injury, the type of symptoms as well as their intensity, and the location of the head injury. Almost all of the questions in the first part of the validated questionnaire were closed ended, and the questions in the matching part, in which we interviewed the participants of the study about biological information as well as about their playing level, were open ended. We excluded all enrolled participants of the study who got injured outside the playing area, or who submitted incomplete data on their questionnaires.

Statistical Methods

For analyses dealing with the case series of injured players, t-tests of association were used to compare proportions in the tables. The Fisher exact test was used when 80% of the expected counts were < 5. Statistical analyses were conducted using SPSS version 12.0 software. The level of significance was set a priori at $p < 0.05$ for t-tests of association.

Results

The case group included 108 athletes aged 6–60 years (median 23 years); the largest part of this group included players between 11 and 25 years of age. This group included 104 male and 4 female patients. The control group included 343 players (323 male and 20 female patients) who had suffered an injury to an extremity or the torso. The age of the players in this group was higher than in the case group; the median age in the control group was 24 years. Consequently, the players in the case group were significantly younger than those in the control group at the time of injury ($p = 0.0204$). The sex of the injured athletes was not significant ($p = 0.280$).

Allocation of Head Injuries

In the case group, 57.4% of all injuries (62 of 108) occurred in the facial area. The zygomatic and the orbital regions were the main focus of facial injuries (33 [53.2%] of 62). The nasal region was the second most frequently injured area of the face (21 [33.9%] of 62). In the neurocranium, the occipital region was the most frequently injured head area (27 [58.7%] of 46; 25% of all head injuries), followed by the parietal, frontal, and temporal regions (Fig. 1).

Symptoms of Head Injuries

The typical symptoms of head injuries were headache (91.7%), vertigo (72.1%), amnesia (51.3%), sleep disorders (49.5%), loss of consciousness (45.9%), nausea (40.5%), and poor concentration (29.1%). The highest intensity occurred in the cases of head and neck pain. The longest duration of symptoms occurred in the cases of sleep disorder (7.1 days) and vertigo (6.3 days).

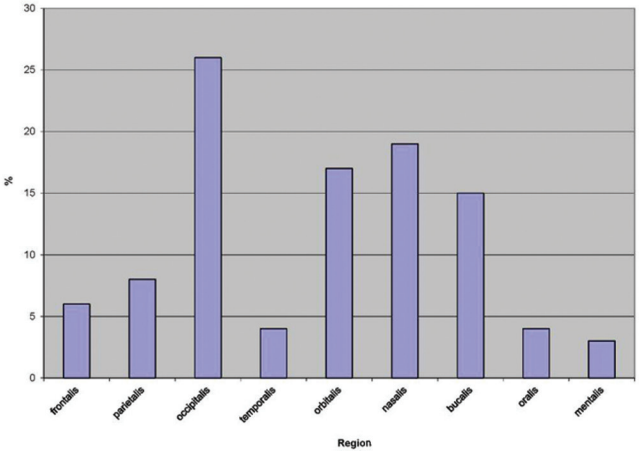

Fig. 1. Bar graph showing head injuries, categorized by the injured part of the head.

Injuries in the Case Group

In the case group, 46 (42.6%) of 108 athletes suffered from collateral injuries. The most common collateral injuries were fractures (35 [76.1%] of 46), mainly of facial bones like the os nasale (17 [37%] of 46) and the os zygomaticum (7 [15.2%] of 46). A Le Fort fracture also occurred occasionally (3 cases). Other injuries were lacerations (8) and contusions (6), which were mostly localized in the facial region (64.3%). A cerebral infarction (relating to the cerebellum and middle cerebral artery) occurred twice as a consequence of a head injury.

Allocation of Injuries in the Control Group

In the control group, the most injured parts of the body were at the lower extremities (241 [70.3%] of 343), with the upper extremities being injured in 88 (25.7%) of 343 cases. A torso injury only occurred in 14 (4%) of 343 cases.

Injuries in the Control Group

The injuries of the lower extremities were mainly at the knee (114 [47.3%] of 241) or localized ruptures of ligaments (88 [36.5%] of 241). The most common ankle joint injuries were bone fractures (31 [12.9%] of 241) or ruptures of ligaments (25 [10.4%] of 241). The most common injuries at the shin were fractures (39 [16.2%] of 241) and ruptures of the Achilles tendon (12 [5%] of 241). The injuries of the upper extremities included distal fractures (42 [47.7%] of 88) and shoulder fractures (19 [21.6%] of 88). Other players suffered lacerations or contusions on their upper extremities.

Injuries Overall

On the whole, the right side of the body was more frequently injured than the left side. An investigation of the results of all injuries showed that the medium-heavy injuries occurred more often in the case group, whereas the heavy injuries occurred more often in the control group (Table 1).

As shown in Table 2, according to the system of Tönnis and Loew,[35] the largest portion of the players of the case group suffered from Level I severity head injuries (48.1%). Loss of consciousness and amnesia occurred in 56 (51.9%) of 108 players in the case group.

Seventy-four (68.5%) of the athletes in the case group and 274 (79.9%) of those in the control group received in-patient care. In the case group, the therapy was performed conservatively (nonsurgically) for 67 (62%) of 108 injuries, but only for 67 (19.5%), a much smaller percentage, of 343 in the control group. In the control group, 276 (80.5%) of the 343 players were treated surgically. Conservative ($p < 0.001$) and outpatient ($p = 0.0279$) treatments were done significantly more often in players in the case group than in those in the control group.

A follow-up treatment was done for 73 (67.59%) of the 108 players in the case group, and for 321 (93.6%) of the 343 players in the control group. Furthermore, subsequent rehabilitation was necessary for 275 (80.2%) of the control group but for only 4 (3.7%) of the case group. In the case group, the occurrence of follow-up treatment ($p = 0.000002$) and rehabilitation ($p < 0.001$) was significantly lower than for the control group.

The comparison of these 2 groups showed no significant differences between the players in height or weight, but the Body Mass Index of the players in the case group was significantly lower than that of the players in the control group ($p = 0.0199$).

The amount of experience playing or time training were not significantly different between groups ($p = 0.413$). However, the number of competitions was significantly different and was decidedly higher in the case group ($p = 0.0377$; see Table 3). The investigation of both groups showed no significant differences in the amount of time devoted to recovery due to a rest from training or from competition (Table 4).

As shown in Table 5, we observed that injuries for the case group occurred more often during a competition, at 91 (84.3%) of 108, than did injuries for the control group, at 239 (69.7%) of 343. The differences are significant ($p = 0.0008$). Our retrospective investigation showed that midfielders suffered an injury more commonly than other players. The results were not significant ($p = 0.4779$).

The investigation of the place of injury showed that head injuries occurred significantly more often in the central part of the field than on the periphery ($p = 0.0093$). Furthermore, head injuries took place significantly more often in the goal and penalty areas than at midfield ($p = 0.0214$). Defensive or offensive halves of the field showed no significant differences ($p = 0.6977$). Almost all inju-

TABLE 1: Outcome of soccer-related injuries in 108 patients in the case (head injury) and 343 in the control (other injuries) groups*

Injury Grade	Case Group (%)	Control Group (%)
marginal: disabled 1–3 days	NA	20 of 343 (5.8)
low: disabled 4–7 days	9 of 108 (8.4)	33 of 343 (9.6)
medium-heavy: disabled 7–31 days	52 of 108 (48.1)	92 of 343 (26.8)
heavy: disabled >31 days	47 of 108 (43.5)	198 of 343 (57.7)

* NA = not applicable.

TABLE 2: Severity of head injuries in the case group*

Level of Severity	Loss of Consciousness	Amnesia	Portion of Players (%)
I	0 min	<30 min	52 of 108 (48.1)
II	<5 min	>30 min	47 of 108 (43.5)
III	>5 min	>24 hrs	9 of 108 (8.4)

* According to the system of Tönnis and Loew.

TABLE 3: Practical experience in case and control groups

Experience	Case Group	Control Group
median time spent training (min/wk)	227.0	235.1
median no. of competitions per season	24.4	10.9

TABLE 4: Injury recovery period in case and control groups

Recovery Period	Case Group	Control Group
median rest from training	4.5 wks	4.1 wks
median rest from competition	8.6 wks	8.9 wks

ries of players in the control group occurred in their own half of the playing area.

Significantly, the occurrence of head injuries almost always involved contact with another player, at 93.5% ($p = 0.0001$; see Table 6). Head injuries occurred more often in striker positions than in defensive positions in the case group, but not in the control group. In the majority of cases, head injuries occurred during ball duels (80.6%). This result was highly significant ($p = 0.0179$). The second mechanism of head injuries was ball shots on the head without an external actor (see Table 7).

In the control group, most of the noncontact injuries were caused by disturbances in the running process. Twisting (36.8%), stumbling (23.8%), and rotation while running (22.8%) were the most common mechanisms of injury in this group.

The development of the injuries is different between the groups. In the case group, they were mostly due to an external actor, but in the control group they were almost equally caused by the players themselves and by other players (30.0% and 31.5%, respectively) (see Table 8).

Additionally, excessive exertion of the players led to injuries for 66.7% of the case group and for 68.5% of the control group. Another reason for the injuries was breaking the rules, for 33.3% of the case group and for 31.5% of the control group. Our investigation of the kind of playing field showed that most injuries occurred on artificial turf, for 65 (60.2%) of 108 patients in the case group and for 256 (74.6%) of 343 patients in the control group.

Discussion

Soccer has not always been perceived to be a high-risk sport for craniofacial injuries. However, in recent years studies have shown that this sports discipline is also a high-risk sport in this regard. Current investigations in Europe have shown that soccer is responsible for approximately 50% of all sport-related injuries.[9] The incidence of head and body injuries is influenced by the popularity of soccer in each country.

TABLE 5: Setting of injury and position of injured player in case and control groups

Factor	Case Group (%)	Control Group (%)
setting of injury		
competition	91 of 108 (84.3)	239 of 343 (69.7)
training	17 of 108 (15.8)	104 of 343 (30.3)
player position		
midfielder	40 of 108 (37.0)	151 of 343 (44.0)
striker	31 of 108 (28.7)	67 of 343 (19.5)
defensive position	20 of 108 (18.5)	93 of 343 (27.1)
goalkeeper	17 of 108 (15.8)	32 of 343 (9.4)

TABLE 6: Occurrence of contact injury and position of other players involved in contact

Factor	Case Group (%)	Control Group (%)
contact injury		
yes	101 of 108 (93.5)	211 of 343 (61.5)
no	7 of 108 (6.5)	132 of 343 (38.5)
player position		
midfielder	40 of 101 (39.6)	90 of 211 (42.6)
striker	28 of 101 (27.7)	36 of 211 (17.1)
defensive position	25 of 101 (24.8)	65 of 211 (30.8)
goalkeeper	8 of 101 (7.9)	20 of 211 (9.5)

Similarly to the results of Andersen et al.,[2] in our investigation the craniofacial region is, at 57%, the most injured part of the head. Furthermore, these authors investigated the mechanisms of head injuries and found, as did our investigation, that 73% of facial injuries were caused by contact with upper extremities. Head-on-head injury in the Andersen et al. investigation, on the other hand, accounted for only 35% of all head injuries, whereas in our investigation it accounted for 41.4%, primarily at the side of the head. Facial injury occurred in the investigation of Andersen et al. in head duels in 31% of cases and in 33.3% of cases in our investigation. Chomiak et al.[10] found that 74.2% of the players suffered a lower-extremity injury during play, whereas it was 70.3% in our investigation. We found that upper-extremity injuries occurred during play in 25.7% of our control group, whereas Chomiak et al. found the same to be true for only 14.4%.

Boden et al.[8] investigated head injuries in 29 players, whereas our group involved 108 players. Boden and colleagues described mild head injuries in 72% of all investigated players, whereas in our investigation 48.1% of players suffered mild, 43.5% had moderate, and 8.4% had severe head injuries. Because the investigation of Boden et al. only involved 29 players and we investigated 108 players, we postulated that the frequency of occurrence of the moderate and severe head injuries in a bigger group is clearly higher. In our investigation, we found that headaches were the main symptom of head injuries in 91.7% of cases, similar to Boden's findings at 97%. Other symptoms were amnesia, poor concentration, vertigo, nausea, and personality change, which were comparable to the study results of Boden et al. (see Table 9).

TABLE 7: Mechanism of head injury

Type of Play	Case Group (%)	Control Group (%)
ball duel	87 of 108 (80.6)	164 of 343 (47.8)
head on head	36 of 87 (41.4)	NA
arm on head	23 of 87 (26.4)	NA
leg on head	14 of 87 (16.1)	NA
head on torso	14 of 87 (16.1)	NA
ball contact w/o ball duel	15 of 108 (13.9)	96 of 343 (27.9)
no ball duel or ball contact	2 of 108 (1.8)	83 of 343 (24.2)
assault	4 of 108 (3.7)	NA

TABLE 8: Cause of injury in the case and control groups

Cause	Case Group (%)	Control Group (%)
another player	57 of 108 (52.8)	108 of 343 (31.5)
own blame	16 of 108 (14.8)	103 of 343 (30.0)
shared blame	16 of 108 (14.8)	26 of 343 (7.6)
accident	14 of 108 (13.0)	54 of 343 (15.7)
location on playing field	5 of 108 (4.6)	52 of 343 (15.2)

TABLE 9: Symptoms of head injury in the present study compared with an earlier investigation in 29 patients

Symptoms of Head Injury	Boden et al.	Present Study
headache	97%	91.7%
amnesia	76%	51.3%
poor concentration	62%	29.1%
vertigo/nausea	52%	72.1%/40.5%
personality change	14%	6.3%

We found that most head injuries occurred among younger players, who suffered a head injury through contact with the ball. Furthermore, older players suffered these injuries through ball duels; similar results were published by Pickett et al.[34] The reason for the number of head injuries at the ages of 6–10 years is probably the fast development of condition, power, and speed at this age, while the coordination of muscles is not able to keep pace.[15] All these factors induce aggressive ways of playing and lead to head injuries among members of this young group.[26] Also, research of the literature shows results that confirm our finding that male players more commonly suffer head injuries.[8,10,26,34]

Age, sex, and play experience were internal factors of the mechanisms of head injuries of the soccer players, adding to the external factors of place, reason for playing, play position, and playing situation. Furthermore, the condition and kind of location have very often been discussed in the literature as an important factor in head injuries. Andersen et al.[2] and Boden et al.[8] postulated that wet artificial turf was most commonly the reason for head injuries. Also Chomiak et al.[10] reported that the type of playing surface was very important in the evaluation of head injuries. In our investigation, the injuries occurred on artificial turf for 60.2% (65 of 108) of the case group and for 74.6% (256 of 343) of the control group.

Our investigation of another external factor, the reason for playing, showed that the frequency of head injury increased during the match (84.2%) and not in training. Boden et al.[8] also found that 79% of all head injuries occurred during the match and not in training. Peterson et al.[33] published findings that almost all head injuries occurred during the match.

In our investigation, the playing position was not significant, which is consistent with previous investigations by Chomiak et al.,[10] Nielsen and Yde,[32] and Hawkins and Fuller.[20] The midfield players had the highest frequency of injury in our investigation (37%), similar to the results of Chomiak et al., at 32.9%. Berbig[5] published the finding that goalkeepers suffered from head injuries more frequently than other players. In our investigation, however, similar to the findings for Chomiak et al., we found that a goalkeeper had suffered head injury in only 15.8% of cases. Therefore, in our opinion, the player in the midfield position is more frequently injured than a defensive or striker player. The frequency of the injuries also depends on their location on the playing field. The high frequency of injury in the midfield and in the defending penalty area has already been published by Andersen et al.,[2] which is confirmed by our investigation, but these results are opposite to the findings of Chomiak et al.[10] and Ekstrand and Gillquist,[15] who postulated a higher frequency of injury on the offensive side. However, in our investigation, head injuries occurred most frequently in the penalty and goal areas, as in the study published by Kirkendall et al.[25] They pointed to ball duels and clashes between strikers and goalkeepers on that small area as a reason for the frequency of head injuries. Furthermore, in our study, the midfielders in the case group were injured on the head significantly more often than other players. Fuller et al.[17] actually found that injuries most often occurred in the offensive half of the playing field but, as found in our results, they postulated that the outside section was very safe.

In our study, 93.5% of head injuries occurred as contact injuries, whereas Nielsen and Yde[32] found that only 74% did. Boden et al.[8] and Pickett et al.[34] found contact head injuries in only 65% of cases. These results are self-explanatory because of the heterogeneity of the age groups and the size of the groups. Fouls are another reason for contact injuries. In our investigation, only 24.3% of contact injuries could be attributed to fouls. However, Chomiak et al.[10] and Peterson et al.[33] found that professional players reported that fouls were the reason for contact injuries (66.7% and 40%, respectively). This higher rate of fouls in the studies of these investigators was probably due to the players' mastery of technique. However, almost the same rate of fouls occurred in the case and control groups. Therefore, fouls were not a significant cause of head injuries.

Arnason et al.[3] reported that most injuries in soccer occurred through personal negligence, and that head injuries happened through contact with other players and through fouls in 52.7% of cases. In our study, duels were the reason and mechanism for 55.9% of all injuries. These mechanisms include an opponent treading on the player or player falls during duels. Bjordal et al.[7] found that 46% of injuries of the cruciate ligament occur through this mechanism. Nielsen and Yde[32] reported ankle joint and knee joint injuries as a consequence of being stepped on. In the case of other injuries, these most often appeared because of running, twisting, stumbling, or rotation of feet.

Only 75% of the injured players in the control group were taking part actively in play during the injury. On the other hand, in the case group, all injured players were taking part in play during the accident.

The most common mechanism of head injuries in our study was header duels, at 51.6%, which is similar to the results of Fuller et al.,[17] who reported that approximately 55% of head injuries occurred through this mechanism

among 248 professional soccer players. Andersen et al.[2] also stated that this mechanism was a cardinal mechanism of head injuries for 58% of professional Norwegian and Icelandic soccer players. In our study, head-on-head was the most common mechanism of injury in duels (41.4%). Upper extremity–to-head only led to injury in 26.4% of cases. Boden et al.[8] (28.0%), Fuller et al.[17] (33.0%), and Pickett et al.[34] (25.5%) also found that head-on-head in duels was the most common mechanism of head injury, not contact between the head and the upper extremities.

However, the main mechanism of all head injuries is the collision of 2 players (93.5%) and, in 51.6% of cases, header duels.

Fuller et al.[17] reported that the use of the upper extremities against the rules leads to significantly more head injuries than other actions during play. Boden et al.[8] published findings that 14% of all collisions and head injuries occurred through the use of upper extremities to the head. According to Andersen et al.,[2] 43% of head injuries were also caused by the upper extremities of the opponent, with 34% using the elbow and 7% using the hand or arm. Head-on-head contact caused 33% of head injuries, as reported in the same investigation. The results were obtained by video analysis of professional players.

Head-on-head contact is the second most common mechanism in the occurrence of head injuries. For only 6% of investigated players, a head injury happened because of the opponent.[34]

We observed in our study that almost the same number of patients in the 2 groups were treated in hospital, although the treatment was different. In the case group, the treatment was almost always conservative, whereas the athletes in the control group got surgical treatment in 80.5% of cases. This suggests a high frequency of fractures of extremities, which have to be treated surgically.

Our study investigated the necessity of rehabilitation and found, significantly, that only 3.7% of the case group players and 80.2% of the control group players needed rehabilitation. This suggests that head injuries need an acute treatment and that rehabilitation is only necessary sporadically. Additionally, the conservative treatment of head injuries allows shorter rehabilitation periods. Hospitalization duration differences were only marginal between the case group and the control group. This kind of investigation has not been published until now. However, the moderate and severe head injuries were common occurrences, as suspected, and deserve more attention in the future.

The recent trend in soccer to protect players from head injuries is to forbid heading, or at least to limit it until an age when young athletes can begin to master the techniques of heading and their skull anatomy is more mature.[24] The second trend is the use of headgear, which is permitted by FIFA (Fédération Internationale de Football Association). There are newly developed national standards for headgear.

Initially, in an experimental study conducted in 2003, Naunheim et al.[31] showed that the use of headgear could reduce impact when heading, and that headbands may play a role in decreasing impact for more forceful blows. In 2006, Al-Kashmiri and Delaney[1] published a report about head and neck injuries. They also postulated after their research that protective headgear may play a role in the prevention of concussions.

Jordan et al.[22] published their findings that males have 0.14 concussions/1000 athletes and females have 0.15 concussions/1000 athletes. Another similar study with a comparable population suggested higher rates: 0.6 concussions/1000 athletes among men and 0.4 concussions/1000 athletes among women.[8]

Some researchers believe that head injuries occur more commonly in certain areas of the playing field,[1] and the penalty area is thought to be a high-risk area, especially when players are competing for a cross-corner kick.[24] Near the midfield, concussions occur from head-to-head impact, especially when players are competing for air balls.[24]

Fuller et al.[17] reported that most of the injuries occurred when players converged on a free ball and not, as usually believed, when one player is in possession of the ball and is tackled by another player. Furthermore, they reported that female soccer players may be more at risk for concussions than their male counterparts. A different study by Delaney et al.[13] and a work by Biros[6] suggested that younger players may be more at risk for concussions because of their thinner skulls or larger heads.

Conclusions

Seemingly trivial soccer head injuries can have a long and complicated course. Nevertheless, the temporary disability is shorter in most patients than in the group of players with an injury in other parts of the body.

Disclosure

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

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References

1. Al-Kashmiri A, Delaney SJ: Head and neck injuries in football (soccer). **Trauma 8:**189–195, 2006
2. Andersen TE, Larsen Ø, Tenga A, Engebretsen L, Bahr R: Football incident analysis: a new video based method to describe injury mechanisms in professional football. **Br J Sports Med 37:**226–232, 2003
3. Arnason A, Gudmundsson A, Dahl HA, Jóhannsson E: Soccer injuries in Iceland. **Scand J Med Sci Sports 6:**40–45, 1996
4. Baroff GS: Is heading a soccer ball injurious to brain function? **J Head Trauma Rehabil 13:**45–52, 1998

5. Berbig R: Die Verletzungsgefährdung im Spitzenfußball aus der Sicht des Sporttraumatologen. **Schwei Ztschr Sportmed Sporttraumat 45:**127–130, 1997
6. Biros MH: Head trauma, in Rosen P, Barkin RM (eds): **Emergency Medicine: Concepts and Clinical Practice, ed 4.** St. Louis: Mosby Year Book, 1998, pp 416–447
7. Bjordal JM, Arnły F, Hannestad B, Strand T: Epidemiology of anterior cruciate ligament injuries in soccer. **Am J Sports Med 25:**341–345, 1997
8. Boden BP, Kirkendall DT, Garrett WE Jr: Concussion incidence in elite college soccer players. **Am J Sports Med 26:** 238–241, 1998
9. Bruzzone E, Cocito L, Pisani R: Intracranial delayed epidural hematoma in a soccer player. A case report. **Am J Sports Med 28:**901–903, 2000
10. Chomiak J, Junge A, Peterson L, Dvorak J: Severe injuries in football players. Influencing factors. **Am J Sports Med 28 (5 Suppl):**S58–S68, 2000
11. Delaney JS, Lacroix VJ, Gagne C, Antoniou J: Concussions among university football and soccer players: a pilot study. **Clin J Sport Med 11:**234–240, 2001
12. Delaney JS, Lacroix VJ, Leclerc S, Johnston KM: Concussions among university football and soccer players. **Clin J Sport Med 12:**331–338, 2002
13. Delaney JS, Puni V, Rouah F: Mechanisms of injury for concussions in university football, ice hockey, and soccer: a pilot study. **Clin J Sport Med 16:**162–165, 2006
14. Dvorak J, Junge A: Football injuries and physical symptoms. A review of the literature. **Am J Sports Med 28 (5 Suppl):** S3–S9, 2000
15. Ekstrand J, Gillquist J: The frequency of muscle tightness and injuries in soccer players. **Am J Sports Med 10:**75–78, 1982
16. Elbe AM, Wenhold F, Müller D: Zur Reliabilität und Validität der Achievement Motives Scale–Sport: Ein Instrument zur Bestimmung des sportspezifischen Leistungsmotivs. **Z Sportpsychol 12:**57–68, 2005
17. Fuller CW, Junge A, Dvorak J: A six year prospective study of the incidence and causes of head and neck injuries in international football. **Br J Sports Med 39 (Suppl 1):**i3–i9, 2005
18. Gerberich SG, Priest JD, Boen JR, Straub CP, Maxwell RE: Concussion incidences and severity in secondary school varsity football players. **Am J Public Health 73:**1370–1375, 1983
19. Gronwall D, Wrightson P: Cumulative effect of concussion. **Lancet 2:**995–997, 1975
20. Hawkins RD, Fuller CW: A prospective epidemiological study of injuries in four English professional football clubs. **Br J Sports Med 33:**196–203, 1999
21. Janda DH, Bir CA, Cheney AL: An evaluation of the cumulative concussive effect of soccer heading in the youth population. **Inj Control Saf Promot 9:**25–31, 2002
22. Jordan SE, Green GA, Galanty HL, Mandelbaum BR, Jabour BA: Acute and chronic brain injury in United States National Team soccer players. **Am J Sports Med 24:**205–210, 1996
23. Junge A, Rösch D, Peterson L, Graf-Baumann T, Dvorak J: Prevention of soccer injuries: a prospective intervention study in youth amateur players. **Am J Sports Med 30:**652–659, 2002
24. Kirkendall DT, Garrett WE Jr: Clinical perspectives regarding eccentric muscle injury. **Clin Orthop Relat Res (403 Suppl):**S81–S89, 2002
25. Kirkendall DT, Jordan SE, Garrett WE: Heading and head injuries in soccer. **Sports Med 31:**369–386, 2001
26. Leininger RE, Knox CL, Comstock RD: Epidemiology of 1.6 million pediatric soccer-related injuries presenting to US emergency departments from 1990 to 2003. **Am J Sports Med 35:**288–293, 2007
27. Matser EJ, Kessels AG, Lezak MD, Jordan BD, Troost J: Neuropsychological impairment in amateur soccer players. **JAMA 282:**971–973, 1999
28. Matser JT, Kessels AG, Jordan BD, Lezak MD, Troost J: Chronic traumatic brain injury in professional soccer players. **Neurology 51:**791–796, 1998
29. Matser JT, Kessels AG, Lezak MD, Troost J: A dose-response relation of headers and concussions with cognitive impairment in professional soccer players. **J Clin Exp Neuropsychol 23:**770–774, 2001
30. Naunheim RS, Bayly PV, Standeven J, Neubauer JS, Lewis LM, Genin GM: Linear and angular head accelerations during heading of a soccer ball. **Med Sci Sports Exerc 35:**1406–1412, 2003
31. Naunheim RS, Ryden A, Standeven J, Genin G, Lewis L, Thompson P, et al: Does soccer headgear attenuate the impact when heading a soccer ball? **Acad Emerg Med 10:**85–90, 2003
32. Nielsen AB, Yde J: Epidemiology and traumatology of injuries in soccer. **Am J Sports Med 17:**803–807, 1989
33. Peterson L, Junge A, Chomiak J, Graf-Baumann T, Dvorak J: Incidence of football injuries and complaints in different age groups and skill-level groups. **Am J Sports Med 28 (5 Suppl):** S51–S57, 2000
34. Pickett W, Streight S, Simpson K, Brison RJ: Head injuries in youth soccer players presenting to the emergency department. **Br J Sports Med 39:**226–231, 2005
35. Tönnis W, Loew F: Einteilung der gedeckten Hirnschädigungen. **Ärztl Prax 36:**13–14, 1953
36. Tucker AM: Common soccer injuries. Diagnosis, treatment and rehabilitation. **Sports Med 23:**21–32, 1997
37. Tysvaer AT: Head and neck injuries in soccer. Impact of minor trauma. **Sports Med 14:**200–213, 1992
38. Tysvaer AT, Løchen EA: Soccer injuries to the brain. A neuropsychologic study of former soccer players. **Am J Sports Med 19:**56–60, 1991
39. Withnall C, Shewchenko N, Gittens R, Dvorak J: Biomechanical investigation of head impacts in football. **Br J Sports Med 39 (Suppl 1):**i49–i57, 2005

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Sports-related chronic repetitive head trauma as a cause of pituitary dysfunction

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Traumatic brain injury (TBI) is recognized as a cause of hypopituitarism even after mild TBI. Although over the past decade, a growing body of research has detailed neuroendocrine changes induced by TBI, the mechanisms and risk factors responsible for this pituitary dysfunction are still unclear. Around the world, sports—especially combative sports—are very popular. However, sports are not generally considered as a cause of TBI in most epidemiological studies, and the link between sports-related head trauma and hypopituitarism has not been investigated until recently. Thus, there is a paucity of data regarding this important concern. Because of the large number of young sports participants with near-normal life expectancy, the implications of undiagnosed or untreated postconcussion pituitary dysfunction can be dramatic. Understanding the pathophysiological mechanisms and risk factors of hypopituitarism caused by sports injuries is thus an important issue that concerns both medical staff and sponsors of sports. The aim of this paper was to summarize the best evidence for understanding the pathophysiological mechanisms and to discuss the current data and recommendations on sports-related head trauma as a cause of hypopituitarism.
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Key Words • **concussion** • **hypopituitarism** • **sports-related concussion** • **traumatic brain injury**

In today's realm of competitive sports environments worldwide, a large number of athletes participate in a wide variety of amateur or professional sports. Closed head injury is an occupational hazard of many sports, such as boxing, kickboxing, ice hockey, football, and many others. Participants in these sports are at risk for concussion, which is considered a type of mild TBI.

During the past decade, numerous studies have shown that TBI is often responsible for pituitary dysfunction such as hypopituitarism,[2,29,55] which seems to be readily present even after mild TBI. Although sports are a well-known cause of concussion, trauma due to sports is generally not considered to be a cause of TBI in most epidemiological studies. Thus, the link between sports-related head trauma and pituitary dysfunction is not well understood, even now. Recent studies have demonstrated that sports-related repetitive head trauma might induce pituitary dysfunction, and in particular, isolated GH deficiency.[38,65,67]

Abbreviations used in this paper: ACTH = adrenocorticotropic hormone; AHA = antihypothalamus antibody; APA = antipituitary antibody; ApoE = apolipoprotein E; GH = growth hormone; ICA = internal carotid artery; IGF-I = insulin-like growth factor–I; TBI = traumatic brain injury.

The aim of this paper was to summarize the best evidence for understanding the pathophysiological mechanisms and to discuss current data and recommendations on sports-related repetitive head trauma as a cause of hypopituitarism.

Sports-Related Concussion

Concussion is considered a clinical syndrome characterized by immediate and transient posttraumatic impairment of neural function, such as alteration of consciousness or disturbance of vision or equilibrium, and other signs due to the involvement of brainstem.[5] Mild TBI (or concussion) may lead to postconcussion syndrome[48] (secondary symptoms), including additional cognitive problems, headaches, anxiety, fatigue, and psychosocial problems.

Concussion is a type of injury that can occur in any sports activity, and is the most frequent type of acute TBI in sports. It is most often associated with boxing, football, ice hockey, and martial arts.[36] The nature of the risk depends on the nature of the game and its specific activities. Individual variables might predispose an athlete to concussion, such as previous concussions, position played, and the sex of the athlete.[24,25]

Although sport is a well-known cause of TBI, it was only in the 1980s that the medical community started to direct research toward the risk of sports-related concussion. In the early 1980s, thanks to the work of Barth et al.[8] and Rimel et al.,[53] a discussion was started among members of the medical community regarding the description, classification, and management of sports-related concussion.[40]

Hypopituitarism and Mild TBI

Traumatic brain injury has been recognized for almost a century as a cause of neuroendocrine dysfunction.[19] However, it was thought to be a rare occurrence,[27] despite the results of autopsy studies that showed pituitary gland necrosis in up to one-third of patients who suffered fatal TBI.[10]

In the past decade, studies reported that 5.4%–69% of hypopituitarism was associated with TBI.[1,4,13,32,39,41,43,45,51,52,56,62] The severity of hormonal disturbance is generally related to the severity of TBI. However, mild TBI can contribute to pituitary dysfunction.[10] Bondanelli et al.[12] noticed hypopituitarism in 37.5% of patients with mild TBI versus 59.3% of patients with severe TBI. Pituitary dysfunction can be partial or complete. Gonadotropin and GH deficiencies appear to be the most common.[3,12]

Recent studies have reported that pituitary function can improve over time in a considerable number of patients. However, it may also worsen over the 1- and 3-year period after TBI.[4,64] Traumatic brain injury may also cause hypothalamic dysfunction and diabetes insipidus.[14,33,37]

Hypopituitarism and Sports

As mentioned earlier, sports-related concussion could be considered as a subgroup of mild TBI. Also, TBI is one of the most important public health problems,[70] and may be associated with pituitary secretion dysfunction, which may contribute to long-term physical, cognitive, and psychological disability.

Almost all of the studies regarding the relationship between TBI and sports published so far in the literature are based on neuropsychological or radiological assessment, and no neuroendocrine changes are investigated. There is a paucity of data regarding the association between sports and hypopituitarism. To our knowledge, only 3 studies and 1 case report investigated the link between pituitary dysfunction and sports-related concussion.[35,38,65,67] Three of these studies were conducted by the same medical team.[38,65,67] The studies only concern 3 sports—boxing,[38,67] kickboxing,[65] and soccer[35]—that are obviously associated with chronic mild TBI.

The first report of pituitary function in boxers was published in 2004 by Kelestimur et al.[38] In this preliminary study, the authors included 11 actively competing or retired male amateur boxers and investigated their GH status compared to a control group. A GH deficiency was found in 45% of these amateur boxers. The mean IGF-I levels in boxers (237 ± 23.3 ng/ml) were significantly lower than in the control group (367 ± 18.2 ng/ml). There was a significant negative correlation both between peak GH levels and boxing duration and between peak GH levels and number of bouts.

In 2007, Tanriverdi et al.[65] investigated the GH status of 22 amateur kickboxers compared with a control group. The serum IGF-I level was significantly lower in kickboxers (276.5 ± 25.9 ng/ml) than in the control group (346.9 ± 20.9 ng/ml). Of the 22 amateur kickboxers, 22.7% had a GH deficiency and 9.1% had an ACTH deficiency. There were negative correlations between serum IGF-I level and age, length of time the athlete has participated in sports, and number of bouts.

Ives et al.[35] reported a case of a 16-year-old high-level junior soccer player who had experienced 4 episodes of mild TBI at different time points over a 4-month period. The first 3 traumas were considered by the athlete to be minor, and thus were not reported to medical personnel. The fourth trauma was a medically diagnosed concussion. Over the next year, the patient presented with failure of physical growth, stagnation of soccer abilities and physical skills, and lower energy levels. After a full battery of endocrine tests, the diagnosis was compatible with hypopituitarism (GH, ACTH, and thyroid-stimulating hormone [TSH] deficiencies).

In 2008, Tanriverdi et al.[67] investigated the pituitary function in 61 retired or active amateur boxers. The findings were similar to those in previous studies, with 15% and 8% of GH and ACTH deficiency, respectively, among boxers. An interesting fact is the lower rate of hypopituitarism in active boxers (18%) than in retired boxers (47%).

All of these findings suggest that sports-related repetitive TBI has a cumulative effect on the development of pituitary dysfunction.

Pathophysiological Mechanisms of Hypopituitarism

Hypopituitarism was first described in 1914 by Simmonds.[57] It is defined as an inability of the pituitary gland to provide sufficient hormones adapted to the needs of the human body. The classic cause of hypopituitarism is pituitary tumor (in 61% of cases).[55]

Anatomical Vulnerability of the Pituitary Gland

The pituitary gland is located in the sella turcica in the skull base. It measures 8 × 10 mm and weighs less than 1 g. It is tethered to the hypothalamus by the pituitary stalk (infundibulum), which inserts on its superior surface. It is separated from the suprasellar cistern by the diaphragma sellae. The ICAs are the primary blood supply for the adenohypophysis and neurohypophysis. Blood reaches the anterior pituitary by means of long hypophysial portal vessels (branches of the ICA and anterior circle of Willis) and short hypophysial portal vessels (branches of the intracavernous ICA and the inferior hypophysial artery) via the pituitary stalk. The long portal vessels provide 70%–90% of its blood supply, whereas the short portal vessels provide less than 30%.

The confined location within the bony sella, the delicate infundibular hypothalamic structures, and the vulnerable blood supply of the pituitary gland may be responsible for the vulnerability of the pituitary to mechanical trauma.

The Most Common Theories

The pathophysiological mechanisms of hypopituitarism after TBI remain incompletely understood. However, several mechanisms have been evoked to explain pituitary dysfunction, such as hypoxic insult or direct mechanical injury to the hypothalamus, pituitary stalk, or pituitary gland; compression from hemorrhage, edema, or increased intracranial pressure; and vascular injury to the hypothalamus or the pituitary gland.

In the 1960s, Ceballos[17] and Kornblum and Fisher[42] provided descriptions of pituitary lesions after fatal head injury. In a total of 202 patients from these 2 reports, 26% of the specimens were normal, 59% had capsular hemorrhage, 31% posterior lobe hemorrhage, 22% anterior lobe necrosis, 17% stalk hemorrhage, and 3% stalk necrosis.

Ischemic injury of the pituitary gland, due to secondary insults from hypotension, hypoxia, anemia, and brain swelling, can occur and compromise the integrity of the gland. Necrosis processes seem to be due to the vulnerability of the long hypophysial portal vessels when they pass through the diaphragma sellae, where they are particularly vulnerable to mechanical compression from both brain and pituitary swelling and direct stalk injury.[21,42]

Direct injury of the pituitary gland, pituitary stalk,[31,47] and/or hypothalamus can be caused by the following mechanisms: rotational and shearing injuries of the brainstem and hypothalamic-pituitary axis, and fractures through the skull base and sella turcica.[11,21] This direct injury also can be reinforced by subsequent hemorrhage into the sella turcica or into the pituitary gland.[20,39]

Antibodies Theory

Goudie and Pinkerton[30] first suggested that autoimmunity could play a role in affecting the pituitary gland, by describing the first case of lymphocytic hypophysitis in 1962. Some studies have shown the presence of antibodies in patients with selective idiopathic hypopituitarism[22,23] and in patients with autoimmune endocrine diseases.[16,46] Also, animal studies have shown a possible role of autoimmunity involving the hypothalamic-pituitary region and triggered by head trauma.[54,59]

In a preliminary study, in 2008, Tanriverdi et al.[61] investigated the presence of APAs in 29 patients with TBI 3 years after head trauma, and in 60 age- and sex-matched normal controls. The APAs were detected in 44.8% of TBI patients but in none of the controls. Furthermore, the hypopituitarism ratio was significantly higher in APA-positive patients (46.2%) than in APA-negative patients (12.5%) ($p = 0.04$). There was also a significant positive correlation ($r = 0.74$) between high APA titer ratio and low peak GH response to the GH-releasing hormone plus GH-releasing peptide–6 test.

Subsequently, the same team[60] investigated the presence of APAs and AHAs in active or retired amateur boxers who were exposed to sports-related repetitive head trauma. The patient population contains the same participants as in one of their previous studies (61 active or retired boxers),[67] plus 60 normal controls of similar age and sex. In boxers, AHAs and APAs were found in 21.3% and 22.9% of cases, respectively. Antibodies were found in none of the control patients. The hypopituitarism ratio was significantly higher in AHA-positive boxers (46.2%) than in AHA-negative boxers (10.4%) ($p = 0.003$). However, in contrast to findings in the study on TBI and APAs,[61] there was no significant difference between APA-positive and APA-negative boxers with regard to hypopituitarism.

So, the nature and clinical significance of these antibodies are controversial. Autoimmunity seems to have an impact on the development of pituitary dysfunction, but the mechanisms of activation of hypothalamic-pituitary autoimmunity after TBI or sports-related injuries remain unclear. However, these antibodies are considered as markers of pituitary impairment when they are detected at a high titer (> 1:8). In healthy people, they are undetectable (< 1:8).[22] Large prospective studies are necessary.

Involvement of Genetic Polymorphisms

Genetic polymorphisms have been recognized as playing a role in CNS disorders.[68,69]

Apolipoprotein E is a key protein that plays an essential role in the repair of cell membrane and spread of neuritis following injury.[9,34,49] This protein is controlled by a gene with 3 primary alleles (ε2, ε3, and ε4). In several studies, ApoE-ε3 has been shown to increase the spread and branching of neuritis, whereas ApoE-ε4 was found to have the opposite effect.[9,49] Many studies have found a positive correlation between the presence of ApoE-ε4 and poor outcome after TBI.[18,26,28,44,58]

Apolipoprotein E is one of the most abundant proteins in the hypothalamic-pituitary region.[50] Tanriverdi et al.,[63] in a preliminary study, investigated the relationship between ApoE polymorphism and TBI-induced hypopituitarism. This study included 93 patients with TBI (61 with sports-related head trauma and 32 in road traffic accidents) and 27 healthy controls. The ratio of hypopituitarism after TBI was significantly lower in patients with ApoE-ε3/ε3 (17.7%) than in those without ApoE-ε3/ε3 (41.9%; $p = 0.01$). Thus, the ApoE-ε3/ε3 genotype may decrease the risk of hypopituitarism after TBI.

Issues, Prevention, and Recommendations

Issues of Mild TBI-Related Hypopituitarism

On the one hand, symptoms of pituitary dysfunction can be masked by identical postconcussion symptoms or overshadowed by other symptoms.[29] Thus, symptoms of hypopituitarism may not show up until several years after trauma. Furthermore, at the time of concussion, only approximately 10% of athletes are rendered unconscious.[7] That is why this complication may be undiagnosed[15] in many patients with mild TBI. Because most sports participants are young adults with near-normal life expectancy, the implications of undiagnosed postconcussion pituitary dysfunction can be dramatic. Although reports have increased, concussions are not recognized as serious injuries or are not consistently identified by athletic training staff.[7]

On the other hand, many questions persist, especially regarding the exact pathophysiological mechanisms of hypopituitarism in sports-related head trauma. The cur-

rent hypotheses seem synergistic, and seem to play a different role in the function of the mechanism and severity of the TBI. Indeed, sella turcica fractures and brain swelling might be related to severe TBI and cause immediate pituitary dysfunction. Severe brain injuries can occur in sports, such as the "knockout" in boxing, but mild TBI is more frequent. Autoimmunity might play more of a role in the long term. Time and repeated trauma seem also to play an important role, because pituitary dysfunction is more frequent in retired boxers than in active ones.

Thus, further multidisciplinary and multicenter studies are warranted to clarify all these possible mechanisms and their role in each type of TBI, and to produce clear recommendations.

Prevention and Recommendations

The potential long-term consequences of repetitive head trauma sustained in high-contact sports have been known for years. Although clearly criticized by the medical community, because of the possible damage to health that can be induced, certain sports such as boxing remain authorized and very common worldwide. This increasing problem has led to the release of several consensus guidelines on concussion management in sports. For instance, the American Collegiate Athletic Association and the American Academy of Neurology have established clear guidelines and management plans for concussion. However, several fundamental issues remain,[6] as follows: the difficulty of the recognition of a concussive event by medical staff; the recognition and understanding of short- and long-term sequelae; the management of each stage; and the establishment of proper action to mitigate effects of the injury. Therefore, sponsors of sports programs should be fully involved and should design and maintain an injury-prevention program. Furthermore, regulatory controls, educating participants, designing specialized protective products, and monitoring injury frequency are evidently necessary.

As mentioned, many means of prevention exist. However, there are no specific recommendations on hypopituitarism in sports-related head trauma; thus, further research is necessary.

Pending these recommendations based on evidence, Tanriverdi et al.[66] recommend the routine investigation of pituitary function in retired athletes who had participated in contact sports, and also in athletes who have a history of concussion or who have clinical findings suggesting pituitary hormone deficiencies.

Conclusions

Current knowledge clearly supports the proposition that sports, especially combat sports, are a cause of hypopituitarism, particularly isolated GH deficiency. Therefore, the medical community should be aware of this, and participants in sports who were exposed to chronic repetitive TBI should be screened. However, further multicenter and multidisciplinary studies are required to explore the details of pathophysiological mechanisms and to produce accurate prevention recommendations and guidelines on hypopituitarism in sports-related head trauma.

Disclosure

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

Author contributions to the study and manuscript preparation include the following. Conception and design: both authors. Acquisition of data: both authors. Analysis and interpretation of data: both authors. Drafting the article: Dubourg. Critically revising the article: both authors. Approved the final version of the manuscript on behalf of all authors: Messerer. Study supervision: both authors.

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References

1. Agha A, Rogers B, Sherlock M, O'Kelly P, Tormey W, Phillips J, et al: Anterior pituitary dysfunction in survivors of traumatic brain injury. **J Clin Endocrinol Metab 89:**4929–4936, 2004
2. Agha A, Thompson CJ: Anterior pituitary dysfunction following traumatic brain injury (TBI). **Clin Endocrinol (Oxf) 64:**481–488, 2006
3. Aimaretti G, Ambrosio MR, Di Somma C, Fusco A, Cannavò S, Gasperi M, et al: Traumatic brain injury and subarachnoid haemorrhage are conditions at high risk for hypopituitarism: screening study at 3 months after the brain injury. **Clin Endocrinol (Oxf) 61:**320–326, 2004
4. Aimaretti G, Ambrosio MR, Di Somma C, Gasperi M, Cannavò S, Scaroni C, et al: Residual pituitary function after brain injury-induced hypopituitarism: a prospective 12-month study. **J Clin Endocrinol Metab 90:**6085–6092, 2005
5. Anonymous: Congress of Neurological Surgeons, Inc. **Clin Neurosurg 14:**424–445, 1966
6. Apuzzo ML: The National Football League: cerebral concussion, peer-review, and the oath of Hippocrates: keynote address—NFL concussion summit, Chicago 2007. **Neurosurgery 62:**202–203, 2008
7. Bailes JE: Sports-related concussion: what do we know in 2009-a neurosurgeon's perspective. **J Int Neuropsychol Soc 15:** 509–511, 2009
8. Barth JT, Macciocchi SN, Giordani B, Rimel R, Jane JA, Boll TJ: Neuropsychological sequelae of minor head injury. **Neurosurgery 13:**529–533, 1983
9. Bellosta S, Nathan BP, Orth M, Dong LM, Mahley RW, Pitas RE: Stable expression and secretion of apolipoproteins E3 and E4 in mouse neuroblastoma cells produces differential effects on neurite outgrowth. **J Biol Chem 270:**27063–27071, 1995
10. Benvenga S, Campenní A, Ruggeri RM, Trimarchi F: Clinical review 113: Hypopituitarism secondary to head trauma. **J Clin Endocrinol Metab 85:**1353–1361, 2000
11. Bistritzer T, Theodor R, Inbar D, Cohen BE, Sack J: Anterior hypopituitarism due to fracture of the sella turcica. **Am J Dis Child 135:**966–968, 1981
12. Bondanelli M, Ambrosio MR, Zatelli MC, De Marinis L, degli Uberti EC: Hypopituitarism after traumatic brain injury. **Eur J Endocrinol 152:**679–691, 2005
13. Bondanelli M, De Marinis L, Ambrosio MR, Monesi M, Valle D, Zatelli MC, et al: Occurrence of pituitary dysfunction following traumatic brain injury. **J Neurotrauma 21:**685–696, 2004
14. Boughey JC, Yost MJ, Bynoe RP: Diabetes insipidus in the head-injured patient. **Am Surg 70:**500–503, 2004
15. Casanueva FF, Leal A, Koltowska-Häggström M, Jonsson P, Góth MI: Traumatic brain injury as a relevant cause of growth

hormone deficiency in adults: a KIMS-based study. **Arch Phys Med Rehabil 86:**463–468, 2005
16. Caturegli P, Newschaffer C, Olivi A, Pomper MG, Burger PC, Rose NR: Autoimmune hypophysitis. **Endocr Rev 26:**599–614, 2005
17. Ceballos R: Pituitary changes in head trauma (analysis of 102 consecutive cases of head injury). **Ala J Med Sci 3:**185–198, 1966
18. Crawford FC, Vanderploeg RD, Freeman MJ, Singh S, Waisman M, Michaels L, et al: APOE genotype influences acquisition and recall following traumatic brain injury. **Neurology 58:**1115–1118, 2002
19. Cyran E: Hypophysenschädigung durch Schädelbasisfraktur. **Dtsch Med Wochenschr 44:**1261–1270, 1918
20. Daniel PM: The pituitary gland and its blood supply, in British Postgraduate Medical Federation (ed): **The Scientific Basis of Medicine–Annual Reviews 1963.** London: Athlone Press, 1963, pp 83–98
21. Daniel PM, Prichard MM, Treip CS: Traumatic infarction of the anterior lobe of the pituitary gland. **Lancet 2:**927–931, 1959
22. De Bellis A, Bizzarro A, Conte M, Perrino S, Coronella C, Solimeno S, et al: Antipituitary antibodies in adults with apparently idiopathic growth hormone deficiency and in adults with autoimmune endocrine diseases. **J Clin Endocrinol Metab 88:**650–654, 2003
23. De Bellis A, Salerno M, Conte M, Coronella C, Tirelli G, Battaglia M, et al: Antipituitary antibodies recognizing growth hormone (GH)-producing cells in children with idiopathic GH deficiency and in children with idiopathic short stature. **J Clin Endocrinol Metab 91:**2484–2489, 2006
24. Delaney JS, Lacroix VJ, Leclerc S, Johnston KM: Concussions among university football and soccer players. **Clin J Sport Med 12:**331–338, 2002
25. Delaney JS, Lacroix VJ, Leclerc S, Johnston KM: Concussions during the 1997 Canadian Football League season. **Clin J Sport Med 10:**9–14, 2000
26. Diaz-Arrastia R, Gong Y, Fair S, Scott KD, Garcia MC, Carlile MC, et al: Increased risk of late posttraumatic seizures associated with inheritance of APOE epsilon4 allele. **Arch Neurol 60:**818–822, 2003
27. Escamilla RF, Lisser H: Simmonds disease. A clinical study with review of the literature; differentiation of anorexia nervosa by statistical analysis of 595 cases, 101 of which were proved pathologically. **J Clin Endocrinol 2:**65–96, 1942
28. Friedman G, Froom P, Sazbon L, Grinblatt I, Shochina M, Tsenter J, et al: Apolipoprotein E-epsilon4 genotype predicts a poor outcome in survivors of traumatic brain injury. **Neurology 52:**244–248, 1999
29. Ghigo E, Masel B, Aimaretti G, Léon-Carrión J, Casanueva FF, Dominguez-Morales MR, et al: Consensus guidelines on screening for hypopituitarism following traumatic brain injury. **Brain Inj 19:**711–724, 2005
30. Goudie RB, Pinkerton PH: Anterior hypophysitis and Hashimoto's disease in a young woman. **J Pathol Bacteriol 83:**584–585, 1962
31. Hellawell DJ, Taylor RT, Pentland B: Cognitive and psychosocial outcome following moderate or severe traumatic brain injury. **Brain Inj 13:**489–504, 1999
32. Herrmann BL, Rehder J, Kahlke S, Wiedemayer H, Doerfler A, Ischebeck W, et al: Hypopituitarism following severe traumatic brain injury. **Exp Clin Endocrinol Diabetes 114:**316–321, 2006
33. Iglesias P, Gómez-Pan A, Diez JJ: Spontaneous recovery from post-traumatic hypopituitarism. **J Endocrinol Invest 19:**320–323, 1996
34. Ignatius MJ, Gebicke-Härter PJ, Skene JH, Schilling JW, Weisgraber KH, Mahley RW, et al: Expression of apolipoprotein E during nerve degeneration and regeneration. **Proc Natl Acad Sci U S A 83:**1125–1129, 1986
35. Ives JC, Alderman M, Stred SE: Hypopituitarism after multiple concussions: a retrospective case study in an adolescent male. **J Athl Train 42:**431–439, 2007
36. Jordan BD: Sports injuries, in **Proceedings of the Mild Brain Injury in Sports Summit.** Dallas: National Athletic Trainers' Association Research & Education Foundation, 1991, pp 43–45
37. Kaufman HH, Timberlake G, Voelker J, Pait TG: Medical complications of head injury. **Med Clin North Am 77:**43–60, 1993
38. Kelestimur F, Tanriverdi F, Atmaca H, Unluhizarci K, Selcuklu A, Casanueva FF: Boxing as a sport activity associated with isolated GH deficiency. **J Endocrinol Invest 27:**RC28–RC32, 2004
39. Kelly DF, Gonzalo IT, Cohan P, Berman N, Swerdloff R, Wang C: Hypopituitarism following traumatic brain injury and aneurysmal subarachnoid hemorrhage: a preliminary report. **J Neurosurg 93:**743–752, 2000
40. Kelly JP, Nichols JS, Filley CM, Lillehei KO, Rubinstein D, Kleinschmidt-DeMasters BK: Concussion in sports. Guidelines for the prevention of catastrophic outcome. **JAMA 266:** 2867–2869, 1991
41. Kokshoorn NE, Smit JW, Nieuwlaat WA, Tiemensma J, Bisschop PH, Groote Veldman R, et al: Low prevalence of hypopituitarism after traumatic brain injury: a multicenter study. **Eur J Endocrinol 165:**225–231, 2011
42. Kornblum RN, Fisher RS: Pituitary lesions in craniocerebral injuries. **Arch Pathol 88:**242–248, 1969
43. Leal-Cerro A, Flores JM, Rincon M, Murillo F, Pujol M, Garcia-Pesquera F, et al: Prevalence of hypopituitarism and growth hormone deficiency in adults long-term after severe traumatic brain injury. **Clin Endocrinol (Oxf) 62:**525–532, 2005
44. Liaquat I, Dunn LT, Nicoll JA, Teasdale GM, Norrie JD: Effect of apolipoprotein E genotype on hematoma volume after trauma. **J Neurosurg 96:**90–96, 2002
45. Lieberman SA, Oberoi AL, Gilkison CR, Masel BE, Urban RJ: Prevalence of neuroendocrine dysfunction in patients recovering from traumatic brain injury. **J Clin Endocrinol Metab 86:**2752–2756, 2001
46. Manetti L, Lupi I, Morselli LL, Albertini S, Cosottini M, Grasso L, et al: Prevalence and functional significance of antipituitary antibodies in patients with autoimmune and nonautoimmune thyroid diseases. **J Clin Endocrinol Metab 92:**2176–2181, 2007
47. Massol J, Humbert P, Cattin F, Bonneville JF: Post-traumatic diabetes insipidus and amenorrhea-galactorrhea syndrome after pituitary stalk rupture. **Neuroradiology 29:**299–300, 1987
48. Miller LJ, Mittenberg W: Brief cognitive behavioral interventions in mild traumatic brain injury. **Appl Neuropsychol 5:**172–183, 1998
49. Nathan BP, Bellosta S, Sanan DA, Weisgraber KH, Mahley RW, Pitas RE: Differential effects of apolipoproteins E3 and E4 on neuronal growth in vitro. **Science 264:**850–852, 1994
50. Nishida Y, Yoshioka M, St-Amand J: The top 10 most abundant transcripts are sufficient to characterize the organs functional specificity: evidences from the cortex, hypothalamus and pituitary gland. **Gene 344:**133–141, 2005
51. Park KD, Kim DY, Lee JK, Nam HS, Park YG: Anterior pituitary dysfunction in moderate-to-severe chronic traumatic brain injury patients and the influence on functional outcome. **Brain Inj 24:**1330–1335, 2010
52. Popovic V, Pekic S, Pavlovic D, Maric N, Jasovic-Gasic M, Djurovic B, et al: Hypopituitarism as a consequence of traumatic brain injury (TBI) and its possible relation with cognitive disabilities and mental distress. **J Endocrinol Invest 27:**1048–1054, 2004
53. Rimel RW, Giordani B, Barth JT, Boll TJ, Jane JA: Disability caused by minor head injury. **Neurosurgery 9:**221–228, 1981

54. Rudehill S, Muhallab S, Wennersten A, von Gertten C, Al Nimer F, Sandberg-Nordqvist AC, et al: Autoreactive antibodies against neurons and basal lamina found in serum following experimental brain contusion in rats. **Acta Neurochir (Wien) 148:**199–205, 2006
55. Schneider HJ, Aimaretti G, Kreitschmann-Andermahr I, Stalla GK, Ghigo E: Hypopituitarism. **Lancet 369:**1461–1470, 2007
56. Schneider HJ, Schneider M, Saller B, Petersenn S, Uhr M, Husemann B, et al: Prevalence of anterior pituitary insufficiency 3 and 12 months after traumatic brain injury. **Eur J Endocrinol 154:**259–265, 2006
57. Simmonds M: Über hypophysisschwund mit todlichem ausgang. **Dtsch Med Wochenschr 40:**322–323, 1914
58. Sorbi S, Nacmias B, Piacentini S, Repice A, Latorraca S, Forleo P, et al: ApoE as a prognostic factor for post-traumatic coma. **Nat Med 1:**852, 1995
59. Stein TD, Fedynyshyn JP, Kalil RE: Circulating autoantibodies recognize and bind dying neurons following injury to the brain. **J Neuropathol Exp Neurol 61:**1100–1108, 2002
60. Tanriverdi F, De Bellis A, Battaglia M, Bellastella G, Bizzarro A, Sinisi AA, et al: Investigation of antihypothalamus and antipituitary antibodies in amateur boxers: is chronic repetitive head trauma-induced pituitary dysfunction associated with autoimmunity? **Eur J Endocrinol 162:**861–867, 2010
61. Tanriverdi F, De Bellis A, Bizzarro A, Sinisi AA, Bellastella G, Pane E, et al: Antipituitary antibodies after traumatic brain injury: is head trauma-induced pituitary dysfunction associated with autoimmunity? **Eur J Endocrinol 159:**7–13, 2008
62. Tanriverdi F, Senyurek H, Unluhizarci K, Selcuklu A, Casanueva FF, Kelestimur F: High risk of hypopituitarism after traumatic brain injury: a prospective investigation of anterior pituitary function in the acute phase and 12 months after trauma. **J Clin Endocrinol Metab 91:**2105–2111, 2006
63. Tanriverdi F, Taheri S, Ulutabanca H, Caglayan AO, Ozkul Y, Dundar M, et al: Apolipoprotein E3/E3 genotype decreases the risk of pituitary dysfunction after traumatic brain injury due to various causes: preliminary data. **J Neurotrauma 25:** 1071–1077, 2008
64. Tanriverdi F, Ulutabanca H, Unluhizarci K, Selcuklu A, Casanueva FF, Kelestimur F: Three years prospective investigation of anterior pituitary function after traumatic brain injury: a pilot study. **Clin Endocrinol (Oxf) 68:**573–579, 2008
65. Tanriverdi F, Unluhizarci K, Coksevim B, Selcuklu A, Casanueva FF, Kelestimur F: Kickboxing sport as a new cause of traumatic brain injury-mediated hypopituitarism. **Clin Endocrinol (Oxf) 66:**360–366, 2007
66. Tanriverdi F, Unluhizarci K, Kelestimur F: Pituitary function in subjects with mild traumatic brain injury: a review of literature and proposal of a screening strategy. **Pituitary 13:**146–153, 2010
67. Tanriverdi F, Unluhizarci K, Kocyigit I, Tuna IS, Karaca Z, Durak AC, et al: Brief communication: pituitary volume and function in competing and retired male boxers. **Ann Intern Med 148:**827–831, 2008
68. Waters RJ, Nicoll JA: Genetic influences on outcome following acute neurological insults. **Curr Opin Crit Care 11:**105–110, 2005
69. Welsh-Bohmer KA, Gearing M, Saunders AM, Roses AD, Mirra S: Apolipoprotein E genotypes in a neuropathological series from the Consortium to Establish a Registry for Alzheimer's Disease. **Ann Neurol 42:**319–325, 1997
70. Whitman S, Coonley-Hoganson R, Desai BT: Comparative head trauma experiences in two socioeconomically different Chicago-area communities: a population study. **Am J Epidemiol 119:**570–580, 1984

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Chronic traumatic encephalopathy in an Iraqi war veteran with posttraumatic stress disorder who committed suicide

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Following his discovery of chronic traumatic encephalopathy (CTE) in football players in 2002, Dr. Bennet Omalu hypothesized that posttraumatic stress disorder (PTSD) in military veterans may belong to the CTE spectrum of diseases. The CTE surveillance at the Brain Injury Research Institute was therefore expanded to include deceased military veterans diagnosed with PTSD. The authors report the case of a 27-year-old United States Marine Corps (USMC) Iraqi war veteran, an amphibious assault vehicle crewman, who committed suicide by hanging after two deployments to Fallujah and Ramadi. He experienced combat and was exposed to mortar blasts and improvised explosive device blasts less than 50 m away. Following his second deployment he developed a progressive history of cognitive impairment, impaired memory, behavioral and mood disorders, and alcohol abuse. Neuropsychiatric assessment revealed a diagnosis of PTSD with hyperarousal (irritability and insomnia) and numbing. He committed suicide approximately 8 months after his honorable discharge from the USMC. His brain at autopsy appeared grossly unremarkable except for congestive brain swelling. There was no atrophy or remote focal traumatic brain injury such as contusional necrosis or hemorrhage. Histochemical and immunohistochemical brain tissue analysis revealed CTE changes comprising multifocal, neocortical, and subcortical neurofibrillary tangles and neuritic threads (ranging from none, to sparse, to frequent) with the skip phenomenon, accentuated in the depths of sulci and in the frontal cortex. The subcortical white matter showed mild rarefaction, sparse perivascular and neuropil infiltration by histiocytes, and mild fibrillary astrogliosis. Apolipoprotein E genotype was 3/4. The authors report this case as a sentinel case of CTE in an Iraqi war veteran diagnosed with PTSD to possibly stimulate new lines of thought and research in the possible pathoetiology and pathogenesis of PTSD in military veterans as part of the CTE spectrum of diseases, and as chronic sequelae and outcomes of repetitive traumatic brain injuries. *(DOI: 10.3171/2011.9.FOCUS11178)*

Key Words • posttraumatic stress disorder • Iraq • chronic traumatic encephalopathy • suicide

In 2002 Dr. Bennet Omalu[22] discovered and described CTE in a football player when he performed an autopsy on Mike Webster. Since 2002, Dr. Omalu, the Brain Injury Research Institute, and other researchers have identified and described CTE in numerous football players, wrestlers, boxers, and ice hockey players, which have been reported in the literature.[16,17,19–22,24,25] Following our elucidation of CTE in athletes, we hypothesized that PTSD in war veterans may belong to the CTE spectrum given that active military personnel are high-risk cohorts for repeated subconcussive and concussive traumatic brain injuries; for example, bomb blasts can cause traumatic brain injuries from primary pressure wave and acceleration-deceleration injury mechanisms.[4,28] We expanded our CTE surveillance and brain tissue analyses to include deceased military veterans who were diagnosed with PTSD.

In 2010 we encountered CTE changes in the brain of a 61-year-old deceased Vietnam war veteran, who died suddenly as a result of coronary atherosclerotic disease. This case was reported in the *Stars and Stripes* news magazine of the Department of Defense.[26] The case was not published because we did not have comprehensive access to the medical records and family and social histories. Approximately 1 year later we have identified CTE changes in the brain of a 27-year-old Iraqi war veteran who was diagnosed with PTSD and committed suicide by hanging.

In our 2010 CTE paper,[20] we had defined CTE as

Abbreviations used in this paper: CTE = chronic traumatic encephalopathy; DSM-IV-TR = *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision*; IED = improvised explosive device; PTSD = posttraumatic stress disorder; TDP-43 = TAR-DNA-binding protein 43; USMC = United States Marine Corps.

a progressive neurodegenerative syndrome caused by single, episodic, or repetitive blunt force impacts to the head and transfer of acceleration-deceleration forces to the brain. Chronic traumatic encephalopathy presents clinically after a prolonged latent period as a composite syndrome of mood disorders and neuropsychiatric and cognitive impairment. Direct brain tissue analysis reveals multifocal or diffuse tauopathy, which may be accompanied by low-grade and multifocal white matter rarefaction, microglial activation, and parenchymal histiocytes. Amyloidopathy may be present; however, the primary proteinopathy in CTE is a tauopathy. Some patients with CTE may not exhibit the classic prolonged latency period before clinical symptoms begin.

Posttraumatic stress disorder in war veterans was first designated in 1978 to describe a condition in Vietnam war veterans, and the syndrome was first recognized by the American Psychiatric Association in the early 1980s.[30] A primary neurodegenerative proteinopathy has not been defined for PTSD in war veterans. Pathognomonic tissue neuropathological features have not been specified. Clinical diagnoses are currently based on presenting clinical symptomatology, based on two diagnostic systems, which continue to evolve as our understanding of PTSD continues to grow.[30,32] Table 1 shows the DSM-IV-TR clinical criteria for PTSD diagnosis,[1] and Table 2 shows the ICD-10 clinical criteria for PTSD diagnosis.[31]

We report this case as a sentinel case of CTE in an Iraqi war veteran diagnosed with PTSD to possibly stimulate new lines of thought and research in the possible pathoetiology and pathogenesis of PTSD in military veterans as it relates to PTSD being part of the CTE spectrum of diseases, and as chronic sequelae and outcomes of repetitive traumatic brain injuries.

Case Report

Premortem History

This subject was a 27-year-old Caucasian man who committed suicide by hanging approximately 8 months after his honorable discharge from the USMC and while he was beginning a divorce process with his wife. His wife for 3 years, a 6-year-old stepson, and a 2-year-old biological son had left him and moved in with her parents. Reportedly, he had performed well in high school, obtaining mostly "A" grades. In college he began binge drinking and barely received passing grades, which he attributed to his binge drinking; however, he received a bachelor's degree in history. Following college he joined the USMC at the age of 23 and was honorably discharged after 4 years (2006–2010) with a rank of corporal. Before entering the military he had worked as a waiter and as a staff member in a national vitamin retail chain.

His listed military occupational specialty was 1833 Amphibious Assault Vehicle Crewman. He served two deployments to Iraq. The first deployment to Iraq was in 2007 for approximately 8 months in Fallujah, where he was assigned to mechanized mobile patrols. The second deployment occurred in 2008 for approximately 5 months in Ramadi, where he was assigned to an entry control point. He experienced combat and reported exposures to mortar blasts and IED blasts less than 50 m away. During the second deployment he was court marshaled twice for acting out, insubordination, fighting, hazing, and assault, and was dropped 1 rank. He described only a few incidents during his deployment that he found bothersome. There was an incident during the 3rd week of his first deployment when he witnessed a vehicle in his patrol blown up, and marines killed and wounded. In another incident, approximately 2 weeks later, while hooking up their disabled vehicle to tow, 2 marines in his section were shot and he helped to patch them up. In yet another incident, he witnessed a school bus full of Iraqi citizens, many of whom were children, blown up by an IED.

After his deployments he was stationed at a base and played football in a base league. During a football game in 2009, approximately 9 months after his second deployment, he reported being hit from the side causing him to fall to the ground. He stood up, stumbled, fell again, and then continued the game. Other players noticed that he was confused and kept asking the count and details of the next play and he had to be removed from the game. The events of the following week were unclear and he reported residual headaches and memory problems. Reportedly a conventional CT scan of the head was performed and showed no significant findings. He reported playing football and hockey for leisure and was never diagnosed with a concussion, although he suffered his "bell rung."

Two days prior to a 2010 neuropsychological evaluation, he was involved in a single motor vehicle crash while he was driving under the influence of alcohol, when he turned a corner and flipped his car. He woke up later hanging upside down in the car. It was not clear whether he lost consciousness from a head injury or from stuporous alcohol intoxication. He noted the following morning that he suffered from headaches and vomited; however, it was not also clear whether these symptoms were alcohol-related or head injury–related. He lost his driver's license after this crash for driving under the influence and refusing a blood-alcohol test. He visited a Veteran Affairs Medical Center the day before he committed suicide and reported having a new job as a football coach with his old high school, and was currently attending a community college. His driving under the influence charges had been dismissed.

In 2010, he was referred for a neuropsychological screening. His wife reported that he forgot dates, conversations, and trivialities of daily living. He also forgot whether he completed tasks, and sometimes confused his wife's and sister's names. He had problems making decisions and therefore avoided them. He believed he snapped at his children too frequently and was increasingly becoming a grumpy person. He admitted to headaches that occurred 3 to 4 times per week, which he described as pressure in his entire head. The headaches were relieved by a nonsteroidal antiinflammatory agent. He experienced bilateral hearing problems and tinnitus, which he dated back to when he had worked on engines in the military. He reported dizziness when he woke up at night to use the bathroom, slept only 4 hours a night, and had trouble falling asleep. Other reported symptoms included irrita-

TABLE 1: Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR), Fourth Edition: Diagnostic Criteria for Posttraumatic Stress Disorder*

A. The person has been exposed to a traumatic event in which both of the following were present:
1. The person experienced, witnessed, or was confronted with an event or events that involved actual or threatened death or serious injury, or a threat to the physical integrity of self or others
2. The person's response involved intense fear, helplessness, or horror. Note: In children, this may be expressed instead by disorganized or agitated behavior
B. The traumatic event is persistently reexperienced in 1 [or more] of the following ways:
1. Recurrent and intrusive distressing recollections of the event including images, thoughts, or perceptions. Note: In young children, repetitive play may occur in which themes or aspects of the trauma are exposed.
2. Recurrent distressing dreams of the event. Note: In children, there may be frightening dreams without recognizable content.
3. Acting or feeling as if the traumatic event were recurring [includes a sense of reliving the experience, illusions, hallucinations, and dissociative flashback episodes, including those that occur on awakening or when intoxicated]. Note: In young children, trauma-specific reenactment may occur.
4. Intense psychological distress at exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event
5. Physiological reactivity on exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event
C. Persistent avoidance of stimuli associated with the trauma and numbing of general responsiveness (not present before the trauma), as indicated by 3 (or more) of the following:
1. Efforts to avoid thoughts, feelings, or conversations associated with the trauma
2. Efforts to avoid activities, places, or people that arouse recollections of the trauma
3. Inability to recall an important aspect of the trauma
4. Markedly diminished interest or participation in significant activities
5. Feeling of detachment or estrangement from others
6. Restricted range of affect (e.g., unable to have loving feelings)
7. Sense of foreshortened future (e.g., does not expect to have a career, marriage, children, or a normal life span)
D. Persistent symptoms of increased arousal (not present before the trauma), as indicated by 2 (or more) of the following:
1. Difficulty falling or staying asleep
2. Irritability or outbursts of anger
3. Difficulty concentrating
4. Hypervigilance
5. Exaggerated startle response
E. Duration of the disturbance (symptoms in Criteria B, C, and D) is more than 1 month.
F. The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.
Specify if:
Acute: if duration of symptoms is less than 3 months
Chronic: if duration of symptoms is 3 months or more
Specify if:
With Delayed Onset: if onset of symptoms is at least 6 months after the stressor

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bility, discomfort with crowds, startle reactions, anhedonia, withdrawal and lack of engagement with the family, emotional numbing, and detachment. Many of his symptoms began after his first and second deployments. He also reported getting angry quickly and losing his temper frequently. He smoked cigarettes and reported drinking rarely; however, when he did drink, he drank a lot. When he got out of the marines he drank weekly. There was minor experimentation with marijuana and other drugs while he was in college. He was not aware of any family history of mental illness or any developmental impairment. His other significant medical history was a nasal bone fracture from an unspecified cause when he was 21 years old.

Neuropsychological Testing

The following neuropsychological tests and procedures were performed: Wechsler Test of Adult Intelligence; California Verbal Learning Test; Rey-Osterrieth Complex Figure Test; Grooved Pegboard Test; Finger Tapping Test; Trail Making Test; Word-list generation tasks; Ruff Figural Fluency Test; Ruff 2&7 Selective Attention Test; Test of Memory Malingering; Beck Depression Inventory-2; and PTSD checklist-military version.

For general cognitive functioning, his baseline intellectual abilities were estimated to fall in the high average to superior range based on demographic variables as well as a word-reading test (Wechsler Test of Adult Intel-

TABLE 2: World Health Organization ICD-10 classification of mental and behavioral disorders. Clinical descriptions and diagnostic guidelines*

PTSD
This arises as a delayed and/or protracted response to a stressful event or situation (either short- or long-lasting) of an exceptionally threatening or catastrophic nature, which is likely to cause pervasive distress in almost anyone (e.g., natural or man-made disaster, combat, serious accident, witnessing the violent death of others, or being the victim of torture, terrorism, rape, or other crime). Predisposing factors such as personality traits (e.g. compulsive, asthenic) or previous history of neurotic illness may lower the threshold for the development of the syndrome or aggravate its course, but they are neither necessary nor sufficient to explain its occurrence.
Typical symptoms include episodes of repeated reliving of the trauma in intrusive memories ("flashbacks") or dreams, occurring against the persisting background of a sense of "numbness" and emotional blunting, detachment from other people, unresponsiveness to surroundings, anhedonia, and avoidance of activities and situations reminiscent of the trauma. Commonly there is fear and avoidance of cues that remind the sufferer of the original trauma. Rarely, there may be dramatic, acute bursts of fear, panic or aggression, triggered by stimuli arousing a sudden recollection and/or re-enactment of the trauma or of the original reaction to it.
There is usually a state of autonomic hyperarousal with hypervigilance, an enhanced startle reaction, and insomnia. Anxiety and depression are commonly associated with the above symptoms and signs, and suicidal ideation is not infrequent. Excessive use of alcohol or drugs may be a complicating factor. The onset follows the trauma with a latency period, which may range from a few weeks to months (but rarely exceeds 6 months). The course is fluctuating but recovery can be expected in the majority of cases. In a small proportion of patients the condition may show a chronic course over many years and a transition to an enduring personality change.
Diagnostic Guidelines
This disorder should not generally be diagnosed unless there is evidence that it arose within 6 months of a traumatic event of exceptional severity. A "probable" diagnosis might still be possible if the delay between the event and the onset was longer than 6 months, provided that the clinical manifestations are typical and no alternative identification of the disorder (e.g., as an anxiety or obsessive-compulsive disorder or depressive episode) is plausible. In addition to evidence of trauma, there must be a repetitive, intrusive recollection or re-enactment of the event in memories, daytime imagery, or dreams. Conspicuous emotional detachment, numbing of feeling, and avoidance of stimuli that might arouse recollection of the trauma are often present but are not essential for the diagnosis. The autonomic disturbances, mood disorder, and behavioural abnormalities all contribute to the diagnosis but are not of prime importance.

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ligence: estimated full-scale intelligence quotient = 119, 90th percentile). For attention, he was able to attend to instructions throughout the evaluation. He occasionally required some limited repetition of directions. A measure of selective attention was in the average range for speed (58th percentile) and accuracy (62nd percentile). His performance improved slightly as the test progressed, which suggested intact sustained attention to the task. For cognitive/motor processing speed, connection of sequentially numbered circles was performed in the average range (25 seconds, 42nd percentile). Fine motor dexterity was in the average range bilaterally. The dominant-hand performance was 1 second slower than the nondominant hand (right 61 seconds, 42nd percentile; left 60 seconds, 69th percentile).

For executive functioning, a task that required the ability to establish and maintain an alternating alphanumeric sequence was performed in the average range (49 seconds, 58th percentile) with no errors. Word-list generation by letter was in the superior range (FAS = 17/18/25, 92nd percentile), while generation by category was high-average (28 animals, 76th percentile). Design fluency was in the low-average range (14th percentile), with a few repetition errors (5 total, 42nd percentile). Approach on the first trial was efficient and productive, but his efficiency declined on the subsequent 4 trials, resulting in a lower total output. Copy of a complex design was impaired (less than first percentile), due largely to a highly segmented approach with no attention to the gestalt of the figure.

For learning and memory, his ability to learn a list of 16 words over 5 consecutive learning trials was in the average range (California Verbal Learning Test-2, 42nd percentile). His performance on a subsequent interference list was in the superior range (93rd percentile), reflecting a benefit from prior exposure to analogous task. Free recall following this interference list was in the average range (12 words, 69th percentile). Cuing did not change his performance (50th percentile). After a longer delay, his recall was at the 31st percentile, and with cuing, at the 14th percentile. He committed 11 intrusion errors across trials (second percentile): about half were the same single word, which he listed on each learning trial and 1 recall trial, and the remainder represented source memory errors. Yes/no recognition revealed a positive response bias (15/16 hits, 50th percentile; 6 false positive errors, most of which were source-recognition errors and all of which were semantically related, 7th percentile). Forced-choice recognition was errorless (16/16). Incidental memory for a complex design was in the impaired range for both immediate (second percentile) and delayed (first percentile) conditions, likely due in large part to his aforementioned fractured approach to the initial copy of the figure.

Diagnosis of PTSD

His final diagnoses were the following: Axis 1: PTSD with hyperarousal (irritability and insomnia) and numbing, alcohol abuse, continuous; Axis 2: deferred; Axis 3: hyperlipidemia; Axis 4: combat deployment, reintegration difficulties, unemployment, recent arrest, marital stress. His prescribed neurotropic medications included

citalopram, trazodone, and prazosin. In a clinic visit 2 months prior to his suicide, he reported persistent PTSD symptoms.

Postmortem History

The subject's parents had requested the police perform a well-being check of their son after they had not heard from him for 2 days. The police found our subject in his residence hanging from a staircase by a leather belt noose around his neck. A full forensic autopsy by the combined technique of Virchow and Rokitansky[14] was performed by the medical examiner. After informed consent was granted by the wife and next-of-kin, his brain was forwarded to the Brain Injury Research Institute for gross neuropathological, histochemical, and immunohistochemical analysis.

Autopsy Findings

At autopsy our subject was unclad, appeared well-developed and well-nourished, weighed approximately 211 pounds, measured approximately 74 inches, and appeared consistent with the stated age of 27 years old. There was a ligature, which was tightly wound around the neck in a noose, and composed of a brown braided leather belt, which was looped through a buckle located on the posterior neck. The underlying ligature indentation mark was situated circumferentially around the neck in a transverse-oblique-ascending fashion. There were no anterior strap muscle hemorrhages or fractures of the hyoid bone or thyroid cartilage.

Dissection of the thoracic and abdominal cavities revealed normally situated viscera, which were grossly and histologically unremarkable. The scalp revealed no contusional hemorrhages. Dissection of the cranial cavity revealed no skull fractures and no intracranial hemorrhages. Toxicological analysis of the fluoridated heart blood and vitreous humor revealed the presence of citalopram only without any other drug detected. A sample of whole heart blood was submitted for apolipoprotein E genotyping in an ethylenediaminetetraacetic acid specimen bottle. The underlying cause of death was determined to be asphyxiation due to hanging and the manner of death was determined to be a suicide.

Gross Neuropathological Findings

The brain was fixed in 10% buffered formaldehyde for 2 weeks before it was grossed. The dura mater revealed no xanthochromia, membranes, or hemorrhages. The weight of the formaldehyde fixed brain was 1624 grams. The cerebral and cerebellar hemispheres appeared symmetrical and exhibited gyral, sulcal, and folial convolutions that appeared normal. There was diffuse global and symmetrical expansion of gyri and compression of sulci, accompanied by symmetrical bilateral grooving of the unci and cerebellar tonsils, with symmetrical compression of the subarachnoidal cisterns. The arachnoid and pia mater appeared smooth and glistening without acute or chronic subarachnoidal hemorrhages. There was no acute or chronic cerebral cortical contusional or ischemic necrosis or hemorrhage. There was no lobar cortical atrophy.

The centrum semiovale and the periventricular white matter revealed very focal decompositional change and Swiss-cheese appearance, accompanied by central red-pink parenchymal discoloration, edema, and congestion. There was no periventricular leukomalacia, demyelinating plaques, necrosis, hemorrhage, or infarct in the subcortical white matter. There was no acute or chronic gliding contusional hemorrhage or necrosis.

The ventricles were symmetrically compressed and showed no acute or chronic intraventricular hemorrhage. The genu, body, and splenium of the corpus callosum revealed no hemorrhages or necrosis. All subcortical nuclei, including the caudate nucleus, putamen, globus pallidus, thalamus, and subthalamic nucleus, on both sides, revealed no atrophy, necrosis, or hemorrhage (Fig. 1 upper). The hippocampus and parahippocampal gyrus, on both sides, were not dysplastic or atrophic (Fig. 1 lower).

The midbrain, pons, and medulla oblongata revealed no atrophy, dorsolateral hemorrhage, or collicular and teg-

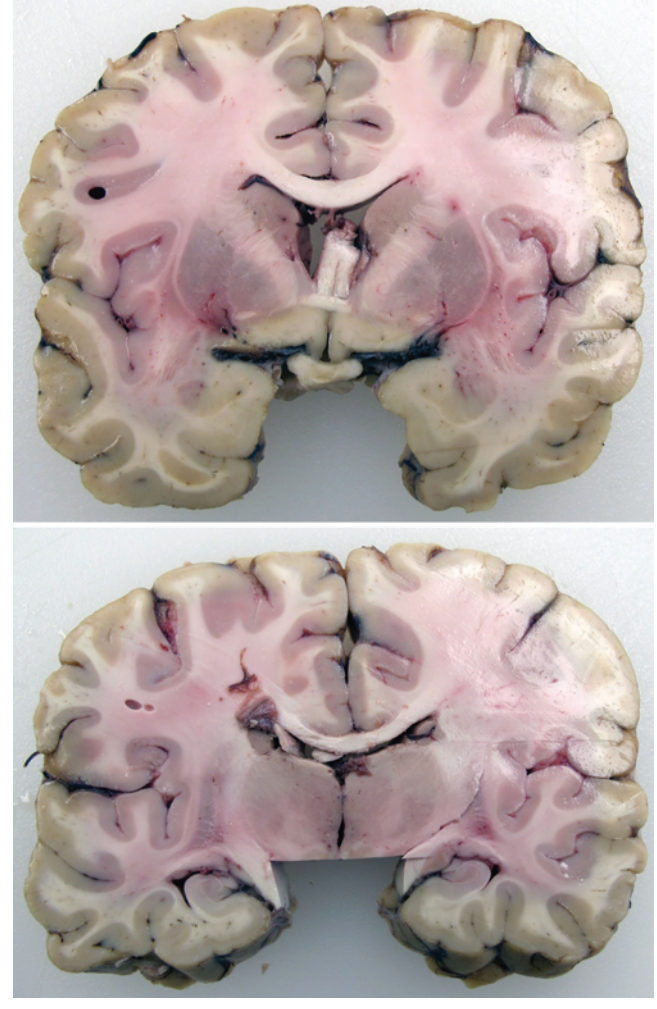

Fig. 1. Gross photographs of the coronal section of the brain at the level of the anterior commissure **(upper)** and hippocampus **(lower)**, showing diffuse parenchymal edema with very focal Swiss-cheese change, without atrophy of the subcortical ganglia **(upper)**, hippocampus or thalamus **(lower)**, or any other focal gross parenchymal gray or white matter lesions.

mental necrosis or hemorrhage. The basis pontis showed no central myelinolysis or Duret hemorrhage. The cerebral peduncles, medullary pyramids, and cerebellar peduncles were not atrophic and showed no hemorrhage or necrosis. The substantia nigra and locus ceruleus were adequately pigmented for age. The inferior olivary nuclei and dentate nuclei revealed normal convolutional patterns. The cerebellar hemispheres revealed no folial atrophy or necrosis. There were no hemorrhages or infarcts in the cerebellar cortex or white matter. The pituitary gland appeared unremarkable. The spinal cord revealed no epidural, subdural, or subarachnoidal hemorrhage. The spinal medulla appeared unremarkable without segmental atrophy, contusional necrosis or hemorrhage, or white matter funicular degeneration or demyelination.

Thirty-one representative topographically selected sections of the dura mater, neocortex, subcortical ganglia, brainstem, pituitary gland, and spinal cord were obtained and stained by the following histochemical and immunohistochemical tissue staining protocols: 1) H & E; 2) tau; 3) β-A4 amyloid; 4) α-synuclein; 5) glial fibrillary acidic protein; 6) CD-68; 7) Luxol fast blue and cresyl violet; 8) ubiquitin; 9) TDP-43; and 10) Bielschowsky silver impregnation stain.

Microscopic Neuropathological Findings

H & E Stains. The frontal, parietal, temporal, occipital, insular, and cingulate cortex revealed the expected columnar and laminar organization without cortical disorganization or dysplasia. There was negligible-to-mild neuronal dropout without eosinophilic neuronal necrosis. There was diffuse perineuronal vacuolation, expansion of Virchow Robin spaces and patchy neuropil microspongiosis of both the gray and white matter. There was marked congestion of the arachnoid and pia mater and the penetrating parenchymal vessels. Multifocal sparse perivascular pigment-laden histiocytes were noted in many Virchow Robin spaces.

The body and splenium of the corpus callosum, internal, external, and extreme capsules revealed no necrosis, hemorrhage, rarefaction, myelinolysis, or axonolysis. The periventricular white matter revealed no leukomalacia or subependymal astrogliosis. The subcortical ganglia including the caudate nucleus, putamen, globus pallidus, hypothalamus, thalamus, amygdala, and basal nucleus of Meynert showed only diffuse neuropil extracellular edema. There was no mineralization of the walls of the vessels of the globus pallidus or cornu ammonis.

The dentate gyrus, cornu ammonis, subiculum, and entorhinal cortex of the hippocampus revealed no significant neuronal dropout, dysplasia, or sclerosis. There was no eosinophilic necrosis of the pyramidal neurons of the stratum pyramidalis. The midbrain, pons, and medulla revealed parenchymal edema without any other focal or diffuse lesions. The neurons of the substantia nigra, locus ceruleus, and dorsal raphe nucleus were adequately pigmented and revealed no neuronal dropout, Marinesco bodies, pale bodies, or Lewy bodies. The cerebellar cortex revealed negligible Purkinje neuron dropout and Bergmann astrogliosis. The internal granule cell layer and dentate nucleus were unremarkable. The cerebellar white matter revealed no rarefaction, necrosis, or infarcts. The dura mater revealed marked intradural congestion without inflammation, or acute or remote hemorrhage. The adenohypophysis revealed focal interstitial fibrosis and marked sinusoidal congestion. The neurohypophysis was congested and edematous. The spinal medulla revealed the normal gray and white matter morphology without significant loss of the anterior horn neurons, which showed sparse nonspecific cytoplasmic neuronal changes. The white matter funiculi and tracts were unremarkable.

Other Histochemical and Immunohistochemical Stains. Tau-immunostained sections revealed none, to sparse, to multifocally frequent band- and flame-shaped neurofibrillary tangles and neuritic threads in the frontal cortex, parietal cortex, temporal cortex, occipital cortex, and cingulate cortex with a skip phenomenon,[20] accentuated in the depths of sulci (Fig. 2). The largest numbers of tangles and threads were noted in the frontal cortex, with none in the occipital cortex. The CA1 region and subiculum revealed none to sparse flame, band, and small globose neurofibrillary tangles and neuritic threads. The entorhinal cortex revealed moderate band- and flame-shaped neurofibrillary tangles and neuritic threads. There was focally sparse to moderate flame- and band-shaped neurofibrillary tangles in the amygdala. The CA2, CA3, and CA4 regions of the cornu ammonis showed no neurofibrillary tangles in the pyramidal neurons and no neuritic threads. The granule neurons of the dentate fascia revealed no neurofibrillary tangles or neuritic threads. There was focally sparse flame- and band-shaped neurofibrillary tangles and neuritic threads in the anterior perforated substance and basal nucleus of Meynert. The thalamus and hypothalamus showed several large globose neurofibrillary tangles and neuritic threads. The substantia nigra showed none to sparse small and large globose neurofibrillary tangles and neuritic threads. The caudate nucleus, insula cortex, putamen, and globus pallidus revealed no neurofibrillary tangles. Only 1 neuritic thread was noted in the globus pallidus. There were no neurofibrillary tangles in the midbrain tegmentum. Few pretangles were noted in neurons of the periaqueductal gray matter. The ventral tegmental pons revealed a single large globose neurofibrillary tangle and several neuritic threads. A single neuron revealed a globose neurofibrillary tangle in the locus ceruleus, accompanied by several neuritic threads. The medullary tegmentum and the inferior olivary nuclei revealed no neurofibrillary tangles or neuritic threads. The Purkinje neurons, internal granule neurons, and the dentate neurons of the cerebellum revealed no neurofibrillary tangles. The anterior horn neurons of the spinal cord revealed no neurofibrillary tangles. There were no neuritic threads in the spinal gray or white matter. Tufted astrocytes, thorn astrocytes, astrocytic plaques, and astrocytic coils and grains were absent in all sections of the brain examined.

There was focal ubiquitin immunopositivity of few neurofibrillary tangles and neuritic threads, otherwise there were no ubiquitin neuronal or glial inclusions in all sections of the brain examined. There were no diffuse or

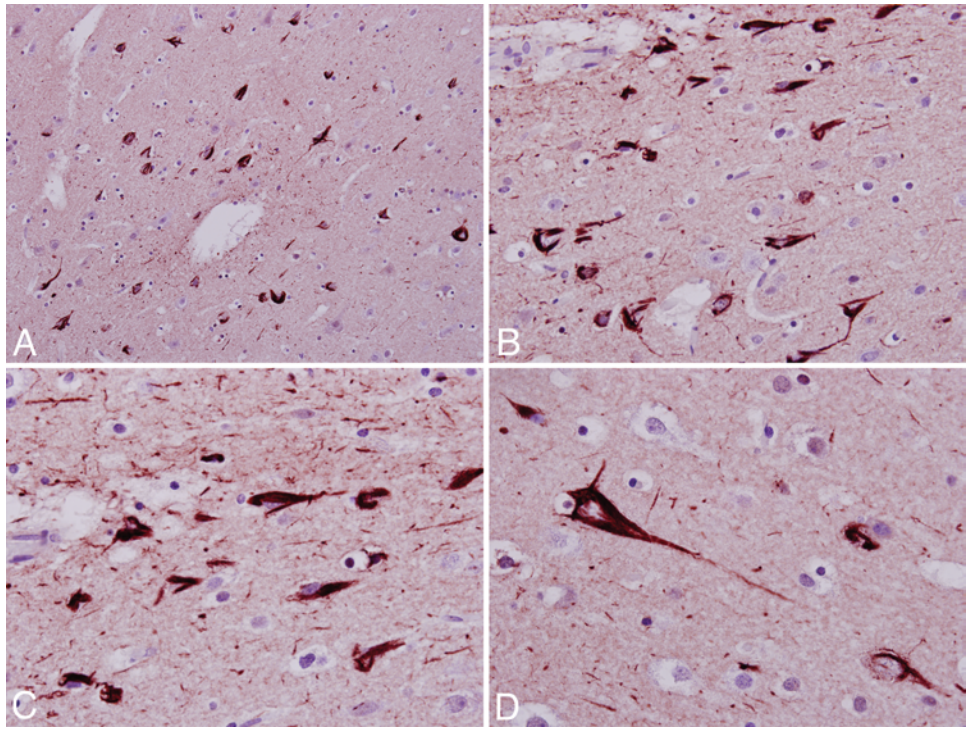

Fig. 2. Photomicrographs of tau-immunostained section of the frontal cortex showing frequent neurofibrillary tangles and neuritic threads **(A and B)**, with higher magnification **(C and D)** showing band- and flame-shaped neurofibrillary tangles and neuropil neuritic threads. Original magnification × 200 **(A)**, × 400 **(B)**, × 600 **(C and D)**.

neuritic amyloid plaques and no cerebral amyloid angiopathy in all examined sections of the cortex, hippocampal formation, subcortical ganglia, brainstem, cerebellum, and spinal medulla. There were no Lewy bodies, Lewy neuritis, or glial alpha-synuclein inclusions in all examined sections of the cortex, hippocampal formation, subcortical ganglia, brainstem, cerebellum, and spinal medulla.

Glial fibrillary acidic protein immunostains revealed multifocal mild perivascular fibrillary astrogliosis and activation of astrocytes in the gray cortex and molecular layer of the frontal cortex, cingulate cortex, parietal cortex, temporal cortex, and occipital cortex. The subcortical gyral white matter of the cortical lobes revealed multifocal and superficial white matter fibrillary astrogliosis and activation of astrocytes. The body and splenium of the corpus callosum showed multifocal and mild fibrillary astrogliosis and activation of astrocytes. There was mild diffuse fibrillary astrogliosis of the hippocampus including the dentate fascia and gyrus, alveus, strata oriens, radiatum, lacunosum, and moleculare. There was mild diffuse astrogliosis of the thalamus, midbrain, pons, and medulla oblongata. There was diffuse astrogliosis of the neurohypophysis. The cerebellum revealed Bergmann astrogliosis with multifocal fibrillary astrogliosis of the superficial folial cerebellar white matter. There was mild diffuse fibrillary astrogliosis of the hypothalamus, amygdala, and anterior perforated substance (basal nucleus of Meynert). The anterior, lateral, and posterior cervical, thoracic, and lumbar funiculi did not show any remarkable astrogliosis.

The CD-68–immunostained sections of the frontal cortex, parietal cortex, temporal cortex, occipital cortex, cingulate gyrus, caudate nucleus, insula cortex, putamen, thalamus, hypothalamus, globus pallidus, cerebellum, corpus callosum, midbrain, pons, and medulla revealed nonspecific multifocal lysosomal astrocytic and microglial staining, accompanied by multifocal immunopositive staining of scattered perivascular histiocytes in and around the Virchow Robin spaces. Small clusters of neuropil histiocytes were present multifocally accentuated in the frontal and parietal subcortical white matter, internal capsule, anterior body of the corpus callosum, splenium of the corpus callosum, anterior commissure, cerebral peduncles, and transverse and longitudinal fascicles of the basis pontis. Similar changes were also noted in the cervical, thoracic, and lumbar anterior, lateral, and posterior funiculi, as well as in the amygdala and anterior perforated substance.

The sections stained with Luxol fast blue and cresyl violet from the frontal, temporal, parietal, and occipital cortex revealed mild diffuse rarefaction of the subcortical white matter, relatively sparing the occipital cortex, and accentuated in the frontal and parietal cortex. There was mild to moderate rarefaction of the subcortical white matter of the cingulate cortex, periventricular white matter of the lateral angle of the lateral ventricle, and of the anterior corpus callosum, as well as of the anterior commissure. There was diffuse mild rarefaction of the internal, external, and extreme capsules. Minimal rarefaction of the cerebral peduncle and medullary pyramids was noted. There was mild to moderate rarefaction of the transverse and longitudinal fasciculi of the basis pontis. There was no rarefaction of the deep cerebellar white matter. The splenium of the corpus callosum revealed mild rarefac-

tion. There was no rarefaction or myelinolysis of the spinal anterior, lateral, or posterior funiculi.

The TDP-43–immunostained sections of the left frontal cortex, parietal cortex, cingulate cortex, insula cortex, claustrum, caudate nucleus, putamen, globus pallidus, and thalamus revealed very sparse and focal TDP-immunopositive cytoplasmic and neuritic inclusions in the frontal cortex and caudate nucleus. The pyramidal neurons of the cornu ammonis and the granule neurons of the dentate fascia revealed no TDP proteinopathy. The midbrain tegmentum, colliculi, periaqueductal gray matter, and substantia nigra showed very sparse and focal TDP nuclear and cytoplasmic inclusions in the substantia nigra. The Purkinje cell layer, internal granule cell layer, and dentate nucleus revealed no TDP proteinopathy or cytoplasmic inclusions. The anterior horn neurons of the spinal medulla revealed sparse and focal nuclear TDP inclusions accompanied by sparse dystrophic TDP-positive neurites.

The specified constellation of gross neuropathological, histomorphological, and immunophenotypical findings in this case are consistent with changes we have described and identified in CTE in American athletes.[20]

Apolipoprotein E Genotyping

Apolipoprotein E genotyping was performed using genomic DNA extracted from peripheral blood leukocytes in whole autopsy blood using the QIAamp DNA Blood Mini kit (Qiagen). The apolipoprotein E genotype was ascertained from the amplified DNA by fluorogenic 5′ nuclease assays (TaqMan SNP genotyping assays; Applied Biosystems). The apolipoprotein E genotype was 3/4.

Discussion

In this report we present the sentinel case of a 27-year-old Iraqi war veteran who was formally diagnosed with PTSD, committed suicide by hanging, and whose brain at autopsy revealed gross, histomorphological, and immunophenotypical findings that we have identified and described in CTE in American athletes.[20] As in our previous CTE cases,[19–25] the primary proteinopathy we identified was a tauopathy in the form of multifocal sparse to frequent topographic neurofibrillary tangles and neuritic threads with a skip phenomenon.[20] Focal secondary TDP proteinopathy was identified. Nonspecific white matter changes accompanied the tauopathy, consisting of nonspecific fibrillary astrogliosis, white matter rarefaction, and perivascular and neuropil infiltration by histiocytes. There was no significant cortical, subcortical, or hippocampal cerebral atrophy. Acute or chronic focal traumatic brain injuries such as lobar contusional necrosis or hemorrhages were absent.

Apolipoprotein E genotype in this case was 3/4. The E3 allele is the most recurrent apolipoprotein E allele we are observing in our CTE cohort,[20] although our sample sizes are small and this observation is preliminary. We currently have no conclusive explanation for this trend that we are observing because the apolipoprotein E4 allele has been reported to be the high-risk allele for Alzheimer disease and adverse outcomes of traumatic brain injury.[2,8] However, we suspect that E4 may not be the primary apolipoprotein E risk allele for CTE although it has been suggested to be associated with chronic traumatic brain injury and cognitive impairment in boxers and professional football players.[11,13,29] Kristman et al.,[12] however, did not find any important association between the E4 allele and the risk of sustaining a concussion in amateur collegiate athletes.

Our subject was a member of a population cohort that can be exposed to repetitive traumatic brain injuries. Traumatic brain injury has been referred to as the signature injury of Operation Enduring Freedom and Operation Iraqi Freedom. It has been estimated that 15%–30% of troops engaged in active combat in Afghanistan and Iraq have suffered concussions and subconcussions, many as a result of blasts from explosive devices. The majority of these patients manifest neuropsychiatric symptoms and cognitive impairments beginning immediately after the injury and lasting for days to weeks to months. Approximately 18%–30% of these patients develop persistent, progressive, and sometimes disabling constellations of neuropsychiatric and cognitive impairments, which are interpreted to represent PTSD.[3] Our subject also played football and hockey for leisure and suffered a remote nasal bone fracture. It is our belief that his eventual CTE risk outcome occurred as a result of his lifetime and cumulative exposure to repeated subconcussive and concussive traumatic brain injuries, with his military exposures being the primary injuries that precipitated CTE.

Our subject manifested persistent symptoms of CTE or PTSD and eventually committed suicide by hanging. We have previously associated parasuicides and suicides with CTE[21] and are observing an overrepresentation of suicides and drug-related accidental deaths in our CTE cohort.[20] According to a 2010 Department of Defense report,[6] the suicide rates in all services increased from about 10 per 100,000 people in 2001 to about 20 per 100,000 people in 2009, while the suicide rate in the general US population remained at approximately 11.5 per 100,000 in 2007. The propositional question that this sentinel case report raises is: What role does CTE play in the increasing incidence of suicides in the US military? This question can only be answered by more forensic observational and translational research focused on CTE and suicides in military veterans.

Members of the armed forces can sustain repetitive traumatic brain injury from training activities, from noncombat professional activities, and from combat activities. Blast exposure is the most common cause of traumatic brain injury in the wars in Iraq and Afghanistan.[7] Explosives like mortar shells, rocket-propelled grenades, and IEDs cause blast injuries via complex physical events, which have the potential to precipitate repetitive subconcussive and concussive brain injuries. The mechanisms of brain damage are not yet well understood, but several neurotrauma mechanisms have been proposed.[4,7,27]

While emphasizing that no human autopsy studies, conducted with current immunohistochemical methods, have been published on blast-related traumatic brain injury in the military, Mac Donald et al.,[15] using diffusion tensor imaging, concluded that blast exposure in US military personnel causes traumatic axonal injury. Follow-up

diffusion tensor imaging 6 to 12 months after preliminary postexposure diffusion tensor imaging confirmed persistence of traumatic axonal injury abnormalities in the brains of their military subjects. Their conclusions and findings support the conclusions, interpretations, and propositional sentinel value of this case report. The primary proteinopathy in CTE is hyperphosphorylated tau, which is a microtubule-associated protein that is involved in the pathophysiological cascades of traumatic axonal injury.[10] We observed nonspecific white matter changes in this case, which accompanied tauopathy.

In addition to tauopathy in this case, we observed a focal and sparse secondary TDP proteinopathy. Such a TDP proteinopathy frequently occurs as a nondiagnostic or nondefining accompaniment and secondary proteinopathy of a variety of primary proteinopathies in a broad spectrum of neurodegenerative diseases, including Alzheimer disease.[5,9,18] Secondary proteinopathies in neurodegenerative diseases is a frequent occurrence, and CTE, as a trauma-induced neurodegenerative disease, may be accompanied by secondary proteinopathies in different cases of CTE.

Conclusions

In this paper we present the case of a 27-year-old USMC Iraqi war veteran who developed persistent impaired neuropsychiatric and cognitive functioning, and mood disorders, following deployments to Iraq and an honorable discharge. He was clinically diagnosed with PTSD and prescribed neurotropic drugs. He eventually committed suicide by hanging. Autopsy, as well as gross and histomorphological examination of his brain, revealed CTE changes similar to the CTE changes we have observed in American athletes. Chronic traumatic encephalopathy is the cumulative outcome of repeated subconcussive and concussive brain injuries, and in this instance, it is our opinion that the decedent sustained repeated subconcussive and concussive brain injuries primarily from exposures from blasts and secondarily from training activities and noncombat activities as a marine. Other possible tertiary nonmilitary contributory factors to his cumulative risk of developing CTE may have included a remote traumatic history of nasal bone fracture and engagement in contact sports such as football and hockey for leisure. This sentinel case highlights the need for forensic, observational, and translational research to further confirm that a proportion of PTSD cases in war veterans may be due to, or contributed to by, CTE caused by repeated subconcussive and concussive traumatic brain injuries.

Disclosure

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

Author contributions to the study and manuscript preparation include the following. Conception and design: Omalu, Hammers, Bailes, Fitzsimmons. Acquisition of data: Omalu, Hammers, Hamilton, Kamboh, Webster. Analysis and interpretation of data: Omalu, Hamilton, Kamboh. Drafting the article: Omalu. Critically revising the article: Omalu, Hammers, Bailes, Fitzsimmons. Reviewed submitted version of manuscript: Omalu, Hammers, Bailes, Webster, Fitzsimmons. Approved the final version of the manuscript on behalf of all authors: Omalu. Administrative/technical/material support: all authors. Study supervision: Omalu, Bailes, Fitzsimmons.

References

1. American Psychiatric Association: **Diagnostic and Statistical Manual of Mental Disorders, ed 4, Text Revision (DSM-IV-TR).** Washington, DC: American Psychiatric Association, 2000
2. Bales KR, Dodart JC, DeMattos RB, Holtzman DM, Paul SM: Apolipoprotein E, amyloid, and Alzheimer disease. **Mol Interv 2:**339, 363–375, 2002
3. Belanger HG, Kretzmer T, Vanderploeg RD, French LM: Symptom complaints following combat-related traumatic brain injury: relationship to traumatic brain injury severity and posttraumatic stress disorder. **J Int Neuropsychol Soc 16:**194–199, 2010
4. Chen Y, Huang W: Non-impact, blast-induced mild TBI and PTSD: concepts and caveats. **Brain Inj 25:**641–650, 2011
5. Chen-Plotkin AS, Lee VM, Trojanowski JQ: TAR DNA-binding protein 43 in neurodegenerative disease. **Nat Rev Neurol 6:**211–220, 2010
6. Department of Defense Task Force on the Prevention of Suicide by Members of the Armed Forces: **The Challenge and the Promise: Strengthening the Force, Preventing Suicide and Saving Lives.** Washington, DC: US Department of Defense, 2010, p 233
7. Elder GA, Mitsis EM, Ahlers ST, Cristian A: Blast-induced mild traumatic brain injury. **Psychiatr Clin North Am 33:** 757–781, 2010
8. Ellison D, Love S, Chimelli L, Harding BN, Lowe J, Vinters HV: **Neuropathology: A Reference Text of CNS Pathology, ed 2.** New York: Mosby, 2004
9. Geser F, Lee VM, Trojanowski JQ: Amyotrophic lateral sclerosis and frontotemporal lobar degeneration: a spectrum of TDP-43 proteinopathies. **Neuropathology 30:**103–112, 2010
10. Greenfield JG, Love S, Louis DN, Ellison D: **Greenfield's Neuropathology, ed 8.** London: Hodder Arnold, 2008
11. Jordan BD, Relkin NR, Ravdin LD, Jacobs AR, Bennett A, Gandy S: Apolipoprotein E epsilon4 associated with chronic traumatic brain injury in boxing. **JAMA 278:**136–140, 1997
12. Kristman VL, Tator CH, Kreiger N, Richards D, Mainwaring L, Jaglal S, et al: Does the apolipoprotein epsilon 4 allele predispose varsity athletes to concussion? A prospective cohort study. **Clin J Sport Med 18:**322–328, 2008
13. Kutner KC, Erlanger DM, Tsai J, Jordan B, Relkin NR: Lower cognitive performance of older football players possessing apolipoprotein E epsilon4. **Neurosurgery 47:**651–658, 2000
14. Ludwig J: **Handbook of Autopsy Practice, ed 3.** Totowa, NJ: Humana Press, 2002
15. Mac Donald CL, Johnson AM, Cooper D, Nelson EC, Werner NJ, Shimony JS, et al: Detection of blast-related traumatic brain injury in U.S. military personnel. **N Engl J Med 364:**2091–2100, 2011
16. McKee AC, Cantu RC, Nowinski CJ, Hedley-Whyte ET, Gavett BE, Budson AE, et al: Chronic traumatic encephalopathy in athletes: progressive tauopathy after repetitive head injury. **J Neuropathol Exp Neurol 68:**709–735, 2009
17. McKee AC, Gavett BE, Stern RA, Nowinski CJ, Cantu RC, Kowall NW, et al: TDP-43 proteinopathy and motor neuron disease in chronic traumatic encephalopathy. **J Neuropathol Exp Neurol 69:**918–929, 2010
18. Neumann M, Sampathu DM, Kwong LK, Truax AC, Micsenyi MC, Chou TT, et al: Ubiquitinated TDP-43 in frontotemporal lobar degeneration and amyotrophic lateral sclerosis. **Science 314:**130–133, 2006

19. Omalu B: **Play Hard, Die Young: Football Dementia, Depression, and Death.** Lodi, CA: Neo-Forenxis Books, 2008
20. Omalu B, Bailes J, Hamilton RL, Kamboh MI, Hammers J, Case M, et al: Emerging histomorphologic phenotypes of chronic traumatic encephalopathy in American athletes. **Neurosurgery 69:**173–183, 2011
21. Omalu BI, Bailes J, Hammers JL, Fitzsimmons RP: Chronic traumatic encephalopathy, suicides and parasuicides in professional American athletes: the role of the forensic pathologist. **Am J Forensic Med Pathol 31:**130–132, 2010
22. Omalu BI, DeKosky ST, Hamilton RL, Minster RL, Kamboh MI, Shakir AM, et al: Chronic traumatic encephalopathy in a National Football League player: Part II. **Neurosurgery 59:** 1086–1093, 2006
23. Omalu BI, DeKosky ST, Minster RL, Kamboh MI, Hamilton RL, Wecht CH: Chronic traumatic encephalopathy in a National Football League player. **Neurosurgery 57:**128–134, 2005
24. Omalu BI, Fitzsimmons RP, Hammers J, Bailes J: Chronic traumatic encephalopathy in a professional American wrestler. **J Forensic Nurs 6:**130–136, 2010
25. Omalu BI, Hamilton RL, Kamboh MI, DeKosky ST, Bailes J: Chronic traumatic encephalopathy (CTE) in a National Football League player: case report and emerging medicolegal practice questions. **J Forensic Nurs 6:**40–46, 2010
26. Robbins S: Doctors study link between combat and brain disease. **Stars and Stripes.** January 23, 2010 (http://www.stripes.com/news/doctors-study-link-between-combat-and-brain-disease-1.98394) [Accessed September 23, 2011]
27. Ropper A: Brain injuries from blasts. **N Engl J Med 364:** 2156–2157, 2011
28. Rosenfeld JV, Ford NL: Bomb blast, mild traumatic brain injury and psychiatric morbidity: a review. **Injury 41:**437–443, 2010
29. Samatovicz RA: Genetics and brain injury: apolipoprotein E. **J Head Trauma Rehabil 15:**869–874, 2000
30. Shalev AY: What is posttraumatic stress disorder? **J Clin Psychiatry 62 (Suppl 17):**4–10, 2001
31. World Health Organization: **The ICD-10 Classification of Mental and Behavioural Disorders: Clinical Descriptions and Diagnostic Guidelines.** Geneva: World Health Organization, 1992
32. Yule W: Posttraumatic stress disorder in the general population and in children. **J Clin Psychiatry 62 (Suppl 17):**23–28, 2001

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Development of an iPhone application for sideline concussion testing

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Professional athletes are taking concussion very seriously, and missed play due to concussion is no longer stigmatized. One fortuitous consequence is increased awareness of the detrimental effects of concussion among student athletes. Whereas professional athletes have access to formal in-competition evaluation and out-of-competition monitoring programs, the majority of student athletes, especially at the middle school and high school levels, do not. The authors therefore set out to create an easy-to-use iPhone application for sideline concussion testing and serial monitoring of these at-risk athletes. *(DOI: 10.3171/2011.8.FOCUS11186)*

Key Words • sports-related concussion • iPhone app • Sport Concussion Assessment Tool

Much of the current discussion regarding sports-related concussion is focused on prevention and return-to-play guidelines. This is appropriate, because prevention of the initial injury and subsequent reinjury is of paramount importance. The model concussion prevention legislation, Washington State's Zachery Lystedt Law, incorporates 3 key aspects, as follows: 1) athletes, parents, and coaches are required to receive yearly concussion education; 2) an athlete suspected of having a concussion must be removed from competition and not returned to play; and 3) a licensed health care professional must clear the athlete to return to play.[3] Implicit is the need for a standardized method to identify the concussed athlete during competition.

To this end, the "Consensus statement on concussion in sport," generated during the 3rd International Conference on Concussion in Sport was widely published in 2009.[2] Germane to the current paper was the recommendation that "Once the first aid issues are addressed, then an assessment of the concussive injury should be made using the SCAT2 or other similar tool." The SCAT2 "represents a standardized method of evaluating injured athletes aged from 10 years or older." The SCAT2 can be used for preseason baseline evaluation, and it is available at no charge for distribution and use.

There are, however, barriers to the use of concussion test instruments like SCAT2: namely, lack of awareness and inconvenience. Lack of awareness regarding sports-related concussion and of the availability of standardized testing platforms are being addressed through legislation and campaigns like the CDC's "Heads Up: Concussion in Youth Sports." The pencil and paper SCAT2 survey is inconvenient to use on the sideline (especially when the weather turns foul), and dissemination of test data is more difficult. A digital format, particularly a smart-phone application, breaches these barriers. In this paper, we report our experience with the development of an iPhone app for concussion testing.

Methods

The app development team included a neurosurgeon (R.A.R.) to provide the medically relevant content, a graphic artist (G.A.C.) to design the user interface, and a computer programmer (N.S.) to translate the vision into the computer's language.

Abbreviations used in this paper: app = application; CDC = Centers for Disease Control; CSV = comma-separated values; SCAT2 = Sport Concussion Assessment Tool 2.

During the planning process, we identified several desirable functions to incorporate into the app, as follows.

1. *SCAT2 elements*. The purpose of our app is to help determine if an injured athlete shows signs and symptoms of concussion. We therefore built the app around test elements from the well-established SCAT2 platform.

2. *Ease of use*. Because the app will be used to evaluate injured athletes in the heat of battle, it has to be easy to use.

3. *Multiple uses*. Many school boards, particularly in rural areas, have budget and personnel limitations that make it difficult to implement a concussion-testing platform like ImPACT (Immediate Post-Concussion Assessment and Cognitive Testing). We planned an app that could be used for preseason baseline evaluations, in-competition testing, and postconcussion monitoring.

4. *Data transmission*. The data generated from our app are compiled in the CSV format so that it can be directly entered into a database or spreadsheet program.

5. *Adaptability*. Although the app is currently based on SCAT2, we anticipate that other sideline concussion test strategies will be validated in the future. We wanted the flexibility to add this functionality to forthcoming versions of the app.

6. *Security and backup*. Because the app will store private health information, security is needed to prevent unwanted data access should the device be borrowed, lost, or stolen. To this end we incorporated a login screen requiring a user name and password. There is a quick test feature allowing access to the test elements (but not private health information) in the event of an emergency. Also planned is a synchronizing feature that will back up test data to a Health Insurance Portability and Accountability Act–compliant server hosted by Marquette General Health System.

Next came the graphic design and programming. Throughout this stage, simulated versions of the app were evaluated to be certain that a product consistent with our desired functionality took shape.

Last, the app was submitted to the Apple app store for testing and approval.

Results

The resulting app is named Concussion Test (Fig. 1). There is no charge to download it through the iTunes store.

In Concussion Test, there are several ways to begin the testing protocol. First, an athlete can be selected from the player roster. In this scenario, an athlete's name and his/her demographic information are stored in the app, along with preseason baseline concussion testing. Second, an athlete's demographic information can be entered right before the concussion test is administered. Third, there is a quick-test option that permits direct entry into the test elements. This is especially helpful if there is a need to evaluate an athlete who is not part of the team. At the end of the test elements, the option of adding the test data to an existing player or to a new player is available.

In addition to general demographic information, the app also stores results of prior concussion tests and the number of previous concussions, general medical information useful in emergency situations, and emergency contact information.

Fig. 1. Logo for the Concussion Test app.

The testing protocol is based on SCAT2 and includes the following elements: concussion symptom survey, modified Maddocks questions, balance test, immediate and delayed word recall, reverse digit recall, and reverse month recall (Fig. 2A and B). To prevent an athlete from memorizing the words and digits through repetitive testing, the app automatically refreshes the word and digits list with each new test. This functionality is not available with the traditional pencil and paper SCAT2.

At the end of the test, a results screen appears, providing a plain-language summary of the data and recommendations regarding return to competition (Fig. 2C). Test results can then be assigned to an existing player or a new player. An email screen (with the test data attached as a CSV file) permits distribution of results to members of the traumatic brain injury team (Fig. 2D).

This app also features a hospital locator—helpful if the team is competing away from home.

Discussion

Concussion recognition and prevention is not a trivial endeavor. Scientific data support the conclusion that repetitive, mild head injury leads to the late sequelae of chronic traumatic encephalopathy.[1] Moreover, it is abundantly clear that the catastrophic second-impact syndrome is both real and preventable.[4,5] It is imperative, then, that the concussed athlete be identified (especially when signs and symptoms are subtle), and then allowed to return to play only after resolution of symptoms. These tasks can best be accomplished by combining clinical judgment with a standardized, validated tool.

Through a combination of legislation and awareness

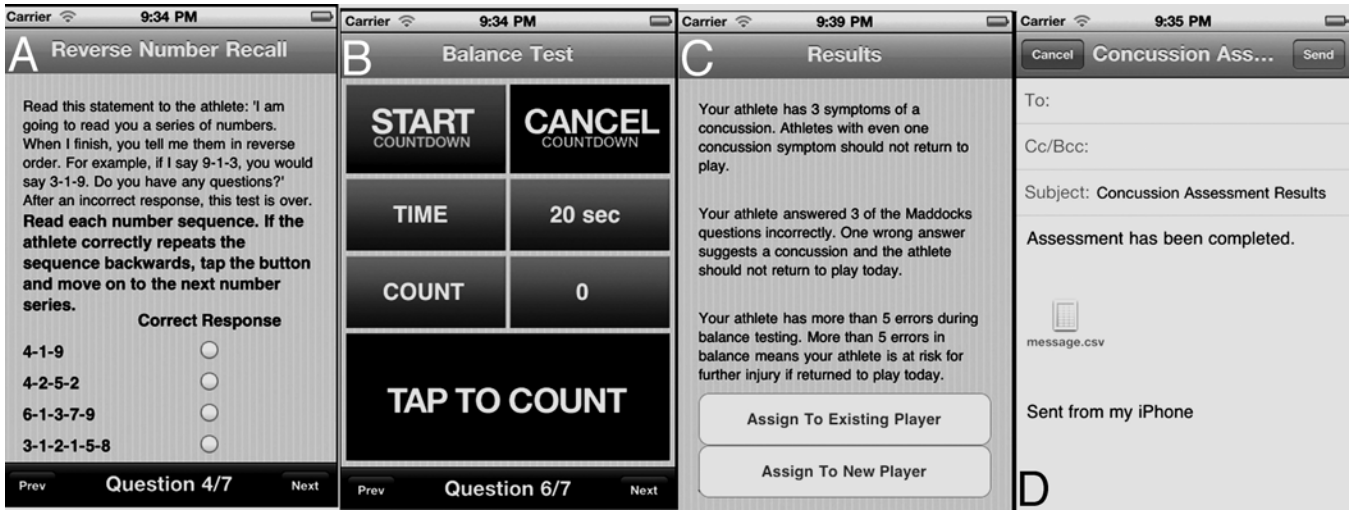

Fig. 2. Selected screen shots demonstrating several of the app's functions. **A:** The Reverse Number Recall screen. To avoid memorization, the number sequences change with each new test. **B:** The Balance Test screen has both a countdown timer and an event timer (tap to count) to record the number of errors made in 20 seconds. **C:** The Results summary screen. Based on test responses, a recommendation regarding return to play is provided in plain language. There is also the option to assign test results to an existing player or to a new player. **D:** The email screen. Test results are automatically attached as a CSV file, and can be sent to appropriate members of the traumatic brain injury program team.

programs, concussion assessment tools will be increasingly used. A digital format is advantageous due to easier data collection, analysis, distribution, and storage. A mobile-phone app offers the ideal combination of functionality and portability. We therefore developed an iPhone app to assist with in-competition diagnosis of concussion and to permit the serial evaluations necessary to inform decisions about return to play.

There are several concerns with a mobile platform: cost, potential for abuse, and validity. The Concussion Test app is free. Nevertheless, the lowest-priced iPod Touch is $229.00, so there certainly is a greater expense compared with the pencil and paper SCAT2 survey. We anticipate that the advantages of the digital format compensate for this initial capital expense. Can sideline concussion assessments be maliciously used to return an injured athlete to competition? Sure. However, that would require the test administrator to actively falsify the athlete's answers. The mobile format is no more vulnerable to this than the pencil and paper version.

Last, does a mobile (or digital) version of a pencil and paper survey need to be additionally validated? Our opinion is that as long as the method of presenting the question does not affect the thought processes leading to the answer, then additional validation is unnecessary. For both the pencil and paper SCAT2 survey and our app, an independent party administers the test elements and, therefore, the method of presentation is the same from the perspective of the athlete being tested. Moreover, there really is not a good way to establish concurrent validity between our app and the pencil and paper version. Sequential administration of the 2 surveys could easily result in different answers because concussion signs and symptoms can fluctuate over time. Also, there is often time pressure to complete the testing, rendering administration of 2 complete surveys impractical.

Ours is not the first concussion-related iPhone app. When we began app development 1 year ago (July 2010), a search of the iTunes store for "concussion" yielded no results. Now (July 15, 2011), the same search produces 5 apps:

- *SCAT2*, Meeuwisse Consulting Services, free
- *SCAT2*, Wayne Hans, $3.99
- *Concussion*, SportSafety Lab, free with $4.99 in app purchases
- *Concussion Recognition and Response: Coach and Parent Version*, Psychological Assessment Resources, $3.99
- *Play it Safe Concussion Assessment*, Concussion Health, free

The first 3 apps are SCAT2-based. The Concussion Recognition and Response app is based on the CDC's "Heads Up: Concussion in Youth Sports" program. The Play it Safe app is based on the Concussion Health program developed by the University of Texas Athletic Department. These apps are all well conceived and executed. Although a detailed review of these apps is beyond the scope of this paper, the growing number of concussion-related iPhone apps is a testament to the feasibility and practicality of the concept.

By providing coaches, parents, athletes, and health and athletic personnel a comprehensive and easy to use tool to identify and monitor student athletes suspected of having a concussion, smart-phone apps will increase awareness of the dangers of concussion and improve decision making with regard to removal from play and return to play. To paraphrase, there should be no doubt when to sit them out.

Conclusions

Mobile-phone apps, due to their popularity and ease of use, offer an excellent platform to promote wellness and prevention agendas such as concussion recognition and second-impact syndrome prevention. Neurosurgeons can and should use their expertise to help develop medically relevant smart-phone apps. Development of an app is a collaborative and cooperative process, and thus plays to neurosurgeons' strengths.

Disclosure

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

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References

1. Gavett BE, Stern RA, McKee AC: Chronic traumatic encephalopathy: a potential late effect of sport-related concussive and subconcussive head trauma. **Clin Sports Med 30:**179–188, xi, 2011
2. McCrory P, Meeuwisse W, Johnston K, Dvorak J, Aubry M, Molloy M, et al: Consensus statement on concussion in sport—the 3rd International Conference on concussion in sport, held in Zurich, November 2008. **J Clin Neurosci 16:** 755–763, 2009
3. NFL Health and Safety: **Lystedt Law Overview.** (http://nflhealthandsafety.com/zackery-lystedt-law/lystedt-law-overview/) [Accessed September 15, 2011]
4. Thomas M, Haas TS, Doerer JJ, Hodges JS, Aicher BO, Garberich RF, et al: Epidemiology of sudden death in young, competitive athletes due to blunt trauma. **Pediatrics 128:**e1–e8, 2011
5. Wetjen NM, Pichelmann MA, Atkinson JL: Second impact syndrome: concussion and second injury brain complications. **J Am Coll Surg 211:**553–557, 2010

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Traumatic brain injury in pediatric patients: evidence for the effectiveness of decompressive surgery

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Traumatic brain injury (TBI) is the current leading cause of death in children over 1 year of age. Adequate management and care of pediatric patients is critical to ensure the best functional outcome in this population. In their controversial trial, Cooper et al. concluded that decompressive craniectomy following TBI did not improve clinical outcome of the analyzed adult population. While the study did not target pediatric populations, the results do raise important and timely clinical questions regarding the effectiveness of decompressive surgery in pediatric patients. There is still a paucity of evidence regarding the effectiveness of this therapy in a pediatric population, and there is an especially noticeable knowledge gap surrounding age-stratified interventions in pediatric trauma. The purposes of this review are to first explore the anatomical variations between pediatric and adult populations in the setting of TBI. Second, the authors assess how these differences between adult and pediatric populations could translate into differences in the impact of decompressive surgery following TBI. *(DOI: 10.3171/2011.8.FOCUS11177)*

Key Words • clinical trial • pediatric • traumatic brain injury • decompressive surgery

An estimated 1 in 10 (5.3 million) of the 54 million Americans living with disabilities have a disability caused by TBI (www.hhs.gov).[49]Approximately 475,000 TBIs occur among children ages 0–14 years old every year in the US (www.cdc.gov), and the current leading cause of death in children more than 1 year old is TBI. Different pediatric age groups experience different causes for their injury. In infants, the most common causes are falls and physical assaults. In toddlers and young children, car accidents and falls are most common. In children and teenagers, car accidents and sports are the most common causes.[30]

Proper management and care of pediatric patients is therefore crucial to improve functional outcome in this population. Recent advances have been made in the more general field of adult TBI, including the conclusion of a controversial trial that analyzed the clinical effectiveness of decompressive craniectomy following TBI in improving outcome in adults.[16] The study did not explicitly target pediatric populations; however, the results of the study do raise important and timely clinical questions regarding the effectiveness of decompressive surgery in pediatric patients. There is still a paucity of evidence regarding the effectiveness of this therapy in this population, and there is an especially noticeable knowledge gap surrounding age-stratified interventions in pediatric trauma.

We have several goals in this review. First, we explore the anatomical variations between pediatric and adult populations in the setting of TBI. Second, we note the epidemiological and physiological differences within age-stratified pediatric populations. Third, we assess how these differences between adult and pediatric populations could translate into differences in the impact of decompressive surgery following TBI by analyzing current knowledge on the utilization of craniectomy as a treatment in these different populations.

Physiology

Pediatric populations represent an important but often under-defined population in the literature investigating TBI. Many adult trials of decompressive craniectomy following TBI include patients as young as 10 or 15 years old in their protocols,[16,26] demonstrating a failure to dis-

Abbreviations used in this paper: CPP = cerebral perfusion pressure; DECRA = DEcompressive CRAniectomy; GCS = Glasgow Coma Scale; ICP = intracranial pressure; RCT = randomized controlled trial; RESCUEicp = Randomised Evaluation of Surgery with Craniectomy for Uncontrollable Elevation of Intra-Cranial Pressure; TBI = traumatic brain injury.

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tinguish between adult and pediatric populations. However, significant physiological, anatomical, and pathological differences exist between the ages of birth and adulthood, resulting in the need for clear definitions in patient populations. For the purposes of this review, the pediatric population includes children from the moment of birth until 18 years of age.

Damage following TBI is the result of primary and secondary injuries on the brain. Primary injury is largely due to shearing forces between brain tissue of different densities, specifically between skull and dura mater, dura mater and gray matter, and white matter and gray matter.[22] Secondary physiological injury, however, is also responsible for a large amount of morbidity and death. Secondary injury can result from impaired cerebral blood flow, regional edema, hemorrhage, elevated ICP and therefore reduced CPP, dysfunction of ion pumps, excessive release of neurotransmitters, cascade of cellular destruction via reactive oxygen species, proteolysis, and inflammation.[19] These processes can often lead to ischemia, infarct, and necrosis.

Intracranial pressure is one of the most important brain physiological variables, especially following TBI.[21,25,30,42] Control of ICP is therefore crucial in preventing secondary injury. Healthy adults normally maintain ICP values below 20–25 mm Hg, although this exact threshold is not clear in any age group.[10]

Anatomy

There are multiple anatomical mechanisms for injury to the brain. In both pediatric and adult populations, the brain is cushioned by a surrounding layer of CSF and infused by layers of vessels that provide structural and nutritional support. The brain is protected further by layers of pia, arachnoid, and dura mater, surrounded by a bony skull. In young children, the skull has not yet calcified completely and is less capable of distributing pressure. By adulthood, the skull has hardened into a sandwich of cortical bone around a spongy diploë and can resist impact fracture at 11 times the force strength of neonates.[38]

Anatomical and mechanical variations between adults and children explain some of the differences between the 2 age groups for severity of and response to TBI. Children have smaller brains than adults, and researchers have demonstrated in chimpanzee models that upon subjection to whiplash, smaller brains are less vulnerable than larger brains to the same amount of angular acceleration inducing injury.[39]

Relative to their body and compared with adults, however, children have large and heavy heads with weaker cervical neck muscles, which allows for a more forceful impact and a more severe injury. In young children, the skull is more pliable and incapable of withstanding bending loads. Cranial sutures are not yet fused, and upon impact, the soft skull deforms into the brain. More severe trauma is associated with plastic deformation and cracking of the skull.[38]

Differences in TBI Within the Pediatric Population

Many elements influence outcome following TBI, including patient age, impact severity, physiological variables, anatomical variations, and especially control of ICP. Although not definitively proven, children are believed to have stratified values of normal ICP based on their age. Infants maintain ICP of 2–4 mm Hg, while older children typically maintain their normal ICP range between 5 and 15 mm Hg.[2] These values of ICP correlate with CPP guidelines implemented in 1997, whereby adequate CPP per age group is defined as ≥ 30 (neonate), ≥ 40 (1 month to 1 year), ≥ 50 (1–4 years), ≥ 60 (5–8 years), and ≥ 70 mm Hg (> 8 years)[11,48] (Table 1). However, a more recent clinical trial has noted lower values for critical minimum CPP, with values of 48, 54, and 64 mm Hg for children ages 2–6, 7–10, and 11–15, respectively (Table 1).[13]

The thresholds for ICP hypertension in children requiring treatment are generally considered to be lower than in adults, with the threshold in infants approximately 15 mm Hg and in young children between 15 and 20 mm Hg.[2] Several clinical studies support these age-stratified values in pediatrics, noting that neurological outcome is improved when medical treatments targeted at maintaining ICP below 20 mm Hg are used.[20,46]

Recent studies, both clinical and basic science, show variation in clinical outcome following TBI between children and adults (Table 2). Worse outcomes might be predicted in pediatrics because children have a higher incidence of edema following TBI and a reduced antioxidative capacity compared with adults.[6,38] Also, children experience a higher incidence of hypotensive episodes following TBI, which decreases CPP.[8] Finally, the young brain normally receives a higher percentage of cardiac output compared with the adult brain, and children are therefore at higher risk for ischemia following TBI due to their dependence on a higher perfusion rate.[45,52] Recent evidence has shown that older teens generally demonstrate better outcomes than younger children, perhaps due to greater vulnerability among younger children to more severe physical injury.[3,4,28,32] Despite these factors, however, children overall tend to have better clinical outcomes following TBI than adults.[3,4,28]

To investigate the reasons for such differences in outcomes, many animal models have been used to compare young versus old age and TBI. Studies have examined the role of biomechanics,[35] metabolism,[43] cell death,[9,25] electrophysiology,[17,44] and glutamatergic neurotransmission[23,24] as possible mechanisms to explain the improved clinical outcome in pediatric compared with adult patients. An additional mechanism by which immature brain tissue may recover better following TBI is due to the presence of low levels of chondroitin sulfate proteoglycan glycoproteins compared with adult tissue. This important matrix component provides rigidity and support to the brain parenchyma, and lower levels of the glycoprotein are associated with increased plasticity. This lower level may be 1 mechanism by which the developing brain can maintain plasticity and rapidly remodel following injury.[5,41,51]

Surgical Interventions for TBI in the Pediatric Population

Because ICP following TBI is widely regarded as an

TABLE 1: Comparison of 2 different clinical measures of minimum adequate CPP

Authors & Year	Age	Adequate CPP (mm Hg)
Bullock et al., 1996	neonate	≥30
	1 month–1 yr	≥40
	1–4 yrs	≥50
	5–8 yrs	≥60
	>8 yrs	≥70
Chambers et al., 2006	2–6 yrs	>48
	7–10 yrs	>54
	11–15 yrs	>60

important physiological determinant of brain function and clinical outcome,[21,25,30,42] many approaches have been developed to control ICP. However, despite the existence of guidelines for management following TBI, there is a lack of data surrounding specific interventions, especially in pediatric populations. Most guidelines focus on ICP control as a means to maintain adequate CPP.[34] Current medical treatments to reduce ICP include hyperosmolar therapy (such as mannitol or hypertonic saline),[53] hyperventilation,[15,47] sedation and paralytics, and head of bed elevation. In addition, barbiturate-induced coma[2] and hypothermia[7] have been shown to reduce cerebral metabolic rate and oxygen demands, offering a protective role.[2] If medical treatment is ineffective in normalizing ICP, several types of surgical intervention are possible. Surgical methods used include hematoma evacuation, ventricular drains, and craniectomy. Hematoma evacuation is indicated in the setting of a hemorrhagic mass lesion, while a ventricular drain is used in the setting of hydrocephalus or when CSF drainage is desired. The third method, craniectomy, is the subject of many current clinical trials. The overall goal is to increase the total volume of the cranial cavity by removing a large portion of the skull, thereby lowering ICP and reducing the incidence of secondary injury to the brain.

Several approaches to decompressive craniectomy exist. In both children and adults, a bilateral frontotemporoparietal craniectomy is often used for diffuse bilateral swelling, while a unilateral frontotemporoparietal craniectomy is chosen for unilateral brain swelling.[21,25,30] A variety of specialized techniques have also been reported in children, including smaller, 4-cm bitemporal craniectomies,[48] larger craniectomies,[25,28] and craniectomy combined with expansion duraplasty.[14,18,42] To date no study has compared the efficacy of the various techniques in improving outcome. Furthermore, the timing of decompressive craniectomy and its effect on clinical outcome have not been clearly established in pediatric patients.

Current opinion on the effectiveness of craniectomy in improving clinical outcome in pediatric patients following TBI is divided. Reduction of refractory ICP has been shown to be a major predictor of mortality following TBI,[21,25,31,42] and many studies have confirmed the positive effects of craniectomy on reducing ICP following TBI.[1,37,49,50] However, few randomized controlled studies exist that specifically address the effectiveness of craniectomy in pediatric populations to improve clinical outcome. In most current adult and pediatric studies, refractory ICP hypertension is often defined as ICP > 20 mm Hg for some defined time period.[14,20,40] However, no standard definition of ICP hypertension exists in pediatric patients, which poses difficult challenges for designing and comparing clinical trials and results.

TABLE 2: Comparison of prognostic factors following TBI between children and adults

Prognosticator	Children	Adults
edema	higher incidence	lower incidence
antioxidative capacity	lesser	greater
hypotensive episodes	higher incidence	lesser incidence
% of cardiac output	higher	lesser
age stratification	younger children more vulnerable to severe injury	younger adults more favorable outcome
basement membrane glycoproteins	lower levels of chondroitin sulfate	higher levels of chondroitin sulfate
physiological variables examined in animals	biomechanics, cell death, metabolism, electrophysiology, glutamatergic neurotransmission	biomechanics, cell death, metabolism, electrophysiology, glutamatergic neurotransmission

Clinical Trials of TBI

Traumatic brain injury trials are inherently challenging. Guidelines recommend prespecified baseline prognostic criteria, broad inclusion criteria, and ordinal statistical analysis to maximize efficiency and generalizability of the results.[33] Additionally, because functional outcome following TBI can change greatly from 1 to 5 years postincident, long follow-up periods greater than 1 year are needed.[12] The 5 trials summarized in Table 3 are the most significant and recent in the field of craniectomy following pediatric and adult TBI. Of these studies, only the Taylor et al.[48] and Kan et al.[27] trials are dedicated pediatric studies. The remaining 3 studies analyze primarily adult populations with overlap into the pediatric age range. All 5 studies were of small sample size, with numbers of patients ranging from 27 to 309. Additionally, 2 of the 5 studies, conducted by Polin et al.[42] and Kan et al.,[27] were retrospective studies.

Taylor et al., 2001

Completed in 2001, the Taylor et al.[48] RCT investigated the clinical effectiveness of very early application of craniectomy in children with TBI. Using functional outcome at 6 months after intervention as the primary outcome and the control of ICP as a secondary outcome, the authors demonstrated positive outcomes in craniectomy patients compared with noncraniectomy patients, as well as a large decrease in ICP.

The inclusion criteria for study participants were refractory intracranial hypertension, defined as sustained ICP during the 1st day after admission (> 20 mm Hg for

TABLE 3: Comparison of 5 clinical studies of decompressive surgery following pediatric mild TBI*

Study Characteristics	Taylor et al., 2001	Polin et al., 1997	Kan et al., 2006	Cooper et al., 2011	Hutchinson et al., TBD
no. of patients	27	70	51	155	309 (to date)
age range or median	10.7 yrs	18.7 yrs	6.6 yrs	23.7 yrs (surgery)/24.6 yrs (SOC) 15–59 yrs	10–65 yrs
methods	RCT; ICP ≤20 mm Hg; surgery <6 hrs postrandomization; 4-cm bitemporal craniotomy via bilateral vertical incision in midtemporal region	retrospective study; bifrontal craniectomy to relieve refractory ICP; surgical patients matched w/ SOC control	retrospective study; decompressive craniectomy performed in children between 1996 and 2005	RCT; ICP maintained ≤20 mm Hg; Marshall criteria; injury severity score; trauma score; treatment <72 hrs postictus	RCT; ICP ≤25 mm Hg
description	clinical effectiveness of very early craniectomy in children w/ TBI	retrospectively compared craniectomy following TBI to appropriately matched controls	analyzed postop mortality and morbidity following TBI in children	clinical effectiveness of craniectomy following brief refractory ICP HTN after TBI	clinical effectiveness of craniectomy following brief refractory ICP HTN after TBI
inclusion criteria	>12 mos old w/ head injury; refractory ICP HTN (>20 mm Hg/30 min, >25 mm Hg/10 min, >30 mm Hg/1 min); evidence of herniation	head injury; refractory ICP HTN (despite mild hyperventilation, elevation of bed, mannitol, or barbiturate); max ICP >20 mm Hg	severe head trauma; elevated ICP	head injury; abnormal CT scan requiring ICP monitoring; ICP >20 mm Hg >15 min despite first-line treatments	head injury; abnormal CT scan requiring ICP monitoring; refractory ICP (>25 mm Hg for 1–12 hrs)
exclusion criteria	none	GCS score >7	none	cerebral mass lesion; successful control of ICP w/ first therapies	bilat fixed and dilated pupils; bleeding diathesis; devastating injury not expected to survive >24 hrs
major findings	positive outcomes in craniectomy group; decreased ICP in surgical group vs SOC	surgery group had better functional outcome vs SOC; decreased ICP in surgical vs SOC; in most favorable pediatric population, large advantage of craniectomy	high rate of mortality in children undergoing decompressive surgery for raised ICP; hydrocephalus and epilepsy common complications of surgery following TBI	better outcome following second-line medical therapy after brief, modest elevation in ICP unresponsive to first-line medical therapy	TBD
limitations	small trial size; no age stratification; long study over 7 yrs, functional outcome only studied at 6 mos	narrow inclusion criteria; low population sample; retrospective protocol; "favorable population" defined narrowly: surgery within 48 hrs, no ICP >40 mm Hg; age of the population	small study population; retrospective protocol; only 6 patients underwent craniectomy for raised ICP only	large screened population w/ narrow inclusion population; baseline patient differences (pupil dilation); inappropriate low threshold for ICP; large crossover rate (23%); interquartile ICP 18–22 mm Hg, questionable if meaningful HTN	TBD

* HTN = hypertension; SOC = standard of care; TBD = to be determined.

30 minutes, > 25 mm Hg for 10 minutes, or > 30 mm Hg for 1 minute), or clinical evidence of herniation, represented by pupil dilation. Of the control group patients, 1 patient had fixed pupils preoperatively, and in the surgery group, 3 patients had fixed pupils preoperatively. Surgery was performed within 6 hours of randomization, with each patient in the surgery group receiving a 4-cm bitemporal craniotomy via a bilateral vertical incision in the midtemporal region. As noted in the Adelson guidelines, this surgical operation is smaller than historical craniectomies for relief of ICP.[25,29] Cranioplasty was performed several months later if indicated.

The study demonstrated positive results for the implementation of early craniectomy in children with TBI. Seven of 13 surgery patients obtained a favorable outcome at 6 months, while only 2 of 14 medical patients obtained a favorable outcome ($p = 0.046$). However, due to the nature of the repeated significance testing performed during the test, a p value < 0.021 was required for statistical significance for this test, per the explanation of the authors. Additionally, surgery patients demonstrated an ICP decrease of 8.98 mm Hg during the 48-hour period following intervention (95% CI 4.987–12.968), while medical patients demonstrated a decrease of 3.39 mm Hg over the same 48-hour period (95% CI −0.435 to 7.807). Finally, surgery patients demonstrated fewer ICP spikes (> 20 mm Hg) than medical patients (107 vs 223, respectively).

In comparison with the recent adult craniectomy trials described below, the Taylor et al. 2001 pediatric trial has important findings and implications. The authors hypothesized that the implementation of an early craniectomy in refractory ICP hypertensive patients would result in better patient outcomes than the historical standard of care, which reserved surgery as a final intervention in refractory ICP hypertension following TBI.[21,42] Based on their preliminary findings, the use of early craniectomy may result in better functional outcomes at 6 months in pediatric patients. However, this trial is limited by several factors. First, there was a small trial size of only 27 patients, and future studies would need to greatly expand the patient number. Second, there are numerous anatomical, mechanical, and physiological differences between the infant brain and the adolescent brain, as explained earlier, and future trials will need to address these issues through age stratification. Third, the study was long, lasting over 7 years, and functional outcome evaluation was only performed early in the recovery period, at 6 months. Another study has noted that long-term recovery following TBI can change greatly from 1 to 5 years postincident.[12]

Kan et al., 2006

A small patient study, the Kan et al.[27] analysis of clinical effectiveness of craniectomy following severe TBI in children was completed in 2006. The study was a retrospective analysis of 51 children's records following craniectomy to either relieve ICP (6 children) or to relieve ICP and evacuate a hematoma (45 children). The authors found that craniectomy for the purpose of relieving ICP alone was associated with high levels of morbidity and mortality; however, strong conclusions cannot be drawn as only 6 patients were included in this specific cohort.

Few additional studies exist that specifically investigate the role of craniectomy in pediatric populations following TBI. The Taylor et al. study is the largest study to date, but has a small sample size of 27 patients. In a smaller study, Cho and colleagues[14] reported significant decreases in ICP in 10 children < 2 years old (from a mean of 59 mm Hg preoperatively to a mean of 12 mm Hg postoperatively).

Polin et al., 1997

Rather than performing a prospective RCT, Polin et al.[42] selected 35 patients who had undergone a bifrontal craniectomy for the purpose of relieving refractory ICP following TBI. Refractory ICP was defined in this study as patients who possessed elevated ICP despite mild hyperventilation, elevation of the head of the bed, mannitol administration, or barbiturate administration. The average patient age was 18.7 years old, all but 2 patients had a maximum ICP > 20 mm Hg, and no patient had a GCS score > 7 at the time of surgery. All surgical patients were matched with appropriately selected controls who had similar characteristics but did not undergo craniectomy. The average reduction in ICP in the surgery group was from 34.9 to 21.6 mm Hg and the average reduction in ICP in the control group was from 33.2 to 29.4 mm Hg ($p = 0.026$), indicating an advantage in the surgical group.

Most importantly, patients undergoing craniectomies demonstrated greater functional outcomes than control patients: 37% of craniectomy patients had a favorable outcome, compared with 16% of control patients ($p = 0.014$). When restricted to pediatric populations (< 18 years old), the difference was 44% versus 22%, respectively, although not statistically significant ($p = 0.079$). Finally, when the patient population was restricted to the most favorable target for craniectomy, defined as surgery performed within 48 hours of injury and no sustained ICP > 40 mm Hg, pediatric populations receiving craniectomies had an 80% favorable outcome but the control group demonstrated a 24% favorable outcome ($p = 0.002$).

Due to the trial's narrow inclusion criteria, low population sample, and retrospective analysis protocol, conclusions are limited. A significant implication, however, is that when the pediatric patient population is appropriately screened for therapeutic benefit as per the definitions of Polin et al., the use of craniectomy appears to improve functional outcome over the standard practice of medical care.

Cooper et al., 2011

The authors of the DECRA trial recently published controversial trial results[16] implicating worse outcome following craniectomy in patients with a brief, modest elevation in ICP following TBI (Table 3).

While it does not target pediatric craniectomy patients, and no pediatric data were reported, this study does raise important, controversial questions for the field. Additionally, the age range of participants in the study overlaps with pediatrics, including people from 15 to 59 years

old. The randomized, nonblinded study suggests that patients with brief, modest elevations in ICP unresponsive to first-line medical therapy who subsequently received second-line medical therapy (and life-saving surgery at the discretion of the surgeon) have better outcomes than those patients who undergo immediate decompression. Completed between December 2002 and April 2010, the trial enrolled 155 participants. The principal comparison was between the effectiveness of decompressive craniectomy and medical standard of care following TBI; endpoints examined were functional outcome at 6 months (primary outcome) and the ICP hypertension index (secondary outcome). The only surgical option considered was a bifrontal temporoparietal craniectomy with bilateral dural opening and without falx cerebri division. Based on the study's definition of inclusion criteria, ICP threshold, patient population characteristics, and intent to treat analysis, a larger unfavorable outcome was associated with surgery over medical therapy in their cohort, which included mostly adult patients. However, the trial did demonstrate reduced hourly ICP and reduced the ICP hypertension index among the surgery patients.

The trial suffers from several limitations, both specifically and for the purposes of this review. The study included confounding variables including baseline patient differences, questionably low threshold for ICP hypertension, and a large crossover rate. The majority of patient characteristics were similar at baseline, with the large exception that the craniectomy group included a higher percentage of participants with nonresponsive pupils than the standard of care group (27% vs 12%, respectively), which is an indicator of more severe prognosis.[36] The authors designated 20 mm Hg as their threshold for intracranial hypertension. While this value is generally accepted in pediatric studies, it has not received a Level I recommendation for the threshold for instituting therapy in adult populations.[10]

For the purpose of this review, the trial is timely and controversial; however, the results must be cautiously interpreted within the pediatric field as no specific conclusions were implemented regarding children. It is difficult to apply the results of the DECRA study to pediatric or any group of patients, due to the variety of confounding variables present in the study.

Hutchinson et al.

A trial in the United Kingdom (RESCUEicp) is currently underway with similar study aims as the DECRA trial but with important differences, such as including participants as young as 10 years old (Table 3). Comparable to the DECRA protocol, the RESCUEicp trial makes provisions for physicians to deliver care in the best interest of the patient, including craniectomy for the medicine group or barbiturates for the surgery group if the situation becomes an emergency (for example, prolonged ICP > 40 mm Hg with compromised CPP). Both studies use multiple-observer readings of CT scans following injury to stage the severity of the injury at baseline, a key component to an efficient trial.[33] The RESCUEicp study has the potential to generate a more powerful analysis of the outcome from craniectomy in both pediatric and adult patients and may be instrumental in directing future standard of care guidelines.

Discussion

Clinical outcomes following craniectomy after TBI in children are controversial. Taylor et al.[48] reported clear favorable outcomes in 7 of 13 patients receiving craniectomy and in only 2 of 14 patients receiving full medical management. Polin et al.[42] compared the rate of favorable outcomes in their pediatric and adult populations and report 44% and 29% favorable, respectively. However, this study had no control group. In a mixed-age prospective study, Guerra et al.[25] used highly restrictive patient selection criteria to analyze the effects of craniectomy on clinical outcome, and the results failed to support young age as a predictor of improved outcome.

Current guidelines are sparse and based on little clinical evidence. Adelson et al.[2] recommend the following criteria for selecting favorable patients for craniectomy in children: 1) diffuse cerebral swelling on cranial CT imaging; 2) within 48 hours of injury; 3) no episodes of sustained ICP > 40 mm Hg before surgery; 4) GCS score > 3 at some point subsequent to injury; 5) secondary clinical deterioration; and 6) evolving cerebral herniation syndrome.

At the moment, thorough investigations examining the clinical effectiveness of craniectomy in pediatric patients suffering from TBI are lacking. An important consideration, which arises from these discussions, is the appropriate selection of patients for craniectomy. The 2003 guidelines for surgical treatment of pediatric intracranial hypertension state "patients who experience a secondary deterioration on the Glasgow Coma Scale (GCS) and/or evolving cerebral herniation syndrome within the first 48 hours after injury may represent a favorable group. Patients with an unimproved GCS of 3 may represent an unfavorable group."

Previous findings[42,48] support a functionally higher outcome in pediatric patients compared with adult patients, especially when recipient patients are appropriately screened for maximum ICP < 40 mm Hg and surgery is implemented within 48 hours. Additionally, the ongoing RESCUEicp study is very similar to the DECRA study but includes patients as young as 10 years old, which may illuminate the effectiveness of the procedure in pediatric populations in the near future. Based on the current paucity of data within the pediatric field, it would be beneficial for the authors to perform subgroup analysis within their pediatric cohort. Additionally, specific goals for future trials might include the following: large pediatric patient database, stratified by age; similar baseline characteristics; minimization of crossover; accordance with guidelines in selecting threshold for refractory ICP hypertension; appropriate selection of long-term (> 5 years) measures of clinical outcome; and specific, nonexcepted exclusion criteria including established characteristics for worse outcome.

Conclusions

To date, decompressive surgery following TBI re-

mains controversial. Current questions that remain include the following. Which patients are appropriate for selection for craniectomy following TBI? Is young age a predictor of improved outcome? How can we optimize the surgical approach to refractory ICP hypertension in pediatric patients? How can we better understand the various physiological measurements (ICP, CPP, cerebral blood flow, and others) to tailor the surgical therapy to the patient? What role, if any, does the age of the patient play in targeting their therapeutic ICP level? Future RCTs are needed to address these and other questions, with special importance being placed on age stratification in pediatric patient populations.

Disclosure

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

Author contributions to the study and manuscript preparation include the following. Conception and design: Appelboom, Zoller, Szpalski, Anderson. Acquisition of data: McDowell. Drafting the article: Appelboom, Zoller, Feldstein. Critically revising the article: Zoller, Piazza, Szpalski, Zacharia, Hickman. Reviewed submitted version of manuscript: Zoller. Administrative/technical/material support: Bruce, D'Ambrosio. Study supervision: Appelboom, Vaughan, D'Ambrosio, Feldstein, Anderson.

References

1. Abdullah JM, Kumaraswamy N, Awang N, Ghazali MM, Abdullah MR: Persistence of cognitive deficits following paediatric head injury without professional rehabilitation in rural East Coast Malaysia. **Asian J Surg 28:**163–167, 2005
2. Adelson PD, Bratton SL, Carney NA, Chesnut RM, du Coudray HE, Goldstein B, et al: Guidelines for the acute medical management of severe traumatic brain injury in infants, children, and adolescents. Chapter 6. Threshold for treatment of intracranial hypertension. **Pediatr Crit Care Med 4 (3 Suppl):**S25–S27, 2003
3. Anderson V, Brown S, Newitt H, Hoile H: Educational, vocational, psychosocial, and quality-of-life outcomes for adult survivors of childhood traumatic brain injury. **J Head Trauma Rehabil 24:**303–312, 2009
4. Anderson VA, Catroppa C, Haritou F, Morse S, Rosenfeld JV: Identifying factors contributing to child and family outcome 30 months after traumatic brain injury in children. **J Neurol Neurosurg Psychiatry 76:**401–408, 2005
5. Aya-ay J, Mayer J, Eakin AK, Muffly BG, Anello M, Sandy JD, et al: The effect of hypoxic-ischemic brain injury in perinatal rats on the abundance and proteolysis of brevican and NG2. **Exp Neurol 193:**149–162, 2005
6. Bauer R, Walter B, Torossian A, Fritz H, Schlonski O, Jochum T, et al: A piglet model for evaluation of cerebral blood flow and brain oxidative metabolism during gradual cerebral perfusion pressure decrease. **Pediatr Neurosurg 30:**62–69, 1999
7. Bayir H, Adelson PD, Wisniewski SR, Shore P, Lai Y, Brown D, et al: Therapeutic hypothermia preserves antioxidant defenses after severe traumatic brain injury in infants and children. **Crit Care Med 37:**689–695, 2009
8. Berger MS, Pitts LH, Lovely M, Edwards MS, Bartkowski HM: Outcome from severe head injury in children and adolescents. **J Neurosurg 62:**194–199, 1985
9. Bittigau P, Sifringer M, Pohl D, Stadthaus D, Ishimaru M, Shimizu H, et al: Apoptotic neurodegeneration following trauma is markedly enhanced in the immature brain. **Ann Neurol 45:** 724–735, 1999
10. Brain Trauma Foundation, American Association of Neurological Surgeons, Congress of Neurological Surgeons, Joint Section on Neurotrauna and Critical Care, AANS/CNS, et al: Guidelines for the management of severe traumatic brain injury. VIII. Intracranial pressure thresholds. **J Neurotrauma 24 (Suppl 1):**S55–S58, 2007
11. Bullock R, Chesnut RM, Clifton G, Ghajar J, Marion DW, Narayan RK, et al: Guidelines for the management of severe head injury. **Eur J Emerg Med 3:**109–127, 1996
12. Carter BG, Taylor A, Butt W: Severe brain injury in children: long-term outcome and its prediction using somatosensory evoked potentials (SEPs). **Intensive Care Med 25:**722–728, 1999
13. Chambers IR, Jones PA, Lo TY, Forsyth RJ, Fulton B, Andrews PJ, et al: Critical thresholds of intracranial pressure and cerebral perfusion pressure related to age in paediatric head injury. **J Neurol Neurosurg Psychiatry 77:**234–240, 2006
14. Cho DY, Wang YC, Chi CS: Decompressive craniotomy for acute shaken/impact baby syndrome. **Pediatr Neurosurg 23:** 192–198, 1995
15. Coles JP, Minhas PS, Fryer TD, Smielewski P, Aigbirihio F, Donovan T, et al: Effect of hyperventilation on cerebral blood flow in traumatic head injury: clinical relevance and monitoring correlates. **Crit Care Med 30:**1950–1959, 2002
16. Cooper DJ, Rosenfeld JV, Murray L, Arabi YM, Davies AR, D'Urso P, et al: Decompressive craniectomy in diffuse traumatic brain injury. **N Engl J Med 364:**1493–1502, 2011
17. D'Ambrosio R, Maris DO, Grady MS, Winn HR, Janigro D: Selective loss of hippocampal long-term potentiation, but not depression, following fluid percussion injury. **Brain Res 786:** 64–79, 1998
18. Dam Hieu P, Sizun J, Person H, Besson G: The place of decompressive surgery in the treatment of uncontrollable post-traumatic intracranial hypertension in children. **Childs Nerv Syst 12:**270–275, 1996
19. Danton GH, Dietrich WD: Inflammatory mechanisms after ischemia and stroke. **J Neuropathol Exp Neurol 62:**127–136, 2003
20. Esparza J, M-Portillo J, Sarabia M, Yuste JA, Roger R, Lamas E: Outcome in children with severe head injuries. **Childs Nerv Syst 1:**109–114, 1985
21. Gaab MR, Rittierodt M, Lorenz M, Heissler HE: Traumatic brain swelling and operative decompression: a prospective investigation. **Acta Neurochir Suppl (Wien) 51:**326–328, 1990
22. Gentry LR, Godersky JC, Thompson B: MR imaging of head trauma: review of the distribution and radiopathologic features of traumatic lesions. **AJR Am J Roentgenol 150:**663–672, 1988
23. Giza CC, Maria NS, Hovda DA: N-methyl-D-aspartate receptor subunit changes after traumatic injury to the developing brain. **J Neurotrauma 23:**950–961, 2006
24. Giza CC, Prins ML: Is being plastic fantastic? Mechanisms of altered plasticity after developmental traumatic brain injury. **Dev Neurosci 28:**364–379, 2006
25. Guerra WK, Piek J, Gaab MR: Decompressive craniectomy to treat intracranial hypertension in head injury patients. **Intensive Care Med 25:**1327–1329, 1999
26. Hutchinson PJ, Corteen E, Czosnyka M, Mendelow AD, Menon DK, Mitchell P, et al: Decompressive craniectomy in traumatic brain injury: the randomized multicenter RESCUEicp study (www.RESCUEicp.com). **Acta Neurochir Suppl 96:** 17–20, 2006
27. Kan P, Amini A, Hansen K, White GL Jr, Brockmeyer DL, Walker ML, et al: Outcomes after decompressive craniectomy for severe traumatic brain injury in children. **J Neurosurg 105 (5 Suppl):**337–342, 2006
28. Keenan HT, Bratton SL: Epidemiology and outcomes of pediatric traumatic brain injury. **Dev Neurosci 28:**256–263, 2006
29. Kerr FW: Radical decompression and dural grafting in severe cerebral edema. **Mayo Clin Proc 43:**852–864, 1968

30. Kraus JF, Rock A, Hemyari P: Brain injuries among infants, children, adolescents, and young adults. **Am J Dis Child 144:** 684–691, 1990
31. Kunze E, Meixensberger J, Janka M, Sörensen N, Roosen K: Decompressive craniectomy in patients with uncontrollable intracranial hypertension. **Acta Neurochir Suppl 71:**16–18, 1998
32. Levin HS, Aldrich EF, Saydjari C, Eisenberg HM, Foulkes MA, Bellefleur M, et al: Severe head injury in children: experience of the Traumatic Coma Data Bank. **Neurosurgery 31:**435–444, 1992
33. Maas AI, Steyerberg EW, Marmarou A, McHugh GS, Lingsma HF, Butcher I, et al: IMPACT recommendations for improving the design and analysis of clinical trials in moderate to severe traumatic brain injury. **Neurotherapeutics 7:**127–134, 2010
34. Madikians A, Giza CC: Treatment of traumatic brain injury in pediatrics. **Curr Treat Options Neurol 11:**393–404, 2009
35. Margulies SS, Thibault KL: Infant skull and suture properties: measurements and implications for mechanisms of pediatric brain injury. **J Biomech Eng 122:**364–371, 2000
36. Marmarou A, Lu J, Butcher I, McHugh GS, Murray GD, Steyerberg EW, et al: Prognostic value of the Glasgow Coma Scale and pupil reactivity in traumatic brain injury assessed pre-hospital and on enrollment: an IMPACT analysis. **J Neurotrauma 24:**270–280, 2007
37. Olivecrona M, Rodling-Wahlström M, Naredi S, Koskinen LO: Effective ICP reduction by decompressive craniectomy in patients with severe traumatic brain injury treated by an ICP-targeted therapy. **J Neurotrauma 24:**927–935, 2007
38. Ommaya AK, Goldsmith W, Thibault L: Biomechanics and neuropathology of adult and paediatric head injury. **Br J Neurosurg 16:**220–242, 2002
39. Ommaya AK, Hirsch AE: Tolerances for cerebral concussion from head impact and whiplash in primates. **J Biomech 4:** 13–21, 1971
40. Pfenninger J, Kaiser G, Lütschg J, Sutter M: Treatment and outcome of the severely head injured child. **Intensive Care Med 9:**13–16, 1983
41. Pizzorusso T, Medini P, Berardi N, Chierzi S, Fawcett JW, Maffei L: Reactivation of ocular dominance plasticity in the adult visual cortex. **Science 298:**1248–1251, 2002
42. Polin RS, Shaffrey ME, Bogaev CA, Tisdale N, Germanson T, Bocchicchio B, et al: Decompressive bifrontal craniectomy in the treatment of severe refractory posttraumatic cerebral edema. **Neurosurgery 41:**84–94, 1997
43. Prins ML, Hovda DA: Mapping cerebral glucose metabolism during spatial learning: interactions of development and traumatic brain injury. **J Neurotrauma 18:**31–46, 2001
44. Reeves TM, Lyeth BG, Povlishock JT: Long-term potentiation deficits and excitability changes following traumatic brain injury. **Exp Brain Res 106:**248–256, 1995
45. Settergren G, Lindblad BS, Persson B: Cerebral blood flow and exchange of oxygen, glucose ketone bodies, lactate, pyruvate and amino acids in anesthetized children. **Acta Paediatr Scand 69:**457–465, 1980
46. Sharples PM, Stuart AG, Matthews DS, Aynsley-Green A, Eyre JA: Cerebral blood flow and metabolism in children with severe head injury. Part 1: Relation to age, Glasgow coma score, outcome, intracranial pressure, and time after injury. **J Neurol Neurosurg Psychiatry 58:**145–152, 1995
47. Stringer WA, Hasso AN, Thompson JR, Hinshaw DB, Jordan KG: Hyperventilation-induced cerebral ischemia in patients with acute brain lesions: demonstration by xenon-enhanced CT. **AJNR Am J Neuroradiol 14:**475–484, 1993
48. Taylor A, Butt W, Rosenfeld J, Shann F, Ditchfield M, Lewis E, et al: A randomized trial of very early decompressive craniectomy in children with traumatic brain injury and sustained intracranial hypertension. **Childs Nerv Syst 17:**154–162, 2001
49. Timofeev I, Czosnyka M, Nortje J, Smielewski P, Kirkpatrick P, Gupta A, et al: Effect of decompressive craniectomy on intracranial pressure and cerebrospinal compensation following traumatic brain injury. **J Neurosurg 108:**66–73, 2008
50. Timofeev I, Kirkpatrick PJ, Corteen E, Hiler M, Czosnyka M, Menon DK, et al: Decompressive craniectomy in traumatic brain injury: outcome following protocol-driven therapy. **Acta Neurochir Suppl 96:**11–16, 2006
51. Tropea D, Caleo M, Maffei L: Synergistic effects of brain-derived neurotrophic factor and chondroitinase ABC on retinal fiber sprouting after denervation of the superior colliculus in adult rats. **J Neurosci 23:**7034–7044, 2003
52. Vavilala MS, Lam AM: Perioperative considerations in pediatric traumatic brain injury. **Int Anesthesiol Clin 40:**69–87, 2002
53. Venkatesh B, Garrett P, Fraenkel DJ, Purdie D: Indices to quantify changes in intracranial and cerebral perfusion pressure by assessing agreement between hourly and semi-continuous recordings. **Intensive Care Med 30:**510–513, 2004

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Definition and measurement of rider-intrinsic physical attributes influencing all-terrain vehicle safety

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Object. All-terrain vehicle (ATV) usage has grown tremendously over the years, reaching 9.5 million vehicles in use in 2007. Accompanying this growth has been a concomitant increase in rider morbidity (including traumatic brain and spine injuries) and death, especially in children. The purpose of this study was to define and measure, through field testing, those physical attributes intrinsic to riders, such as height, weight, and wingspan, which may have implications for ATV riders' safety.

Methods. Three field tests (J-hook, brake, and bump) were developed and performed to allow direct measurement of the lateral, longitudinal, and vertical dynamics in 5 riders of varying heights, weights, and wingspans. Two ATVs, a utility and a sport model, were tested for further comparisons. Data were acquired using a comprehensive data acquisition system attached to the ATVs. Assignment of individual rider/ATV test safety ratings and a rider/ATV Total Safety Rating were made from the results of these field tests.

Results. The J-hook test results demonstrated that larger rider wingspans positively influence ATV rider safety and mitigate against lateral instability. From the brake test it was determined that a 10-in (25.4-cm) longitudinal displacement, such as that experienced during a sharp deceleration, for a rider of any height or weight, breached the level of defined safety. As rider weight increased, displacement decreased. The bump test provided evidence that increased rider weight also mitigates against vertical displacement.

Conclusions. Individuals with light weights and small wingspans, such as those in the pediatric population, are under considerable risk of injury when operating an ATV due to lateral, longitudinal, and vertical operational instability. *(DOI: 10.3171/2011.9.FOCUS11176)*

Key Words • all-terrain vehicle • safety legislation • wingspan • pediatric traumatic brain injury

Since their introduction to the North American market by Honda in 1971, ATVs have become very popular for use in both utility and recreational applications. It has been estimated that in 2007 there were 9.5 million 4-wheel ATVs in use in the US—a 126% increase from just the year 2000 (4.2 million).[23] With this dramatic increase in ATV proliferation has come a significant amount of rider morbidity and mortality.

Epidemiological studies have demonstrated that head and spinal column injuries account for a significant portion of ATV-related injuries.[11,20] In a recent series of 741 reported ATV-related injuries, > 200 patients with head injuries required care in the ICU, and 57 additional patients underwent immediate surgery on admission. Furthermore, 59 patients arrived with a Glasgow Coma Scale score of 3. In such series, 49 patients sustained a spinal cord injury.[4] Moreover, in addition to neurological morbidity, several other types of injuries have also been reported, such as orthopedic, abdominal, and ophthalmological injuries.[12,19]

All-terrain vehicles are 3- or 4-wheeled, motorized, gasoline-powered vehicles with a weight typically between 300 and 600 lbs (135 and 270 kg), a high center of gravity, motorcycle-style handlebars for steering control, a seat designed to be straddled from atop a platform, and oversized, low-pressure knobby tires optimized for off-road, nonpaved terrain traversal.[21] With engine sizes ranging from approximately 50–750 cm^3 of displacement, ATVs are now capable of achieving speeds up to 75 mph (120 km/h).[22] In 1988, the US Department of Transportation and ATV manufacturers negotiated a 10-year consent decree in federal district court that included a ban on the marketing of 3-wheeled ATVs.[24]

Abbreviations used in this paper: ATV = all-terrain vehicle; CPSC = Consumer Product Safety Commission; TSR = total safety rating. *Conversion of units used in this paper:* 1 in = 2.54 cm; 1 ft = 30.48 cm; 1 lb = 0.453 kg; 1 mph = 1.6 km/h.

With only 2 exceptions, in each successive year between 1991 and 2007 an increase was witnessed in the US in the number of injuries derived from ATV operation or usage that required hospital emergency department medical treatment—from 58,100 to 150,900 injuries.[23] Moreover, a minimum of 8995 riders have lost their lives in ATV-associated accidents between 1982 and 2007. Of this total, 28% (2497) were children < 16 years of age, and 12% (1062) were < 12 years of age.

According to a US CPSC study on ATV-related injuries and frequency of use (Table 1), from 1997 to 2001 the number of ATV riders increased from 12 to 16.3 million (36%), the total number of riding hours went from 1.575 to 2.364 billion (50%), and the number of ATVs in use rose from 4 to 5.6 million (40%).[10] However, revised CPSC data show an incongruent rise in the number of ATV-related injuries treated in hospital emergency departments over this 5-year period: from 52,800 to 110,100 (an increase of 109%).[2,10,23] These results suggest that the CPSC's exposure metrics are unable to fully account for the 109% rise in ATV-associated injuries.

Given the alarming rate of increase in ATV-related injuries, some investigations have sought to define vehicle-intrinsic factors that affect rider safety (that is, engine size, number of wheels). Others have focused on rider characteristics (age, sex, and ATV experience) and specific risk factors identified as correlative to ATV safety (that is, training, geography, operator vs passenger, and occupational vs recreational usage). The aim of this investigation, however, was to define and measure, through field testing, those physical attributes intrinsic to riders that may influence their safety while operating an ATV (that is, rider height, weight, and wingspan).

Methods

Research on vehicle dynamics was conducted to ascertain those dynamic factors deemed most influential on rider safety. Lateral acceleration, longitudinal acceleration, weight distribution, and roll moment arm were accepted as key influencers.[7,15] Three tests were developed for direct measurement of rider movements in the 3 translational coordinate directions (lateral, longitudinal, and vertical) and to draw conclusions about rider safety. These tests were the J-hook, brake, and bump tests (which are described in detail below and illustrated by Video 1).

Video 1. Video clip showing ATV riders executing the J-hook, brake, and bump tests. Click here to view with Windows Media Player. Click here to view with Quicktime.

TABLE 1: Frequency of use and ATV-related injury data in the US for 1997 and 2001

Parameter	1997	2001	% Increase
no. of ATV riders	12 million	16.3 million	36
hrs of ATV riding	1.575 billion	2.364 billion	50
no. of ATVs in use	4 million	5.6 million	40
ATV-related injuries*	52,800	110,100	109

* Based on revised CPSC data not available during original study.

To conduct these tests it was necessary to design a data acquisition system capable of dynamically locating the center of gravity of both the ATV and rider while in the field simulating riding events. The center of gravity is the point at which gravity can be said to act. On a dynamic vehicle, the roll moment acts through this point. However, the location of the center of gravity of an ATV shifts as the vehicle bounces, tilts, and deflects its shocks and tires through riding events. Moreover, unlike in an automobile, the shifting of rider weight to maintain balance on an ATV is necessary. Therefore, the data acquisition system needed to be capable of deriving the position of the rider in relation to the ATV. By tracking the center of gravity of the rider, the movement of the rider's weight force can be traced. Knowing the center of gravity of both the ATV and rider then allows for the determination of weight distribution, lateral, longitudinal, and vertical displacements, and an estimation of rollover risk.

The data acquisition system we designed was developed for direct attachment to the ATVs. It comprised an AiM Sports EVO3 data logger (with 2 internal accelerometers, 3 linear potentiometers, 1 magnetic pick-up speed sensor, and 2 inclinometers); a Bumblebee2 Stereoscopic Vision Camera (Point Grey Research, Inc.) for tracking rider movements and location with respect to the ATV; and custom MATLAB software code for translating the camera data into coordinates and combining all of the collected data from the EVO3 (that is, shock displacements, lateral and longitudinal accelerations, ATV speed, longitudinal and lateral inclination angles, and ATV and rider centers of gravity) for evaluation.

The EVO3 data logger was placed atop the ATV so the sensors could be easily plugged in. The 3 linear potentiometers were placed parallel to the ATV's suspension by attaching them to metal brackets connected to the vehicle. The magnetic speed sensor was attached to one of the ATV's wheels by using silicone caulk on the wheel and a metal bracket attached to the frame. The Bumblebee2's specifications required the camera to be located 3 ft (91 cm) away from the target it was tracking. An angled boom and bracket was designed, stress analyzed, built, and attached to the back of the ATV so that it positioned the camera at least 3 ft (91 cm) from the rider's back. The 2 inclinometers were attached to this boom and bracket. Additionally, to pull images from the camera it was necessary to place a laptop on the ATV during testing. A padded box was fabricated that allowed the safe attachment of a laptop to the back of the ATV during testing.

For field testing, 2 ATVs were ridden through identical events within the parameters of the 3 tests developed to measure and compare the dynamics of 5 adult riders of varying heights, weights, and wingspans. To ensure that comparable speeds were used by each rider during testing, and thus that reliable evaluations could be drawn, speed data were captured throughout each riding event. The ATVs used were a Polaris Trailblazer 250 (a sport ATV) and a Honda FourTrax 250 (a utility ATV). Both ATVs had 250 cm^3 displacement engines and weights of approximately 480 lbs. However, each had a very different suspension design; the Polaris used a MacPherson strut front suspension, whereas the Honda had a typical

4-bar-linkage front suspension. Both ATVs had a solid rear axle.

After all field testing was concluded and the results were analyzed, a test safety rating was assigned to each individual rider/ATV combination in each of the 3 tests. Based on these results, a TSR was calculated for each rider/ATV combination. These ratings allowed for further evaluation and comparison of factors affecting ATV rider safety.

In relation to the different baseline driving capabilities of the participants, which might have significantly influenced the results, all 5 drivers were matched for such confounding factors as follow: 1) none of them had ever driven an ATV vehicle before the instructions and training that were provided in the laboratory; and 2) all 5 participants were grossly matched regarding their driving skills. All riders were students at Bradley University in Peoria, Illinois, who were > 18 years of age and had no professional driving training; although all of them reported having the general skills for car driving expected for the adult population at such an age.

The J-Hook Test

The J-hook test concept is used by the National Highway Traffic and Safety Administration to measure a vehicle's dynamic movements in sharp turning. From this performance, a rating of resistance to rollover is assigned. We adapted this test, which involved the rider turning to the left and then suddenly veering to the right (Fig. 1), to gauge the lateral movement of an ATV rider under centrifugal forces. The results of this test are important because a common mechanism of ATV injury for younger riders is the rolling over of their ATV onto its side while on level terrain.[7]

A few assumptions had to be made to enable calculations predicting rider movement during a turn. First, it was assumed that the dimension of the rider's arm segments and the distance between the shoulder joints were equal, and that the rider's torso alignment was always perpendicular to the seat (Fig. 2A). This assumption was supported by the arm dimensions of the 5 riders. Second, it was assumed that the rider's arm was fully extended, aligning the 2 arm segments (Fig. 2B). Third, the system was said to be a 4-bar linkage comprising the shoulder-to-shoulder distance, the distance between the rider's center of gravity and the fulcrum of the handlebars, and the fully extended arm (Fig. 2C). Last, it was assumed that the rider shifted his or her weight a fixed distance into the J-hook to compensate for the centrifugal force (Fig. 2D). This fixed value was derived from examining the J-hook test data and finding the average inward lateral shift of the riders, which was approximately 3 in.

The rider/ATV geometry was modeled as a 4-bar linkage. The loop closure equation for this system is

$$\bar{r}_M + \tfrac{1}{2}\cdot\bar{r}_4 - \bar{r}_5 - \bar{r}_6 - \tfrac{1}{2}\cdot\bar{r}_1 = 0 \qquad \text{[Eq. 1]},$$

where r_M is the vector between the rider's center of gravity and the fulcrum of the handlebars; r_4 is the vector between the 2 handlebar grips (hands); r_5 is the vector

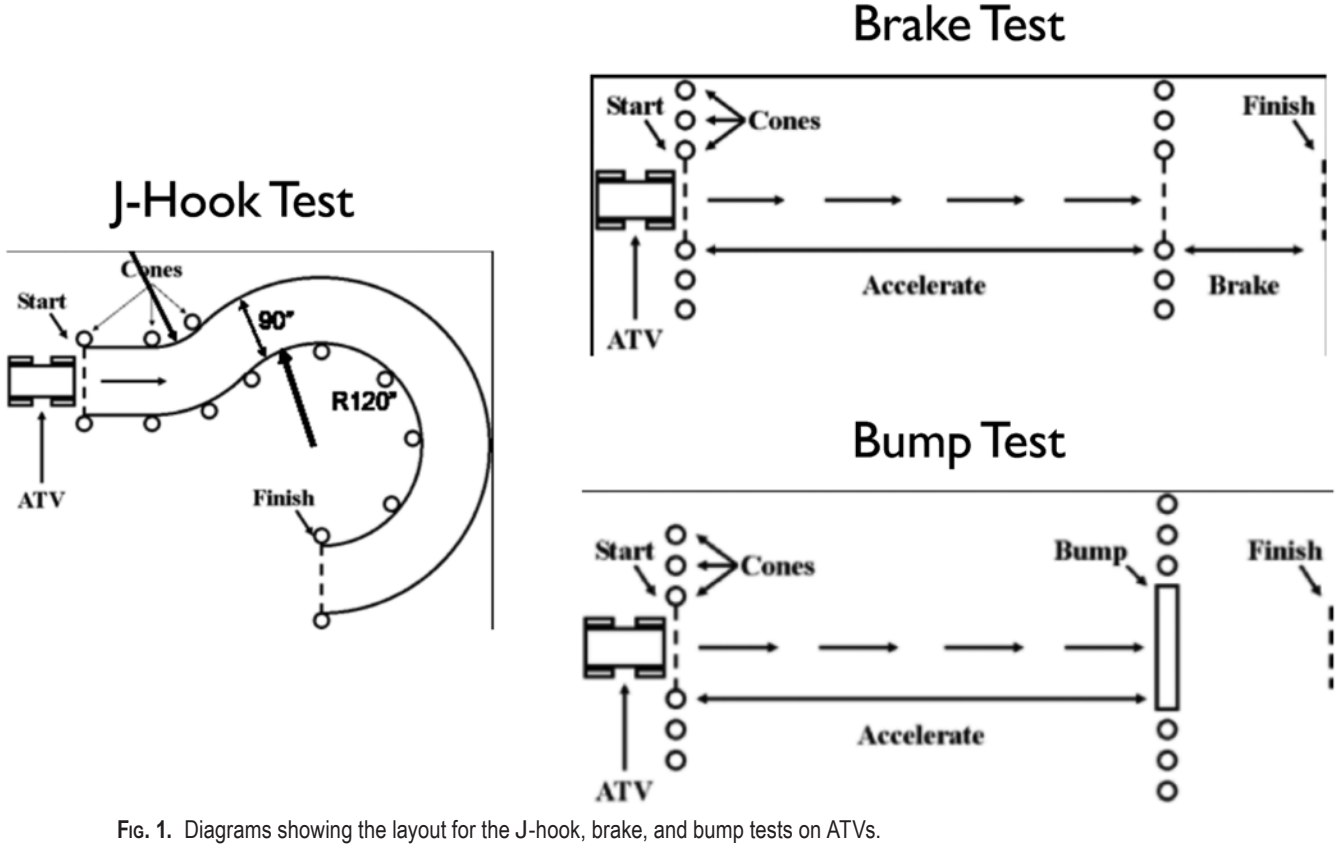

Fig. 1. Diagrams showing the layout for the J-hook, brake, and bump tests on ATVs.

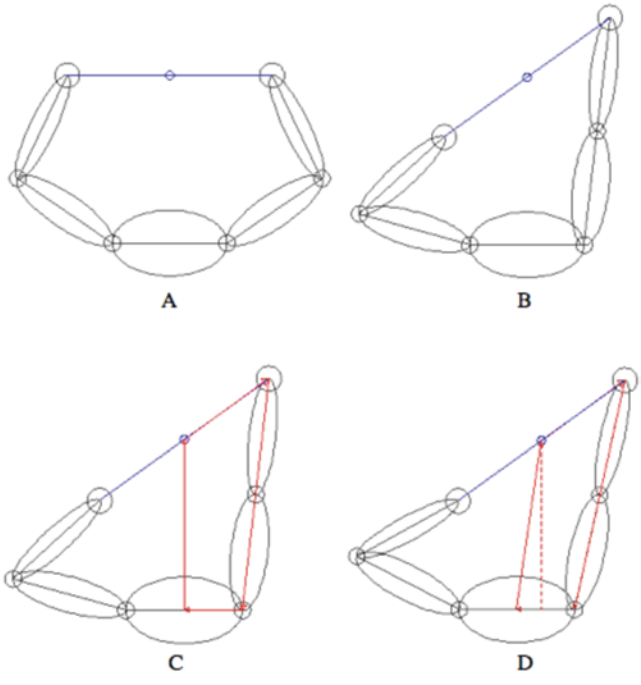

Fig. 2. Schematics showing assumptions used to make calculations predicting rider movement during a turn on an ATV. **A:** The rider's arm segments and the distance between the shoulder joints are equal, and the rider's torso alignment is always perpendicular to the seat. **B:** Rider's arm is fully extended, aligning the 2 arm segments. **C:** The system was said to be a 4-bar linkage comprising the shoulder-to-shoulder distance, the distance between the rider's center of gravity and the fulcrum of the handlebars, and the fully extended arm. **D:** The rider shifts his or her weight a fixed distance into the J-hook maneuver to compensate for the centrifugal force.

between a handlebar grip (hand) and corresponding elbow joint; r_6 is the vector between an elbow joint and corresponding shoulder joint; and r_1 is the vector between the 2 shoulder joints.

Equation 1 can be simplified using the above-listed assumptions, as follows:

$$l_M \angle\theta_M + \tfrac{1}{2}\cdot l_H \angle\theta_4 - 2\cdot l_A \angle\theta_{56} - \tfrac{1}{2}\cdot l_A \angle\theta_1 = 0 \quad \text{[Eq. 2]},$$

where l_M is the magnitude of the vector between the rider's center of gravity and the fulcrum of the handlebars; θ_M is the direction of the vector between the rider's center of gravity and the fulcrum of the handlebars; l_H is the length of the vector between the 2 handlebar grips (hands); θ_4 is the direction of the vector between the 2 handlebar grips (hands); l_A is the magnitude of the vector between the rider's center of gravity and the fulcrum of the handlebars; θ_{56} is the direction of the vector between the handlebar grip (hand) and the shoulder joint; and θ_1 is the direction of the vector between the 2 shoulder joints.

For the Polaris ATV, the grip-to-grip distance and the distance between the rider's center of gravity and the fulcrum of the handlebars were measured to be 25 in (63.5 cm) and 20.5 in (52.1 cm), respectively. For the Honda ATV, the analogous values were 26 in (66 cm) and 20.5 in (52.1 cm).

The Brake Test

The brake test was developed and performed to measure the longitudinal displacement of a rider experiencing sudden deceleration (Fig. 1). Starting from a stop, the rider accelerated along a linear path, reaching a certain minimum speed. On reaching a preset location, the rider would immediately apply the brakes as if in an emergency braking situation. The longitudinal displacement of the rider during the deceleration event was measured.

Without seat belts, which are used to control occupants in an automobile, an ATV rider can easily be thrown forward and sustain injuries in cases of sharp deceleration.

The Bump Test

The bump test was conducted to observe and measure a rider's vertical bounce when driving over a 3.5 in (8.9 cm) high bump (Fig. 1). This test is based on the premise that riders' safety is severely compromised once they are in a position in which their legs are completely extended. Any displacement beyond this boundary could cause their feet to leave the foot pegs—a condition conducive to ATV/rider separation. Similarly to longitudinal displacement, without restraint, excessive vertical displacement from the ATV seat can easily lead to rider injury.

The maximum allowable bounce for a particular rider was determined as his or her change in position from sitting to standing with legs at full extension. Figure 3 shows the basic geometry of a rider in the sitting position.

Equations 3, *4*, and *5* were used to calculate the inseam of the riders based on their height through the golden ratio and proportions presented by Leonardo da Vinci's Vitruvian Man (http://en.wikipedia.org/wiki/Vitruvian_Man [Accessed October 7, 2011]).

$$DistFromSeatToNavel = \frac{1}{12}\cdot RiderHeight \quad \text{[Eq. 3]}$$

$$GoldenRatio\phi = \frac{RiderHeight}{HeightToNavel} \cong \frac{1+\sqrt{5}}{2} \quad \text{[Eq. 4]}$$

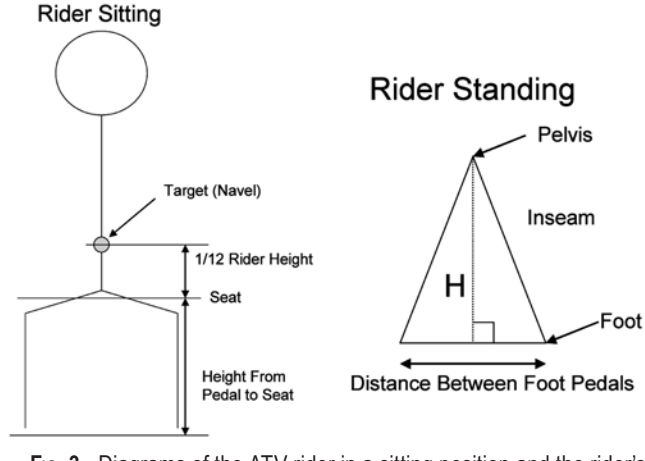

Fig. 3. Diagrams of the ATV rider in a sitting position and the rider's legs when standing.

$$Inseam = HeightToNavel - DistFromSeatToNavel = RiderHeight \cdot (\frac{1}{\phi} - \frac{1}{12}) \quad \text{[Eq. 5]}$$

Figure 3 also shows a diagram of an ATV rider in a standing position, legs fully extended. The vertical change in the position of the pelvic area of the rider is equivalent to the change in position of the tracked target (center of gravity) on the rider. Therefore, defining the change in height from the sitting to standing position will define the maximum allowable bounce deemed safe.

Equations 6, *7*, and *8* describe the derivation of the maximum allowable bounce through the measurable dimensions of rider height, distance between ATV foot pegs, and distance from the foot pegs to the seat of the ATV.

$$H = \sqrt{Inseam^2 - (\frac{DistB/nFootPedals}{2})^2} \quad \text{[Eq. 6]}$$

$$AllowableBounce = H - HeightFromPedalsToSeat \quad \text{[Eq. 7]}$$

$$AllowableBounce = \sqrt{Inseam^2 - (\frac{DistB/nFootPedals}{2})^2} - HeightFromPedalsToSeat \quad \text{[Eq. 8]}$$

Although a rider at a given weight can be one of many heights, the bounce experienced by an ATV rider is dependent on his or her weight and the ATV's suspension engineering. Therefore, the safety of a rider experiencing vertical displacement must be evaluated within the context of the weight variable. For the purposes of defining the safety of a rider, the typical height ranges corresponding to a particular weight were found using health insurance documentation from BlueCross BlueShield for juvenile and adult health underwriting guidelines.[8] For the minimum and maximum heights at each weight, *Equation 8* was used to calculate the maximum allowable bounce of the rider.

The TSR

The 5 riders chosen for field testing of the ATVs were selected based on desired variances in height, weight, and wingspan combinations. This allowed for the observation and examination of how each intrinsic physical attribute may be responsible for rider safety.

Table 2 outlines the key physical features of each test rider. Rider A simulates a child due to her short height and light weight. Rider B is nearly the same height as Rider A, but weighs considerably more. Riders C and D are of approximately the same weight as Rider B, but with greater height and wingspan. Therefore, they were used to evaluate the effect of increased height on rider safety. Rider E is close in height to Riders C and D, but is > 90 lbs (40.8 kg) heavier than both.

The TSR was derived from the safety ratings of the individual tests, which were combined through the following equation:

$$TotalSafety = 60\% \cdot J\text{-}HookSafety + 25\% \cdot BumpSafety + 15\% \cdot BrakeSafety \quad \text{[Eq. 9]}$$

Each test's safety rating was weighted according to what rider movements were deemed most influential to rider safety.

Results

The J-Hook Test Results

With the aid of *Equation 8* and the measured values for grip-to-grip distance, the distance between the rider's center of gravity and the fulcrum of the handlebars, and the lateral rider shift, a plot was made using a range of wingspans from 24 in (61 cm) to 84 in (213.4 cm), in 0.5-in (1.3-cm) increments, for the 2 ATV models (Fig. 4).

Analysis of this plot allowed certain adjustments to be made. Because a rider must be able to shift his or her body into a turn to maintain balance, any handlebar angle $< 0°$ was filtered out, on the assumption that if the rider is unable to shift at least 3 in (7.6 cm) into a turn and maintain a handlebar angle $\geq 0°$, the rider is unsafe. Additionally, any handlebar angle $> 60°$ was filtered out, on the assumption that if the rider is able to shift at least 3 in (7.6 cm) into a turn and maintain a handlebar angle $\geq 60°$, any increase in wingspan is superfluous.

Applying these adjustments, a plot of the calculated J-hook test safety values (five-thirds of handlebar angle) as a function of wingspan was developed (Fig. 5). As shown, there are minimum and maximum values for each data set. These values represent the 0% and 100% values, respectively. Any wingspan that has a handlebar angle $\leq 0°$ will have a corresponding safety value of 0%. This safety value represents the range of wingspans that cannot shift and turn adequately on the ATV. In contrast, any wingspan that has a handlebar angle $\leq 60°$ will have a corresponding safety value of 100%. This safety value represents the range of wingspans that should not have difficulty shifting and turning adequately on the ATV. For example, an individual with a 60-in (152.4-cm) wingspan can turn the handlebars 7.27° and 5.96° on the Polaris and Honda, respectively, with corresponding safety values of 12.12% and 9.93%.

The J-hook test results clearly demonstrate that rider wingspan can have direct implications for ATV safety. Much of this effect is probably due to the need for the ATV rider to actively engage the vehicle to mitigate against rollovers. To maintain stability, a rider must coordinate the counterintuitive shifting of his or her body weight in the opposite direction of a turn. For these procedures, a large wingspan is certainly beneficial.

The Brake Test Results

Figure 6 shows the exponential curve fit for the experimental data collected from the brake test. Through measurements of the geometry of each ATV, it was determined that a 10-in (25.4-cm) position change for a rider of any height or weight would be unsafe. This 10-in value was true for both the Polaris and Honda. All rider weights that moved 10 in or more were given a safety rating of 0%. As rider weight increased, the change in position decreased, and the difference between the unsafe value of 10 in and actual rider displacement was used to find the safety rating. A diagram of the measured rider displacement is shown in Fig. 7, and a plot summarizing the safety ratings of various weighted riders is illustrated in Fig. 8. These results illustrate a key takeaway: heavier weights can significantly aid in ATV rider safety by effectively

TABLE 2: Key physical characteristics in ATV test riders and TSRs for ATV/rider combinations

Rider	Weight	Height	Wingspan	% Polaris TSR	% Honda TSR
A	93.4 lbs/42.3 kg	60 in/1.52 m	58.0 in/1.47 m	9.8	15.9
B	168.8 lbs/76.5 kg	62 in/1.57 m	59.5 in/1.51 m	46.1	45.5
C	167.8 lbs/76.1 kg	68 in/1.72 m	68.0 in/1.72 m	64.7	64.1
D	164.2 lbs/74.4 kg	70 in/1.77 m	71.0 in/1.80 m	66.5	65.9
E	257.6 lbs/116.8 kg	71 in/1.80 m	71.5 in/1.81 m	73.1	72.5

buffering longitudinal displacement experienced during sharp deceleration.

The Bump Test Results

The calculated maximum allowable bounce for riders of specific weights at minimum and maximum heights, as defined by the BlueCross BlueShield data, is shown in Fig. 9 upper for the Honda ATV. As shown, a rider weighing 100 lbs (45.3 kg) would be expected to bounce a distance of 9 in (22.9 cm) on the Honda when driving over a 3.75-in (9.5-cm) bump. However, a tall 100-lb (45.3-kg) rider is capable of bouncing 13 in (33 cm) before becoming unsafe, and a short 100-lb rider can bounce only 5 in (12.7 cm) before the same effect occurs. Therefore, not every 100-lb rider should be expected to be safe when riding under congruent conditions. Assuming the typical population has a height between the minimum and maximum values described by BlueCross BlueShield, only 45%–50% of 100-lb persons could be expected to withstand a 9-in (22.9-cm) vertical displacement without leaving the foot pegs. Figure 9 lower illustrates the bounce and bump test safety of a rider with respect to his or her weight on the Honda.

Maximum allowable bounce and safety rating profiles were also calculated for the Polaris (Fig. 10). Notable differences are evident between the 2 ATV bounce plots. The Honda, being a utility ATV, has a more stable design. It sits lower to the ground and has firmer suspension. The Polaris, a sport ATV, is made for rugged entertainment and sits higher off the ground, with more bounce in its suspension. In addition, the Polaris has a greater distance from its foot pegs to its seat than the Honda. As a result, riders' legs are more extended in the sitting position. Consequently, a rider will have less allowable bounce before their legs will reach full extension on the Polaris. These stability factors influenced the location and shape of the "predicted bounce" lines of each bounce plot. Under equal conditions, a rider would bounce less on the Honda than on the Polaris. To illustrate, a weight of 75 lbs (34 kg) marks the weight value at which a rider becomes unsafe on the Honda when riding over a 3.75-in (9.5-cm) bump. On the Polaris, however, this weight value is 85 lbs (38.5 kg).

The TSR

Table 2 documents the TSRs derived for each rider on the Polaris and Honda ATVs. As can be seen in relation to Rider A, the greater weight of Rider B produced a significant increase in the TSR: approximately 35 percentage points for the Polaris and 30 percentage points for the Honda. However, due to the relatively short wingspans of Riders A and B, they were still both rated considerably less safe than the other riders.

The greater heights and wingspans of Riders C and D contributed to a TSR increase of approximately 20 percentage points over Rider B on both machines. The heavier weight of Rider E led to a 7–8 percentage point TSR increase over Riders C and D. This underwhelming increase illustrates 2 key points: short height, light weight, and small wingspan traits dramatically and detrimentally influence rider safety. In addition, there are physical attribute thresholds whereupon the TSR begins to taper, and further increases do not significantly augment rider safety.

Discussion

The implications of this study are enormous, especially for the pediatric population, because they have been found to be 4–12 times more likely to be injured riding an

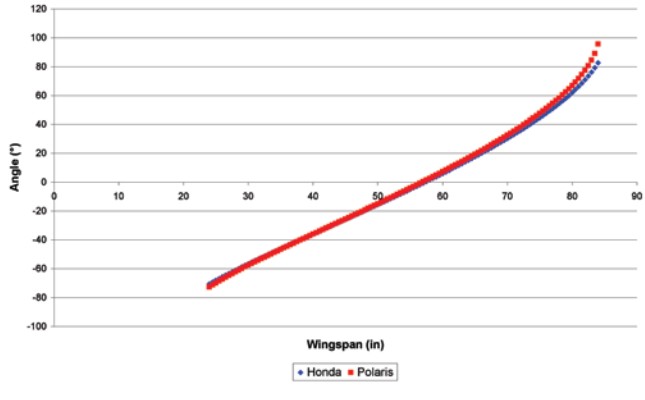

Fig. 4. Graph showing wingspan versus angle of handlebars in ATV riders tested.

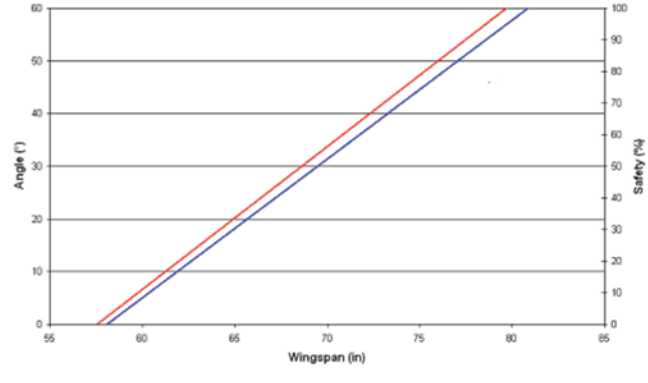

Fig. 5. Graph showing wingspan versus angle of handlebars (3 in filtered; see *The J-Hook Test Results*) in ATV riders, with safety values.

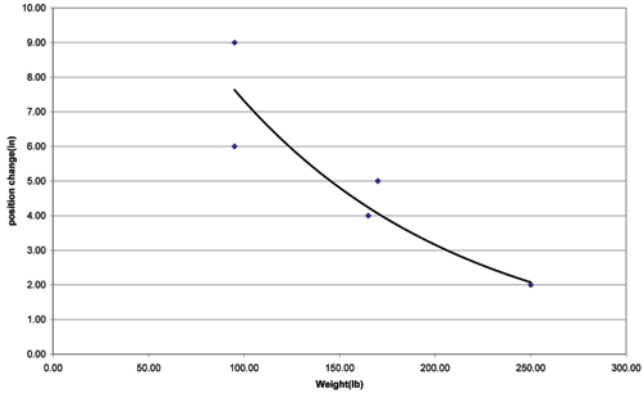

Fig. 6. Graph showing exponential curve fit of brake test data for ATV riders.

ATV than adult riders.[9,13,18] In 2001, there were 2.8 million ATV riders < 16 years old, of whom only 1% received any formal ATV rider training.[1,10] Despite having made up only 17% of all ATV riders, children < 16 years of age disproportionately accounted for 31% of ATV-associated injuries and 26% of ATV-associated fatalities in 2001.[16] Within this pediatric population, 85% rode adult-sized ATVs (> 90 cm^3 displacement) and, not surprisingly, 87% of those injured were riding on such a vehicle.[21] An analysis of ATV fatalities from 1999 to 2000 by the CPSC noted that 93% of ATV-related fatalities in children < 16 years old occurred on adult-sized machines. Moreover, an analysis of the hazard patterns associated with these fatalities demonstrates that many were due to the vehicle overturning (40%) and ejection from the vehicle (15%).[25] Compounding matters, children have a high incidence of trauma to the CNS, defined as intracranial or spinal cord injury, when involved in ATV crashes.[3,22] In fact, adolescent ATV riders have more head injuries than other age groups, and these injuries tend to be more severe in nature.[22]

For all age groups, head trauma is the leading cause of death from ATV-associated injuries.[10] It has been reported that 80% of all ATV-associated deaths involved a CNS injury.[2] Despite this fact, the helmet usage rate among ATV riders is dismal. Only 35% of riders reported always wearing a helmet, whereas 32% reported never wearing one. A recent survey of typical ATV usage patterns in rural Illinois youth revealed that most never used safety gear, including helmets (61.4%), and few (14.6%) had received safety education.[6] A statistical analysis revealed that helmet usage among all ATV riders would reduce the risk of death by 42% and nonfatal head injuries by 64%.[9] In Ireland, for example, where helmet usage is required and compliance is quoted at 78%, head injuries are rare, and most injuries are orthopedic in nature.[22] Moreover, by extrapolating motorcycle-associated health care costs to ATVs, the cost of care for unhelmeted ATV patients could be 23%–60% greater than for helmeted patients.[8,17]

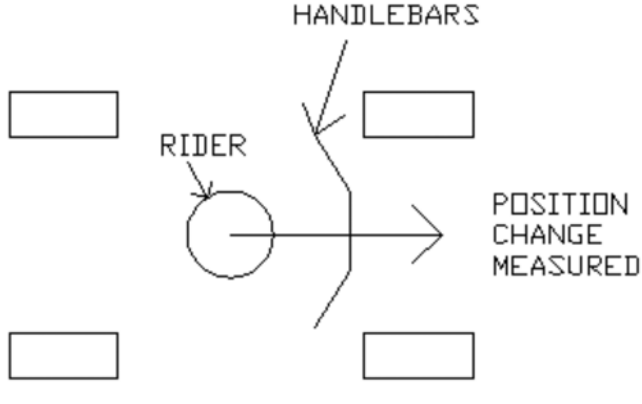

Fig. 7. Diagram of measured ATV rider displacement in the brake test.

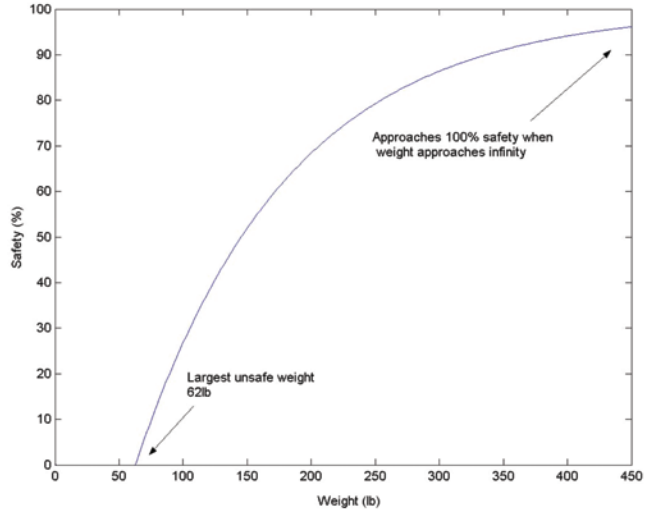

Fig. 8. Graph showing safety ratings of ATV riders at different weights in the brake test.

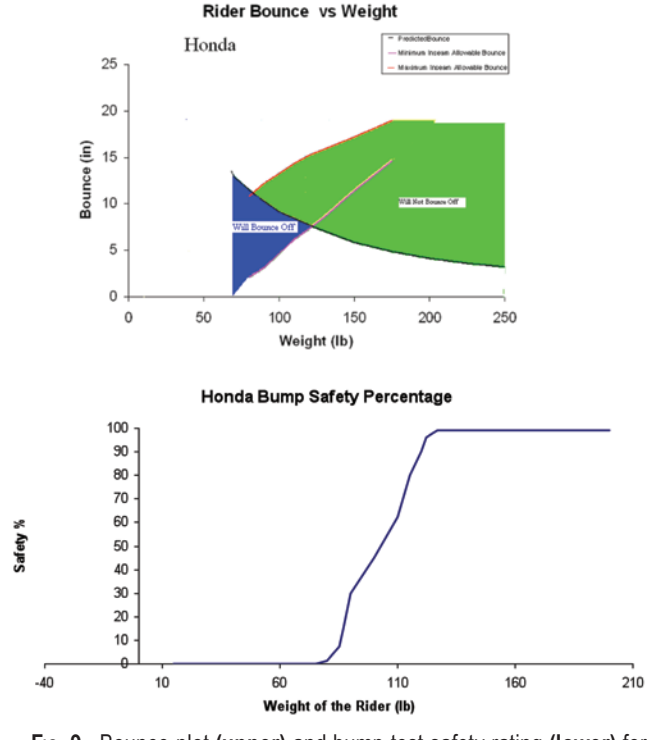

Fig. 9. Bounce plot **(upper)** and bump test safety rating **(lower)** for the Honda ATV.

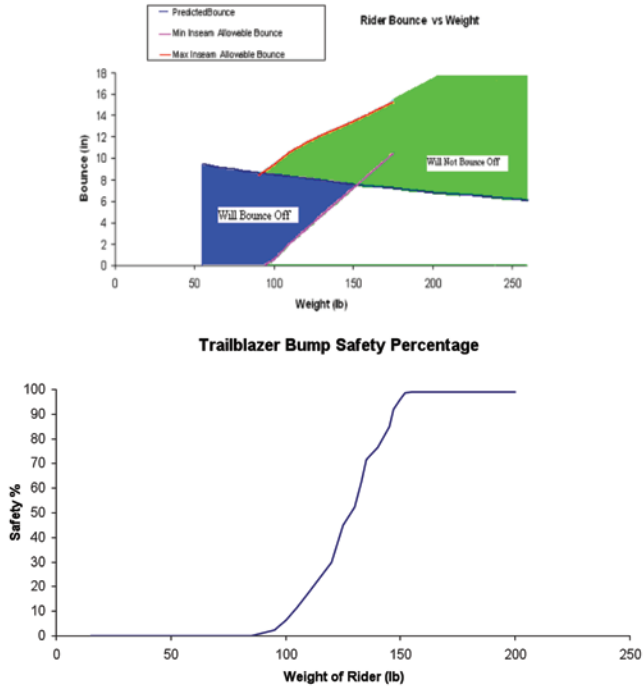

Fig. 10. Bounce plot **(upper)** and bump test safety rating **(lower)** for the Polaris ATV.

Helmeted or unhelmeted, ATV-associated injuries are not without considerable financial consequences. Expenses related to ATV injury can include medical and legal bills, disability payments, and lost economic productivity. The American Academy of Orthopedic Surgeons estimated that in the year 2000 alone, ATV-related injuries in the US cost society $6.5 billion.[2] The bulk of these expenses are borne by private insurers.[9,14]

Between 1988 and 1998, the CPSC instituted federal marketing and manufacturing requirements for ATV usage in children. Integral to these requirements was the stratification of ATV engine size (and thus vehicle size and power) for riders at specific ages (< 70 cm^3 for riders < 12 years old, 70–90 cm^3 for those 12–16 years old, and > 90 cm^3 for operators > 16 years old). After the consent decree expired, the major ATV manufacturers agreed to continue the program voluntarily. In 2008, due to public outcry over rising ATV injuries and fatalities in children, the CPSC published a new and final rule adopting the ATV industry standard, ANSI/SVIA-1–2007, as a mandatory consumer product safety standard, effective April 13, 2009.[5] The mandatory standard defines youth-sized ATVs by their maximum speeds, rather than by engine size, as under the prior consent decree. Under this standard, maximum speed capabilities are provided for 4 types of youth-sized models. These capabilities depend on the rider's age. In addition, the standard requires that all youth-sized models be equipped with a speed governor that limits the vehicle to a certain speed, but can be adjusted or removed with the use of a tool or other specialized device so that the vehicle can then go faster.

Although top speed is an important characteristic for vehicle stability and safety, it does not address the total size of the vehicle. Prior CPSC evaluation of the specification data of ATV models available from manufacturers was not specific enough to show a discrepancy in the anthropometric characteristics of children and the physical size of ATVs intended for children < 16 years old. However, CPSC mechanical engineers have noted that a child who is too light or too heavy for the vehicle may affect the center of gravity and lead to instability.[25] As demonstrated by our testing, ATV rider stability and safety are also based on rider wingspan and weight, which may not be adequately addressed in the new top speed–related age requirements. Based on our findings, we recommend that ATVs designed for children should have both size and weight limitations for riders included as a stratification measure.

Because most states unfortunately lack legislation governing the size or use of ATVs for children, most children ride adult-sized ATVs, even with federal manufacturer and distributor regulations in place. It is imperative that parents become fully educated about the inherent safety risks presented to children on ATVs. Parents who choose to allow their children to use ATVs must be aware of the mechanical differences and proportional safety risks that are present on adult-sized ATVs used by youth. Current child-sized ATV stratifications determined using regulated engine speed may not be enough to mitigate risks associated with the size and weight of these vehicles.

Although this work focused strictly on the rider's physical profile that increases the risk of ATV-related injuries, parents should be aware that there is plenty of scientific evidence against the use of ATVs by the pediatric population, because other cognitive and judgment factors are known to decrease a rider's safety in this population.[1–4,11,12,16]

Conclusions

Injuries related to ATVs continue to be a great concern for public health as well as safety regulatory agencies. Through our comprehensive field testing and safety rating assignments, we were able to provide substantial evidence supporting the belief that individuals with short wingspans and light weights are under considerable risk while operating an ATV.

Disclosure

There is no conflict of interest, because funding for this study was not received from any ATV manufacturer, and no author has a business or professional affiliation with any ATV manufacturer.

Author contributions to the study and manuscript preparation include the following. Conception and design: Bond. Acquisition of data: Bond, Hafner, Morris, Travis, Webster. Drafting the article: Mattei. Critically revising the article: Mattei. Approved the final version of the manuscript on behalf of all authors: Mattei. Statistical analysis: Hannah. Administrative/technical/material support: Hafner, Morris, Travis, Webster. Study supervision: Lin.

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References

1. Brown RL, Koepplinger ME, Mehlman CT, Gittelman M, Garcia VF: All-terrain vehicle and bicycle crashes in children: epidemiology and comparison of injury severity. **J Pediatr Surg 37:**375–380, 2002
2. Carr AM, Bailes JE, Helmkamp JC, Rosen CL, Miele VJ: Neurological injury and death in all-terrain vehicle crashes in West Virginia: a 10-year retrospective review. **Neurosurgery 54:**861–867, 2004
3. Cvijanovich NZ, Cook LJ, Mann NC, Dean JM: A population-based assessment of pediatric all-terrain vehicle injuries. **Pediatrics 108:**631–635, 2001
4. Finn MA, MacDonald JD: A population-based study of all-terrain vehicle-related head and spinal injuries. **Neurosurgery 67:**993–997, 2010
5. Government Accountability Office: All-terrain vehicles: how they are used, crashes, and sales of adult-sized vehicles for children's use. GAO-10-418, April 8, 2010 (http://www.gao.gov/products/GAO-10-418) [Accessed September 22, 2011]
6. Hafner JW, Hough SM, Getz MA, Whitehurst Y, Pearl RH: All-terrain vehicle safety and use patterns in central Illinois youth. **J Rural Health 26:**67–72, 2010
7. Haney P: **The Racing and High-Performance Tire: Using the Tires to Tune for Grip and Balance.** Springfield, IL: TV Motorsports and SAE, 2003
8. Humphries RL, Stone CK, Stapczynski JS, Florea S: An assessment of pediatric all-terrain vehicle injuries. **Pediatr Emerg Care 22:**491–494, 2006
9. Killingsworth JB, Tilford JM, Parker JG, Graham JJ, Dick RM, Aitken ME: National hospitalization impact of pediatric all-terrain vehicle injuries. **Pediatrics 115:**e316–e321, 2005
10. Levenson MS: **All-Terrain Vehicle 2001 Injury and Exposure Studies.** Washington: US Consumer Product Safety Commission, 2003
11. Long G, Thompson TM, Storm B, Graham J: Cranial impalement in a child driving an all-terrain vehicle. **Pediatr Emerg Care 27:**409–410, 2011
12. Mayercik VA, Eller AW, Stefko ST: Ocular injuries in all-terrain-vehicle accidents. **Injury** [epub ahead of print], 2011
13. McBride AS, Cline DM, Neiberg RH, Westmoreland KD: Pediatric all-terrain vehicle injuries: does legislation make a dent? **Pediatr Emerg Care 27:**97–101, 2011
14. Merrigan TL, Wall PL, Smith HL, Janus TJ, Sidwell RA: The burden of unhelmeted and uninsured ATV drivers and passengers. **Traffic Inj Prev 12:**251–255, 2011
15. Milliken WF, Milliken DL: **Race Car Vehicle Dynamics.** Warrensville, PA: Society of Automotive Engineers, 1995
16. Murphy N, Yanchar NL: Yet more pediatric injuries associated with all-terrain vehicles: should kids be using them? **J Trauma 56:**1185–1190, 2004
17. Myers ML, Cole HP, Mazur JM: Cost effectiveness of wearing head protection on all-terrain vehicles. **J Agromed 14:**312–323, 2009
18. Rodgers GB, Adler P: Risk factors for all-terrain vehicle injuries: a national case-control study. **Am J Epidemiol 153:** 1112–1118, 2001
19. Sawyer JR, Kelly DM, Kellum E, Warner WC Jr: Orthopaedic aspects of all-terrain vehicle-related injury. **J Am Acad Orthop Surg 19:**219–225, 2011
20. Scott A, Dansey R, Hamill J: Dangerous toys. **ANZ J Surg 81:**172–175, 2011
21. Shults RA, Wiles SD, Vajani M, Helmkamp JC: All-terrain vehicle-related nonfatal injuries among young riders: United States, 2001-2003. **Pediatrics 116:**e608–e612, 2005
22. Smith LM, Pittman MA, Marr AB, Swan K, Singh S, Akin SJ, et al: Unsafe at any age: a retrospective review of all-terrain vehicle injuries in two level I trauma centers from 1995 to 2003. **J Trauma 58:**783–788, 2005
23. Streeter RA: **2007 Annual Report of ATV-Related Deaths and Injuries.** Washington: U.S. Consumer Product Safety Commission, 2008
24. *United States v. Am. Honda Motor Co.*, 143 F.R.D. 1 (D.D.C. 1992)
25. United States Consumer Product Safety Commission: **Analysis of ATV-Related Fatality Data for CPSC Petition CP 02-4/HP 02-1.** (http://www.cpsc.gov/library/foia/foia05/brief/atvpt3.pdf) [Accessed September 26, 2011]

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Cervical spinal stenosis and sports-related cervical cord neurapraxia

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Cervical cord neurapraxia is a common sports-related injury. It is defined as a transient neurological deficit following trauma localizing to the cervical spinal cord and can be caused by hyperextension, hyperflexion, or axial load mechanisms. Symptoms usually last less than 15 minutes, but can persist up to 48 hours in adults and as long as 5 days in children. While a strong causal relationship exists between cervical spine stenosis and cervical cord neurapraxia in adult patients, this association has not been observed in children. Likewise, while repeated episodes of neurapraxia can be commonplace in adult patients, recurrences have not been reported in the pediatric population. Treatment is usually supportive, but in adults with focal cervical lesions or instability, surgery is an option. Surgery for neurapraxia in children is rarely indicated. *(DOI: 10.3171/2011.7.FOCUS11173)*

Key Words • **neurapraxia** • **cervical spine** • **spinal cord** • **cervical stenosis** • **sports**

Cervical cord neurapraxia is defined as a transient neurological deficit following cervical spinal cord trauma.[24] It is a common sports-related injury, occurring in 1.3–6 per 10,000 athletes, but there have been few studies that thoroughly describe the phenomenon.[5,24] In a large series of 110 patients with cervical cord neurapraxia, the vast majority of cases (87%) occurred during football.[22] In the largest pediatric series with cervical cord neurapraxia (13 patients), once again football was the most common sport (4 cases, 31%).[6] Case reports do exist of cervical cord neurapraxia following nonsports-related injury.[1] The mechanism of injury is typically hyperextension, but cervical cord neurapraxia can occur after hyperflexion and axial loading as well.[5,24]

There is a wide range of clinical presentations of cervical cord neurapraxia. Sensory symptoms (paresthesias) can include burning pain, numbness, or tingling, and can involve both arms (upper), both legs (lower), ipsilateral arm and leg (hemi), or all 4 extremities (quad).[22] Motor symptoms can occur in a similar anatomical distribution and ranges from weakness (paresis) to complete paralysis (plegia).[22] Symptoms generally resolve in less than 15 minutes, but have been reported to persist for up to 48 hours after injury.[24] By definition, a patient with neurapraxia completely returns to their baseline neurological functional status with no residual weakness or paresthesias. Torg et al.[22] developed a grading system based on duration of symptoms: Grade I (< 15 minutes), Grade II (15 minutes to 24 hours), and Grade III (> 24 hours). The following sections will describe current information regarding the contribution of cervical spinal stenosis to cervical cord neurapraxia in the adult and pediatric athlete.

Pathophysiology of Neurapraxia

Underyling the motor/sensory manifestations of neurapraxia is a temporary derangement of axonal permeability.[25] Hyperextension or hyperflexion causes a mechanical injury that depolarizes the axon membrane in a reversible but sustained manner. Laboratory studies reveal that the rapid stretch experienced by the strained axon results in calcium influx, hyperpolarization, then prolonged depolarization, during which the axon is no longer excitable. In addition, anatomical strain experienced during this type of insult can result in microvascular constriction and vasospasm. As a result, local and regional blood flow is altered and the threat of ischemia becomes prominent. The transient nature of these physiological changes distinguished neurapraxia from irreversible neurological damage.

Cervical Spinal Stenosis

Cervical spinal stenosis is common in pediatric and adult athletes.[4] Several methods to screen for cervical spinal stenosis in the setting of cervical cord neurapraxia have been proposed. Sagittal spinal canal diameter can be measured on lateral cervical plain radiographs and compared with standard measurements (< 14 mm in the adult

cervical spine is considered stenotic).[11] The Torg ratio is calculated as the ratio of the spinal canal diameter to the vertebral body diameter at the C3–7 levels as measured on lateral plain radiographs of the cervical spine (Fig. 1).[19] It was developed as a measure of congenital spinal canal stenosis that theoretically minimizes the effect of variations in landmarks and radiographic technique. A Torg ratio < 0.8 is considered evidence of congenital stenosis. A criticism of this technique is that it does not take into consideration disproportionate differences in vertebral body size; football players commonly have larger vertebral bodies relative to the other spinal elements.[10] Magnetic resonance imaging has surpassed plain radiographs and is the accepted method for evaluating spinal stenosis. Magnetic resonance imaging provides visualization of the vertebral column and intervertebral discs in relationship to the spinal cord, nerve roots, and surrounding CSF within the spinal canal. Magnetic resonance imaging demonstrates bone and discogenic encroachment on the spinal canal and spinal cord compression. The "functional reserve" of the spinal canal is indicated by the presence or absence of CSF signal surrounding the spinal cord.[13] This can be quantified by subtracting the spinal cord diameter on a midsagittal MR image from the disc-level spinal canal diameter (Fig. 2).[22] Dynamic flexion and extension cervical spine MR imaging modalities have been proposed to evaluate functional stenosis, although not all centers may be capable of performing these studies.[2]

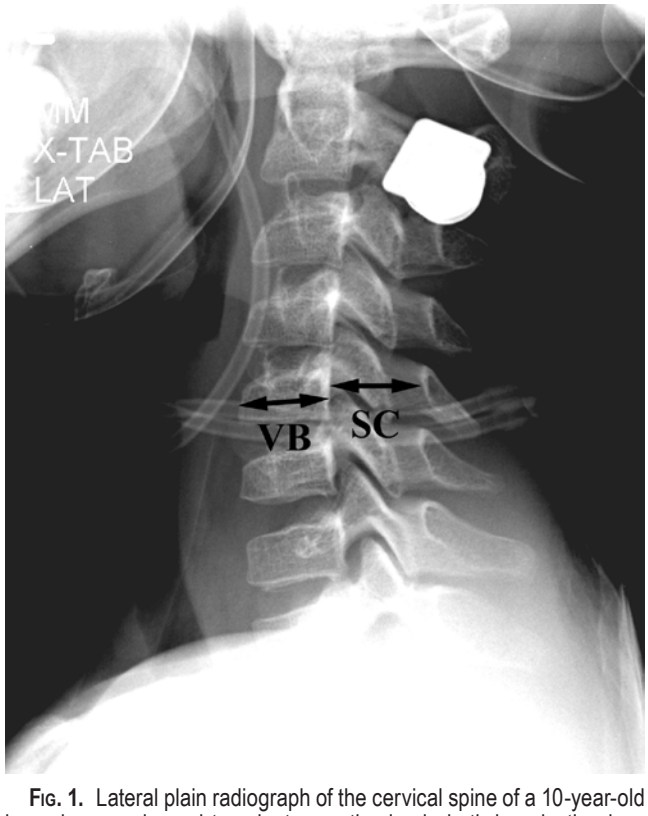

Fig. 1. Lateral plain radiograph of the cervical spine of a 10-year-old boy who experienced transient paresthesias in both legs lasting less than 24 hours after a hyperextension injury during football practice. The Torg ratio is calculated as the ratio of the spinal canal diameter (SC, distance from the midpoint of the posterior vertebral body to the nearest point on the spinolaminar line) to the vertebral body diameter (VB). The Torg ratio at C-4 in this patient is > 0.8 and therefore demonstrates no evidence of cervical spinal stenosis.

Cervical Cord Neurapraxia in Adult Athletes

A large epidemiological study[23] compared athletes who reported an episode of cervical cord neurapraxia to athletes and nonathletes who had never experienced neurapraxia and found that those with previous neurapraxia had significantly smaller cervical spinal canals and lower Torg ratios, suggesting an association between stenosis and neurapraxia. A smaller series[21] of 9 rugby players with cervical cord neurapraxia demonstrated 4 athletes with Torg ratios < 0.8 and an additional 2 athletes with congenital vertebral body fusions. Another series[12] of 2 professional football players, each with an episode of cervical cord neurapraxia, reported normal Torg ratios in both, but significant stenosis on myelography. In the largest series to date of cervical cord neurapraxia in athletes,[22] 110 patients were evaluated after 1 episode of cervical cord neurapraxia. In this series, 80% presented with symptoms in all 4 extremities, and 40% were completely plegic; 74% were Grade I (symptoms lasting < 15 minutes). On subsequent evaluation of 104 radiographs of the athletes with cervical cord neurapraxia, 86% had Torg ratios < 0.8,[22] and of these patients, 53 underwent MR imaging. More than 81% of these patients had evidence

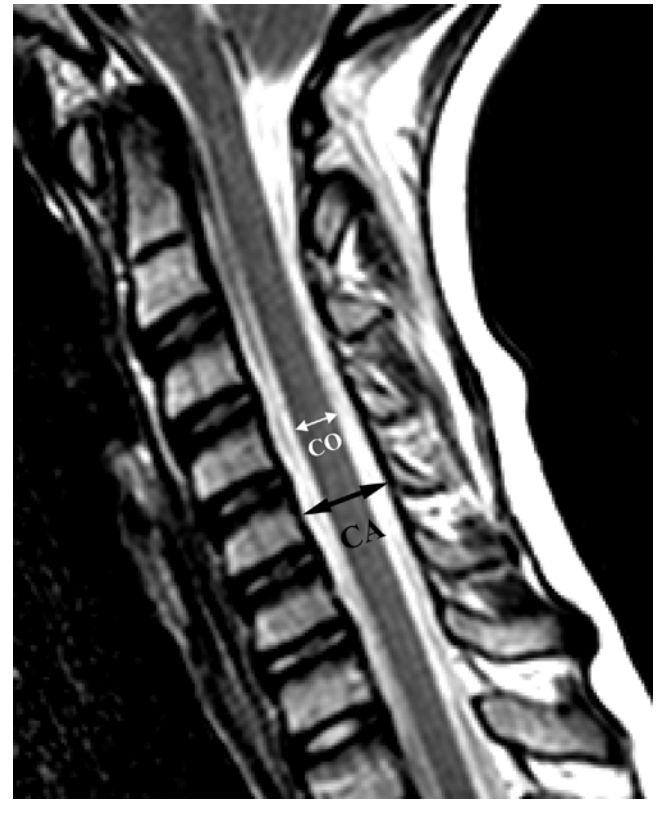

Fig. 2. Midsagittal MR image of the patient in Fig. 1. There is no evidence of a structural lesion. Presence of CSF signal surrounding the spinal cord indicates good "functional reserve" of the spinal cord. Quantification can be performed by subtracting the diameter of the spinal canal (CA) from the diameter of the spinal cord (CO).

of cervical disc herniation, 25% had evidence of effacement of the thecal sac, and 34% had frank cervical cord compression. In the largest modern series,[2] 10 athletes who experienced cervical cord neurapraxia underwent MR imaging that demonstrated cervical stenosis in all patients and frank cord compression in 3 (33%).

For patients with cervical cord neurapraxia, surgery should be considered in the setting of focal lesions and associated cord compression or instability on plain radiographs and MR imaging. In two combined series, 12 (8.5%) of 142 patients underwent surgery for cord compression or spinal instability.[22,24] The authors did not make general recommendations regarding surgical decision-making as they believed the number of patients was too small. Instead, they proposed that the decision to pursue surgery should be individualized based on imaging findings and patient wishes. Maroon et al.[13] reported a series of 5 professional-level athletes who underwent cervical decompressive surgery and fusion for focal cord compression after an episode of cervical cord neurapraxia. All 5 returned to sports, but 2 subsequently developed career-ending adjacent-level disease. The authors suggest that it is safe for athletes to return to previous levels of activity after a single-level, radiographically confirmed fusion, but close attention should be paid as these patients may develop recurrence at the level above or below.

A previous episode of cervical cord neurapraxia may predispose athletes to recurrent episodes, but the risk of recurrence is determined by a complex interplay between the patient's cervical spine anatomy and the type of athletic activity. One series reports 52 patients who returned to sports after cervical cord neurapraxia, 32 (62%) of whom experienced a subsequent episode. Of the athletes who returned to previous levels of activity after an episode of cervical cord neurapraxia in previous large series, none subsequently developed a permanent neurological injury.[24] Conversely, of the athletes who sustained permanent neurological injury, none reported a previous episode of neurapraxia, leading the authors to suggest that cervical cord neurapraxia does not necessarily confer an increased risk of permanent injury. However, 1 case report describes a football player who became quadriparetic from a subsequent injury 1 year after an episode of cervical cord neurapraxia.[7] Consequently, some practitioners would consider a single episode of cervical cord neurapraxia to be a contraindication to return to sports. With respect to cervical stenosis, Bailes'[2] report included 4 athletes with cervical stenosis who returned to play after an episode of cervical cord neurapraxia. None of these athletes experienced a subsequent episode and, interestingly, all 4 had intact "functional reserve" (CSF signal surrounding the spinal cord) on MR imaging.

In 1962, Penning[20] described a "pincers mechanism" by which extension of the cervical spine can cause myelopathy that can also be applied to the mechanics of cervical cord neurapraxia. Penning studied lateral flexion-extension radiographs and developed a model of spinal cord "pinching" between the posterior inferior aspect of the superior vertebral body and the anterior superior aspect of the inferior lamina during extension. In addition, loss of tension on the dura and the ligamentum flavum caused these structures to protrude into the spinal canal, further decreasing the canal reserve with the neck extended. Torg et al.[22] extrapolated these findings to explain that, during flexion, the spinal cord is compressed between the lamina of the superior level and the posterior superior aspect of the inferior vertebral body. In the stenotic canal of an adult, the pincer mechanism is likely more profound. Experimental studies in a giant squid axon model of cord deformation demonstrated that during injury there was an increase in intracellular calcium.[25] Depending on the strength and duration of the injury, the chemical disturbance can be either reversible or irreversible, leading to permanent cellular damage. This can be applied to the phenomenon of sports-related cervical neurapraxia that results from a short duration injury of moderate magnitude that causes the spinal cord to be deformed by the "pincers mechanism," which causes reversible chemical changes in the spinal cord below the level of injury. This is expressed symptomatically as a transient neurological deficit.

Cervical Cord Neurapraxia in Pediatric Athletes

In the large Torg et al.[22] series, 7 patients had normal Torg ratios (that is, no evidence of cervical spinal stenosis), and the mean age of these patients was 17 years old. Only 1 study[6] has specifically evaluated the association between cervical spinal stenosis and cervical cord neurapraxia in pediatric patients. Boockvar et al.[6] retrospectively reviewed 13 children younger than 16 years of age who presented to the Children's Hospital of Philadelphia with cervical cord neurapraxia. The most common mechanism of injury was hyperflexion (38%). There were significant differences in symptomatology relative to adult athletes. In contrast to adult patients, the majority of children (77%) reported neck pain and decreased cervical range of motion. The distribution of deficits was most commonly upper-extremity paresis (38%), followed by quadriparesis (31%), hemiparesis (23%), and lower-extremity paresis (8%). The duration of symptoms was longer than in adults, with a mean duration of 26 hours, with 1 patient experiencing quadriparesis and paresthesias for 5 days. The majority of patients had combined motor and sensory disturbances (85%). No patients were completely plegic.

Torg ratios were calculated for all 13 patients.[6] Interestingly, all patients had Torg ratios > 0.8 indicating that none had cervical spinal stenosis by traditional radiographic criteria. Magnetic resonance imaging was performed within 24 hours of injury and none of the patients demonstrated evidence of spinal cord or extraneural pathology, which often appear in adults. No patients were treated with cervical spine surgery. Neurological symptoms resolved in all patients. Follow-up flexion-extension radiographs confirmed cervical stability. Ten of 13 patients had long-term follow-up, and all of these patients had returned to previous levels of activity including sports. None reported recurrence of neurapraxia symptoms. None had experienced a subsequent permanent neurological injury. Although the number of patients is small, this evidence suggests that children can safely return to athletic activities after an episode of cervical cord neurapraxia. Similarly, in the series by Torg et al.,[22]

3 of the 7 patients with cervical cord neurapraxia and normal Torg ratios returned to contact sport activity with no recurrence. Future large-scale studies are needed to confirm that cervical cord neurapraxia does not incur an increased risk of future neurological injury.

The observation that cervical cord neurapraxia in children is not associated with cervical spinal stenosis is indicative of a different mechanism of neurological deficit in this unique population. In contrast to adults, the pediatric cervical spine is more mobile, likely due to more compliant ligaments,[3] underdeveloped paraspinal musculature,[18] increased water content of intervertebral discs,[9] and immature facet joints.[8] It was proposed that in this setting, the mobility of the spine allows the spinal cord to stretch past its tolerance or allows the spinal cord to forcibly contact the bony elements of the spine resulting in transient neurological symptoms. Therefore, even in the absence of cervical spinal stenosis, injury can occur. The phenomenon of spinal cord injury without radiographic abnormality describes the potential consequence of this increased mobility.[17] Spinal cord injury without radiographic abnormality is generally associated with extreme forces such as a motor vehicle accident. Cervical cord neurapraxia in children can be considered a mild form of spinal cord injury without radiographic abnormality in which the forces that deform the spine are sufficient to cause reversible perturbation of spinal cord physiology without permanently damaging the cord.

Guidelines for Return to Play After Cervical Neurapraxia

Clearance of athletes for resumption of physical and athletic activity is a highly controversial topic and one that is often without consensus opinion.[15] Fundamental requirements for returning to athletic activity after a cervical injury with neurapraxia should include normal strength, painless range of motion, and a stable vertebral column.[14] Bailes[2] suggests that patients with MR imaging evidence of CSF signal surrounding the cervical cord may be safe to return to play. Further considerations should be the mechanism of the original injury, objective physical examination and radiographic findings, and the athlete's recovery response.[26] Page and Guy[16] recommend that absolute contraindications for return to play after cervical neurapraxia are ligamentous instability, a single neurapraxic event with evidence of cord damage, multiple events, and/or events with symptoms lasting longer than 36 hours.

Conclusions

Cervical cord neurapraxia is common in adult and pediatric athletes. Cervical cord neurapraxia is associated with cervical spinal stenosis in adult athletes but not in the pediatric population. This observation likely highlights a mechanistic difference in the injury in the two different age groups. In adults, a stenotic canal will predispose patients to cervical cord injury at the level of stenosis following an extension, flexion, or axial load injury. Therefore, surgery should be considered for a focal lesion causing cord compression. In comparison, the pediatric spine demonstrates increased mobility, predisposing the spinal cord to contact with bony elements with stretching even in the absence of a focal stenosis. Although symptoms invariably resolve, recurrences are not uncommon, most notably in adults. Patients should be advised of this risk when considering return to sports-related activities.

Disclosure

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

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References

1. Andrews FJ: Transient cervical neurapraxia associated with cervical spine stenosis. **Emerg Med J 19:**172–173, 2002
2. Bailes JE: Experience with cervical stenosis and temporary paralysis in athletes. **J Neurosurg Spine 2:**11–16, 2005
3. Bailey DK: The normal cervical spine in infants and children. **Radiology 59:**712–719, 1952
4. Berge J, Marque B, Vital JM, Sénégas J, Caillé JM: Age-related changes in the cervical spines of front-line rugby players. **Am J Sports Med 27:**422–429, 1999
5. Boden BP, Tacchetti RL, Cantu RC, Knowles SB, Mueller FO: Catastrophic cervical spine injuries in high school and college football players. **Am J Sports Med 34:**1223–1232, 2006
6. Boockvar JA, Durham SR, Sun PP: Cervical spinal stenosis and sports-related cervical cord neurapraxia in children. **Spine (Phila Pa 1976) 26:**2709–2713, 2001
7. Cantu RC: Cervical spine injuries in the athlete. **Semin Neurol 20:**173–178, 2000
8. Cattell HS, Filtzer DL: Pseudosubluxation and other normal variations in the cervical spine in children. A study of one hundred and sixty children. **J Bone Joint Surg Am 47:**1295–1309, 1965
9. Henrys P, Lyne ED, Lifton C, Salciccioli G: Clinical review of cervical spine injuries in children. **Clin Orthop Relat Res (129):**172–176, 1977
10. Herzog RJ, Wiens JJ, Dillingham MF, Sontag MJ: Normal cervical spine morphometry and cervical spinal stenosis in asymptomatic professional football players. Plain film radiography, multiplanar computed tomography, and magnetic resonance imaging. **Spine (Phila Pa 1976) 16 (6 Suppl):**S178–S186, 1991
11. Kessler JT: Congenital narrowing of the cervical spinal canal. **J Neurol Neurosurg Psychiatry 38:**1218–1224, 1975
12. Ladd AL, Scranton PE: Congenital cervical stenosis presenting as transient quadriplegia in athletes. Report of two cases. **J Bone Joint Surg Am 68:**1371–1374, 1986
13. Maroon JC, El-Kadi H, Abla AA, Wecht DA, Bost J, Norwig J, et al: Cervical neurapraxia in elite athletes: evaluation and surgical treatment. Report of five cases. **J Neurosurg Spine 6:**356–363, 2007
14. Morganti C: Recommendations for return to sports following cervical spine injuries. **Sports Med 33:**563–573, 2003
15. Morganti C, Sweeney CA, Albanese SA, Burak C, Hosea T, Connolly PJ: Return to play after cervical spine injury. **Spine (Phila Pa 1976) 26:**1131–1136, 2001
16. Page S, Guy JA: Neurapraxia, "stingers," and spinal stenosis in athletes. **South Med J 97:**766–769, 2004

17. Pang D: Spinal cord injury without radiographic abnormality in children, 2 decades later. **Neurosurgery 55:**1325–1343, 2004
18. Pang D, Wilberger JE Jr: Traumatic atlanto-occipital dislocation with survival: case report and review. **Neurosurgery 7:** 503–508, 1980
19. Pavlov H, Torg JS, Robie B, Jahre C: Cervical spinal stenosis: determination with vertebral body ratio method. **Radiology 164:**771–775, 1987
20. Penning L: Some aspects of plain radiography of the cervical spine in chronic myelopathy. **Neurology 12:**513–519, 1962
21. Scher AT: Spinal cord concussion in rugby players. **Am J Sports Med 19:**485–488, 1991
22. Torg JS, Corcoran TA, Thibault LE, Pavlov H, Sennett BJ, Naranja RJ Jr, et al: Cervical cord neurapraxia: classification, pathomechanics, morbidity, and management guidelines. **J Neurosurg 87:**843–850, 1997
23. Torg JS, Naranja RJ Jr, Pavlov H, Galinat BJ, Warren R, Stine RA: The relationship of developmental narrowing of the cervical spinal canal to reversible and irreversible injury of the cervical spinal cord in football players. **J Bone Joint Surg Am 78:**1308–1314, 1996
24. Torg JS, Pavlov H, Genuario SE, Sennett B, Wisneski RJ, Robie BH, et al: Neurapraxia of the cervical spinal cord with transient quadriplegia. **J Bone Joint Surg Am 68:**1354–1370, 1986
25. Torg JS, Thibault L, Sennett B, Pavlov H: The Nicolas Andry Award. The pathomechanics and pathophysiology of cervical spinal cord injury. **Clin Orthop Relat Res (321):**259–269, 1995
26. Vaccaro AR, Klein GR, Ciccoti M, Pfaff WL, Moulton MJ, Hilibrand AJ, et al: Return to play criteria for the athlete with cervical spine injuries resulting in stinger and transient quadriplegia/paresis. **Spine J 2:**351–356, 2002

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Spinal injury patterns among skiers and snowboarders

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Object. Skiing and snowboarding injuries have increased with the popularity of these sports. Spinal cord injuries (SCIs) are a rare but serious event, and a major cause of morbidity and mortality for skiers and snowboarders. The purpose of this study is to characterize the patterns of SCI in skiers and snowboarders.

Methods. The authors queried the Nationwide Inpatient Sample for the years 2000–2008 for all patients admitted with skiing or snowboarding as the mechanism of injury, yielding a total of 8634 patients. The injury patterns were characterized by the ICD-9 diagnostic and procedure codes. The codes were searched for those pertaining to vertebral and skull fracture; spinal cord, chest, abdominal, pelvic, and vessel injuries; and fractures and dislocations of the upper and lower extremity. Statistical analysis was performed with ANOVA and Student t-test.

Results. Patients were predominantly male (71%) skiers (61%), with the average age of the skiers being older than that of snowboarders (39.5 vs 23.5 years). The average length of stay for patients suffering from spine trauma was 3.8 days and was increased to 8.9 days in those with SCI. Among hospitalized patients, SCI was seen in 0.98% of individuals and was equally likely to occur in snowboarders and skiers (1.07% vs 0.93%, $p < 0.509$). Cervical spine trauma was associated with the highest likelihood of SCI (19.6% vs. 10.9% of thoracic and 6% of lumbar injuries, $p < 0.0001$). Patients who were injured skiing were more likely to sustain a cervical spine injury, whereas those injured snowboarding had higher frequencies of injury to the lumbar spine. The most common injury seen in tandem with spine injury was closed head injury, and it was seen in 13.4% of patients. Conversely, a spine injury was seen in 12.9% of patients with a head injury. Isolated spine fractures were seen in 4.6% of patients.

Conclusions. Skiers and snowboarders evaluated at the hospital are equally likely to sustain spine injuries. Additionally, participants in both sports have an increased incidence of SCI with cervical spine trauma.
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Key Words • **epidemiological study** • **skiing** • **snowboarding** • **spine injury**

Skiing and snowboarding are commonly enjoyed as winter sports and are growing in popularity, which has coincided with an increase in injuries in both sports.[2] With injury rates ranging from 2 to 6 per 1000 days of skiing or snowboarding, both sports are considered to be fairly safe. Despite the low incidence, there tends to be high morbidity associated with these injuries, because they typically occur at high speeds. Accidents involving collision with trees or other obstacles are the most common causes of injury and death seen in skiers,[7,11] whereas intentional jumping over obstacles higher than 2 m is consistently reported as the most frequent cause of injury in snowboarders.[1,13] Most injuries in skiers occur in the lower extremities, most commonly as a tibial fracture or anterior cruciate ligament strain.[2,7,13] Snowboarding accidents lead to fewer lower-extremity injuries, but to more wrist fractures and a higher incidence of splenic injuries.[1]

Although SCIs are a relatively rare event,[12] they result in significant morbidity and mortality when they do occur. One study suggested that snowboarders had a higher risk of spine injuries.[1] The inherent differences in skiing and snowboarding, such as stance, preferred terrain, and maximum speeds attained, probably account for the differences in severity and patterns of injury. A later study suggests that the risk of SCI is approximately equal in both skiers and snowboarders.[12]

In the present study, the NIS was used to attain a large study population, and our investigation aimed to describe SCI patterns in those who suffer a vertebral column fracture while skiing or snowboarding. This study is a descriptive analysis of spinal column injuries and SCIs seen in skiers and snowboarders who sustained trauma requiring hospital evaluation.

Methods

The NIS was obtained from the Agency for Healthcare Research and Quality for the years 2000–2008. The NIS represents the largest database of hospital admissions

Abbreviations used in this paper: NIS = Nationwide Inpatient Sample; SCI = spinal cord injury; SCIWORA = SCI without radiographic abnormality.

in the US, includes all payers, and with approximately 8 million entries per year, it accounts for a stratified sample of approximately 20% of all inpatient admissions. For the year 2008, the NIS contains data obtained from 1056 hospitals in 42 states. The authors queried the NIS for the years 2000–2008 for all patients admitted with skiing or snowboarding as the mechanism of injury, yielding a total of 8634 patients. Patient sex, age, in-hospital death, and the day of the week and month of injury were recorded. The injury patterns were characterized using the ICD-9 diagnostic and procedure codes. The codes were searched for those pertaining to vertebral and skull fracture; spinal cord, chest, abdominal, pelvic, and vessel injuries; and fractures and dislocations of the upper and lower extremity. Procedure codes for fusion of any vertebral level, laminectomy, or any surgical spine repair were extracted from the sample set (Table 1). The length of stay, need for repeat surgery, and presence of thromboembolic complication were assessed for each patient. Statistical software (SPSS, Inc.) was used to search the database for each of the above-mentioned codes. Statistical analysis was completed using the Fisher exact, chi-square, ANOVA, and Student t-tests in GraphPad Prism (GraphPad Software, Inc.).

Results

Patient Demographic Data

Patients were predominantly male (71%) skiers (61%), with the average age of the skiers being older than that of snowboarders (39.5 vs 23.5 years). Nine (25%) of 36 snowboarders with an SCI were younger than 18 years of age, compared with 9 (18.3%) of 49 skiers (Table 2). The

TABLE 1: List of ICD-9 codes queried for injuries, procedures, and complications related to ski and snowboard accidents

Description of Injury	ICD-9 Injury Codes
cause of injury	
fall from skis	E885.3
fall from snowboard	E885.4
closed head injury	
concussion, contusion, intracranial bleed	850–854
skull fracture	
skull vault	800
skull base	801
facial fracture	802
other skull fracture	803, 804
nervous system injury	
SCI	806
cranial nerve injury	950, 951
peripheral nerve injury	955, 956
SCIWORA	952
spine fracture	
cervical vertebrae/dislocation	805.0, 805.1, 806.0, 806.1, 839.0, 839.1
thoracic vertebrae/dislocation	805.2, 805.3, 806.2, 806.3, 839.21, 839.31
lumbar vertebrae/dislocation	805.4, 805.5, 806.4, 805.5, 839.20, 839.30
sacral	805.6, 805.7, 806.6, 806.7
pelvic fracture	
anywhere in pelvis	808
limb injury	
upper extremity	810–819, 880–887, 840–842
lower extremity	820–829, 890–897, 843–846
dislocations	830–838
chest injury	
internal chest injuries	860–862
rib fracture	807
abdominal injury	
internal injuries	863–868
vessel injury	
carotid artery	900

TABLE 2: Demographic data for patients with ski and snowboard injuries obtained from the NIS

Characteristic	No. (%)		
	Total	Skiers	Snowboarders
no. of patients	8634 (100)	5277 (61)	3357 (39)
sex			
M	6096 (71)	3423 (65)	2673 (80)
F	2538 (29)	1854 (35)	684 (20)
average age in yrs	33.3	39.5	23.5
range	2–99	2–99	2–97
no. of injuries			
vertebral	510 (5.9)	433 (8.2)	77 (2.3)
SCI	85 (0.98)	49 (0.93)	36 (1.07)

majority of injuries occurred in February, followed by March and January (23.1%, 21.8%, and 21.2%, respectively; $p < 0.002$). Among hospitalized patients, SCI was seen in 0.98% of the patient sample and was equally prevalent in snowboarders and skiers treated in the hospital (1.07% vs 0.93%; $p < 0.509$ [not significant]). Of the subset of patients who suffered an SCI, 57.6% were injured while skiing and 42.4% were injured while snowboarding.

Surgical Intervention

Isolated spine injuries were seen in 4.6% of patients. Skiers had higher rates of fracture or dislocation in the cervical spine, followed by thoracic and lumbar spine (3%, 2.7%, and 2.5%, respectively; $p < 0.0001$ between cervical and lumbar spine). Snowboarders were more likely to suffer lumbar or thoracic injury than trauma in the cervical spine (4.9% lumbar, 3.9% thoracic, 2.6% cervical; $p < 0.0001$). In the analysis of patients who suffered a spine injury with or without SCI, skiers were more likely to injure the cervical spine (40.7% in skiers vs 25.3% in snowboarders; $p < 0.0001$), whereas snowboarders were more likely to injure the lumbar spine (47.6% in snowboarders vs 35.2% in skiers; $p < 0.0007$). However, in both groups, cervical spine trauma was associated with the highest likelihood of SCI (19.6%, vs 10.9% of thoracic and 6.0% of lumbar injuries; $p < 0.0001$) (Fig. 1 upper). This held true when analyzed according to sport as well, as seen in Fig. 1 lower.

Of those who sustained a cervical SCI, 66.6% underwent surgical fixation compared with 56.7% of those with a thoracic injury and 55.6% of those with a lumbar injury. Patients with SCI associated with a thoracic fracture were more likely to undergo surgical fusion than those without SCI (OR 11.93, 95% CI 5.171–27.54). Similarly, in patients with a lumbar spine fracture, there was a significantly increased tendency to undergo spinal fusion if there was an associated SCI (OR 13.4, 95% CI 4.847–37.25). Patients with cervical spine injuries were equally likely to have fusion with and without SCI (OR 1.3, 95% CI 0.707–2.540) (Fig. 2).

Of note, SCIWORA, although rarely occurring, was seen almost twice as frequently in skiers as in snowboarders (1.17% vs 0.54%, $p < 0.0001$).

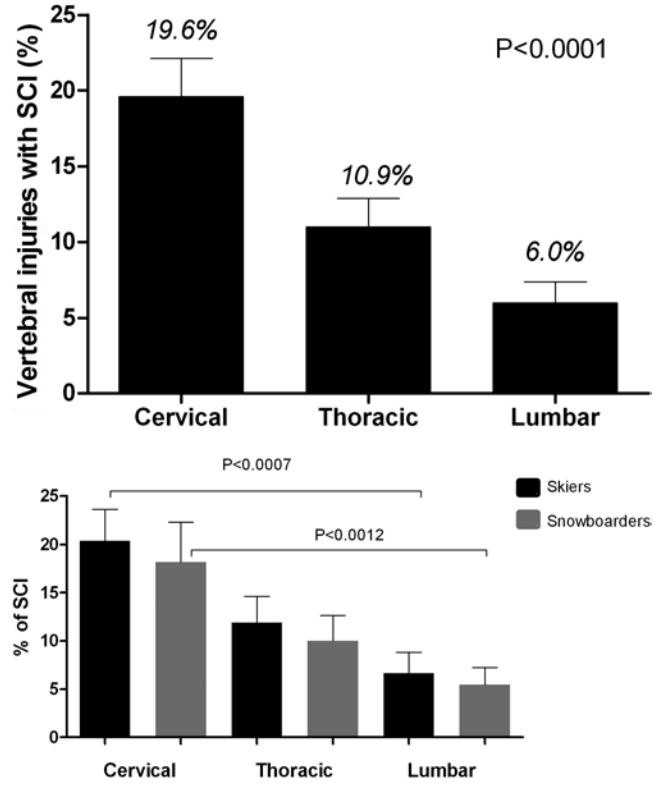

Fig. 1. Bar graphs showing associations between trauma level and SCI. **Upper:** The SCI level among injured skiers and snowboarders was significantly correlated with the vertebral level of the trauma. Cervical spine trauma had the highest incidence of SCI, at 19.6%, compared with 10.9% in thoracic, and 6.0% in lumbar spine fractures ($p < 0.0001$). **Lower:** The association between higher vertebral level and increased likelihood of SCI was maintained across both skiers and snowboarders.

Multiply Injured Patients

The most common injury seen in tandem with spine injury was closed head injury, and it was seen in 13.4% of patients. Conversely, a spine injury was seen in 12.9% of patients with a head injury. Among those with spine injuries, snowboarders had almost twice as many closed head injuries compared with their skiing counterparts (14.0% and 7.8%, $p < 0.0001$).

Excluding head injuries, skiers who had thoracic and lumbar vertebral injury were more likely to have multiorgan trauma than skiers who had cervical spine injuries (48% for thoracic and 46% for lumbar vs 19% for cervical; $p < 0.004$). A similar trend was seen in snowboarders; 24% with thoracic trauma, 27% with lumbar trauma, and 14% with cervical trauma had multiple injuries ($p < 0.06$).

In general, snowboarders were more likely to have injuries to their upper extremities, (48% vs 13% of skiers, $p < 0.0001$), whereas skiers had significantly more lower-extremity injuries (58.6% vs 22.3% of snowboarders, $p < 0.0001$). Patients who had an SCI were more likely to be discharged to a rehabilitation facility than those with injuries not including SCI (OR 17.02, 95% CI 11.02–26.30).

Length of Stay and Hospital Charges

On average, patients who suffered an SCI stayed 5.8

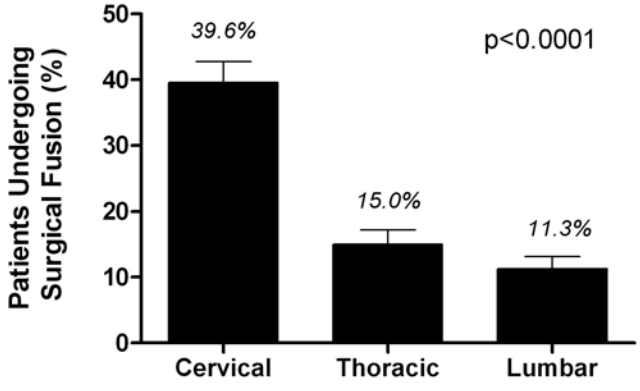

Fig. 2. Bar graph showing that operative stabilization was performed most frequently among skiers and snowboarders with cervical fractures (39.6%) compared with thoracic (15.0%) and lumbar spine injuries (11.3%), with or without SCI.

days longer than their counterparts without SCI (8.8 days vs 3.0 days, $p < 0.0001$). Patients who suffered an SCI also had higher costs associated with their hospitalizations ($94,795 vs $29,479; $p < 0.0001$). Patients with SCI were more than 8 times as likely to be discharged to a rehabilitation facility versus home or self-care after their hospital stay (53% of patients with SCI vs 6.2% patients without SCI, $p < 0.0001$). Skiers and snowboarders had similar length of hospital stays and charges accrued (2.9 vs 3.2 days and $20,175 vs $22,824, respectively).

Discussion

As skiing and snowboarding become more popular sports, with growing numbers of occasional participants, the rates of injuries are increasing.[3,6,7] Two studies suggested that the rates of spinal column (vertebral) injury and SCI are decreasing in overall frequency, but have changed in regard to the level injured.[5,7] The rates of neurological injury are typically in the range of 1 in 100,000 skier days, and complications leading to death are as low as 1 in 2 million skier days.[5,11–13] This study aimed to examine, on a large scale, how spinal column injuries and SCIs differed in participants in these 2 sports. As with other studies, minor injuries may not have been brought to the attention of the ski patrol or may not have required evaluation in an emergency department.[10]

Using the dataset, we calculated an estimate of the national burden of spine injuries among skiers and snowboarders. Because the NIS represents a rotating sample of approximately one-fifth of hospitalizations in the US annually, we performed an approximation of the annual injury burden by calculating the mean injury rate within the NIS for the years in question and then estimating a national average of the types of injuries in patients admitted to hospital. Between the years 2000 and 2008, there was a mean of 81.3 (range 18–126) spine injuries, leading to an estimate of 406.5 spine fractures sustained nationally by skiers and snowboarders each year. Of these spine injuries, an estimated 105.5 required surgical treatment each year nationwide.

In this population, SCI was not seen more frequently in skiers or snowboarders, which is divergent from the study by Tarazi et al.,[13] but mirrors the results seen by Sacco et al.[12] The difference may be due a deficiency in our study due to inability to calculate the total number of skiing participants or because of the substantially larger patient population in this study.

Cervical injuries were seen more frequently in skiers than in snowboarders, which is congruent with the mechanism of injury previously described for each sport.[1,7,11] The majority of injuries sustained while skiing occur due to falls or collision and have been associated with cervical injuries, whereas snowboarding injuries are more likely to occur as a result of intentional jumping and subsequent landing on the buttocks or with increased force on the legs.[4,6,9,11,13,14,16] This is contrary to what was seen in an analysis of injuries treated in a Utah hospital,[15] where the most frequent level injured was the thoracic and lumbar spine. As stated above, our data suggest that cervical spine injuries lead to more SCI and that skiers more frequently injure their cervical spine. However, it did not show statistically significantly more SCIs in skiers. We reported a higher surgical intervention rate than in previous studies[5] and noted that SCI substantially increased the incidence of fusion in lumbar and thoracic injury. Cervical fusion rates were not dependent on SCI. The high occurrence of fusion in cervical injuries without SCI could be due to surgeons' comfort level with cervical fusions compared with the thoracic spine. In our study, the exact vertebral injury level was not assessed, nor was injury to the thoracolumbar junction, which was noted to be the most common location by other authors.[4,13] Also, the type of fracture could not be assessed in this study due to the inability to review patient charts.

The proximal cause of death was not available because charts were not reviewed. However, the number of deaths in patients admitted to the hospital with ski or snowboard injuries is extremely low. Other authors have quoted the incidence of fatal ski or snowboarding injury to be as low as 1 in 2 million skier days.[5] Also, similarly to Sacco et al.,[12] a higher number of fatal injuries was seen in primarily male skiers.

The younger age of snowboarders is a trend that has been noted in several other studies.[6,8,12,14–16] Although in this study we were not able to assess skill levels in the injured participants, others have noted that most injuries occurred while patients were skiing or snowboarding at their skill level, and individuals sustaining injuries were typically intermediate or expert skiers.[11,14] This has been attributed to the higher likelihood of "jump failure" in the expert groups; these individuals are taking more risks than those in the beginner groups.

The most common mechanism of injury in snowboarders is associated with jumping.[1,16] With aerial maneuvers being cited as the culprit for increased SCIs in snowboarders,[16] it is not unreasonable to believe that more skiers will sustain injuries as they begin pushing vertical boundaries. The addition of more extreme skiing and snowboarding into the Olympics and other international competitions will serve to push those limits further.

The increased incidence of SCIWORA in skiers is interesting, because it is typically associated with pediatric injuries due to the laxity of the spinal ligaments.[8] This

differs from what would be expected when the younger population typically consists of more snowboarders than skiers. It is likely that the flexion and extension injuries seen in skiers accounts for this difference.

We did not find a difference in length of hospital stay between skiers and snowboarders, unlike others.[13,15] Length of hospital stay was dictated more by the presence of SCI, which is probably reflective of the more severe nature of the injury. The overall cost of hospitalization was not different between sports, which is expected if the length of stay is primarily determined by other variables. Similar to what was found by Sacco et al.,[12] February was the month most associated with injuries.

Limitations of the Study

As a retrospective study, there may have been some information that was not included in the initial collection of data. Coding for similar injuries may have been inconsistent because the patient population was formed using a nationwide database. In such a large, heterogeneous population, there is no way to know the total number of ski/snowboard days, and thus there is no true denominator. Also, the use of protective equipment was not recorded in the database.

Skiers and snowboarders who suffered injuries that did not necessitate an emergency room workup were not part of this study group, thus eliminating less severe injuries from this cohort. To counteract this, those who died while still at the resorts were also not included, which leads to an underestimation of fatalities in this study.

Conclusions

The prevalence of SCIs is similar among skiers and snowboarders evaluated in hospital. Additionally, participants of both sports have an increased incidence of SCI with cervical spine trauma. The most predominant difference between the groups is at what level the fracture occurred; snowboarders had more lumbar fractures and skiers had more cervical injuries. This is probably attributed to the mechanism of injury, which has been described in other studies.

Disclosure

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

Author contributions to the study and manuscript preparation include the following. Conception and design: Rughani, Dumont. Acquisition of data: Rughani, Hubbard. Analysis and interpretation of data: Rughani, Hubbard. Drafting the article: Hubbard. Critically revising the article: Rughani. Reviewed submitted version of manuscript: Rughani, Hubbard, Dumont. Approved the final version of the manuscript on behalf of all authors: Rughani. Statistical analysis: Rughani, Hubbard. Administrative/technical/material support: Jewell, Dumont. Study supervision: Jewell, Dumont.

References

1. Abu-Laban RB: Snowboarding injuries: an analysis and comparison with alpine skiing injuries. **CMAJ 145:**1097–1103, 1991
2. Ackery A, Hagel BE, Provvidenza C, Tator CH: An international review of head and spinal cord injuries in alpine skiing and snowboarding. **Inj Prev 13:**368–375, 2007
3. Chissell HR, Feagin JA Jr, Warme WJ, Lambert KL, King P, Johnson L: Trends in ski and snowboard injuries. **Sports Med 22:**141–145, 1996
4. Deady LH, Salonen D: Skiing and snowboarding injuries: a review with a focus on mechanism of injury. **Radiol Clin North Am 48:**1113–1124, 2010
5. Floyd T: Alpine skiing, snowboarding, and spinal trauma. **Arch Orthop Trauma Surg 121:**433–436, 2001
6. Franz T, Hasler RM, Benneker L, Zimmermann H, Siebenrock KA, Exadaktylos AK: Severe spinal injuries in alpine skiing and snowboarding: a 6-year review of a tertiary trauma centre for the Bernese Alps ski resorts, Switzerland. **Br J Sports Med 42:**55–58, 2008
7. Hagel BE, Goulet C, Platt RW, Pless IB: Injuries among skiers and snowboarders in Quebec. **Epidemiology 15:**279–286, 2004
8. Legome E, Shockley LW: **Trauma: A Comprehensive Emergency Medicine Approach.** Cambridge: Cambridge University Press, 2011
9. Levy AS, Smith RH: Neurologic injuries in skiers and snowboarders. **Semin Neurol 20:**233–245, 2000
10. Macnab AJ, Cadman R: Demographics of alpine skiing and snowboarding injury: lessons for prevention programs. **Inj Prev 2:**286–289, 1996
11. Prall JA, Winston KR, Brennan R: Spine and spinal cord injuries in downhill skiers. **J Trauma 39:**1115–1118, 1995
12. Sacco DE, Sartorelli DH, Vane DW: Evaluation of alpine skiing and snowboarding injury in a northeastern state. **J Trauma 44:**654–659, 1998
13. Tarazi F, Dvorak MF, Wing PC: Spinal injuries in skiers and snowboarders. **Am J Sports Med 27:**177–180, 1999
14. Wakahara K, Matsumoto K, Sumi H, Sumi Y, Shimizu K: Traumatic spinal cord injuries from snowboarding. **Am J Sports Med 34:**1670–1674, 2006
15. Wasden CC, McIntosh SE, Keith DS, McCowan C: An analysis of skiing and snowboarding injuries on Utah slopes. **J Trauma 67:**1022–1026, 2009
16. Yamakawa H, Murase S, Sakai H, Iwama T, Katada M, Niikawa S, et al: Spinal injuries in snowboarders: risk of jumping as an integral part of snowboarding. **J Trauma 50:**1101–1105, 2001

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Direct surgical repair of spondylolysis in athletes: indications, techniques, and outcomes

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Object. Athletes present with back pain as a common symptom. Various sports involve repetitive hyperextension of the spine along with axial loading and appear to predispose athletes to the spinal pathology spondylolysis. Many athletes with acute back pain require nonsurgical treatment methods; however, persistent recurrent back pain may indicate degenerative disc disease or spondylolysis. Young athletes have a greater incidence of spondylolysis. Surgical solutions are many, and yet there are relatively few data in the literature on both the techniques and outcomes of spondylolytic repair in athletes. In this study, the authors undertook a review of the surgical techniques and outcomes in the treatment of symptomatic spondylolysis in athletes.

Methods. A systematic review of the MEDLINE and PubMed databases was performed using the following key words to identify articles published between 1950 and 2011: "spondylolysis," "pars fracture," "repair," "athlete," and/or "sport." Papers on both athletes and nonathletes were included in the review. Articles were read for data on methodology (retrospective vs prospective), type of treatment, number of patients, mean patient age, and mean follow-up.

Results. Eighteen articles were included in the review. Eighty-four athletes and 279 nonathletes with a mean age of 20 and 21 years, respectively, composed the population under review. Most of the fractures occurred at L-5 in both patient groups, specifically 96% and 92%, respectively. The average follow-up period was 26 months for athletes and 86 months for nonathletes. According to the modified Henderson criteria, 84% (71 of 84) of the athletes returned to their sports activities. The time intervals until their return ranged from 5 to 12 months.

Conclusions. For a young athlete with a symptomatic pars defect, any of the described techniques of repair would probably produce acceptable results. An appropriate preoperative workup is important. The ideal candidate is younger than 20 years with minimal or no listhesis and no degenerative changes of the disc. Limited participation in sports can be expected from 5 to 12 months postoperatively. *(DOI: 10.3171/2011.9.FOCUS11180)*

Key Words • pars interarticularis repair • lumbar spondylolysis • pedicular screw • surgery • outcome • technique • athlete • sports

Spondylolysis is usually an asymptomatic pars interarticularis defect caused by a stress fracture in one or both sides of the neural ring. These fractures can lead to stimulation of the free nerve endings and cause significant back pain, mostly in young athletes ages 12–16 years.[1,6] The goals of treatment are the alleviation of pain and the restoration of stability. Conservative management with activity restriction for pain control followed by 3–6 months of lordotic bracing is recommended.[10] Despite changes in their daily activities and secession from all strenuous sports, some patients will continue to experience low-back pain. Although the incidence of unmanageable back pain in these competitive athletes is low, some individuals experience debilitating symptoms that could prevent them from pursuing their passion for sports.[32] Direct surgical repair of spondylolysis is well documented as an effective treatment in young patients in whom nonoperative treatment fails.[1,6,7,28,39]

Abbreviation used in this paper: rhBMP-2 = recombinant human bone morphogenetic protein–2.

Surgical Repair of Spondylolysis

Indications for Surgical Repair

Surgical repair of spondylolysis is indicated in cases in which low-back pain has not resolved after at least 6 months of activity modification and other nonoperative treatment modalities. Increasing pain, worsening neurological problems, and progressive listhesis also are indications for surgical consideration.[10,17] Historically, patients with high-grade spondylolisthesis are considered for multilevel fusions whereas lower grade slips or spondylolysis

without any slips is suited for direct repair.[32,34] Athletes younger than 20 years are treated with direct repair of the pars defect. Once their pain is controlled, these athletes can start muscle strengthening and range of motion exercises that, with aggressive rehabilitation programs, will have the best chance of returning them to their desired sports.

Preoperative Management

Preoperative studies should illustrate a lytic defect, minimal spondylolisthesis, healthy disc, and negligible movement of the vertebra. Plain anteroposterior and lateral flexion and extension radiographs can clearly demonstrate any degree of slippage or any motion abnormality in the vertebrae.[35] These studies should be supplemented with CT scans to define the bony anatomy of the pars.[6,10] The addition of SPECT scanning allows the detection of an occult and acute stress fracture that would otherwise be missed on plain radiographs.[10] Moreover, it would ensure the presence of metabolic activity in the lysis as the cause of pain, a factor that would increase the surgical chances of osseous union.[6] Finally, proper evaluation of the disc is done via MR imaging, which allows further investigation of the canal to rule out any other soft tissue causes of back pain. The routine use of discography is not recommended given the inherit risks of the technique, the fact that the integrity of the disc can be evaluated with MR imaging studies, and the young age of candidates for direct repair. Disc space is considered acceptable if its height is at least two-thirds its normal height and if the slippage is < 10 mm.[12] Once the above studies are obtained, direct repair of lumbar spondylolysis is ruled out if there is 1) Grade 2 or higher spondylolisthesis; 2) dysplastic lamina, which could make fixation unlikely; 3) significant disc degeneration at the level of the lysis; and 4) a patient age > 20 years.[6,17]

Overview of Surgical Techniques

In 1968, Kimura[23] reported on bone grafting without internal fixation for spondylolytic defects. Although in 1968 Scott began using a wiring technique to augment bone grafting of the lytic defect, his results were not published until 1986.[28,36] Many authors use the Scott wiring method, whereas others have modified the technique to include pedicle screws or cable instead of wire.[39] In 1970, Buck[7] documented the use of a lag screw across the lysis, and many authors have described their outcomes following this technique. In 1984, Morscher et al.[27] reported that the Buck technique of using a 3.5-mm lag screw did not work well with a thin or dysplastic lamina, and they advocated using laminar fixation with a hook screw device specially made for this purpose. That device, a modified Harrington hook that accepts a bone screw, is no longer available from the original manufacturer.[6] Other authors have reported using pedicle screws to secure the lamina with either a rod-hook construct or a V-shaped rod under the spinous process.[1,12,29,39]

Basic Surgical Technique. A standard midline approach to the lumbar spine is performed with care taken to preserve the multifidi attachment to the lateral capsules of the L4–5 and L5–S1 facet joints (unless pedicle screws will be used) and to keep the supraspinous and interspinous ligaments intact. The pars defect is exposed and fibrous tissue is removed. Direct exposure of the pars defect is unnecessary if the lytic defect is in the coronal plane. Internal fixation is applied next. Through a 3-cm window over the posterior inferior iliac spine, a small amount of cancellous bone can be harvested from the iliac crest. Some have reported harvesting cancellous bone from the ala of the sacrum, whereas others use cancellous allograft or off-label rhBMP-2 (Medtronic Sofamor Danek).[9,29] The graft is placed as an onlay at the pars defect with care taken not to place the graft ventral to the defect, a location which could compromise the exiting nerve root. Resection of the caudal 3–5 mm of the inferior facet joints of the cephalad vertebra is recommended no matter what internal fixation is selected. Theoretically, this resection reduces the possibility of the inferior facets impinging into the pars region when the patient stands or loads the spine, particularly during hyperextension.

Single Lag Screw Fixation (Buck). After exposing the pars defect and lamina bilaterally, the inferior edge of the lamina is squared off using a bur.[7] A drill is introduced at this edge and is directed upward, forward, and slightly outward to pass through the pars and across the pars defect. Direct visualization should confirm the passing of the drill through the pars defect, and the drill trajectory should remain wholly within bone. A screw of appropriate length is placed through this path, and again the screw must be seen to pass through the pars defect. The screws are partially withdrawn, and an autologous bone graft from the iliac crest is placed in the pars defect. The screws are readvanced forward through the pars defect, securing the bone grafts in place and stabilizing the construct (Fig. 1). A less invasive modification of the Buck method involves stereotactic navigation using the O-arm (Medtronic, Inc.).[5] After exposing the spinous process, a trajectory across the pars defect is determined via navigation. A Kirschner wire is passed through this trajectory, and a cannulated screw is placed over the wire across the defect.

Hook Screw Fixation (Morscher). Autologous cancellous bone grafts from the iliac crest are first placed into the pars defects.[27] Then, 2.5-mm holes are drilled at

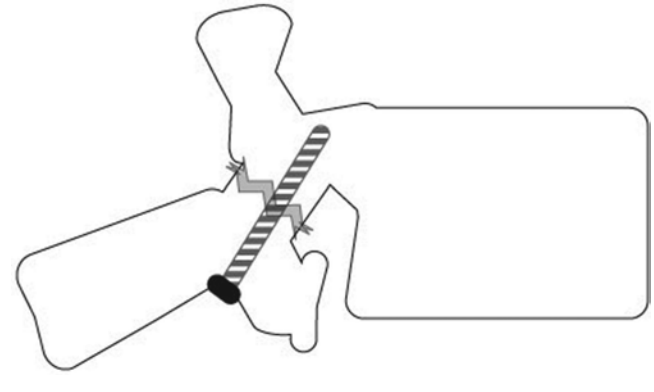

Fig. 1. Drawing showing single lag screw fixation as described by Buck, 1970.

the bases of the superior articular processes bilaterally. Special screws whose tips consist of a cancellous thread and whose bases consist of a machine thread are used. The screw head is designed to allow attachment to a hook that hooks over the lamina. The screws are inclined so that they form a 40° angle with the superior vertebral endplate. The screws are also placed approximately 20° divergent from each other. The hooks are attached to the screw heads and fastened via a lock nut. The distal end of the hook is hooked underneath the lamina. The lock nut is tightened to achieve appropriate compression over the defect (Fig. 2).

Cerclage Wire Fixation (Scott). The pars defects, laminae, and transverse processes are exposed.[28,36] The soft tissues on the anterior edge of the transverse processes are freed from the transverse processes. The sclerotic margins of the pars defects are drilled down to expose healthy bone, and the transverse process, superior facet, and lamina are decorticated. A 2-mm hole is drilled in the base of each transverse process, and a 4-mm hole is drilled in the base of the spinous process. A wire is passed through the hole in the transverse process and draped superiorly over the top of the transverse process. The other end of the wire is passed through the hole of the spinous process and draped inferiorly around the bottom of the spinous process. The same procedure is performed on the contralateral side. Autologous cancellous bone from the iliac crest is used to fill the pars defect, and strips of corticocancellous bone from the iliac crest are laid over the pars defect beneath the wires, extending from the base of the transverse process to the lamina. The wires are tightened, providing compression and stabilization across the pars defects (Fig. 3).

Pedicle Screw Cable Fixation (Songer). The bone graft is inserted into the pars defect between the pedicle above and the lamina below.[38] Pedicle screws are placed with the entry point just below the facet joint. A cable is passed underneath the left laminae, threaded through the right pedicle screw head, and finally draped around the cranial end of the spinous process. The 2 ends of the cable are tied together. A second cable is passed in a similar fashion under the right laminae and through the left pedicle screw head. The ends of the cables are crimped to apply tension to the cables (Fig. 4).

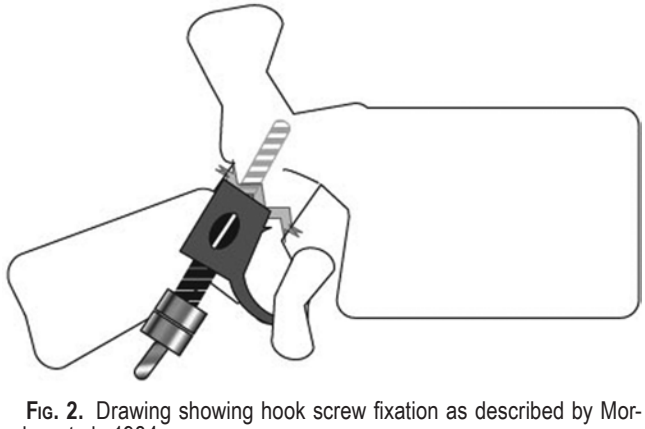

Fig. 2. Drawing showing hook screw fixation as described by Morscher et al., 1984.

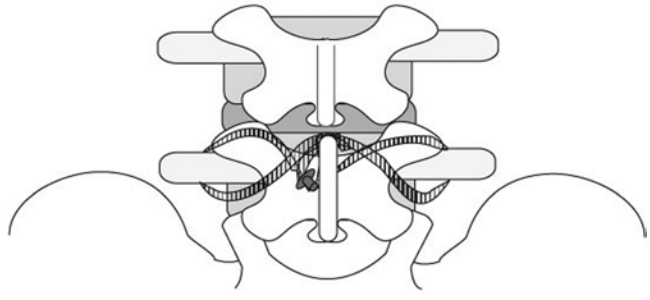

Fig. 3. Drawing depicting wire fixation as described by Scott, 1987.

Pedicle Screw Hook Fixation. Using biplanar fluoroscopy, the intersection of the transverse process, superior articular process, and pars interarticularis is cannulated with an awl for a pedicle screw. The provisional tract is tapped and a screw is placed. A high-speed bur is used to decorticate the area of the pars defect, removing the callus formation. Custom iliac crest allograft or off-label rhBMP-2 is fashioned and carefully tapped into place with a mallet. Sublaminar hooks are applied bilaterally and connected to the pedicle screw using a titanium rod. The rod is affixed to the construct using set screws, which are then tightened to the manufacturer's recommended torque (Fig. 5).[39]

A minimally invasive modification of this method involves placing the pedicle screws under fluoroscopy by using Jamshidi needles and cannulated screws.[29] Laminar hooks are placed underneath the lamina and connected to the pedicle screws.

Pedicle Screw Rod Fixation. After exposing the pars defect, transverse process, and lamina, these areas are decorticated with a high-speed drill. Pedicle screws are placed bilaterally using a standard technique. The autologous bone grafts from the iliac crests are placed in the pars defects. A U-shaped rod is placed underneath the spinous process of the affected level, and the ends of the rod are secured to each pedicle screw. Tightening the rod to the screws provides compression and stabilization across the pars defects (Fig. 6).[12]

Postoperative Routine

The postoperative course and radiographic studies are managed and tailored by each surgeon and his or her rehabilitation team. In the early postoperative period, most authors have recommended plain radiographs for asymptomatic patients and CT or MR imaging studies only if symp-

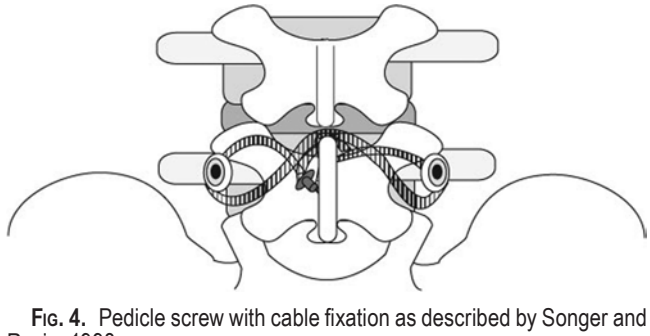

Fig. 4. Pedicle screw with cable fixation as described by Songer and Rovin, 1998.

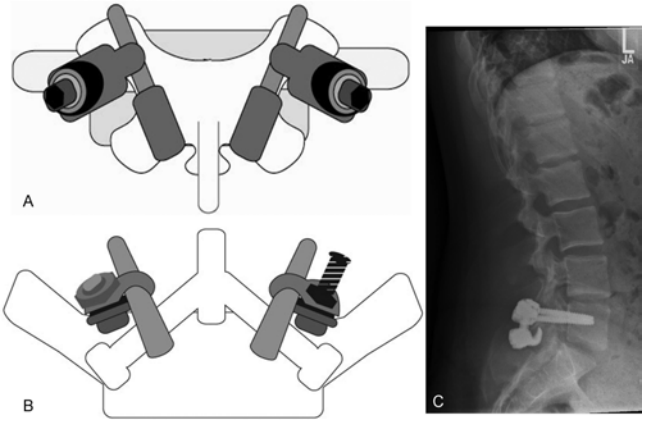

Fig. 5. Posterior **(A)** and inferior **(B)** views of the pedicle screw hook fixation as described by Tokuhashi and Matsuzaki, 1996. Postoperative lateral radiograph **(C)** showing bilateral pedicle screw hook construct at L-5 to correct a pars defect.

toms persist or new symptoms appear; however, fusion and attenuation of pars fractures are mostly assessed 3–6 months from surgery by performing CT scanning.[5,12,29,32] Early mobility and ambulation, as well as the avoidance of heavy lifting and strenuous activities for the first 3 months, are highly encouraged.[5,12,29] Additionally, patients are discouraged from any hyperextension or flexion movement in the first 3 postoperative months.[6,12] As regards to postoperative physical therapy, the literature does not provide enough reliable data; some authors recommend avoiding any physical therapy exercises as long as 3 months from the operation.[6,29] As most patients are younger adult and teenage athletes, however, they typically have a fast recovery and are able to return to sports conditioning within 6 months of their operations.[5,12,29,32]

At our institution, we follow up patients with plain anteroposterior/lateral and oblique radiographs 4 and 10 weeks from the surgery and with CT scanning in 6 months to confirm pars stabilization. Additionally, we gradually ease them into physical therapy exercises within 4 weeks after surgery while a team of physiatrists strictly monitors their progress. A team of pain specialists weans them from all narcotics within the first 2 months and, in general, by 10 weeks after surgery they are eased back into their sport activities but with limitations. They are fully released to competitive activities within 5–6 months postoperation.

In the literature, many studies outline the natural history, nonoperative treatment, and prevalence of lumbar spondylolysis in athletes. However, the literature lacks a review article on the most common surgical techniques for pars fractures and their respective outcomes in athletes. The objective in the present study was to review the surgical techniques for and outcomes of the treatment of symptomatic spondylolysis in athletes.

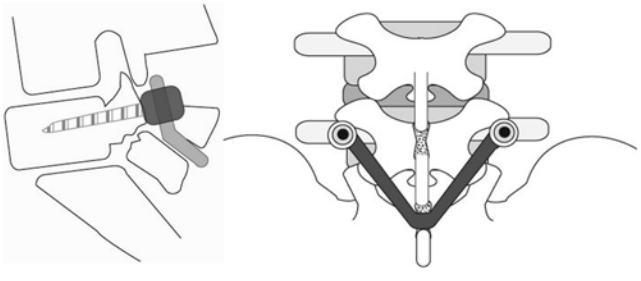

Fig. 6. Sagittal **(left)** and axial **(right)** positioning of the pedicle screw rod instrumentation as described by Gillet and Petit, 1999.

Methods

Literature Review

Both the PubMed and MEDLINE databases were searched to identify articles that had been published between 1950 and 2011 which were pertinent to the methods and outcomes of the surgical treatment of spondylolysis in athletes. The key words used in the search were "spondylolysis," "pars fracture," "repair," "sports," and/or "athlete." Inclusion criteria were full-length English-language papers or abstracts, surgical treatment, athlete outcomes, prospective studies, and retrospective studies. Exclusion criteria were non–English-language papers or abstracts and inadequate information about outcomes and/or surgical treatment. The efficacy of the surgical treatment of spondylolysis in athletes was clarified. The primary end points were descriptions of the procedures in and the outcomes of surgical treatment.

An initial search using the key words "spondylolysis" and/or "sport" returned 262 articles. The search was further limited to the English-language literature (223 articles) and a patient age of 24 years or younger (123 articles). Two separate authors (D.D. and A.S.) reviewed abstracts from these articles, yielding 123 articles for detailed review.

Of these 123 articles, 105 were excluded from analysis because they failed to meet the surgical treatment criterion or to report postoperative outcomes. The remaining 18 articles were included in our analysis. Articles were reviewed for data on methodology (retrospective vs prospective), type of treatment, number of patients, mean patient age, and mean follow-up. Specifics on the patient and the fracture included which competitive sport, level of fracture, degree of spondylolisthesis, complications, and need for reoperation. Clinical outcome data, based on patient reporting and preoperative evaluation, were also recorded when available. In addition, we used modified Henderson criteria that subjectively assessed the patient's pain and ability to return to sports (Table 1).[16]

Statistical Analysis

All descriptive statistics were calculated using JMP 7.02 (SAS Institute). Averages for age, duration of follow-

TABLE 1: Subjective assessment of pain and the ability to return to work and sporting activities

Grade	Description
excellent	no pain; return to normal occupation & normal sport
good	occasional pain after strenuous activity; return to normal occupation & less strenuous sport
poor	pain persists; unable to return to occupation & participate in sport

up, and Henderson criteria were calculated. Additionally, studies documenting patient-reported outcomes were summarized.

Illustrative Cases

History and Examination. A 23- and a 19-year-old male professional athlete both presented with several-year histories of ongoing and worsening low-back pain. At that time, both patients were being considered for competition at the highest level, and they described their symptoms as worsening with increased activity and training. The main symptom was primarily axial midlumbar back pain with occasional radiation into the gluteal region. It was aggravated by lumbar hyperextension and rotation positions and was relieved by rest. Prior to our evaluation, both patients had undergone nonoperative treatment including exhaustive physical therapy, core body strengthening, transcutaneous electrical nerve stimulation, massage, bracing, and trigger point injections. Both denied experiencing any weakness, radicular symptoms, or bowel or bladder incontinence. Each had been treated with intermittent extended periods of rest and inactivity but the symptoms would recur once training was restarted. In light of their high performance sports demands, each patient refused any invasive needle injections and permanent activity limitations or modifications. Subsequent advanced imaging was performed, and CT scans of the lumbar spine in both patients demonstrated bilateral L-5 pars fractures (Fig. 7). A SPECT CT scan confirmed increased activity at the bilateral pars fractures. Lateral flexion and extension lumbar spine radiographs showed no evidence of significant dynamic spondylolisthesis, and MR imaging showed preserved discs at L4–5 and L5–S1. Additionally, there were no signs of significant central or foraminal stenosis.

After a discussion of each patient's high functional demands and an explanation of the procedure's risks and benefits, we agreed to perform an intrasegmental bilateral pars repair using a pedicle screw rod fixation technique in each patient.

Operation. Using a standard limited midline approach centered over the L-5 lamina, we exposed the bilateral pars pseudarthroses with care not to disturb the facet capsule of L4–5. Under microscopic visualization, we used a high-speed drill bit to resect the sclerotic bony ends of the pars fracture. The underlying L-5 nerve root was identified and decompressed as it traversed the foramen. Pedicle screws were inserted into the bilateral L-5 pedicles in a laterally based trajectory to avoid facet abutment. An appropriately contoured curved rod was passed across the midline under the interspinous ligament and spinous process of L-5. The rod was affixed to each screw, causing direct compression across the pars facture sites. Once the instrumentation was secured, the bilateral pars region was grafted with locally harvested autograft (and demineralized bone matrix, if necessary).

Postoperative Course. Following the operation, each patient gradually returned to regular activity within 4 weeks under a strict physical therapy regimen. Follow-up radiographs were obtained (Fig. 8). Both patients were completely weaned off narcotic medications and returned to sports conditioning for the next 8–10 weeks. A full return to competitive sports occurred at approximately 5 months posttreatment.

Results

We identified 9 studies that specifically included athletes. One of these studies was a case report, whereas the rest were case series. One was a prospective study and the rest were retrospective. All studies involved surgical treatment. Nine studies of nonathletes were used to compare the results of the techniques applied to repair spondylolysis; all of these studies were retrospective case series involving surgical treatment. Tables 2 and 3 show the outcomes of surgical treatment in athletes and nonathletes. Eighty-four athletes and 279 nonathletes with a mean age of 20 and 21 years, respectively, were reviewed (Fig. 9). Cricket, soccer, and baseball were the most common athletics described in the studies reviewed (Fig. 10).

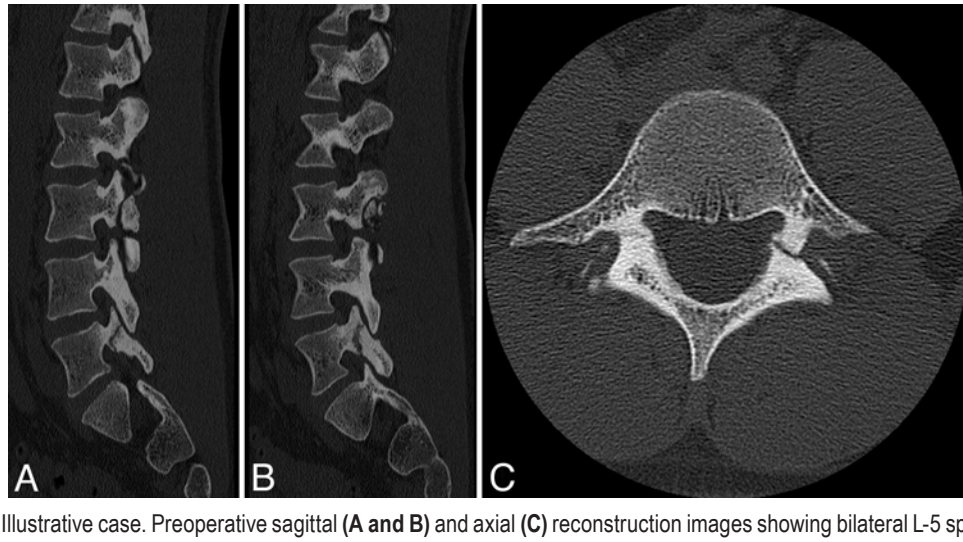

Fig. 7. Illustrative case. Preoperative sagittal **(A and B)** and axial **(C)** reconstruction images showing bilateral L-5 spondylolytic defects.

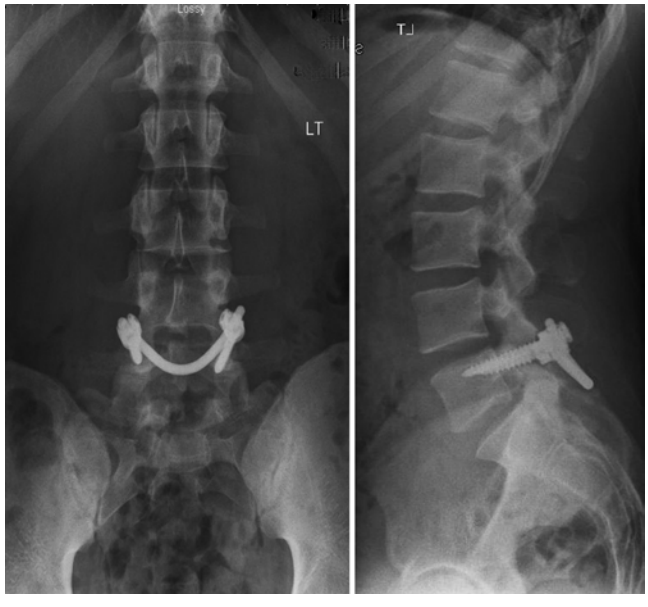

Fig. 8. Illustrative case. Postoperative anteroposterior **(left)** and lateral **(right)** radiographs showing positioning of the pedicle screw rod instrumentation.

The studies did not consistently document when a spondylolisthesis was present, but among the studies that did, there seemed to be a trend toward a higher likelihood of spondylolisthesis in nonathletes. Most of the fractures were at L-5 in both patient groups, specifically 96% in athletes and 92% in nonathletes. The average follow-up period was 26 months for athletes and 86 months for nonathletes. According to the modified Henderson criteria, 84% (71 of 84) of the athletes returned to their sports activities (Fig. 11). The time interval until their return ranged from 5 to 12 months. Of the remaining athletes, 7 returned to a less strenuous sport and 6 did not return to any sports in the study period.

Age-Related Outcomes

Direct surgical repair of spondylolysis has largely focused on patients younger than 30 years old. Most authors theorize that the outcomes are better for younger patients because their discs are less degenerative and more suitable for direct repair. The study by Ivanic et al.[17] is most often cited as evidence for a 20-year age cutoff. In that study, 113 patients were treated using the Morscher technique. Of the 20 treated patients older than 20 years of age, 35% had pseudarthroses, whereas only 8.6% of the 93 patients younger than 20 years had pseudarthroses. There was persistent postoperative pain in 4 of the 20 patients older than 20 years and in only 4 of the 93 patients younger than 20 years. In a case series by Nozawa et al.,[30] among 20 athletes treated using a Scott wiring technique, 13 were older than 20 years of age. Among the treated patients, 70% of those older than 20 years of age had an excellent clinical outcome, whereas 86% of the patients 20 years old or younger had an excellent clinical outcome.

Johnson and Thompson[21] used a modified Scott technique and found "satisfactory" results for all 19 patients under 25 years of age. In the patients older than 25 years, 2 of 3 had "poor" results. Debusscher and Troussel[9] discussed their case series in which they used pedicle screw hook fixation with 12 patients 30 years of age or younger and 11 patients older than 30 years. In the younger group, all patients had a good or excellent clinical outcome, whereas only 73% of patients in the older group had a similar outcome.

Outcomes of Specific Techniques

Single Lag Screw Fixation (Buck). Outcomes following the Buck fixation have been widely published for both nonathletes and professional athletes in a variety of sports. The published outcomes for athletes in our extensive literature search consistently showed that > 90% of patients eventually return to their preoperative sports performance.[5,8,14,31,32] Outcomes for nonathletes show similar but slightly lower success rates, possibly because athletes are often known to be highly motivated in their recovery process.[7]

Hook Screw Fixation (Morscher). The literature is lacking in data on outcomes following hook screw fixation in athletes, but there are several reports on the procedure in nonathletes. In a retrospective study of 113 patients, Ivanic et al.[17] found that > 90% of patients had excellent clinical outcomes after hook screw fixation with a mean follow-up of 10.9 years.

Cerclage Wire Fixation (Scott). Many authors have published outcomes of the Scott method in both athletes and nonathletes. The data consistently show outcomes that are not as favorable as those for other direct repair techniques. Many studies show between 60% and 80% of patients having an excellent clinical outcome.[3,21,30,35]

Pedicle Screw Cable Fixation (Songer). In 1998, Songer and Rovin[38] published a small study showing the outcomes associated with a new pedicle screw cable fixation technique. The study involved 7 patients, 5 of whom had excellent clinical outcomes. Bozarth et al.[4] modified the Songer technique in 3 patients and found that they all had excellent clinical outcomes. Our literature search did not yield further data on this technique.

Pedicle Screw Hook Fixation. Data from several retrospective studies on pedicle screw hook fixation with small numbers of nonathlete patients have been published. Noggle et al.[29] showed an excellent clinical outcome in 5 of 5 patients. Two slightly larger studies, one by Kakiuchi[22] and another by Debusscher and Troussel,[9] showed about 80% of patients having an excellent clinical outcome.

Pedicle Screw Rod Fixation. Data on pedicle screw and rod fixation have been published by 2 different authors. Gillet and Petit[12] used a V-shaped rod, whereas Altaf et al.[1] used a U-shaped rod. Both were small studies of 10 and 20 patients, respectively. Gillet and Petit found excellent clinical outcomes in only 60% of patients, whereas Altaf et al. documented excellent outcomes in 90% of patients. It is difficult to draw conclusions about this technique with only 2 small studies.

TABLE 2: Literature review of athletes with spondylolysis who underwent direct surgical repair*

Authors & Year	No. of Pts	Direct Repair Method (no. of pts)†	Sport	Mean Age in Yrs (range)	% Pts w/ Bilat Defects	% Pts w/ Spondylolisthesis	No./Vertebral Level	Average FU in Mos (range)	Outcome in % of Pts
Hardcastle et al., 1992	10	Buck	cricket	20.9 (15–25)	50	10	9 L-5, 1 L-4	17.9 (6–47)	90‡
Ranawat et al., 2003	9	Buck	cricket	21.7	50	NA	NA	68 (22–120)	100‡
Reitman & Esses, 2002	4	Buck	soccer, gymnastics, baseball	17.8 (13–22)	NA	25	L-5	26 (21–38)	100‡
Noggle et al., 2008	5	pedicle screws, rod & laminar hook	variety	15.8 (15–17)	100	NA	L-5	7.2 (6–9)	100§
Debnath et al., 2003	22	modified Buck (19) & Scott (3)	variety	20.2 (15–34)	68	NA	NA	7	95 (Buck),‡ 0 (Scott)‡
Brennan et al., 2008	1	modified Buck	baseball	17	100	100	L-5	6	100‡
Bozarth et al., 2007	3	Scott & Songer	soccer, baseball	NA	NA	NA	NA	NA	100‡
Nozawa et al., 2003	20	Scott	variety	23.7 (12–37)	NA	NA	19 L-5, 2 L-4, 1 L4–5	3.5 (1.3-8.6)**	75¶
Gillet & Petit, 1999	10	pedicle screws, V-shaped rod	tennis††	26 (16–48)	NA	0	9 L-5, 1 L-4	35 (7–64)	60§

* FU = follow-up; NA = not available; Pts = patients.
† If no number is listed in parentheses, all patients underwent listed method of repair.
‡ Returned to preoperative sports performance (athletes).
§ Asymptomatic or with minimal symptoms.
¶ Excellent outcome by Henderson criteria, that is, no pain; return to normal occupation and normal sport.
** Values expressed in years.
†† One athlete.

TABLE 3: Literature review of nonathletes with spondylolysis who underwent direct surgical repair

Authors & Year	No. of Pts	Direct Repair Method	Mean Age in Yrs (range)	% Pts w/ Bilat Defects	% Pts w/ Spondylolisthesis	No./Vertebral Level	Average FU in Yrs (range)	Outcome in % of Pts
Ivanic et al., 2003	113	Morscher	16.9 (7.5–39)	NA	95	111 L-5, 1 L-4, 1 L4–5	10.9 (1–15.5)	93*
Johnson & Thompson, 1992	22	modified Scott	15.5	91	82	21 L-5, 1 L-4	4	73†
Schlenzka et al., 2006	25	Scott	18.2	NA	NA	20 L-5, 3 L-4, 2 L-3	14.8 (11–16)	64*
	23	segmental fusion	16.1	NA	NA	all L-5	15 (13–19)	87*
Songer & Rovin, 1998	7	pedicle screws & cables	20.5 (12–32)	NA	43	all L-5	25.5 (19–37)‡	71*
Buck, 1970	16	Buck	NA	NA	NA	NA	NA	81*
Kakiuchi, 1997	16	pedicle screws, rod & laminar hook	32.4 (12–60)	NA	38	13 L-5, 1 L2–3, 1 L3–4, 1 L-3	25.2 (24–28)‡	81*
Debusscher & Troussel, 2007	23	pedicle screws, rod & laminar hook	34 (16–52)	100	52	20 L-5, 3 L-4	59 (6–113)‡	100§ ≤30 yrs old (12 pts); 73§ >30 yrs old (11 pts)
Altaf et al., 2011	20	pedicle screws, U-shaped rod	13.9 (9–21)	NA	45	all L-5	4 (2.3–7.3)	90§
Askar et al., 2003	14	Scott	17.4 (13–24)	100	36	12 L-5, 1 L-4, 1 L-3	10.9 (8–15)	43*

* Asymptomatic or minimal symptoms.
† Excellent outcome according to Henderson criteria, that is, no pain; return to normal occupation and normal sport.
‡ Values expressed in months.
§ Excellent according to Oswestry Disability Index criteria (0%–20% disability index).

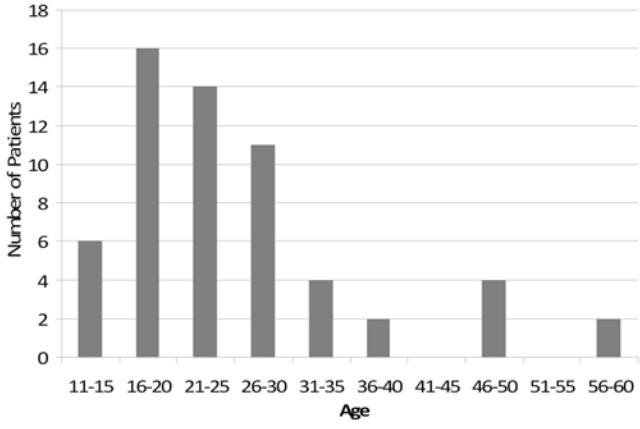

Fig. 9. Bar graph illustrating age distribution (in years) of athletic patients who underwent direct surgical repair in the reviewed studies.

Discussion

Sports involving repetitive hyperextension of the spine along with axial loading seem to most predispose athletes to spondylolysis. Specifically, these actions are thought to overload the posterior elements, leading to pars fractures.[24] Nearly all sports carry some elevated risk of spondylolysis. The sports with relatively high incidences of such injuries include gymnastics, football, hockey, diving, wrestling, baseball, volleyball, racquet sports, and weightlifting, with gymnastics and football generally considered to have the highest risk.[2,6,8,10,11,18–20,24–26,28,30] Two studies have estimated the incidence of low-back pain in athletes with spondylolysis to be 79.8% in high school football players, 72.5% in high school rugby players, and 80.5% in college football players.[18,19] Soler and Calderón[37] documented the prevalence of spondylolysis in Spanish professional athletes, and the results are summarized in Table 4. According to these

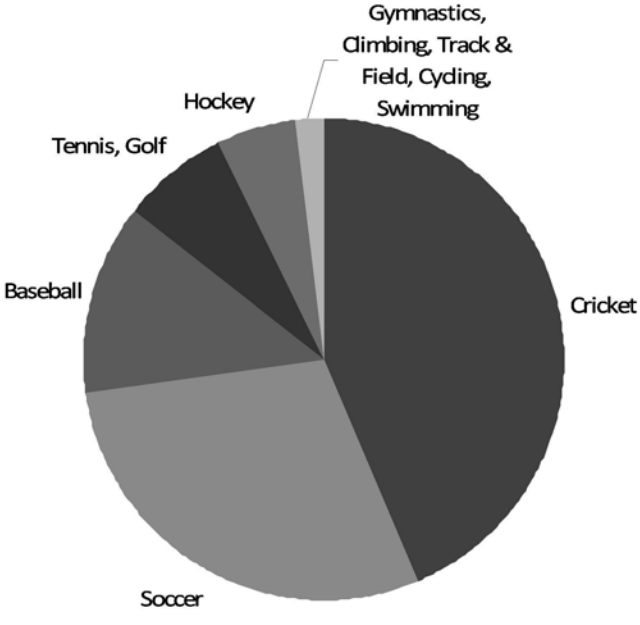

Fig. 10. Pie graph illustrating the distribution of the types of sports played by patients who underwent direct surgical repair in the reviewed studies.

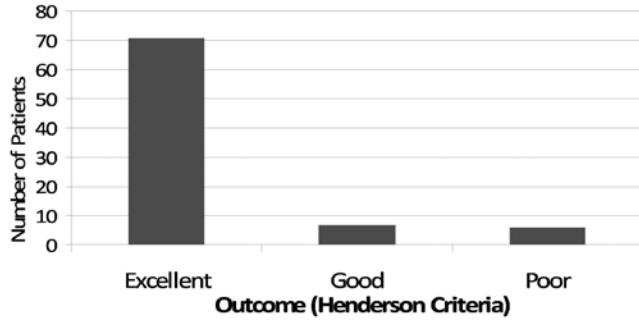

Fig. 11. Bar graph illustrating the subjective assessment of pain and the ability to return to work and sporting activities (from Henderson, 1966) in the reviewed studies.

data, activities such as rowing, gymnastics, volleyball, weightlifting, and track and field carry the highest risk of symptomatic spondylolysis.

Gymnastics is widely considered to carry one of the highest risks for pars fractures, probably because gymnasts regularly combine hyperextension of the spine with large axial forces. Studies have shown the incidence of spondylolysis among gymnasts to be anywhere from 11% to 14%.[20,37] Hall[13] recorded the impact forces produced by collegiate gymnasts executing various acrobatics using a force pad. Back handsprings led to the greatest mean lumbar curvature, as well as to a high mean force of vertical impact. Other maneuvers, such as front and back walkovers and front handsprings, resulted in large lumbar curvatures with front handsprings carrying a particularly high impact force.[13]

The incidence of pars fractures has been found to be up to 15% in college football players.[26] Although various positions are affected, linemen seem to be at particular risk for spondylolysis. Ferguson et al.[11] hypothesized that this finding was due largely to the motions of blocking, specifically extension of the lumbar sacral spine combined with the axial force of collision. Unlike other positions, linemen experience these stresses regularly and repeatedly with every play, ostensibly placing them at particular risk.

In cricket, fast bowlers seem to be most at risk for spondylolysis. One study found that the incidence of pars interarticularis defects among young fast bowlers was 55%.[14] In this case, hyperextension, lateral flexion, and thoracolumbar rotation in combination with the jerk force of bowling were hypothesized to cause spondylolysis.[31] Repeated as many as 500 times per week, these motions put immense stresses on the spine.[15]

Tennis also carries an elevated risk of pars injury. Hyperextension is thought to be the cause of these injuries, specifically those that occur during a serve.[25] In attempts to "top spin" a serve, this hyperextension may be even more pronounced. The modern forehand shot and two-handed play with its repetitive rotation also result in additional hyperextension.[2]

Baseball has also been associated with pars fractures. In professional Japanese baseball players, the incidence of pars fractures has been anywhere from 27.5% to 53.5%, far higher than in the general population.[34] Similar to injuries seen in cricket, the pitching motion is hypoth-

TABLE 4: Prevalence of asymptomatic and symptomatic spondylolysis in elite athletes from Spain

Sport	No. of Athletes	Prevalence of Spondylolysis (no. [%])	Athletes w/ Spondylolysis Who Were Symptomatic (no. [%])
track & field (races, heptathlon, & others)	685	69 (8.9)	32 (52.5)
gymnastics	235	33 (14.0)	19 (57.6)
combat sports (boxing, karate, & others)	207	23 (11.1)	9 (39.1)
swimming	176	18 (10.23)	6 (33.3)
weightlifting	85	11 (12.9)	6 (54.5)
rowing	77	13 (16.88)	8 (61.5)
volleyball	70	7 (10.0)	4 (57.1)

esized to cause most pars fractures, probably as a result of hyperextension of the spine and the rapid rotation associated with launching the ball.

Return to Athletics

For the athlete undergoing direct repair of spondylolysis, a return to preinjury athletic performance is often the primary goal. Authors reporting on surgical outcomes in athletes with these injuries use a variety of methods to measure outcome. In 1966, Henderson[16] proposed a set of criteria to measure the outcome in patients undergoing surgical treatment for spondylolisthesis (Table 1). These criteria are still used today and provide an overall view of outcomes in a large patient population.

Although the literature contains limited data on athletes, the available data reveal that professional athletes can undergo various methods of surgical treatment and achieve excellent outcomes and a return to their previous athletic performance (Fig. 12). Buck's is a popular technique for the surgical treatment of spondylolysis in athletes, with half of the studies on athletes utilizing this technique. The procedure also yields consistently high outcomes, with > 90% of athletes able to return to their previous athletic performance.[5,8,15,31,32] Other methods, such as pedicle screw hook fixation, show potential, but the limited available data make it difficult to draw conclusions.

There is no consensus on when an athlete can return to play. In a survey, Scoliosis Research Society members reported greatly varied recommendations regarding a return to sports following surgery for scoliosis.[33] The most common recommendation for a noncontact, low-impact sport was after 6 months. For contact sports, the most common recommendation was after 12 months; however, 13% of responding clinicians recommended never returning. For collision sports, 60% of responding clinicians did not favor ever resuming the sport.

After fusion for spondylolisthesis surgery, the recommendations of the Scoliosis Research Society members were similar to those made following scoliosis surgery. The most common recommendation for a noncontact, low-impact sport was after 6 months. For contact sports, the most common recommendation was after 12 months, although 14% of responding clinicians who had treated low-grade slips and 21% of responding clinicians who had treated high-grade slips recommended never returning. For collision sports, between 49% and 58% of responding clinicians did not favor ever resuming the sport.

This survey illustrates the wide differences of opinion regarding the timing and safety of returning an athlete to his or her sport following spinal surgery. Factors that must always be considered include the amount of time that has elapsed since surgery, the instrumentation that has been used, and the degree of contact or collision common to the athlete's sport, which could endanger the surgical repair and, consequently, the athlete's safety.

Complications of the Techniques

Authors report a variety of potential complications with these techniques. Hardware failure is uncommon but has been reported with all of the techniques, including screw breakages, wire and cable fractures, and wires pulled out from the transverse process.[30,32,38] For example, Ranawat et al.[31] reported the case of a professional fast bowler who had undergone L3–S1 fusion after conservative treatment had failed. During play the next season, the patient noticed swelling in the lumbar region. It was discovered that a screw had broken, and the patient was taken to surgery to remove the screw. Screw breakage happened a second time during the season, and thus all hardware from the fusion was removed.

Nonunion has been reported in several cases.[8,17,21] Pseudarthroses have been reported not uncommonly, with Ivanic et al.[17] finding that 15 of 113 patients treated with the Morscher technique had pseudarthroses and 5 required second surgeries. Rarely, authors report persistent low-back pain after surgery.[38] In general, the available studies on direct repair techniques are too small to accurately gauge complication rates.

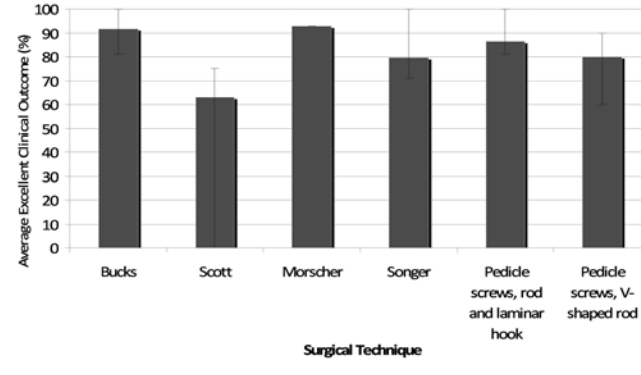

Fig. 12. Bar graph illustrating the published results of techniques used to repair spondylolysis in athletes.

Conclusions

For a young athlete with a symptomatic pars defect, any of the described techniques of repair will probably produce acceptable results. An appropriate preoperative workup is important. The ideal candidate is younger than 20 years of age with minimal or no listhesis and no degenerative changes of the disc. Limited participation in sports can be expected from 5 to 12 months postoperatively. Familiarity with the various fixation techniques and anticipation of the anatomical variations will allow the surgeon to select the most appropriate surgical technique for repairing lytic defects in the lumbar spine.

Disclosure

Dr. Rasouli is a consultant for DePuy Spine and Synthes. Dr. Kim is a consultant for EBI/Biomet. Dr. Johnson is a consultant for Alphatec, Pioneer, and Spine Wave.

Author contributions to the study and manuscript preparation include the following. Conception and design: Drazin. Acquisition of data: Drazin, Jeswani, Ching, Rosner, Rasouli. Analysis and interpretation of data: Drazin, Shirzadi, Ching, Rasouli, Pashman. Drafting the article: Drazin, Shirzadi, Jeswani, Ching, Rosner, Kim. Critically revising the article: Johnson, Drazin, Shirzadi, Jeswani. Reviewed submitted version of manuscript: Drazin, Johnson, Rasouli, Kim, Pashman. Study supervision: Johnson.

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References

1. Altaf F, Osei NA, Garrido E, Al-Mukhtar M, Natali C, Sivaraman A, et al: Repair of spondylolysis using compression with a modular link and screws. **J Bone Joint Surg Br 93:**73–77, 2011
2. Alyas F, Turner M, Connell D: MRI findings in the lumbar spines of asymptomatic, adolescent, elite tennis players. **Br J Sports Med 41:**836–841, 2007
3. Askar Z, Wardlaw D, Koti M: Scott wiring for direct repair of lumbar spondylolysis. **Spine (Phila Pa 1976) 28:**354–357, 2003
4. Bozarth GR, Fogel GR, Toohey JS, Neidre A: Repair of pars interarticularis defect with a modified cable-screw construct. **J Surg Orthop Adv 16:**79–83, 2007
5. Brennan RP, Smucker PY, Horn EM: Minimally invasive image-guided direct repair of bilateral L-5 pars interarticularis defects. **Neurosurg Focus 25(2):**E13, 2008
6. Brigham CD: Direct repair of lumbar spondylolysis in athletes. **Oper Tech Sports Med 13:**108–113, 2005
7. Buck JE: Direct repair of the defect in spondylolisthesis. Preliminary report. **J Bone Joint Surg Br 52:**432–437, 1970
8. Debnath UK, Freeman BJ, Gregory P, de la Harpe D, Kerslake RW, Webb JK: Clinical outcome and return to sport after the surgical treatment of spondylolysis in young athletes. **J Bone Joint Surg Br 85:**244–249, 2003
9. Debusscher F, Troussel S: Direct repair of defects in lumbar spondylolysis with a new pedicle screw hook fixation: clinical, functional and Ct-assessed study. **Eur Spine J 16:**1650–1658, 2007
10. Dunn IF, Proctor MR, Day AL: Lumbar spine injuries in athletes. **Neurosurg Focus 21(4):**E4, 2006
11. Ferguson RJ, McMaster JH, Stanitski CL: Low back pain in college football linemen. **J Sports Med 2:**63–69, 1974
12. Gillet P, Petit M: Direct repair of spondylolysis without spondylolisthesis, using a rod-screw construct and bone grafting of the pars defect. **Spine (Phila Pa 1976) 24:**1252–1256, 1999
13. Hall SJ: Mechanical contribution to lumbar stress injuries in female gymnasts. **Med Sci Sports Exerc 18:**599–602, 1986
14. Hardcastle P, Annear P, Foster DH, Chakera TM, McCormick C, Khangure M, et al: Spinal abnormalities in young fast bowlers. **J Bone Joint Surg Br 74:**421–425, 1992
15. Hardcastle PH: Repair of spondylolysis in young fast bowlers. **J Bone Joint Surg Br 75:**398–402, 1993
16. Henderson ED: Results of the surgical treatment of spondylolisthesis. **J Bone Joint Surg Am 48:**619–642, 1966
17. Ivanic GM, Pink TP, Achatz W, Ward JC, Homann NC, May M: Direct stabilization of lumbar spondylolysis with a hook screw: mean 11-year follow-up period for 113 patients. **Spine (Phila Pa 1976) 28:**255–259, 2003
18. Iwamoto J, Abe H, Tsukimura Y, Wakano K: Relationship between radiographic abnormalities of lumbar spine and incidence of low back pain in high school and college football players: a prospective study. **Am J Sports Med 32:**781–786, 2004
19. Iwamoto J, Abe H, Tsukimura Y, Wakano K: Relationship between radiographic abnormalities of lumbar spine and incidence of low back pain in high school rugby players: a prospective study. **Scand J Med Sci Sports 15:**163–168, 2005
20. Jackson DW: Low back pain in young athletes: evaluation of stress reaction and discogenic problems. **Am J Sports Med 7:**364–366, 1979
21. Johnson GV, Thompson AG: The Scott wiring technique for direct repair of lumbar spondylolysis. **J Bone Joint Surg Br 74:**426–430, 1992
22. Kakiuchi M: Repair of the defect in spondylolysis. Durable fixation with pedicle screws and laminar hooks. **J Bone Joint Surg Am 79:**818–825, 1997
23. Kimura M: [My method of filling the lesion with spongy bone in spondylolysis and spondylolisthesis.] **Seikei Geka 19:**285–296, 1968 (Jpn)
24. Lawrence JP, Greene HS, Grauer JN: Back pain in athletes. **J Am Acad Orthop Surg 14:**726–735, 2006
25. Maquirriain J, Ghisi JP: The incidence and distribution of stress fractures in elite tennis players. **Br J Sports Med 40:**454–459, 2006
26. McCarroll JR, Miller JM, Ritter MA: Lumbar spondylolysis and spondylolisthesis in college football players. A prospective study. **Am J Sports Med 14:**404–406, 1986
27. Morscher E, Gerber B, Fasel J: Surgical treatment of spondylolisthesis by bone grafting and direct stabilization of spondylolysis by means of a hook screw. **Arch Orthop Trauma Surg 103:**175–178, 1984
28. Nicol RO, Scott JH: Lytic spondylolysis. Repair by wiring. **Spine (Phila Pa 1976) 11:**1027–1030, 1986
29. Noggle JC, Sciubba DM, Samdani AF, Anderson DG, Betz RR, Asghar J: Minimally invasive direct repair of lumbar spondylolysis with a pedicle screw and hook construct. **Neurosurg Focus 25(2):**E15, 2008
30. Nozawa S, Shimizu K, Miyamoto K, Tanaka M: Repair of pars interarticularis defect by segmental wire fixation in young athletes with spondylolysis. **Am J Sports Med 31:**359–364, 2003
31. Ranawat VS, Dowell JK, Heywood-Waddington MB: Stress fractures of the lumbar pars interarticularis in athletes: a review based on long-term results of 18 professional cricketers. **Injury 34:**915–919, 2003
32. Reitman CA, Esses SI: Direct repair of spondylolytic defects in young competitive athletes. **Spine J 2:**142–144, 2002
33. Rubery PT, Bradford DS: Athletic activity after spine surgery in children and adolescents: results of a survey. **Spine (Phila Pa 1976) 27:**423–427, 2002
34. Sakai T, Sairyo K, Suzue N, Kosaka H, Yasui N: Incidence

and etiology of lumbar spondylolysis: review of the literature. **J Orthop Sci 15:**281–288, 2010
35. Schlenzka D, Remes V, Helenius I, Lamberg T, Tervahartiala P, Yrjönen T, et al: Direct repair for treatment of symptomatic spondylolysis and low-grade isthmic spondylolisthesis in young patients: no benefit in comparison to segmental fusion after a mean follow-up of 14.8 years. **Eur Spine J 15:**1437–1447, 2006
36. Scott JH: The Edinburgh repair of isthmic (Group II) spondylolysis. **J Bone Joint Surg Br 69:**491, 1987
37. Soler T, Calderón C: The prevalence of spondylolysis in the Spanish elite athlete. **Am J Sports Med 28:**57–62, 2000
38. Songer MN, Rovin R: Repair of the pars interarticularis defect with a cable-screw construct. A preliminary report. **Spine (Phila Pa 1976) 23:**263–269, 1998
39. Tokuhashi Y, Matsuzaki H: Repair of defects in spondylolysis by segmental pedicular screw hook fixation. A preliminary report. **Spine (Phila Pa 1976) 21:**2041–2045, 1996

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Axillary nerve injury associated with sports

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Object. The aim of this retrospective study was to present and investigate axillary nerve injuries associated with sports.

Methods. This study retrospectively reviewed 26 axillary nerve injuries associated with sports between the years 1985 and 2010. Preoperative status of the axillary nerve was evaluated by using the Louisiana State University Health Science Center (LSUHSC) grading system published by the senior authors. Intraoperative nerve action potential recordings were performed to check nerve conduction and assess the possibility of resection. Neurolysis, suture, and nerve grafts were used for the surgical repair of the injured nerves. In 9 patients with partial loss of function and 3 with complete loss, neurolysis based on nerve action potential recordings was the primary treatment. Two patients with complete loss of function were treated with resection and suturing and 12 with resection and nerve grafting. The minimum follow-up period was 16 months (mean 20 months).

Results. The injuries were associated with the following sports: skiing (12 cases), football (5), rugby (2), baseball (2), ice hockey (2), soccer (1), weightlifting (1), and wrestling (1). Functional recovery was excellent. Neurolysis was performed in 9 cases, resulting in an average functional recovery of LSUHSC Grade 4.2. Recovery with graft repairs averaged LSUHSC Grade 3 or better in 11 of 12 cases

Conclusions. Surgical repair can restore useful deltoid function in patients with sports-associated axillary nerve injuries, even in cases of severe stretch–contusion injury. *(DOI: 10.3171/2011.8.FOCUS11183)*

Key Words • axillary nerve • stretch–contusion injury • sports • deltoid muscle

Contact and low-intensity sports can result in axillary nerve injury,[16] resulting in loss of deltoid function. This muscle is the major abductor of the shoulder. The axillary nerve arises from the posterior cord of the brachial plexus and contains fibers derived from C-5 and C-6 spinal nerve roots via the posterior division of the upper trunk. It passes through the quadrilateral space along with the posterior circumflex artery just distal to the shoulder joint. The nerve then curves around the posterolateral surface of the humerus deep to the deltoid and divides into anterior and posterior branches, both of which innervate that muscle. The nerve is tethered posteriorly as a result of the overlying muscle, making it susceptible to stretch. Etiologies include not only fracture and dislocation of the humerus, but less frequently compensation due to prolonged pressure arising from common sports-related activities. The purpose of this paper is to analyze indications for surgical repair and to access outcomes in patients with solitary axillary nerve injury associated with sports.

Abbreviation used in this paper: LSUHSC = Louisiana State University Health Science Center.

Methods

Patient Population

Sports-related isolated axillary nerve injuries in our patient population were associated with the following sports: skiing (12 cases), football (5), rugby (2), baseball (2), ice hockey (2), soccer (1), weightlifting (1), and wrestling (1) (Table 1). Each patient's function was evaluated using the grading scale established by the senior authors (D.G.K. and D.H.K.) for axillary nerve palsy and deltoid muscle weakness (the LSUHSC grading scale; Table 2). Seventeen patients had complete loss of axillary activity (Grade 0); 9 had incomplete but severe functional loss (Grade 1–2), without significant spontaneous recovery. Patients who did not recover after 6–8 months of conservative therapy were treated surgically.

Electromyography was performed in all patients during their baseline evaluation. This study was repeated after an interval of several months of conservative therapy. Surgery was performed only in cases in which poor recovery of deltoid function was evidenced by both clinical and electromyographic examination. Patients selected for surgery had undergone radiographic studies and some-

TABLE 1: Sports associated with axillary nerve injury in 26 cases

Sport	No. of Cases (%)
skiing	12 (46)
football	5 (19)
rugby	2 (8)
baseball	2 (8)
ice hockey	2 (8)
soccer	1 (4)
weightlifting	1 (4)
wrestling	1 (4)

times MR imaging of the shoulder. In addition, these patients were often evaluated by orthopedists to rule out conditions resulting in comparable functional impairments, such as rotator cuff injury. In this way, a definitive diagnosis of axillary nerve injury could be established.

Surgical Procedure

An anterior infraclavicular surgical approach was used in all of our patients. In those cases in which the lesion was determined to extend significantly distally, a posterior approach was added to the anterior approach so that adequate exposure could be achieved. The patients were positioned supine and anesthetized with general endotracheal anesthesia. The table was then inclined to place the patient in an approximately 20° reverse Trendelenburg position. The patient's arm was abducted approximately 10°–20°.

The mildly curvilinear incision extended from the level of the clavicle to the axilla. At the deltopectoral groove, the pectoralis major muscle was incised and split in the direction of its fibers. A self-retaining retractor was used to hold the muscle edges apart. Laterally and medially, bundles of split pectoralis major were set aside for subsequent repair.

The deltopectoral vein and associated vascular branches were ligated at the superior aspect of the wound, and the underlying pectoralis minor muscle was divided transversely. The self-retaining retractor was then repositioned to a deeper level and placed along both edges of the pectoralis minor.

After the lateral cord was identified and dissected free from adipose and scar tissue, it was traced distally to and through its contribution to the median nerve. Next, the cord was followed past its branches to the coracobrachialis, and finally, on to its continuation as the musculocutaneous nerve. The latter was freed up several inches distal to the coracobrachialis branches by splitting the overlying biceps and brachialis muscles. The axillary artery was dissected out and traced distally along with the overlying median nerve. Often, a large venous branch crossing the median nerve was encountered. This branch was usually isolated and divided.

The posterior cord was then exposed by gently retracting the lateral cord; on some occasions, the more proximal portion of the axillary artery, and even the medial portion of the medial cord, required gentle retraction by means of application of a Penrose drain to allow visualization of the posterior cord (Fig. 1). The thoracodorsal branch extends from the posterior aspect of the posterior cord and was usually found at right angles to the cord, coursing toward the latissimus dorsi muscle. Distally, subscapularis branches were usually seen leaving the posterior cord close to its bifurcation into the axillary and radial nerves. By retracting the more distal axillary artery medially, and by displacing the musculocutaneous nerve superiorly and either medially or laterally, access to the deeper axillary nerve, including that in the quadrilateral space, could be gained.

TABLE 2: The LSUHSC grading scale for axillary nerve palsy and deltoid muscle weakness*

Grade	Definition
0	no deltoid contraction
1	trace of deltoid contraction
2	some abduction of shoulder beyond 30° w/ gravity eliminated
3	abduction of shoulder against gravity & mild pressure; lateral abduction usually 60°–90°
4	abduction of shoulder against gravity & moderate pressure; lateral abduction usually >90°
5	abduction of shoulder against gravity & great amount of pressure; lateral abduction usually >110°

* Reproduced with permission from Kline DG, Kim DH: **J Neurosurg 99:**630–636, 2003.

On occasion, posterior circumflex vessels were encountered and had to be ligated and divided. At this level, the axillary nerve passes lateral to the profundus branch of the axillary artery, whereas the radial nerve is located on its medial side (Fig. 2).

To reach the quadrilateral space, the axillary nerve follows a slightly oblique course laterally and posteriorly. Exposure of the nerve down to the level of the quadrilateral space was sometimes assisted by deep placement of 1 or 2 small self-retaining retractors. Just distal to the inferior shoulder joint capsule, the nerve passes through the quadrilateral space along with the posterior circumflex humeral artery. The quadrilateral space is bounded by the teres minor above, the long head of the triceps medially, the teres major and latissimus dorsi muscles below, and the humeral neck laterally. Posteriorly, the axillary nerve divides into anterior and posterior branches. The posterior branch supplies the posterior portions of the deltoid muscles, and a small branch from this posterior division innervates the teres minor. The posterior branch of the axillary nerve also supplies sensation to the skin overlying the deltoid muscle. The anterior branch curls around the surgical neck of the humerus under the deltoid muscle and branches as it extends to the anterior border of the deltoid. The branches further divide to innervate this muscle (Fig. 3). The deltoid is composed of 3 bundles of muscle fibers that contract in concert to raise the arm laterally. Contraction of the anterior bundles, however, provides a more significant contribution to anterior or forward abduction. This motion is aided by the pectoralis major muscle and sometimes by the long head of the biceps, depending on its relationship to the glenohumeral joint.

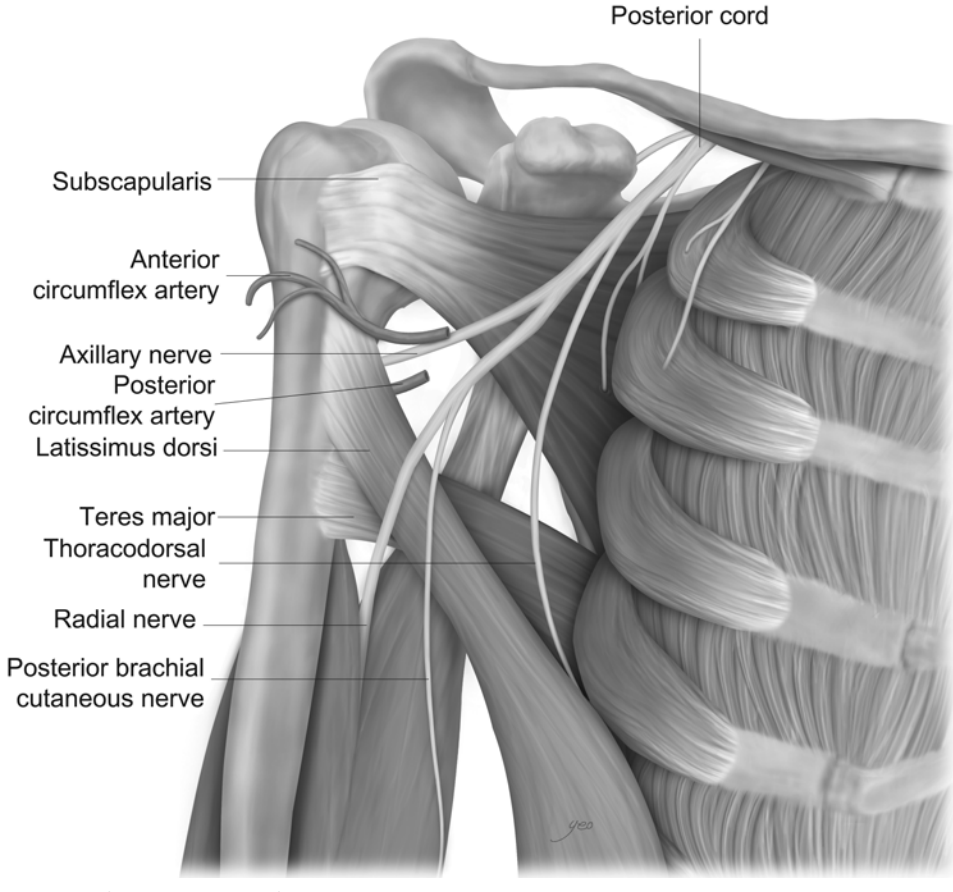

Fig. 1. Illustration of an anterior view of the posterior cord and its major branch relationships between the radial nerve, axillary nerve, and thoracodorsal nerve, and the anterior and posterior circumflex arteries.

Intraoperative nerve action potential recordings guided the method of surgical repair of the nerve. If a nerve action potential was recorded through and distal to the stretch–contusion injury, then the epineurial scar was removed, but the lesion in continuity was not resected. If the lesion was irregular, neurolysis was performed along the full extent of the lesion. The skeletonized nerve was then examined. If a portion of that nerve on cross-section appeared worse than the rest, that specific portion was resected across the lesion. The nerve was subsequently repaired, usually with grafts, leaving the rest of the nerve intact. If there was no action potential conducted across the lesion, the lesion was resected and the gap was usually repaired by means of grafts—either sural or antebrachial cutaneous nerves—using a technique similar to that described by Millesi.[19] If a lesion in continuity was not significantly long, then suture repair was performed.

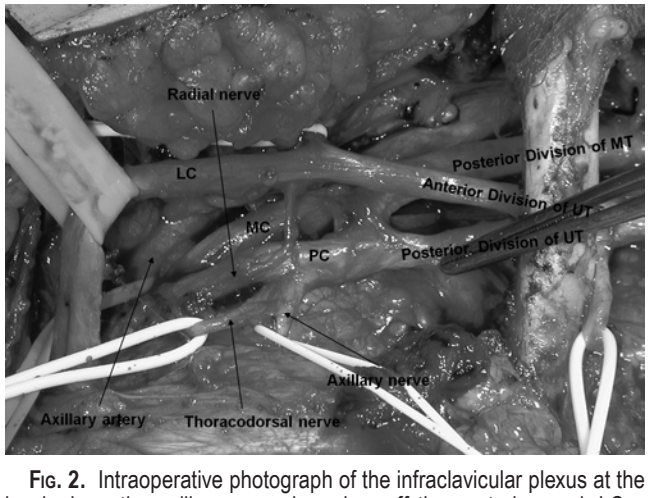

Fig. 2. Intraoperative photograph of the infraclavicular plexus at the level where the axillary nerve branches off the posterior cord. LC = lateral cord; MC = medial cord; MT = middle trunk; PC = posterior cord; UT = upper trunk.

Follow-Up

Postoperatively deltoid function was again assessed by means of both clinical examination and electromyography. All patients were evaluated at least once during the follow-up period (range 16 months–6 years, mean 20 months).

Results

Twenty-six cases were surgically explored via an infraclavicular approach. After a mean follow-up of 20 months, functional recovery appraised by means of the LSUHSC grading system was quite satisfactory. Nine patients with partial nerve loss Grade 1 or 2 preoperatively, underwent exploratory surgery. In this subset of patients, neurolysis was performed on the axillary nerves.

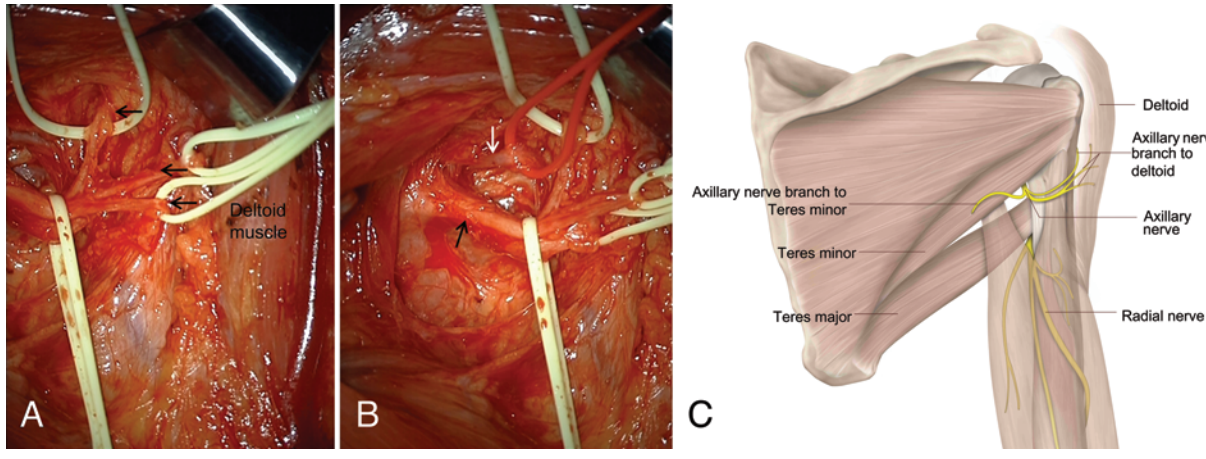

Fig. 3. Intraoperative photographs **(A and B)** of the posterior approach to the axillary nerve and artist's illustration of the posterior view of the shoulder joint **(C)**. The axillary nerve courses around the humerus with the posterior circumflex artery to branch into the deep aspect of deltoid muscle. The *black arrows* in **A** and **B** indicate the axillary nerve branches to the deltoid muscle. The *white arrow* in **B** indicates the posterior circumflex artery.

Postoperatively, the nerves recovered satisfactorily, and a mean grade of 4.2 was attained. Another subset of 3 patients with stretch injuries in continuity were found to have complete nerve loss preoperatively. Intraoperatively, nerve action potentials were able to be conducted across the lesion. These nerves were treated with neurolysis alone and eventually a grade of 3–4 was attained in each of these cases (Table 3). In 2 patients, resection and suture repairs were performed for relatively focal lesions. Nerve action potentials could be conducted across the lesions. The mean outcome grade in these cases was 3–4. In 12 patients, graft repairs were made for lengthier lesions in continuity that required resection; the mean outcome grade was 3 in 3 patients, 3.5 in 8 patients, and 4 in 1 patient. Specifically, outcomes of neurolysis from partial loss (1 case) and resection with graft from complete loss (1 case) revealed extraordinarily good results with postoperative grades of 4.2 and 4, respectively.

Discussion

Incidence in Various Sports

Most axillary nerve injuries described to date in the literature have been part of a combined brachial plexus injury. In 1999, however, Oberle et al.[22] presented a case of axillary nerve paralysis after excessive squash playing. In 2003, repair of axillary nerve–associated shoulder injury either with or without fracture/dislocation was reported by the senior authors in our group (D.G.K. and D.H.K).[16] For the first time, isolated axillary nerve palsy was credited with a dominant role in shoulder dysfunction. Previously, our senior authors showed that tailoring the surgical repair to restore the axillary nerve yielded satisfactory results. This current paper takes the repair of the axillary nerve one step further by categorizing the nuances of the surgical repair and assigning them to be applied to specific subsets of patients, as determined from nerve action potential measurements across the lesion.

TABLE 3: Outcome of axillary nerve repair in 26 cases*

Condition & Treatment	No. of Cases	Mean Postop Grade
partial loss & positive NAP†		
neurolysis	9	4.2
complete loss & positive NAP‡		
neurolysis	3	3–4
resection & suture	2	3–4
resection & graft	3	3
	8	3.5
	1	4

* NAP = nerve action potential.
† The mean preoperative grade was in the range of 1–2.
‡ The preoperative grade in each case was 0.

In our series, skiing and football were the sports most often associated with axillary nerve injury—involved, respectively, in 46% and 19% of the injuries in our case series. Krivickas and Wilbourn[17] reported a very large series of over 200 sports-related nerve injuries. Wrestling, football, rugby, and weightlifting were the 4 sports in which the highest numbers of peripheral nerve injuries were identified. The absolute number of injuries seen as a result of participation in a particular sport suggests that the risk of nerve injury is higher in certain sports and that the risk is also influenced by a specific referral base for a particular patient population. The most common upper-extremity injuries were cervical radiculopathies and brachial plexopathies, usually the result of severe burners (also referred to as stingers) sustained while wrestling or playing football.[17] A related study by Hirasawa[12] also reported some cases of nerve palsies, but the causes were limited to injuries sustained during mountain climbing, gymnastics, and baseball. The reported prevalence of nerve injury after anterior dislocation of the shoulder ranges from 5% to 25%,[6] and it tends to increase with advancing age. In adolescents and young adults who have already had one shoulder dislocation, the prevalence of recurrent shoulder dislocation is about 40%.[13–15]

Another sport deserving of attention is ice hockey. The peripheral nerves of the upper extremity are exposed

to acute and chronic mechanical injuries in ice hockey players. This is due to frequent repetitive motions, large muscular forces, and extreme shoulder positions. Colak et al.[8] evaluated the effect of ice hockey playing on the axillary, musculocutaneous, and radial nerves crossing the upper arm region. In this study, the values of distal motor latency of the axillary, musculocutaneous, and radial nerves were found to be significantly prolonged in ice hockey players compared with controls. Hence, ice hockey and similar sports can repetitively stress the upper extremity through repeated hard throwing motions

Mechanism of Injury

Axillary nerve injuries occur both with and without shoulder dislocation. When the shoulder is not dislocated, blunt trauma to the anterior aspect of the shoulder has been implicated in causing axillary nerve injury. This occurs in sports such as football, wrestling, and ice hockey as a result of blows from helmeted heads of other players to the shoulder.[3,11,16] A more frequent athletic injury, however, is nerve injury resulting from anterior shoulder dislocation (Fig. 4).[4,11] The incidence of nerve palsy after acute dislocations of the shoulder ranges from 9% to 18%.[4,23] The prevalence of electrophysiologically documented nerve damage associated with closed shoulder trauma is reported to be up to 62% and most frequently involves the axillary nerve.[10] Occasionally, the axillary nerve is damaged during reduction of a shoulder dislocation. After glenohumeral dislocation, transient axillary numbness is often noted. This usually reflects neurapraxia rather than a more severe nerve injury.

Initially, the mechanism of axillary nerve injury in sports was described by Bateman,[2] in 1967, as being the result of an upward driving force directly into the armpit. A traumatic insult such as this has the ability to crush the posterior cord against the inferior aspect of the glenohumeral joint, resulting in axillary nerve palsy. An alternative mechanism was also described that attributed falling backward to result in traumatization of the axillary nerve. As far as sports such as baseball, skiing, ice hockey, and football are concerned, stretch injuries of the nerve rank highest on the list of causes of axillary nerve injury. This is mainly due to the repetitive motions specific to individual sports, which engage the same, distinct muscles repeatedly, leading to overuse of particular muscles or muscle groups and adverse effects on the associated nerves.

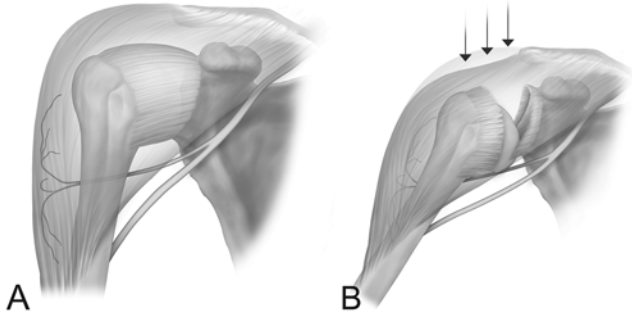

Fig. 4. Schematic illustration of the axillary nerve being stretched across the humeral head following shoulder dislocation. The capsule is represented as completely disrupted or greatly elongated. **A:** Intact shoulder joint and axillary nerve. **B:** Shoulder joint dislocation and stretched axillary nerve.

Many proposed mechanisms of injury are described in the literature in regard to blunt trauma to both the shoulder and the deltoid muscle. Perlmutter and colleagues described compression of the axillary nerve by a helmeted head blow as it travels on the deep surface of the deltoid.[24–26] Direct blunt trauma to the anterolateral aspect of the shoulder via the same mechanism has also been noted to cause axillary nerve injury. There may also be a traction component associated with this injury, as scarring of the axillary nerve distal to the quadrilateral space, as well as scarring to the intramuscular portion of the nerve, has been identified.

A direct blow to the anterior lateral deltoid muscle is another main cause of the injury for athletes playing contact sports.[3] The proximity and route of the axillary nerve along the deltoid muscles make it vulnerable to the injuries sustained from contact sports. Furthermore, the short length of the axillary nerve renders it vulnerable to stretch injuries, especially during shoulder dislocation.[11]

Quadrilateral space syndrome represents a chronic compression syndrome of the axillary nerve in athletes whose sports of choice require repetitive throwing motions.[11,18,28] Teres minor is the superior margin of the quadrilateral space. If fibrous bands develop at the inferior edge of the teres minor, they can compress both the axillary nerve and the posterior humeral circumflex artery. Quadrilateral space syndrome can cause complete denervation of the deltoid and teres minor muscles by compressing the axillary nerve. Compression could be more severe in the abducted, externally rotated (throwing) position.[28] Cheung et al.[7] described the effects of shoulder position on axillary nerve positions during the split lateral deltoid approach.

Clinical Presentation and Examination

Surprisingly, many patients with axillary nerve injury may be asymptomatic even though they have sustained complete or incomplete lesions.[29] Pain is not a prominent complaint, and deltoid weakness is often masked by compensation from surrounding muscle groups.[1] In the acute setting, the athlete classically presents with weakness in abduction, decreased sensation along the deltoid muscle insertion, progressive atrophy of the deltoid muscle, and subluxation of the glenohumeral joint.

When affected athletes exercise, however, they may fatigue quickly. This is especially noted with overhead activity and heavy lifting. Affected persons may also notice reduced strength with abduction or an inability to raise the arm. Furthermore, numbness of the lateral arm may be noted. The presence of trauma, dislocation, or fracture should be documented. The physical examination of athletes with axillary nerve injury should include evaluation for range of motion (passive and active) and strength in all planes. External rotation strength is important to assess, because 45% of external rotation strength is from the teres minor. This is an important distinction, because global strength, especially abduction and forward elevation, is predominantly provided by the deltoid, the prime mover of the shoulder. When a patient is seen late in the course of the affliction, muscular atrophy, particularly of the deltoid and teres minor, should be noted, as it is of

great importance in localizing the lesion. If the posterior deltoid and teres minor are spared, then the lesion is distal to the quadrilateral space. A complete neurovascular examination should be performed to assess sensation over the upper lateral arm and to rule out other lesions such as thoracic outlet syndrome and brachial plexus and cervical spine lesions. It is also important to note that complete deltoid muscle deficit may occur in the presence of normal sensation to the upper lateral arm and shoulder. Several reports have documented the unreliability of sensory changes after injury.[4]

Axillary nerve injury resulting from a direct blow to the anterolateral shoulder, however, has a less optimistic prognosis. Perlmutter et al.[24–26] noted persistent paralysis of the deltoid with such injuries in 11 athletes at 2.5–23 years after injury.

Acute axillary nerve palsy is often seen in the athletic setting, and careful physical examination and EMG evaluation are necessary to make an accurate diagnosis. Evaluation of strength is typically hampered by pain. The injured athlete can often elevate the arm using the pectoralis and supraspinatus muscles, and subluxation can be prevented by the supraspinatus and long head of the biceps muscles. Active arm elevation by compensatory muscles is seen in up to 60% of athletes after axillary nerve injury.

Careful manual muscle testing is important in identifying gross weakness in abduction. Patients with chronic compression of the axillary nerve in quadrilateral space syndrome typically present with tenderness in the posterior shoulder area in the quadrilateral space, which is exacerbated by placing the arm in the throwing position and resisting internal rotation.[28] Symptoms, however, are often vague, consisting of a dull ache in the shoulder with progressive use, and may be difficult to differentiate from the internal impingement also seen in the throwing athlete.

Treatment

When electromyography reveals an axillary nerve lesion, the athlete should be reevaluated at monthly intervals for signs of nerve regeneration. Because the axillary nerve is relatively short, in the case of a second-degree injury (axonotmesis), recovery should be seen between the 3rd and 4th month after injury. Surgical exploration and possible nerve grafting are generally recommended if no return of function has occurred by 4–6 months after injury.[1,3,9,11,27]

In our series, all patients who were deemed to be appropriate surgical candidates did not show adequate signs of spontaneous recovery (greater than LSUHSC Grade 2) even after 6–8 months of conservative therapy. Thus, surgical intervention was believed to be a reasonable treatment option. The patients in our series were further subdivided into 3 groups: neurolysis-only group, resection-and-direct-suture-repair group, and resection-and-graft-repair group; after surgical intervention, patients in all 3 groups showed satisfactory results.

The site of nerve injury is sometimes in the quadrilateral space, requiring both anterior and posterior surgical approaches. Cable grafts are commonly required if the axillary nerve ends cannot be repaired without tension, but despite this, the results of surgery are generally good, with restoration of 3–4/5 strength in the deltoid in more than 90% of patients.[27] Chronic axillary nerve compression in quadrilateral space syndrome generally responds to conservative management, including changing pitching mechanics, which has been reported to be successful in 75%–90% of patients.[9,11] Surgical intervention for quadrilateral space syndrome is indicated if conservative management fails and involves release of fibrous bands of the teres minor muscle and any aberrant bands crossing the quadrilateral space.[9,28] Timing of surgery, however, remains controversial. In a thorough review of 146 cases, Bonnard et al.[5] demonstrated a dramatic reduction in the number of successful outcomes with increasing delay between the time of injury and intervention. By contrast, Moor et al.[20] showed the positive results in delayed axillary nerve reconstruction with interposition of sural nerve grafts and concluded that even delayed axillary nerve grafting may lead to satisfactory functional results with a low morbidity and should therefore be done in selected patients.

Preventive Measures

Proper conditioning and preventive measures in young athletes are important to avoid axillary nerve injuries around the shoulder. Most of these injuries occur in inexperienced athletes, and poor conditioning plays a large role.[21,31] Preventing these types of injuries includes the following: adequate preseason examination; matching competitors for age, weight, and skill level; proper conditioning; avoiding excessive training at too early an age; thorough rehabilitation of the injured athlete before return to sports; appropriate and properly maintained equipment and playing fields; adequate supervision; and rule changes as necessary. Although in the US the highest injury rates are noted in adolescents participating in football, basketball, gymnastics, baseball, and roller skating, in Japan nerve injury occurs most commonly from mountain climbing—as a result of excessive backpack weight.[2,30] Rather than condemning certain sports as unsafe because of a high incidence of injuries, proper training techniques should be emphasized in all sports to reduce the risk of these injuries.

Conclusions

Direct attacks and blows to the shoulder area are common for athletes playing contact sports. In addition, the axillary nerve has characteristic anatomical features such as its short length and a specific route beneath the deltoid muscle that predispose it to injury. As such, axillary nerve injury associated with sports has been a relatively common occurrence, with or without humerus fracture and dislocation. Favorable functional outcomes in those with axillary nerve injury are essential, especially for athletes who wish to continue engaging in their sport of choice. Therefore, surgical repair of the axillary nerve by means of neurolysis and nerve grafting should be considered in the management of these cases. In our patient population, surgical intervention appropriate to the extent of nerve injury has proven to be a reliable method of regaining deltoid function in severe cases of sports-related stretch–contusion injuries.

Disclosure

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

Author contributions to the study and manuscript preparation include the following. Conception and design: Kim, Lee. Acquisition of data: Lee, Saetia. Analysis and interpretation of data: Kim, Lee, Saetia, Saha. Drafting the article: Lee, Saetia. Critically revising the article: Kim, Saha, Kline. Reviewed submitted version of manuscript: Lee, Saha, Kline. Approved the final version of the manuscript on behalf of all authors: Kim. Administrative/technical/material support: Kim, Saetia, Kline. Study supervision: Kline.

References

1. Alnot JY: Traumatic brachial plexus palsy in the adult. Retro- and infraclavicular lesions. **Clin Orthop Relat Res (237):**9–16, 1988
2. Bateman JE: Nerve injuries about the shoulder in sports. **J Bone Joint Surg Am 49:**785–792, 1967
3. Berry H, Bril V: Axillary nerve palsy following blunt trauma to the shoulder region: a clinical and electrophysiological review. **J Neurol Neurosurg Psychiatry 45:**1027–1032, 1982
4. Blom S, Dahlbäck LO: Nerve injuries in dislocations of the shoulder joint and fractures of the neck of the humerus. A clinical and electromyographical study. **Acta Chir Scand 136:** 461–466, 1970
5. Bonnard C, Anastakis DJ, van Melle G, Narakas AO: Isolated and combined lesions of the axillary nerve. A review of 146 cases. **J Bone Joint Surg Br 81:**212–217, 1999
6. Brown HA, Brown BA: Treatment of peripheral nerve injuries. **Rev Surg 24:**1–8, 1967
7. Cheung S, Fitzpatrick M, Lee TQ: Effects of shoulder position on axillary nerve positions during the split lateral deltoid approach. **J Shoulder Elbow Surg 18:**748–755, 2009
8. Colak T, Bamaç B, Alemdar M, Macit Selekler H, Ozbek A, Colak S, et al: Nerve conduction studies of the axillary, musculocutaneous and radial nerves in elite ice hockey players. **J Sports Med Phys Fitness 49:**224–231, 2009
9. Cormier PJ, Matalon TA, Wolin PM: Quadrilateral space syndrome: a rare cause of shoulder pain. **Radiology 167:**797–798, 1988
10. de Laat EA, Visser CP, Coene LN, Pahlplatz PV, Tavy DL: Nerve lesions in primary shoulder dislocations and humeral neck fractures. A prospective clinical and EMG study. **J Bone Joint Surg Br 76:**381–383, 1994
11. Duralde XA: Neurologic injuries in the athlete's shoulder. **J Athl Train 35:**316–328, 2000
12. Hirasawa Y: [Mechanism of regeneration and repair of the peripheral nerve.] **Nippon Seikeigeka Gakkai Zasshi 64:**99–111, 1990 (Jpn)
13. Hoelen MA, Burgers AM, Rozing PM: Prognosis of primary anterior shoulder dislocation in young adults. **Arch Orthop Trauma Surg 110:**51–54, 1990
14. Hovelius L, Eriksson K, Fredin H, Hagberg G, Hussenius A, Lind B, et al: Recurrences after initial dislocation of the shoulder. Results of a prospective study of treatment. **J Bone Joint Surg Am 65:**343–349, 1983
15. Hovelius L, Lind B, Thorling J: Primary dislocation of the shoulder. Factors affecting the two-year prognosis. **Clin Orthop Relat Res (176):**181–185, 1983
16. Kline DG, Kim DH: Axillary nerve repair in 99 patients with 101 stretch injuries. **J Neurosurg 99:**630–636, 2003
17. Krivickas LS, Wilbourn AJ: Peripheral nerve injuries in athletes: a case series of over 200 injuries. **Semin Neurol 20:** 225–232, 2000
18. McKowen HC, Voorhies RM: Axillary nerve entrapment in the quadrilateral space. Case report. **J Neurosurg 66:**932–934, 1987
19. Millesi H: Nerve grafts: indications, techniques and prognosis, in Omer GE Jr, Spinner M (eds): **Management of Peripheral Nerve Problems.** Philadelphia: WB Saunders, 1980, pp 410–430
20. Moor BK, Haefeli M, Bouaicha S, Nagy L: Results after delayed axillary nerve reconstruction with interposition of sural nerve grafts. **J Shoulder Elbow Surg 19:**461–466, 2010
21. O'Neill DB, Micheli LJ: Overuse injuries in the young athlete. **Clin Sports Med 7:**591–610, 1988
22. Oberle J, Kuchelmeister K, Schachenmayr W, Richter HP: [Axillary nerve paralysis after playing squash. A case report.] **Nervenarzt 70:**750–753, 1999 (Ger)
23. Pasila M, Jaroma H, Kiviluoto O, Sundholm A: Early complications of primary shoulder dislocations. **Acta Orthop Scand 49:**260–263, 1978
24. Perlmutter GS: Axillary nerve injury. **Clin Orthop Relat Res (368):**28–36, 1999
25. Perlmutter GS, Apruzzese W: Axillary nerve injuries in contact sports: recommendations for treatment and rehabilitation. **Sports Med 26:**351–361, 1998
26. Perlmutter GS, Leffert RD, Zarins B: Direct injury to the axillary nerve in athletes playing contact sports. **Am J Sports Med 25:**65–68, 1997
27. Petrucci FS, Morelli A, Raimondi PL: Axillary nerve injuries—21 cases treated by nerve graft and neurolysis. **J Hand Surg Am 7:**271–278, 1982
28. Redler MR, Ruland LJ III, McCue FC III: Quadrilateral space syndrome in a throwing athlete. **Am J Sports Med 14:**511–513, 1986
29. Safran MR: Nerve injury about the shoulder in athletes, part 1: suprascapular nerve and axillary nerve. **Am J Sports Med 32:**803–819, 2004
30. Sicuranza MJ, McCue FC III: Compressive neuropathies in the upper extremity of athletes. **Hand Clin 8:**263–273, 1992
31. Stein CJ, Micheli LJ: Overuse injuries in youth sports. **Phys Sportsmed 38:**102–108, 2010

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Peroneal nerve injury associated with sports-related knee injury

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Object. This study analyzes 84 cases of peroneal nerve injuries associated with sports-related knee injuries and their surgical outcome and management.

Methods. The authors retrospectively reviewed the cases of peroneal nerve injury associated with sports between the years 1970 and 2010. Each patient was evaluated for injury mechanism, preoperative neurological status, electrophysiological studies, lesion type, and operative technique (neurolysis and graft repair). Preoperative status of injury was evaluated by using a grading system published by the senior authors. All lesions in continuity had intraoperative nerve action potential recordings.

Results. Eighty-four (approximately 18%) of 448 cases of peroneal nerve injury were found to be sports related, which included skiing (42 cases), football (23 cases), soccer (8 cases), basketball (6 cases), ice hockey (2 cases), track (2 cases) and volleyball (1 case). Of these 84 cases, 48 were identified as not having fracture/dislocation and 36 cases were identified with fracture/dislocation for surgical interventions. Good functional outcomes from graft repair of graft length < 6 cm (70%) and neurolysis (85%) in low-intensity peroneal nerve injuries associated with sports were obtained. Recovery from graft repair of graft length between 6 and 12 cm (43%) was good and measured between Grades 3 and 4. However, recovery from graft repair of graft length between 13 and 24 cm was obtained in only 25% of patients.

Conclusions. Traumatic knee-level peroneal nerve injury due to sports is usually associated with stretch/contusion, which more often requires graft repair. Graft length is the factor to be considered for the prognosis of nerve repair. *(DOI: 10.3171/2011.9.FOCUS11187)*

Key Words • **peroneal nerve** • **stretch/contusion** • **sports-related injury** • **graft repair** • **neurolysis**

Common peroneal nerve palsy is a debilitating complication, and its incidence due to sports-related knee injury has been reported to be as high as 50%.[6] The mechanism for peroneal nerve injury as a group includes laceration, stretch/contusion, entrapments, iatrogenic, compression, or gunshot wounds. However, peroneal nerve injuries caused by sports are found to be frequently associated with severe ligamentous knee injuries. Most of the peroneal nerve injuries sustained by players come under the category of stretch/contusion injuries. The occurrence of peroneal nerve injury in association with knee dislocations has been reported to be between 14% and 40%, with most studies reporting an incidence in the range of 25%–35%.[10] Reports show that the most common cause of knee injury is motor vehicle accidents, followed by those that are sports related;[14] however, recent studies have shown an increase in knee injuries associated with sports.[1,2] Peroneal nerve injuries may occur in as many as 23% of patients with knee dislocations. Nearly one-half of the patients with peroneal nerve injuries have a permanent deficit.[25]

The type of sport plays a major role in defining the mechanism and frequency of peroneal nerve injuries. In the 84 sports-related peroneal nerve injuries represented in this series, skiing, football, soccer, basketball, ice hockey, and volleyball were found to be responsible for the knee injuries leading to peroneal nerve palsy. The major sports that were found to be highly responsible for peroneal nerve injuries were skiing (50%) as the most frequent, followed by football (27%). The reasons for this are not only the frequency with which they are prolonged but also biomechanical forces involved and the chances of a direct blow to the knee. Some of the low-intensity sports

Abbreviations used in this paper: CPN = common peroneal nerve; NAP = nerve action potential; PCL = posterior cruciate ligament.

like basketball, track, and volleyball require repeated usage of muscles and ligaments, frequent twists and turns, and on occasion sudden jerk to the knee, which makes the peroneal nerve vulnerable to either injury or compression.

Methods

Patient Population

Retrospectively, 448 cases of peroneal nerve injuries were identified, which were surgically managed between 1970 and 2010. The mechanism of injury for these cases included stretch/contusion without fracture/dislocation (215 cases), stretch/contusion with fracture/dislocation (57), tumor (62), laceration (47), entrapment (53), and gunshot wound (14; Fig. 1). Most of the 272 patients who had stretch/contusion from high-velocity injuries were involved in motor vehicle accidents. Of 272 stretch/contusion cases, 84 (approximately 30%) were identified as sports-related (Table 1), which included skiing (42 cases), football (23), soccer (8), basketball (6), ice hockey (2), track (2), and volleyball (1). The 84 sports-related peroneal nerve injuries were categorized into those without fracture/dislocation (48 cases) and those with fracture/dislocation (36 cases). All 84 of these cases underwent surgical exploration. Postoperative evaluations were performed over an average follow-up duration of 16 months (range 1–6 years).

TABLE 1: Summary of 84 cases identified as sports-related peroneal nerve injuries

Sport	Cases (%)
skiing	42 (50)
football	23 (27)
soccer	8 (10)
basketball	6 (7)
ice hockey	2 (2)
track	2 (2)
volleyball	1 (1)

Surgical Anatomy

The CPN originates as the sciatic nerve divided into the CPN and the tibial nerve at the mid- to distal-third of the thigh. The CPN descends obliquely over the proximal gastrocnemius muscle from the apex of the popliteal fossa to the lateral popliteal fossa and usually lies beneath the medial aspect of the lateral hamstring muscle. It then curves around the proximal peroneus longus muscle to travel toward the anterior lower leg, where it divides into deep and superficial branches (Fig. 2).

The deep branch of the CPN quickly divides after passing beneath the fibrous lateral edge of the peroneus longus muscle. The initial branch supplies the anterior tibial muscle, and subsequent branches supply the extensor digitorum longus, extensor hallucis longus, and peroneus tertius muscles. The deep branch is divided further as it approaches the foot into a medial sensory branch and a lateral motor branch. The medial branch supplies a small area of skin over the first dorsal web space of the foot, whereas the lateral branch innervates the extensor digitorum brevis and extensor hallucis brevis muscles.

The superficial peroneal branch supplies the peroneus longus and then the brevis muscles as it descends in a straight course between them, becoming gradually more superficial in the distal third of the lower leg. It then branches in front of the ankle joint into the medial and lateral branches. The superficial peroneal branch supplies cutaneous sensation to the anterolateral lower leg and ankle and also supplies sensation to the dorsum of the foot.

Surgical Exposure

The patient is placed prone with the leg slightly flexed at the knee. An S-shaped incision begins in the lower thigh, medial to the long head of the biceps femoris muscle (lateral hamstring). A lateral curvilinear extension crosses the surgical neck of the fibula and continues toward the proximal lateral surface of the leg (Fig. 3). As the proximal portion of the incision is deepened, the lateral hamstring is moved away from the underlying peroneal nerve. The peroneal nerve is in the fatty tissue behind the long

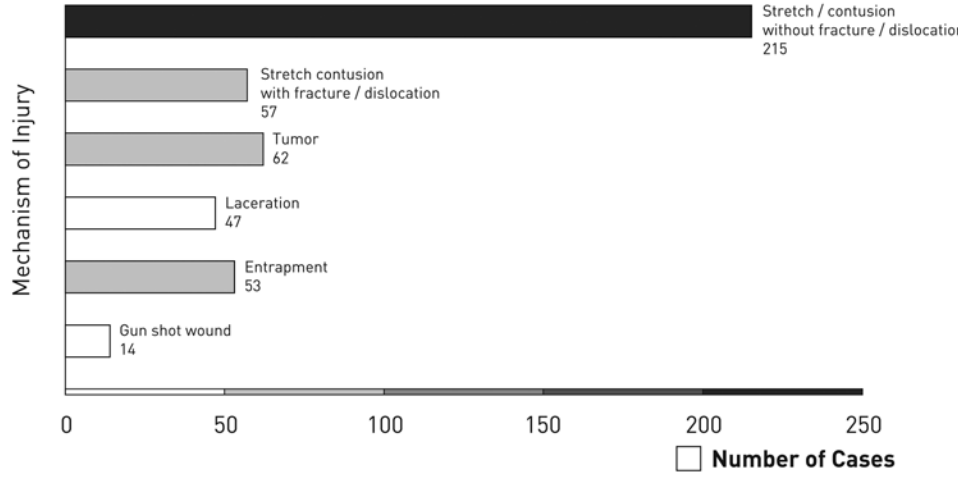

Fig. 1. Mechanism of nerve injury in 448 cases of peroneal nerve injuries.

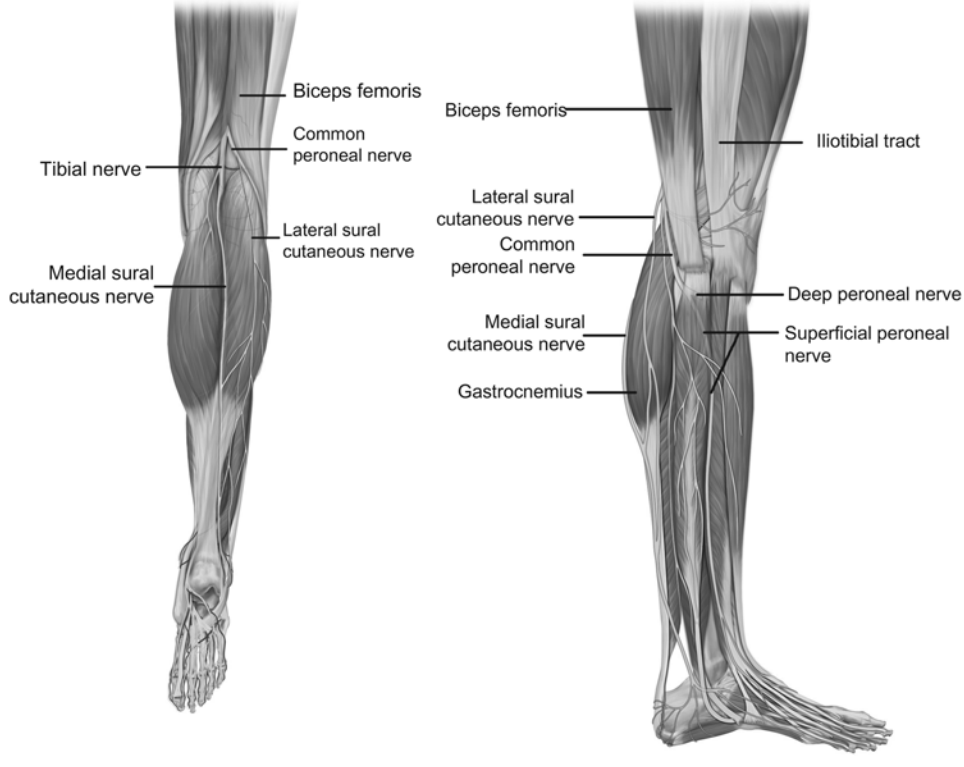

Fig. 2. Anatomy of the peroneal nerve. **Left:** The CPN originates as the sciatic nerve divides into the CPN and the tibial nerve at the mid- to distal-third of the thigh (posterior view). **Right:** The CPN descends obliquely across the plantaris muscle from the apex of the popliteal fossa to the lateral popliteal fossa and curves around the proximal peroneus longus muscle. It then travels toward the anterior lower leg, where it divides into deep and superficial branches (lateral view).

biceps (lateral hamstring) head. Dissection is then carried toward the lateral popliteal space. Usually in the popliteal fossa, a large sensory branch, the lateral sural cutaneous nerve, originates from the peroneal nerve. The nerve then curves around the surgical neck of the fibula and divides into the deep and superficial peroneal branches. By splitting the peroneal nerve, branches are exposed distal to the surgical neck and fibular head. The deep branch is traced around the surgical neck of the fibula using a plastic loop. The more distal deep branches can be traced only a short distance into the anterior compartment. Small vessels are coagulated and fine neural branches running to the knee joint can be sacrificed. In this series, NAP stimuli were used to evaluate the lesion in continuity. If NAP stimulus traces were flat, the lesion in continuity was resected and the nerve ends were prepared for graft repair. If NAPs were transmitted across the lesion or lesions, then neurolysis was usually performed. The head of the fibula was usually leveled off using Leksell rongeurs and bone wax was applied. A split hamstring muscle was reapproximated with 2-0 or 3-0 suture, as was the more distal portion of the peroneus longus and brevis muscles.

Surgical Methods

Each patient was initially followed up from 3 to 6 months and checked for spontaneous functional recovery. When there was no significant positive functional outcome as confirmed by electromyography and clinical examination, surgical exploration and repair was recommended. The grading system for knee-level CPN injuries (Table 2), previously published by the senior authors,[11] was used to evaluate preoperative and postoperative function. Neurolysis was performed on lesions in continuity when intraoperative NAP could be recorded across the lesion. However, graft repair was performed on lesions that were not in continuity or on lesions in continuity with no intraoperative NAPs across the lesion. After resection of proximal and distal stumps or a segment of neuroma, healthy appearing fascicles could be observed under high magnification on both the nerve ends. Length of the graft needed (usually sural nerve) was determined by the nerve gap as measured during surgical exploration. For graft repair, 3 different ranges of the graft lengths were used: < 6 cm, 6–12 cm, and 13–24 cm.

Results

Eighty-four cases of sports-related traumatic CPN injury with stretch/contusion injury underwent surgical repair. Mechanisms of injury included 48 cases without fracture/dislocations and 36 with fracture/dislocations of stretch/contusions (Table 3). A total of 58 graft repairs were performed, using 3 ranges of graft length (< 6 cm, 6–12 cm, and 13–24 cm).

Stretch/Contusion Without Fracture/Dislocation

Of the 48 cases (57%) with stretch/contusion without fracture/dislocation, 17 patients (35%) underwent neu-

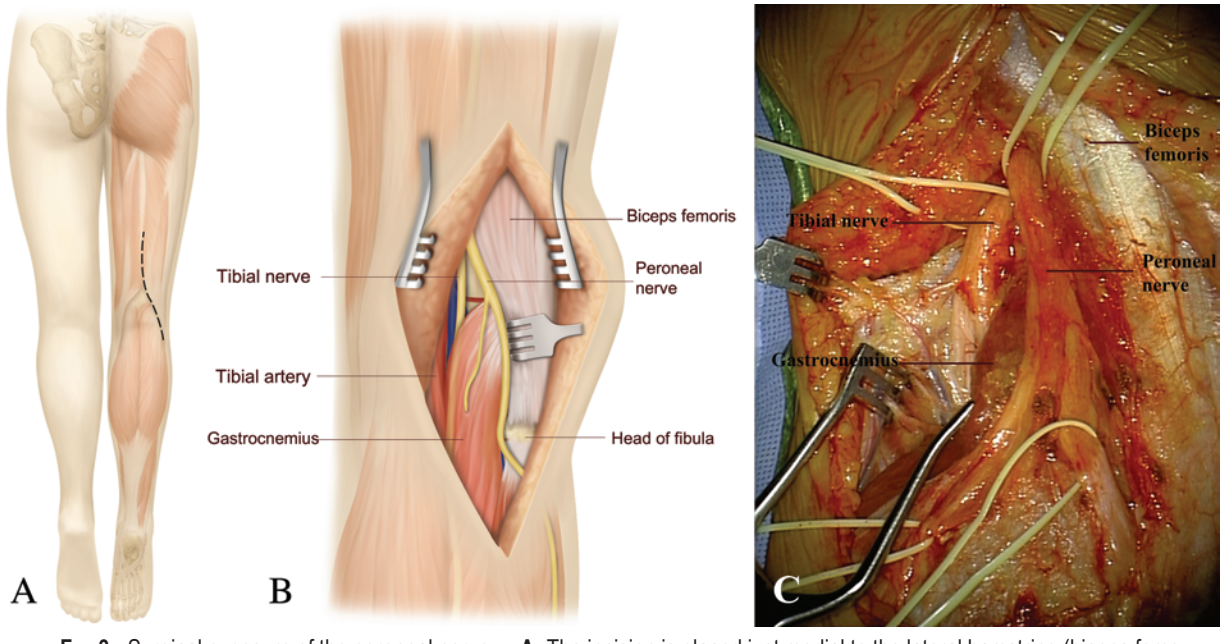

Fig. 3. Surgical exposure of the peroneal nerve. **A:** The incision is placed just medial to the lateral hamstring (biceps femoris) in the leg in an S-shaped curve in the region of the proximal lateral and anterior compartment. **B and C:** Exposure of the proximal peroneal nerve is observed beneath the medial edge of the lateral hamstring. The tibial nerve and popliteal vessels are observed deep in the popliteal fossa.

rolysis because of an intraoperative NAP recorded distal to the lesion, whereas 31 patients (65%) underwent sural nerve graft repair because of negative NAP across the lesion or due to complete disruption of the CPN where proximal and distal stumps were found. These injuries were presented with complete or severe loss of CPN function, that is, loss or severe weakness of dorsiflexion of the foot and toes and of eversion of the foot.

Despite complete functional loss, careful neurolysis was performed across the lesions in continuity where a NAP could be recorded. Functional recovery of Grade 3 or better was achieved in the neurolysis group in 85% of cases, which was attributed to a lower severity of injury compared with those in which graft repair was required.

Stretch/Contusion With Fracture/Dislocation

Thirty-six (43%) of 84 patients with CPN lesions underwent operative intervention for stretch/contusion injuries associated with fibular or tibial fractures or dislocations. Nine (25%) of these 36 patients with stretch/contusions had transmittable NAPs across their lesions in continuity and thus underwent neurolysis. Twenty-seven patients (75%) with severe nerve lesions required graft repairs. Graft length depended on the nerve gap between the 2 stumps of the nerve after neuromas had been removed. Ten (17%) of the 58 graft repairs performed were less than 6 cm in length, and 7 (70%) of 10 achieved functional grades between 3 and 4. Forty of the 58 patients undergoing graft repairs had graft lengths between 6 and 12 cm, and 17 (43%) of these 40 achieved functional recovery grade of 3 or better. If the length of the graft was between 13 and 24 cm, which was used in 8 (14%) of the 58 grafts, only 2 (25%) of the 8 patients showed functional outcome of Grade 3 or 4. Not surprisingly, the functional outcomes were higher for the short grafts than for the longer ones.

TABLE 2: Grading scale of the Louisiana State University Health Sciences Center for peroneal nerve palsy*

Grade	Definition
0	no palpable muscle contraction
1	palpable contraction of peronei or anterior tibial muscles
2	peronei or anterior tibial muscles contract against gravity
3	peronei & anterior tibial muscles contract against gravity & some resistance
4	peronei & anterior tibial muscles contract against moderate resistance
5	peronei & anterior tibial muscles contract w/ full strength

* From Kim et al., 2004.

TABLE 3: Peroneal nerve injuries as a result of sports and surgical results*

Stretch/Contusion Injury	No. of Pts	Neurolysis	Graft Length (cm)		
			<6	6–12	13–24
w/o fracture/dislocation	48	17	8	21	2
w/ fracture/dislocation	36	9	2	19	6
total	84	26	10	40	8
no. w/ postop Grade 3 or better results (%)		22 (85)	7 (70)	17 (43)	2 (25)

* Pts = patients.

Discussion

The highest number of CPN injuries at the knee level fall into the category of stretch/contusion. For example, there is a large incidence of CPN injuries reported with dislocation of the knee.[18] Knowledge of the mechanisms of injuries involved in sports enables analysis and classification as to location and type, guides surgery, and facilitates assessment. The reasons that peroneal nerve injuries are associated with sports is because of extreme biomechanical demand by the body while playing, such as stretching, twisting, jumping, running, falling, and direct blows in the contact sports. Different sports require different physical demands, thereby making peroneal nerve injuries specific to some and not as common to others. A series of cases of peroneal nerve injuries from football, skiing, soccer, and basketball and other sports has been presented in this paper. Peroneal nerve injuries are most commonly found to occur when the posterolateral corner structures of the knee are also injured.[10] Considerable connective tissue damage may occur with severe stretch injuries, which can lead to both intraneural and extraneural scar formation. In this series, peroneal nerve stretch/contusion injury that occurred during sports was associated with knee joint soft-tissue injuries and ligamentous and/or cartilaginous stretches or tears caused by impact-induced knee adduction and torsion. Other than this, compression injury is also found in participants in contact sports. Babwah[1] reported a case of CPN injury in a soccer player because of excessive cooling with ice.

Most studies[8,10] have reported an incidence of 25%–36% of peroneal nerve palsy in knee dislocations. Recent studies show that the occurrence rate of CPN palsy associated with knee dislocation or bicruciate ligament injury ranges from 10% to 40%.[3–5] Palsy of the CPN was associated with dislocation of the knee in 25% of patients as reported by Niall et al.,[16] exclusively with dislocations involving a disruption of the PCL and posterolateral corner. It is important to note that in dislocations with disruption of the PCL and posterolateral corner, the incidence is greater and may be as high as 45%. Fractures of either the distal femur or proximal tibia are present in 16% of peroneal nerve injury cases.[14] Marginal avulsion fractures of the lateral tibial plateau may be observed in some cases, indicating significant capsular, collateral, and cruciate disruption. Fractures of the anteromedial tibial plateau in particular are associated with the presence of disruption of the PCL and posterolateral corner. A significant number (43%) of stretch/contusion cases was found to be associated with fracture/dislocation.

Timing of Surgery

The time from trauma to repair has been found to have a profound influence on positive functional outcome of the injured peroneal nerve.[11] Recommendations regarding the timing of surgical intervention differ, but most peripheral neurosurgeons emphasize careful clinical follow-up after the initial injury by physical examination and electromyography for approximately 3 months postinjury before operating.[9,10]

Vulnerability of the CPN to Injury

Several anatomical factors predispose the CPN to injury. The CPN passes lateral to the surgical neck of the fibula, at which point it is superficial and relatively fixed. At this point, it is vulnerable to direct external compression, but is also vulnerable to stretch due to relative fixation. The severity of CPN injuries depends on the direction and force of the displacement causing the various ligament ruptures, which in turn depends on intensity of the trauma: the neurological lesion, like the popliteal vascular lesion, is part of regional trauma sustained by the knee (Fig. 4). There is consensus as to the parallel nature of the neurological and ligamentous lesions: the more extensive and severe the latter, the more frequent and severe the CPN palsy.

As the second branch of the sciatic nerve, the tibial nerve is less prone to injury associated with knee injuries. It lies deep in the posterior compartment of the leg, is not as tethered in the leg as the CPN, and is therefore less vulnerable to injury than the CPN. The tibial nerve is most often injured in association with knee dislocation or very severe injuries to the posterior knee capsule. Anatomically, as explained by Sunderland,[22] the number of fascicles doubles and the percentage of connective tissue decreases distally in the lower extremities. Thus, the maximum load that the CPN may sustain before reaching its elastic limit is less than that of the tibial nerve in this same region. This suggests that when the CPN is subjected to the same force, it is more susceptible to injury than the posterior tibial nerve in the same location; that is, the internal arrangement of the nerve makes it less able to absorb axially directed forces.

Manifestations of Peroneal Nerve Injuries

Foot drop is a common and significant manifestation of peroneal nerve injury. This condition results from the loss of motor innervations of the tibialis anterior muscle and causes significant gait impairment and disability. The complaint of numbness or tingling over the anterolateral leg, foot, or first web space is very common in athletes. Athletes may complain of foot drop or weakness of ankle dorsiflexion.

Injury to the CPN or to both its superficial and deep branches causes weakness of the deep branch-innervated ankle dorsiflexors (the anterior tibialis and peroneus tertius) and toe extensors, including the extensor digitorum brevis and longus and the extensor hallucis brevis and longus, which extend the second through fifth toes and the great toe, respectively. There is also paresis of the superficial branch-innervated ankle evertors, the peroneus longus and brevis muscles. Deep branch involvement results in decreased sensation in the area between the great and second toes while superficial branch involvement results in numbness in the anterolateral calf and the dorsum of the foot. If there is involvement of only one of the branches, it is usually the deep branch that is involved.

Associated Vascular Injuries

The incidence of vascular injuries in association with knee dislocation varies between reports, ranging from 7% to 64%.[18,20] Stretch/contusion to the CPN from high-im-

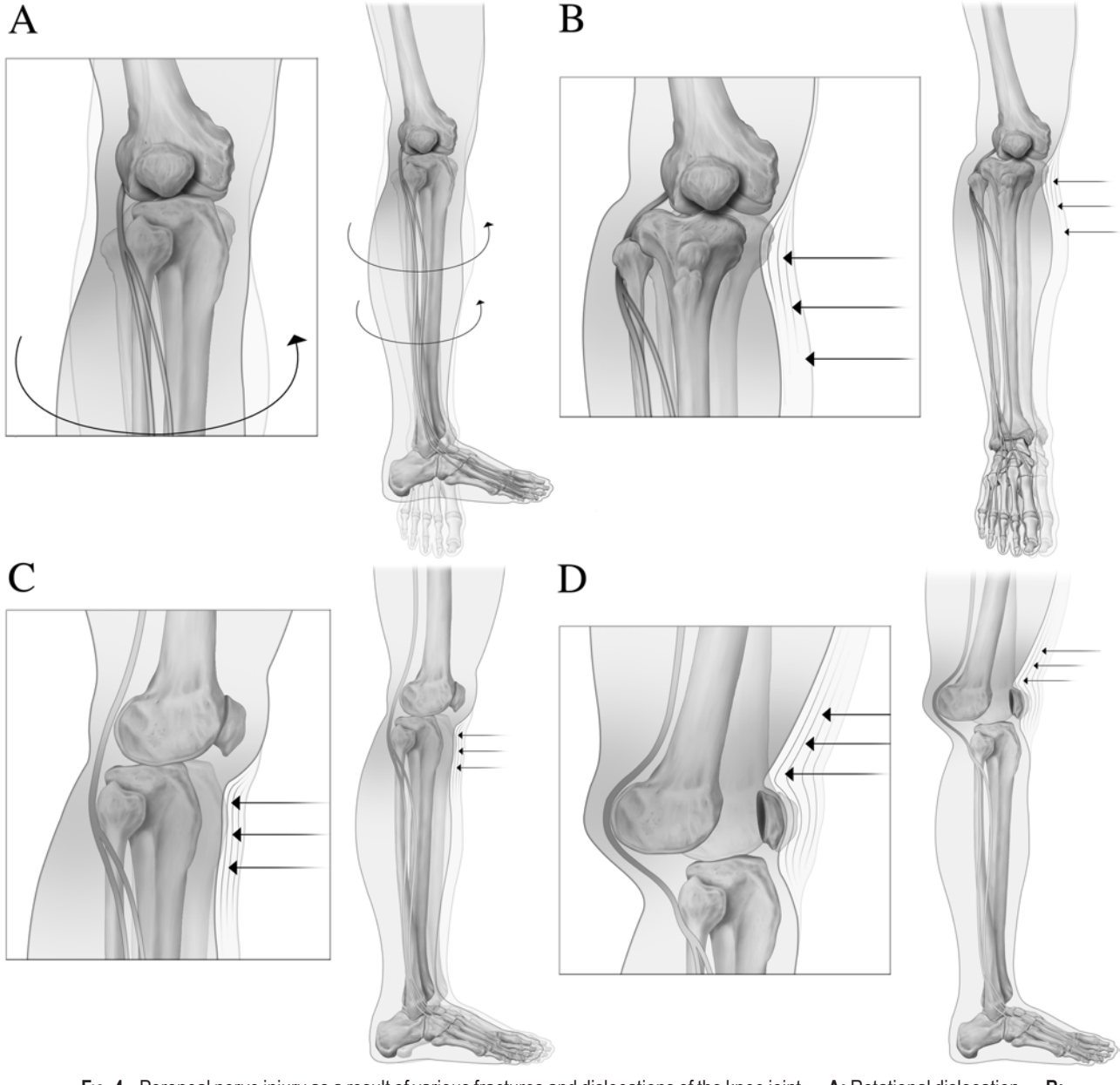

Fig. 4. Peroneal nerve injury as a result of various fractures and dislocations of the knee joint. **A:** Rotational dislocation. **B:** Lateral dislocation. **C and D:** Anterior-posterior dislocation.

pact sports injury not only damages the nerve but also can rupture the vasa nervorum, which causes bleeding into the nerve sheath and a compressive hematoma resulting in ischemia.[10] A high prevalence (4%–20%) of disruption of the popliteal vascular supply in patients with knee dislocations has been reported.[3] The majority of the acute sports-related combined neuronal and vascular injuries are associated with contact sports such as rugby, football, and ice hockey.[13]

Surgical Approach

Recently, Giuseffi et al.[8] reviewed the future prospects of surgical treatment of peroneal nerve palsy after knee dislocation. Nerve regeneration following CPN repair is poor compared with other peripheral nerves,[12] and this can explain the reluctant attitude of many physicians toward exploration and repair of this nerve. One factor explaining the poor outcome of CPN reinnervation might be the imbalance between the functioning flexors and the weakly innervated extensors that can result in fixed equinism of the foot with associated heel cord (Achilles tendon) shortening.

Other surgical options besides direct nerve repairs include nerve transfers and tendon transfers. Nerve transfer involves taking a branch from a less important lower leg muscle in the tibial distribution and connecting it to nerve to the muscle that lifts the foot in the peroneal distribution. A tendon transfer involves taking a tendon that moves the foot inward, and connecting it to the top of the foot so that it now lifts the foot up and out, thereby resolving the foot

drop. Successful functional outcomes of foot drop by nerve transfers to deep peroneal nerve have been reported in the literature.[12,15,21] In this series, either neurolysis and/or graft repair was selected for the 84 patients with CPN injuries due to sports. Follow-up showed 85% of positive functional outcomes of Grade 3 or more in cases of neurolysis based on a positive NAP across the lesion.

Tibialis Posterior Tendon Transfers

Tendon transfer procedures for CPN palsies due to stretch are promising.[7] Yeap et al.[26,27] concluded that tibialis posterior tendon transfers as a group produced 83% excellent or good results in terms of patient satisfaction. These procedures can be recommended to patients who would prefer to discard an ankle/foot orthosis. The results are likely to be more successful if the nerve lesion causing the drop foot is at the common peroneal level rather than at the sciatic level, where posterior tibialis may be paralyzed and in men less than 30 years of age, which is the usual case with sports injuries. Posterior tibial tendon transfer procedures have had reasonable success in allowing patients to return to ambulation without assistive devices; however, dorsiflexion strength on the affected side has been reported as only 30% that of the normal contralateral side, and return to activities more strenuous than walking has not been reported.[16] Vigasio et al.[23] reported the outcomes of combined posterior tibial tendon/flexor digitorum longus transfers for CPN palsy in 16 patients and concluded that their procedure effectively restored balance to foot dorsiflexion and gait without the use of orthosis. It should be noted, however, that tendon transfer can lead to flatfoot and/or hind foot valgus, which can hinder full functional recovery.[2]

Recent surgical reports for peroneal nerve palsies of various origins confirm success for nerve grafting without tendon transfer in 75% of patients with a nerve gap of < 6 cm and 16% with a nerve gap of > 6 cm.[11] By contrast, success (Grade M3 to M4+) of 85%–90% has been reported recently when tendon transfer is added to the nerve grafting.[7]

Length of Graft

Functional outcome of nerve grafting result is dependent on nerve graft length necessary to close the gap, with documented recovery rates of only 44% for nerve grafts longer than 6 cm.[8,24] Sedel and Nizard[19] also reported results of nerve grafting for traction injury of the CPN. Of the 17 patients who underwent grafting for nerve gaps ranging from 7 to 20 cm, only 6 had a functionally satisfactory result. The authors attributed these poor graft results to the significant length of their traction injuries (up to 15 cm).

As reported by Prasad et al.,[17] the reason for failure of nerve grafting for CPN disruption due to stretch/traction is that the zone of injury has transformed the normal intercalation of the terminal motor axons and muscle into a region of collagen, thereby not allowing even an expertly performed interfascicular interposition nerve graft from having regenerating axon branches reach the target end-organ, the denervated muscle. Conceptually, this problem might be solved by direct neurotization of the muscle, instead of performing interfascicular interposition nerve grafting.

Conclusions

Common peroneal nerve palsy is an incapacitating complication, and its incidence, because of sports-related knee injury, is relatively common. Recent studies have shown an increase in knee injuries associated with sports. Traumatic knee-level peroneal nerve injury is usually due to stretch/contusion, which often requires graft repair. Surgical repair using neurolysis if NAPs across the lesion are positive, and nerve grafting to the nerve, can rescue dorsiflexion of the foot but only when grafts are relatively short (< 6 cm). Graft length is one of the major factors to be considered in the prognosis of peroneal nerve repair. Tendon transfer, either in addition to CPN repair or without it, is a reliable procedure for restoring dorsiflexion, even though not with full strength.

Disclosure

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

Author contributions to the study and manuscript preparation include the following. Conception and design: Kim, Kline. Acquisition of data: Cho, Saetia, Lee. Analysis and interpretation of data: Kim, Cho, Saetia, Lee. Drafting the article: Cho, Saetia, Lee. Critically revising the article: Kline. Reviewed submitted version of manuscript: Kim, Kline. Approved the final version of the manuscript on behalf of all authors: Kim. Statistical analysis: Cho, Saetia. Administrative/technical/material support: Kim, Cho. Study supervision: Kim.

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References

1. Babwah TJ: Incidence of football injury during international tournaments. **Res Sports Med 17:**61–69, 2009
2. Bodily KD, Spinner RJ, Bishop AT: Restoration of motor function of the deep fibular (peroneal) nerve by direct nerve transfer of branches from the tibial nerve: an anatomical study. **Clin Anat 17:**201–205, 2004
3. Bonnevialle P, Chaufour X, Loustau O, Mansat P, Pidhorz L, Mansat M: [Traumatic knee dislocation with popliteal vascular disruption: retrospective study of 14 cases.] **Rev Chir Orthop Reparatrice Appar Mot 92:**768–777, 2006 (Fr)
4. Bonnevialle P, Dubrana F, Galau B, Lustig S, Barbier O, Neyret P, et al: Common peroneal nerve palsy complicating knee dislocation and bicruciate ligaments tears. **Orthop Traumatol Surg Res 96:**64–69, 2010
5. Bonnevialle P, Pidhorz L, Membres du Groupe d'Etude en Traumatologie Ostéoarticulaire (GETRAUM): [Dislocation and fractures around the knee with popliteal artery injury: a retrospective analysis of 54 cases.] **Rev Chir Orthop Reparatrice Appar Mot 92:**508–516, 2006 (Fr)
6. Cush G, Irgit K: Drop foot after knee dislocation: evaluation and treatment. **Sports Med Arthrosc 19:**139–146, 2011
7. Garozzo D, Ferraresi S, Buffatti P: Surgical treatment of common peroneal nerve injuries: indications and results. A series of 62 cases. **J Neurosurg Sci 48:**105–112, 2004

8. Giuseffi SA, Bishop AT, Shin AY, Dahm DL, Stuart MJ, Levy BA: Surgical treatment of peroneal nerve palsy after knee dislocation. **Knee Surg Sports Traumatol Arthrosc 18:**1583–1586, 2010
9. Goitz RJ, Tomaino MM: Management of peroneal nerve injuries associated with knee dislocations. **Am J Orthop 32:**14–16, 2003
10. Johnson ME, Foster L, DeLee JC: Neurologic and vascular injuries associated with knee ligament injuries. **Am J Sports Med 36:**2448–2462, 2008
11. Kim DH, Murovic JA, Tiel RL, Kline DG: Management and outcomes in 318 operative common peroneal nerve lesions at the Louisiana State University Health Sciences Center. **Neurosurgery 54:**1421–1429, 2004
12. Kline DG: Surgical repair of peripheral nerve injury. **Muscle Nerve 13:**843–852, 1990
13. Koffler KM, Kelly JD IV: Neurovascular trauma in athletes. **Orthop Clin North Am 33:**523–534, vi, 2002
14. Krivickas LS, Wilbourn AJ: Peripheral nerve injuries in athletes: a case series of over 200 injuries. **Semin Neurol 20:** 225–232, 2000
15. Nath RK, Lyons AB, Paizi M: Successful management of foot drop by nerve transfers to the deep peroneal nerve. **J Reconstr Microsurg 24:**419–427, 2008
16. Niall DM, Nutton RW, Keating JF: Palsy of the common peroneal nerve after traumatic dislocation of the knee. **J Bone Joint Surg Br 87:**664–667, 2005
17. Prasad AR, Steck JK, Dellon AL: Zone of traction injury of the common peroneal nerve. **Ann Plast Surg 59:**302–306, 2007
18. Robertson A, Nutton RW, Keating JF: Dislocation of the knee. **J Bone Joint Surg Br 88:**706–711, 2006
19. Sedel L, Nizard RS: Nerve grafting for traction injuries of the common peroneal nerve. A report of 17 cases. **J Bone Joint Surg Br 75:**772–774, 1993
20. Seroyer ST, Musahl V, Harner CD: Management of the acute knee dislocation: the Pittsburgh experience. **Injury 39:**710–718, 2008
21. Strazar R, White CP, Bain J: Foot reanimation via nerve transfer to the peroneal nerve using the nerve branch to the lateral gastrocnemius: case report. **J Plast Reconstr Aesthet Surg 64:**1380–1382, 2011
22. Sunderland S: The anatomy and physiology of nerve injury. **Muscle Nerve 13:**771–784, 1990
23. Vigasio A, Marcoccio I, Patelli A, Mattiuzzo V, Prestini G: New tendon transfer for correction of drop-foot in common peroneal nerve palsy. **Clin Orthop Relat Res 466:**1454–1466, 2008
24. Wilkinson MC, Birch R: Repair of the common peroneal nerve. **J Bone Joint Surg Br 77:**501–503, 1995
25. Wood MB: Peroneal nerve repair. Surgical results. **Clin Orthop Relat Res 267:**206–210, 1991
26. Yeap JS, Birch R, Singh D: Long-term results of tibialis posterior tendon transfer for drop-foot. **Int Orthop 25:**114–118, 2001
27. Yeap JS, Singh D, Birch R: Tibialis posterior tendon dysfunction: a primary or secondary problem? **Foot Ankle Int 22:** 51–55, 2001

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