Structural brain injury in sports-related concussion

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Object. Sports-related concussions (SRCs) represent a significant and growing public health concern. The vast majority of SRCs produce mild symptoms that resolve within 1–2 weeks and are not associated with imaging-documented changes. On occasion, however, structural brain injury occurs, and neurosurgical management and intervention is appropriate.

Methods. A literature review was performed to address the epidemiology of SRC with a targeted focus on structural brain injury in the last half decade. MEDLINE and PubMed databases were searched to identify all studies pertaining to structural head injury in sports-related head injuries.

Results. The literature review yielded a variety of case reports, several small series, and no prospective cohort studies.

Conclusions. The authors conclude that reliable incidence and prevalence data related to structural brain injuries in SRC cannot be offered at present. A prospective registry collecting incidence, management, and follow-up data after structural brain injuries in the setting of SRC would be of great benefit to the neurosurgical community.

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epidural hemorrhage • subdural hemorrhage • malignant cerebral edema •
second-impact syndrome

CONCUSSION, also termed mild TBI, is broadly defined as a transient, trauma-induced physiological disruption of brain function.^{44,79} According to the international body of experts making up the Concussion in Sport Group, concussion results in the rapid onset of short-lived impairment of neurological function that resolves spontaneously with no abnormality seen on standard structural neuroimaging studies.⁵⁹ Sports-related concussions comprise nearly 9% of all injuries sustained during athletics, and emergency department visits for SRCs among children and adolescents have increased by 60% in the last decade.^{32,36} About one-third of SRCs are sustained by high school football players alone.³⁶ While collision and contact sports like football, ice hockey, soccer, and lacrosse garner much attention, concussion rates in other sports are significant. A recent nationwide

study identified girls' soccer, girls' basketball, and boys' wrestling as the second, third, and fourth most implicated sports, respectively, in concussion incidence.⁵⁴ Female athletes seem to be at greater risk for concussion than their sport-matched male counterparts.^{48,54} Similarly, younger athletes appear to not only be more susceptible to concussion, but also take longer to recover.^{38,105}

While most concussions produce symptoms that resolve spontaneously, more severe injuries, reflected by positive imaging studies, are seen in a small subset of athletes with SRC. The spectrum of sports-related concussion with structural brain injury (SRCsBI) includes the following: 1) SAH; 2) EDH; 3) SDH; and 4) malignant cerebral edema, dubbed second-impact syndrome, or SIS.^{10,14,15,57,58,64,101} All cases are medical emergencies.

Due to their infrequency, epidemiological and long-term data are lacking for the more severe SRCsBI cases. The objective of our article is to review the literature on sports-related structural head injuries and to offer perspective on how to learn more about these rare injuries and improve treatment, outcomes, and prevention. We also include comments about our own institutional experience with SRCsBI, which has not been previously published.

Abbreviations used in this paper: AC = arachnoid cyst; ASDH = acute subdural hemorrhage; CSDH = chronic SDH; EDH = epidural hemorrhage; GCS = Glasgow Coma Scale; LOC = loss of consciousness; MLS = midline shift; SAH = subarachnoid hemorrhage; SCI = spinal cord injury; SIS = second-impact syndrome; SRC = sports-related concussion; SRCsBI = SRC with structural brain injury; TBI = traumatic brain injury; tSAH = traumatic SAH.

Methods

MEDLINE and PubMed records were searched to identify all studies pertaining to structural injuries in the setting of SRC in the last half-decade. The proceeding terms were used as Medical Subject Heading terms and text words: sport-related head injury, concussion, catastrophic head injury, diffuse axonal injury, epidural hemorrhage, subarachnoid hemorrhage, subdural hemorrhage, malignant cerebral edema, and second impact syndrome. The reference lists of these articles were examined to identify additional relevant research. Of note, we defined “sport-related” as any endeavor in which an individual was performing an athletic activity alone or in competition against an opposing person or team. Traditional team sports were included, such as basketball, football, and soccer, in addition to individual sports such as snowboarding or equestrian events.

Results

We found a total of 40 articles containing reports of SRCSBI. The study types included 28 case reports, 10 case series, 1 observational cohort design, and 1 case-control study. We provide a summary of all sports implicated in reported cases of SRCSBI (Table 1). We present these data based on the type of structural damage seen. When overlap between intracranial injuries existed in a single publication, the paper was categorized based on the most relevant lesion discussed.

Subarachnoid Hemorrhage

Subarachnoid hemorrhage is defined as bleeding into the subarachnoid space, between the arachnoid membrane and pia mater surrounding the brain. Subarachnoid

TABLE 1: Sports implicated in cases of sport-related structural brain injuries

Sport
basketball
boxing
cycling
equestrian
football
golf
ice hockey
jump training
kickboxing
martial arts
race walking
rugby
skateboarding
snow skiing
snowboarding
soccer
weightlifting

hemorrhage in SRCSBI may be divided into categories based on the site or origin and pattern of bleeding. Isolated tSAH is an uncommon sport-related head injury; the literature is limited to small series and case reports (Table 2).^{33,77} This form of SAH is a consequence of momentary oscillation of the brain as a result of rotational acceleration of the head and subsequent shearing of small cortical vessels.²⁸ Often diagnosed on CT scans as a hyperdense focus along the convexity, this type of brain injury has been described as an isolated finding in mild TBI or as a component of additional injuries that includes traumatic axonal injury, SDH, or other structural injuries.^{29,80}

Other forms of SAH in athletes may be due to trauma-induced large-vessel damage or aneurysm rupture precipitated by a traumatic event. Blows to the neck or base of the skull caused by a puck (hockey),⁸⁰ collision with a tackler (rugby),⁶¹ and an opponent's foot (martial arts, kickboxing)^{21,53} have all been described as causing vertebral artery injury and subsequent hemorrhage. Subarachnoid hemorrhage caused by aneurysm rupture in the setting of athletic play has also been described in soccer,¹¹ hockey,⁸¹ and weightlifting,³⁹ sometimes blurring the distinction between tSAH and traditional aneurysmal SAH. In these scenarios, an underlying vascular lesion is subjected to extreme external or hemodynamic forces, precipitating catastrophic hemorrhage.

As in non-sport-related trauma, the finding of a culprit aneurysm portends a worse prognosis, whereas tSAH from cortical vessel rupture alone often involves a benign recovery. In patients with mild TBI, Deepika and associates²⁶ demonstrated no difference in outcome between patients with isolated SAH and those with a normal CT scan. In patients with severe TBI, however, there is debate about the risk of tSAH-related cerebral vasospasm and whether antivasospastic therapy should be employed. In a retrospective review of 117 patients with tSAH, Lin et al.⁴⁷ found extensive tSAH to be an independent risk factor for vasospasm, as were age and initial GCS score. Conversely, a study by Shahlaie and coauthors⁸⁸ found only parenchymal contusions and fever—not the presence of SAH—to be associated with posttraumatic vasospasm.

In our clinical practice, SAH is rarely seen after sport-induced mild TBI. In patients with convexity tSAH associated with mild TBI—sports related or otherwise—and reassuring examination findings, antiplatelet or anticoagulation medication is withheld for 1 week, at which point a repeat CT scan is obtained to confirm the evolution and nonexpansion of subarachnoid blood. Furthermore, episodes of tSAH may occur undetected after SRC even in cases of transient concussive symptoms. A high index of suspicion is essential in these situations.

Overall, the literature on sport-related SAH is limited to case report and case series data only. Several reports have cited aneurysm rupture incited by sport-related trauma, whereas others have noted tSAH with no associated aneurysmal involvement.^{11,83,40} The general neurosurgery literature is helpful in extrapolating trends and outcomes, but sport-specific data are lacking.

Epidural Hemorrhage

An EDH occurs when there is bleeding between the

TABLE 2: Sport-related brain injuries leading to SAH*

Authors & Year	Study Type	Sport	Injury	Neurosurgical Management	Details
Brunelle et al., 2012	case report	soccer	tSAH w/ aneurysm	surgical clipping	small aneurysm at ICA bifurcation
Rughani et al., 2011	case series	snow skiing	tSAH, EDH, SDH	unspecified	11 SAHs; no difference in incidence of SAH w/ or w/o helmet use
Cohen et al., 2010	case report	martial arts	tSAH, VA dissection	endovascular VA occlusion & PICA reconstruction	GCS Score 3 on arrival; discharged at 1 mo w/ mild dizziness & gait ataxia
Depreitere et al., 2004	case series	cycling (leisure & competitive)	tSAH, SDH	varied	majority related to MVCs; 45 tSAHs; SAH greater predictor of worse outcome than skull fracture, EDH, SDH, or ICH
Malek et al., 2000	case report	kickboxing	tSAH, dissecting VA aneurysm	endovascular pseudoaneurysm coiling	presented 6 days after event w/ LOC, nystagmus, CN VI palsy; asymptomatic at 3-mo follow-up
McCrory et al., 2000	case series	rugby	tSAH, VA trauma	unspecified	4 fatal tSAHs; 3 related to VA injury
Sahjapaul et al., 1998	case report	hockey	tSAH, ruptured VA aneurysm	none	died hrs after struck by puck; massive posterior fossa SAH w/ ruptured VA aneurysm
Haykowsky et al., 1996	case series	weightlifting	SAH, aneurysm rupture w/ valsalva	surgical clipping	3 SAHs in setting of biceps curls & leg press; uneventful clipping w/ good outcomes
Raschka et al., 1995	case report	soccer	tSAH, IVH, atlantal fracture	unspecified	extensive SAH & IVH leading to rapid death
Fekete, 1968	case report	hockey	tSAH, EDH, SDH	none	16-yr-old helmeted & body checked to ice; died 2 hrs after event

* CN = cranial nerve; ICA = internal carotid artery; IVH = intraventricular hemorrhage; MVC = motor vehicle collision; PICA = posterior inferior cerebellar artery; VA = vertebral artery.

skull and the dura mater. Classically, these intracranial lesions occur secondary to calvarial fractures within the temporal or temporoparietal bone region.⁸ Extradural bleeding most commonly results from tearing of the middle meningeal artery but may be caused by laceration of the middle meningeal vein, diploic veins, or venous sinuses.^{13,62} Clinical presentation of EDH is varied as patients can present during the so-called lucid interval after a brief period of unconsciousness or arrive comatose upon admission. Neurological deficits can range from minimal to severe with cranial nerve palsies and signs of intracranial herniation.

The most common causes of EDH are traffic-related accidents (53%), falls (30%), and assaults (8%); however, EDH has also been described as a result of sports-related injuries including golf, skiing, basketball, soccer, and skateboarding (Table 3).^{12,25,67} Rughani et al.⁸⁰ reported finding EDHs, often occurring in unhelmeted athletes, in 3 pediatric skiers, all in the acute setting. Bruzzzone and colleagues¹² reported a delayed EDH in a 19-year-old soccer player, in whom there was radiographic evidence of an EDH first at 8 hours posttrauma, with negative imaging 80 minutes after the collision. Wang et al.⁹⁹ performed a retrospective analysis of children who had been struck in the head with a golf club while watching another person swing. They found that all patients sustained depressed skull fractures, with 6 of 13 resulting in acute EDH. In these children, all 6 required craniotomy and clot evacuation. Only 1 child experienced long-term neurological sequelae. Overall, the epidemiology and prognosis of patients developing EDH secondary to sports-related injuries has not been well studied.

With respect to sport-related EDH, the current case studies in the literature offer little insight into trends and outcomes. There are as many data on injured sports observers (for example, golfing victims) as there are on athletes. As seen in the next section, SDH is the more common injury in SRCSBI. Although there is likely significant overlap between both injuries, information specific to EDH is difficult to ascertain.

Subdural Hemorrhage

Subdural hemorrhage, defined as the presence of blood between the outermost dura and arachnoid layer covering the brain, is the most common cause of death due to catastrophic sport-related head injury (<http://www.unc.edu/depts/nccsi/>).¹⁶ Often resulting from tearing of bridging veins as they cross the subdural space, SDH can cause a rapid increase in intracranial pressure and subsequent neurological deficit or death. In contrast to EDH, in which secondary injury is often the culprit, the magnitude of impact in SDH damage is greater, leading to significant parenchymal injury, as well as compression with MLS.^{3,769} Both ASDH and CSDH are often diagnosed after a noncontrast head CT scan is acquired. With ASDH, a crescent-shaped mass of hyperdensity is seen along a convexity or the tentorium. Chronic SDH is diagnosed by depiction of a hypodensity along the cerebral convexity, designating chronic blood.

As first reported in the early 20th century, 18 players died during the 1905 college football season, nearly all

TABLE 3: Sport-related brain injuries leading to EDH*

Authors & Year	Study Type	Sport	Injury	Neurosurgical Management	Details
Wang et al., 2011	case series (n = 13)	golf	acute temporal EDH	craniotomy	only 1 patient had long-term neurological sequelae after EDH evacuation
Nemecek et al., 2005	case report	skateboard- ing	delayed vertex EDH	no surgical intervention	EDH located overlying the SSS; no op intervention due to increased risk to sinus tear during EDH evacuation; patient now w/o neurological deficits
Bruzzone et al., 2000	case report	soccer	acute frontoparietal EDH	craniotomy	9-mo follow-up w/ no seizures & neurophysiological testing w/in normal range
Datti et al., 1995	case report	basketball	acute temporal EDH	craniotomy	patient discharged w/o neurological deficits; basketball has a low risk of nervous system injuries

* SSS = superior sagittal sinus.

due to SDH.¹⁶ The more popular sports, such as football, soccer, and hockey, have been implicated in SDH, as have lesser practiced athletic endeavors, such as snowboarding, martial arts, and race walking.^{17,35,76,95,96} The majority of SDHs are acute, and immediate hospitalization and neurosurgical treatment are indicated; however, CSDHs have been reported. A 43-year-old man suffered a CSDH due to a basketball injury, returning to the hospital weeks later with debilitating headaches.⁴³ A 65-year-old man receiving warfarin presented with left leg weakness and headache, and imaging showed a chronic SDH; the authors reported the bleed was likely due to the jarring impact of race walking.¹⁷

The literature on SDH due to sport-related head injury includes the highest level of evidence, a case-control study, rather than single reports or small series (Table 4).^{52,55,74,94} Kushi and associates⁴⁶ compared 10 boxers with ASDH compared with 26 nonboxers with ASDH. They found that boxing-related ASDHs occurred in younger patients who experienced lucid intervals, without cerebral contusions. No between-group differences were noted for GCS score, MLS, or overall prognosis.⁴⁶

Within the existing case reports, an interesting theme emerged in that several athletes reported a recent head injury prior to the inciting impact leading to SDH.^{45,49,51} Not to be confused with diffuse cerebral edema or SIS, in which the brain loses its ability to autoregulate and massively swells, malignant edema is not seen in these cases. Kersey⁴⁵ reported on a 19-year-old football player with acute ASDH 35 days after sustaining a mild concussion. The patient underwent a craniotomy and recovered without neurological deficit, despite presenting with decerebrate posturing and a fixed and dilated left pupil. The second impact, involving a presumptive minor head injury immediately prior to SDH, could not be recalled by the player or by spectators. Logan et al.⁵¹ similarly described 2 unreported episodes of head trauma preceding an ASDH in an 18-year-old football player who underwent medical management. The specific head injury precipitating the ASDH could not be identified, yet the athlete recalled 2 prior unreported head impacts that caused symptoms of headache and nausea. In 1995, Litt⁴⁹ reported on a 16-year-old football player with concussion

and symptom resolution 16 days postinjury. The athlete was cleared to play 30 days after the initial concussion. During his first game back, the player collided with an opposing player, was taken out of the game, and quickly experienced deterioration on the sideline. Attending neurosurgeons at a local emergency department diagnosed ASDH, and a bur hole craniotomy was performed. Lastly, in their critical review of sudden death in young, competitive athletes, Thomas et al.⁹³ found that 17% of the 138 football players who died of head/neck trauma had a history of concussion several days to 4 weeks prior to injury.

This theme is echoed in our own institutional experience. In the last 3 years, 2 high school football players presented to our pediatric emergency department with ASDH, one 15-year-old managed medically and one 17-year-old requiring craniotomy. Both players and coaches reported head trauma within the 2 weeks preceding their presentation to medical attention. Neither player was diagnosed with a concussion during the game where the original injury was thought to have occurred. Of note, both players' helmets were tested under standard National Operating Committee for the Safety of Athletic Equipment guidelines.⁶⁶ Both helmets passed the aforementioned guidelines, deeming them adequate to protect against the accepted minimum levels of linear acceleration.

A second theme among the SDH case reports links arachnoid cysts to an increased risk of sport-related CSDH.⁷⁵ Arachnoid cysts occur in 1% of the population and can be complicated by intracystic, subdural, or epidural bleeding caused by damage to surrounding veins.^{19,34,40,75,98,100} The annual risk of cyst hemorrhage has been reported to be less than 0.1%.^{71,87} Treatment for problematic arachnoid cysts includes fenestration of the cyst with or without excision, or shunting the cyst contents to the peritoneal cavity.⁷¹

Prabhu and Bailes⁷⁵ reported the case of a 16-year-old girl who was struck in the head by a soccer ball and presented 1 month later with headaches and numbness on the right side of her face. She was found to have a large, chronic and subacute SDH and underwent craniotomy and cyst fenestration. Zeng et al.¹⁰³ reported 2 similar cases. In the first, a 14-year-old boy suffered a residual headache of several weeks' duration after vigorous jumping during

TABLE 4: Sport-related brain injuries leading to SDH*

Authors & Year	Study Type	Sport	Injury	Neurosurgical Management	Details
Galvez & Hecht, 2011	case report	martial arts	subacute SDH w/ 7-mm MLS	bur hole craniotomy	noncontact head trauma: patient felt popping sensation upon kick against pad
Zeng et al., 2011	case report case report	jump training soccer	CSDH w/ signif MLS CSDH w/ mod MLS	craniotomy bur hole craniotomy	AC predisposed to sport-related SDH; asymptomatic at postop clinic visit AC predisposed to SDH in setting of frequent ball heading; good neurological recovery
Kushi et al., 2009	case-control study (n = 10)	boxing	ASDH	decompressive craniectomy	boxing-related ASDH not assoc w/ cerebral contusion or contralat brain injury; no diff in GCS, MLS, or prognosis vs controls
Lutfi et al., 2009	case report	soccer	ASDH w/ 4-mm MLS	nonop; prophylactic anticonvulsant meds	healthy child w/o underlying CNS abnormality; SDH after heading a soccer ball; full recovery
Matsuda et al., 2008	case report	football	ASDH w/ mod MLS	bur hole craniotomy	blow to occipital region; delayed-onset SDH 6 days after initial normal head CT; full recovery
Türkoğlu et al., 2008	case report	martial arts	CSDH	bur hole craniotomy	denied specific traumatic event; complained of 6-mo HA; resolved postop
Demetriades et al., 2004	case report	soccer	CSDH	bur hole craniotomy	AC predisposed to SDH in setting of repeated heading of soccer ball; complete neurological recovery
Rajan & Zellweger, 2004	case report	snowboarding	SDH w/ massive MLS	bur hole craniotomy	HA w/ short-term memory deficits; complete recovery at 2-wk follow-up
Tsuzuki et al., 2003	case report	basketball	CSDH w/ minimal MLS	bur hole craniotomy	CSDH assoc w/ AC; no direct head injury identified
Uzura et al., 2003	case series	snowboarding	CSDH (1): 15-mm MLS; CSDH (2): slight MLS	bur hole craniotomy	gradual HA leading to vomiting; full neurological recovery
Carmont et al., 2002	case report	race walking	CSDH with signif MLS	bur hole craniotomy	chronic anticoagulation for cardiac condition; unilat weakness resolved postop
Prabhu & Bailes, 2002	case report	soccer	CSDH & subacute SDH	craniotomy & AC fenestration	SDH assoc w/ AC after hit in head by ball; full recovery
Logan et al., 2001	case report	football	ASDH	medical management	small SDH; conserv mgt; residual HA only at 1-mo follow-up
Powell & Barber-Foss, 1999	observational cohort study	football	SDH	unspecified	4 cases of SDH of 773 cases of mild TBI; no deaths reported
Keller & Holland, 1998	case report	basketball	bilat chronic SDH	bilat bur hole craniotomy	persistent HAs led to op consent despite patient's initial reluctance; asymptomatic at final follow-up
Kersey, 1998	case report	football	acute-on-chronic SDH w/ massive MLS	bur hole craniotomy	pupils fixed & dilated w/ decerebrate posturing; full neurological recovery 19 days postop
McAbee & Ciminera, 1996	case series (n = 2)	equestrian	ASDH w/ EDH (1); ASDH (2)	craniotomy	both sustained serious intracranial injury, 1 w/ sustained sequelae; both wore helmets
Litt, 1995	case report	football	ASDH w/ intraop evidence of old SDH	bur hole craniotomy	underwent 2nd impact; athlete did not report symptoms from initial injury; full neurological recovery
Saunders & Harbaugh, 1984	case report	football	ASDH	hemicraniectomy	resulted in death; autopsy revealed widespread anoxic changes; mod nos. of reactive astrocytes w/o acute inflammation

* AC = arachnoid cyst; assoc = associated; conserv mgt = conservative management; diff = difference; HA = headache; mod = moderate; signif = significant.

youth sport activities. He was then seen in an outpatient primary care setting and found to have an arachnoid cyst. Several weeks later, the patient presented with worsening headache and projectile vomiting, and he was found to have intracystic bleeding and a left-side CSDH with significant shift. A craniectomy was performed to evacuate the CSDH, and the cyst was fenestrated. In the second case, a 16-year-old male soccer player had a 4-week history of headache and nausea. He denied history of trauma other than frequent heading of the soccer ball. Magnetic resonance imaging showed CSDH and left middle fossa arachnoid cyst despite the absence of neurological deficit. A bur hole craniotomy was performed, and the CSDH was evacuated. Similarly, Demetriades et al.²⁷ reported the case of a 24-year-old soccer player with a 6-week history of headache and nausea and no history of head trauma except for regular heading during soccer practice. The patient was found to have a CSDH overlying a portion of arachnoid cyst and underwent craniotomy.

The earliest link between arachnoid cysts and CSDH was noted in 1971.¹⁰⁴ Chronic SDH complicates approximately 20% of arachnoid cysts. One theory states that because arachnoid cysts are less compliant than normal brain tissue, less cushioning is provided during minor trauma, increasing the likelihood of bridging vein rupture.¹⁰³ Bilginer et al.⁵ studied 132 patients with arachnoid cysts and found that the risk of associated SDH was 2.27% (3 cases). Mori and colleagues⁶³ reported this rate to be 1.5% in 541 patients. The risk of arachnoid cyst hemorrhage with SDH secondary to a sports-related head trauma is unknown; however reports have been published on boxing, basketball, racing, jumping, and football.^{27,75,103} Given the anecdotal increased risk of SDH in young patients with arachnoid cysts, screening may be indicated for those involved in sports who have sustained repetitive head trauma. Only in extreme cases may it be prudent for a careful risk-benefit discussion between the surgeon and the family of certain athletes concerning surgical removal. There have been no reports of prophylactic surgery for arachnoid cysts in the setting of competitive sports.

Overall, SDH is the first structural injury to offer a case-control study and themes within the case reports. There is a propensity for SDH after a minor head trauma, oftentimes associated with multiple subconcussive impacts. Furthermore, an arachnoid cyst may increase a young athlete's risk for acute or chronic SDH.

Diffuse Cerebral Edema

As he first described in his 1973 book, *Head and Neck Injuries in Football: Mechanisms, Treatment, and Prevention*, neurosurgeon Richard Schneider reported an initial concussion in 2 young athletes who subsequently died after a second, minor head injury that produced diffuse cerebral edema.^{85,101} The term "second-impact syndrome," or SIS, was coined 11 years later by Saunders and Harbaugh,⁸³ in their description of a 19-year-old college football player who collapsed and died on the field 4 days after a concussion. Today, SIS is defined as occurring when "[a]n athlete who has sustained an initial head injury, most often a concussion, sustains a second head injury before symptoms associated with the first have fully

cleared" and when the second, minor injury, perhaps only involving a blow to the torso, results in catastrophic brain swelling and death.^{14,42,60,83,91} Second-impact syndrome occurs primarily in male adolescents and/or teenagers, rarely in females, most often during football, but has been seen in boxing, martial arts, and skiing.^{15,58,64} The pathophysiology of SIS involves a rapid loss of autoregulation, combined with a catecholamine surge, leading to a paralyzed arterial tree unable to respond to rapid escalation of blood pressure.^{9,10,42,50,89,91,101} Furthermore, a key finding in SIS is absence of an expanding intracranial hematoma causing compression. A thin, acute SDH may be seen, but without mass effect; rapid, malignant cerebral edema is the major culprit in SIS.^{15,64}

Since its initial description, the existence of SIS has been marred with controversy. Several experts support the underlying pathophysiology,^{14,15} whereas another contingent has argued against its existence, positing that it is simply a higher-magnitude SDH with edema and poorer outcome.^{57,58} The epidemiology of SIS is largely based on anecdotal case reports, complicated by the debate over whether SIS exists (Table 5).^{73,101} McCrory⁵⁷ believes that by the strict definition of SIS, only 5 cases meet criteria for "probable SIS," with several reports being excluded. The US National Center for Catastrophic Sport Injury Research cited 145 cases of catastrophic cerebral injury over a 26-year period with only a single case of possible SIS (<http://www.unc.edu/depts/nccsi/>). However, Cantu and Gean¹⁵ recently reported 10 cases of SIS associated with a slim, acute SDH. They maintained that the inciting acceleration forces required to produce a small SDH reach the threshold to produce SIS in the setting of previously damaged brain tissue. Mori et al.⁶⁴ reported a similar case series of 18 patients, but they hypothesized that the "more impressive" brain swelling was likely due to the thin SDH.

A debate over the existence of SIS is beyond the scope of this article. However, it is inarguable that malignant brain swelling with catastrophic outcome has been documented and that reliable epidemiological data are needed to correctly manage minor concussive sport-related head injury. The fear of SIS continues to drive return-to-play guidelines and concussion safety protocols.⁵⁸ Albeit SIS is rare, knowing its true risk is crucial for neurosurgeons and providers to establish reliable safety parameters. Dutiful reporting of diffuse cerebral edema cases is needed.

Discussion

Concussion is a common sports injury, seen at the youth, high school, college, and professional level. An army of health care providers is involved in the treatment of individuals with these mild brain injuries, including certified athletic trainers, internists, pediatricians, family practitioners, neuropsychologists, neurologists, and neurosurgeons. With proper knowledge of concussive injury and management and an evidence-based approach to return to play, many health care providers can successfully manage SRCs. However, only one population possesses the training to treat structural brain injuries requiring acute surgical intervention. In the setting of these life-

TABLE 5: Sport-related brain injuries leading to diffuse cerebral edema*

Authors & Year	Study Type	Sport	Injury	Neurosurgical Management	Details
Potts et al., 2012	case report	football	ASDH; DCE	craniectomy	superb neurological outcome after treating DCE; patient functioning at baseline academically w/o neurological deficits
Cantu & Gean, 2010	case series (n = 10)	football	ASDH; DCE	craniotomy or hemicraniectomy or no surgical intervention	38% of athletes who had ASDH were still symptomatic from a prior head injury; clinical outcomes in the 10 patients ranged from minimal neurological deficits to death
Mori et al., 2006	case series (n = 8)	multiple	ASDH; DCE	craniotomy or bur hole	prior head injury w/o resolution of symptoms increased risk of SDH & DCE after a 2nd impact
Cantu & Mueller, 2003	case report	rugby	DCE	no surgical intervention	cerebral edema occurred after patient's helmet was impacted by another player; resulted in death
McCorry, 2001	case report	football	DCE	no surgical intervention	head CT scan confirmed subfalcine herniation due to massive swelling
Kelly et al., 1991	review	boxing	DCE	no surgical intervention	DCE is associated w/ structural brain injury (e.g., ASDH)
	case report	football	ASDH; DCE	no surgical intervention	resulted in death; individuals symptomatic from concussion, even w/o LOC, at risk for DCE after a 2nd impact

* DCE = diffuse cerebral edema.

altering injuries, where permanent neurological deficits or death occur 60% of the time, neurosurgeons are called on to intervene.⁶

Whereas concussions are transient and lack positive neuroimaging findings, severe TBI can lead to permanent neurological deficit and have definitive imaging findings. The international Concussion in Sport Group defines concussion as a “complex pathophysiologic process affecting the brain, induced by traumatic biomechanical forces.”⁵⁹ Because concussion is typically a functional injury that is often unaccompanied by identifiable structural insults, postconcussion treatment is largely observational, behavioral (for example, rest and the avoidance of noxious environmental stimuli that can exacerbate symptoms), and preventative of secondary injury. Beyond rest and recovery, there have been modest efforts to introduce pharmacological agents into the postconcussion treatment realm. Recent reviews of the role of pharmacology in SRC have been provided by Petraglia and coworkers⁷² and by Solomon and Sills.⁹⁰ A recent retrospective case-control study suggests that the use of amantadine may improve reported symptoms, verbal memory, and reaction time in patients suffering from a recent SRC.⁷⁸

Our review demonstrates the paucity of current data and suggests that much work remains to be done. In the subsequent sections we explore the potential mechanisms that may be used to better address this problem from a public health perspective. These strategies include the use of a national registry, continued emphasis on education and prevention programs, and changes in public policy.

National Registries

A patient registry is defined as “an organized system that uses observational study methods to collect uniform data to evaluate specified outcomes for a population defined by a particular disease, condition or exposure, which serves predetermined scientific, clinical or policy purpose(s).”³⁷ With strong methodology and systematic research questioning, registries can provide longitudinal patient outcome data, improving our knowledge of specific health states and allowing critical evaluation of interventions.^{37,70} Key points to consider when designing a registry include formulating a question, choosing a study design, translating question of clinical interest into measurable outcomes, choosing patients, and deciding how many patients need to be studied for how long.³⁷ An in-depth discussion of registry science is outside the scope of this article, but rich literature exists describing the design and implementation of patient registries.³⁷

In the realm of SRC/SBI registries, the National Center for Catastrophic Sports Injury Research has served as the leader in recording these rare injuries. Since 1965, it has collected valuable information on demographic patterns, mechanism, and injury timing, with the most recent 2011 Survey of Catastrophic Football Injuries highlighting the summary data.⁶⁵ However, its focus remains on epidemiological data, rather than prospective, outcomes-based registry information. Treatment and long-term follow-up data are outside the scope of the efforts of the National Center for Catastrophic Sports Injury Research.

Outside of sports, several robust patient registries

exist collecting TBI and SCI data. Researchers in Korea established the Korean Brain Rehabilitation Registry for patients with stroke, TBI, brain tumors, and other brain disorders.¹⁰² Over an approximately 2-year period, they enrolled 1697 patients and recorded measurable functional improvement outcomes throughout the rehabilitation process. Similarly, in the US, researchers in Philadelphia have established a registry for post-stroke or -TBI patients by recording their cognitive rehabilitation progress for several years after the initial injury.⁸⁶ With respect to SCI registries, the Rick Hansen Spinal Cord Injury Registry was launched in 2004 to improve outcomes in patient with SCIs.⁷⁰ Based in Canada, this registry created a standardized set of clinical information to record outcomes from several international centers. For each patient, 265 data elements are recorded and patients are contacted up to 10 years from the date of injury, with regular 5-year contacts thereafter. In the US, the National Spinal Cord Injury Database has similarly aimed to further our understanding of Spinal Cord Injury and predictors of quality of life.^{30,42,70} Internationally, the European Multicenter Study about Spinal Cord Injury has evaluated SCI injury scales and recovery patterns.²⁴ It is imperative to highlight the emphasis these registries place on recovery and outcomes rather than epidemiology alone.

The neurosurgical community has invested heavily in registry-based initiatives. Recently, the American Association of Neurological Surgeons created a prospective data collection tool that includes case data from surgeons throughout the country. The National Neurosurgery Quality and Outcomes Database, or N²QOD, is a national registry that serves to include quality and outcomes data in an effort to define the quality of care delivered.⁶⁸ It is appropriate that the management of SRCSBI be addressed in a similar manner. In this way, decisions regarding the safety of our athletes will no longer be based upon case reports, small series, and anecdotal evidence but, rather, upon a large prospective tool accessible to health care personnel, coaches, and policymakers alike.

Education and Injury Prevention Programs

Bramley et al.⁷ recently demonstrated that high school soccer players with education training in concussions are more likely to notify their coach of a suspected concussion than players with no such training. Education is crucial in changing the SRCSBI landscape. The existing SRC management education literature serves as a useful model. In response to the public health problem of SRC, several organizations have risen to the occasion.

The Center for Disease Control and Prevention, through the Children's Health Act of 2000, implemented a national concussion education and awareness program.⁸² In 2005, "Heads Up: Concussion in High School Sports," directed toward high school coaches, athletic directors, and certified athletic trainers, was launched.⁸⁴ The tool kit included a guide for coaches and videos, clipboard stickers, wallet cards, and fact sheets for parents and athletes.⁸⁴ Within 3 months, the Center for Disease Control and Prevention distributed over 20,000 tool kits nationwide. The most recent iteration of the tool kit is available at www.cdc.gov/Concussion.

Chrisman et al.²⁰ randomly surveyed 414 physicians across the US; 183 received the Heads Up tool kit and 231 served as a controls. Physicians who received the tool kit were significantly less likely to recommend next-day return to play after SRC. Similarly, Sarmiento et al.⁸² administered the Heads Up tool kit to high school coaches and found favorable changes in knowledge, attitudes, and practices toward the prevention and management of concussions. Covassin et al.²³ distributed the same educational material to 340 youth sport coaches, who were found to be better able to prevent, recognize, and respond to SRC, with nearly three-quarters of coaches now educating others on SRC. Efforts have also been made to reach out to school personnel and special education staff who play an equally important role in identifying the effects of concussive symptoms through the initiative "Heads Up to Schools: Know Your Concussion ABCs."⁸²

The Sports Legacy Institute Community Educators, or SLICE, curriculum for student athletes seeks to educate student athletes on concussion diagnosis and management.⁴ Its program features medically trained students who deliver interactive presentations to their peer student athletes. Bagley et al.⁴ administered quizzes before and after the SLICE presentation to 636 students ages 9–18 years and found significant improvement in concussion knowledge in all subgroups. Presentations lasted 40–60 minutes and were highly interactive, including PowerPoint slides, videos, audience participation, case studies, and personal testimonials. In Canada, the ThinkFirst program has similarly succeeded in furthering concussion injury prevention by teaching mechanisms, consequences, and prevention of brain injuries in sports, most often to hockey players.^{18,22} The program has also been expanded to kids in kindergarten to 6th grade.⁹⁷

The advent of social media provides another avenue for concussion education. Ahmed et al.² recently analyzed the use of social networking sites for SRC by analyzing Facebook postings. From the 17 Facebook groups studied, the authors found that people most often shared their concussion experience online and also sought or offered advice on concussion management. They pointed out the evolving nature of health education and highlighted the crucial peer-to-peer interaction that takes place.² Sullivan et al.⁹² also sought to determine the context of mild TBI in the online population. The authors identified and classified 3488 concussion-related Tweets and categorized them appropriately, highlighting the powerful potential of Twitter to serve as broadcast medium for sports concussion information and education. At our own institution, we have developed a smartphone application for concussion safety to be used on the sidelines.

Sports-related concussions with structural brain injury are exceedingly rare, which makes education a challenge. Similar modules and tool kits would serve to increase awareness of these devastating injuries, but the condition presents a greater challenge than SRC management. Sports-related concussions are ubiquitous—virtually every coach has witnessed a player suffer an SRC, and every athlete has a teammate who has sustained a concussion. With SRCSBI, however, coaches, parents, school personnel, and players may be less enthusiastic

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about learning because of the injury's rarity. Organized efforts to educate and alert the sports community about the devastating and potentially irreversible consequences of failure to identify these injuries can ameliorate the educational gap.

Policy Changes

Effective concussion prevention requires legislation in addition to education.¹ The prototype model of a "concussion law" has been the Zackery Lystedt Law, passed in Washington State in 2006. In 2003, Zack was playing in a high school football game and suffered a concussion. He returned to play in that same game. Several plays later, he received another blow to the head that caused life-threatening bilateral SDH, leading to permanent neurological deficits and necessitating the use of a wheelchair. The new legislation proposed prompted by his injury contained several key provisions: 1) informed consent must be given in writing by parents of youth athletes prior to the first practice; 2) any athlete with a suspected concussion must be taken out of the game; and 3) a player cannot return to play until cleared by a licensed health care provider trained in concussion management.^{1,31}

Since the enactment of the Lystedt law in 2009, national organizations, including the American College of Sports Medicine and the National Football League, have joined forces to promote legislation regarding concussion education, prevention, and management. Over 40 states have either laws or bills pending that address concussion in youth sports.⁴

Extending legislation to SRCSBI should also be considered. Sports-related concussion laws will certainly help prevent devastating structural brain injuries, but additional legislation specific to SRCSBI may be needed. In our opinion, this begins with mandatory notification to a national injury registry. Armed with these data, patterns of injury can be tracked over time and the benefits of any such future sports-related legislation can be objectively measured. Other examples of possible legislation include debriefing with players and coaches after a catastrophic injury occurs, assessment of equipment involved in injury, and harsh penalties to coaches and/or schools who do not adhere to appropriate return-to-play policies. While coaches are generally expected to act in the best interest of the player, there is often a direct conflict of interest when weighing the decision to withhold a player from the field in the heat of a competitive game. The consequences for such decisions are amplified in SRCSBI, which may initially be perceived as an uncomplicated concussion.

Conclusions

Our review of the literature demonstrates the essential absence of prospective studies assessing the epidemiology and outcome of structural injury in SRC. The current structural injury literature is composed of small series, case reports, and one case-control study. Given the infrequency of these specific injuries, the state of the literature, although appreciated, is understood as limited. We conclude that reliable incidence and prevalence data related to SRCSBI cannot be offered at present.

Thus, we propose that the field of neurosurgery address this gap in the knowledge base of structural injury in SRC. As athletes at all levels become stronger and faster, and with the size of playing fields unchanged, physics and biomechanics would suggest that neurosurgeons may be seeing more cases of SRCSBI in the future. With respect to football, there are approximately 2000 professional players, 75,000 college players, and 1.2 million high school players, with an additional 250,000 competing at the Pop Warner level (<http://www.popwarner.com/football.htm>).⁶ Although the professional level receives ubiquitous media attention and financial support, specifically the formation of the National Football League Head, Neck, and Spine Committee (<http://www.nflrevolution.com>), the scope of this problem is most critical at the youth level. With decreased funding for equipment refurbishment and adequately trained sports medicine clinicians on the sidelines, many sports injuries may go unnoticed and/or unreported.

It is incumbent on the field of neurosurgery to provide comprehensive information in an effort to offer empirical epidemiological, risk, and follow-up data. Such information should include the incidence of structural injury, type of structural injury, whether the injury was managed medically or surgically, possible risk factors for the structural injury (for example, arachnoid cyst, multiple concussions, and so on), and several years of follow-up. Given the current climate of media focus, and the need to intervene in this public health problem, it would seem beneficial that neurosurgeons participate in reporting these cases, furthering SRCSBI education and helping to pass appropriate legislation.

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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