Second Impact Syndrome or Cerebral Swelling after Sporting Head Injury

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Abstract Second impact syndrome is believed to be the catastrophic consequence of repeated head injury in sport. The scientific evidence to support this concept is nonexistent, and belief in the syndrome is based upon the interpretation of anecdotal cases more often than not, lacking sufficient clinical detail to make definitive statements. The fear of this condition has driven many of the current return-to-play guidelines following con-cussion. Diffuse cerebral swelling (DCS) following a head injury is a well-recognized condition, more common in children than in adults, and usually has a poor outcome.

Introduction

The phenomenon of the second impact syndrome (SIS) continues to appear in the medical literature in spite of the lack of systemic evidence for its existence. Over a decade ago, the existence of this syndrome was brought into question, and since that time, other authors have begun to raise similar concerns as to the underlying entity (1,3,15,19). SIS has been defined as occurring when "an athlete who has sustained an initial head injury, most often a concussion, sustains a second head injury before symptoms asso-ciated with the first have fully cleared" (4,15,22). This second injury, it is believed, results in catastrophic brain swelling and a usually fatal outcome. The key question that remains controversial is whether a repeated head injury or blow is required to cause this entity or whether the brain swelling is the result of a single blow to the head, which is a well-recognized sequela of head injury.

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tinues to drive anecdotal sports concussion guidelines and to be cited by many clinicians as the basis for the use of computerized neuropsychological tests in determining return to play for concussed athletes. The premise that neurocognitive testing prevents the likelihood of complications such as diffuse brain swel-ling remains firmly opinion-based.

The fear of the existence of SIS con-

What Is the Evidence for SIS? The concept sts on the int

of anecdotal case reports. The majority of reported cases, including the index case, of this entity actually have not incident in the constraint of the china constraints and the involved a "second" impact (22). In the only systematic review of this topic, a total of 17 cases of reported SIS were identified in the world literature (12,15). Of these, only five cases actually involved a repeated injury, and all these occurred within 7 d of the initial injury Even in these cases, it was not clear that the initial injury played a contributory. role insofar as providing evidence that the athlete had obje tive evidence of ongoing symptoms and/or injury prior to the putative second impact. Of the 14 cases in this series that purance second impact. Or the 14 cases in this series that went to autopsy, 11 had evidence of other structural brain injuries such as intracranial bleeding (typically an acute subdural hematoma) in addition to the cerebral swelling. A ology of the more recent literature review of the pathophysiology of the "thin subdural hematoma + brain swelling" syndrome, involving a total of 18 cases, confirmed the presence of thin subdural hematomas on all cases in which computed tomographic (CT) scanning had been obtained (17). This of subdural hematomas also has been noted in ath-). While it may be understandable that a structural letes (5). injury, such as a subdural hematoma, may cause brain swelling, the traditional view of SIS is that this entity occurs in

the absence of structural injury. How Common Is SIS?

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If SIS actually exists, then its occurrence vanishingly is are. The only anecdotal reports of SIS are from the North American literature. As mentioned previously, a recent report from Japan noted 18 cases of brain swelling associated with CT-diagnosed subdural hematomas (17). It is

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surprising that there are no reports of SIS in the European or Australian literature, in particular, from sports such as Australian football, which has both a high participation rate and a high concussive injury rate (approximately 15 times that of American football (16)). An Australian study examining all sports-related deaths over a 35-year period did not find a single case of SIS (16). The 2010 report from the U.S. National Center for

Catastrophic Sport Injury Research (based at the University of North Carolina) cited 145 cases of catastrophic cerebral injury during the period of 1984 to 2010 with only a single case of a "possible" SIS noted in a case that actually pre-sented an acute subdural hematoma (18). Based upon an annual participation rate of 1.8 million subjects in U.S. high chool and collegiate sports, Randolph and Kirkwood (20) have estimated that this corresponds to one instance of "possible" SIS for every 205,000 player seasons. For a squad size of 50 players, this would translate to one such injury every 4,100 seasons (19,20). Given that virtually all of these injuries occur as the result of the immediate consequences of a single traumatic brain injury that typically results in an intracranial hematoma, the support for SIS from the recent data available from this source is limited.

What is the Underlying Pathophysiology?

It is known that a single brain impact can result in and blood volume that, in turn, is se

to a failure of cerebral vascular autoregulatory mechanisms. The increased cerebral blood volume results in cerebral swelling (2.21). Death from raised intracranial pressure usually follows rapidly because of transtentorial brainstem herniation. Animal models, as well as evidence from human cases, demonstrate the extreme rapidity by which these vascular changes can occur (8,28). One human case report demonstrated catastrophic brain swelling within 20 min of a single head impact (28). While there is no doubt that brain swelling may occur in

response to a head injury, the issue of whether recurrent concussive injury is a risk factor for this condition is dis-puted. In the SIS literature, McCrory and Berkovic (15) found 12 reports that clearly describe sport-related cata-strophic brain injury associated with unexplained cerebral

situations largely rests on the interpretation of anecdotal

in boxers should be made. With the likelihood of numer-ous head impacts during a single bout, it makes it almost impossible to determine what is, in fact, a "first" or "second" impact. If SIS is a real entity, the repet impacts in boxing should make boxers much mo ffer from this entity. It is well understood that boxers ffer more frequently from other forms of catastrophic

other sports (14,29,30). Given that significant head impact pisodes occur in virtually every bout fought, why then is IS not more frequently seen in boxing? In a published case series of boxers aged 17 to 24 years

such as subdural hematoma, than athletes in

described as having SIS, there was a variety of diagnostic issues (4). In one case, there was no CT or postmortem performed, so it is uncertain whether death was secondary) stracranial hemorrhage or edema. In two of the case lting in multiple eight counts before the onset of cere edema and death. It is highly probable that any of th vs that triggered an eight count was of sufficient force to e acute malignant cerebral edema, with nvoke the concept of a second impact. Finally, the other two cases both demonstrated brain swelling and subdura

ma on CT. Outside of boxing, only two cases of "probable" SIS exist, one in a 16-year-old ice hockey player and the other in a 17-year-old gridiron football player (15). In the pediatric literature, this same phenomenon has been labeled "malignant brain edema" (2). Additional case series of diffus cerebral swelling in children following a single minor brain trauma have been reported by Snoek *et al.* (23) and Mandera et al. (10,11). This phenomenon may be related to a calcium channel subunit receptor gene mutation (9). Similarly, Cantu and Gean (5) have described 10 cases of a cal

summary, cantu and ucan (2) nave described 10 cases of trenagers playing American tootball (aged 13 to 19 years) who developed a small subdural hematoma and cerebrah edema. In nearly every case, a significant impact to the head preceded the injury, and the outcome was universally poor, with five dead and the other five left with severe neurological deficit. While brain swelling was noted, it is more likely that the magnitude of force required to produce the

subdural hematoma was sufficient to induce cerebral edema, independent of any earlier concussive injury (7).

Can Cerebral Swelling Be Prevented?

The risk factors for posttraumatic acute brain swelli re not understood at present. Our current limited know de not independent of a present our entries independent edge suggests that children and adolescents are at a higher risk, and increased clinical vigilance and perhaps more aggressive investigational strategies may be necessary after ead injuries in this age group. Certainly, the presence of concussive symptoms should be an important factor npting further medical assessment.

symptoms should mandate restriction from furthe sports participation until the symptoms fully resolve. There is a large body of research on the neuropsychological effects f sport-related concussions indicating that the principal ognitive deficits in the postinjury period relate to reduced eaction times and impaired speed of information process-

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of the impairment of cellular energetic metabolism. Evidence from animal and human proton magnetic resonance spectroscopy suggests that metabolic change persists up to 15 d following concussive injury (24–27). While not directly addressing SIS *per se*, it does raise indirect evidence that the possibility of a further concussive injury during this period may result in a more severe injury due to impaired cerebral

Arbitrary exclusion periods based on a fear of a nor existent entity are not the answer in this era of evidence

existent entity are not the answer in this era of evidences based medicine. We need to assess return-to-play strategies prospectively on the basis of symptom resolution and co-nitive recovery, and determine that the outcomes of this approach are safe for the player concerned and appropriate for the sport played (13). (Unit) such studies are performed, the management of concussion should follow the experience of most team physicians who study rear concused ath-letes with a combination of good common sense and clinical the state.

Conclusions

The critical questions are how can we prevent the onset of cerebral swelling and can we predict which children or athletes are at risk of developing this condition and treat them aggressively to reduce the morbidity and mortality? To date, we do not have the answers to these questions. The rarity of this condition of DCS suggests that it is more likely to be due to an underlying genetic susceptibility than simply a response to impact alone. The rarity of this condition also creates a significant barrier to effective research to identify the exact cause. In the interim, it is inherent upon all those involved in the treatment of children following head injury to be aware of the potential for delayed deterioration and to e at their disposal the appropriate facilities and personnel to manage this condition in a timely fashion.

The authors declare no conflict of interest and do not have any financial disclose

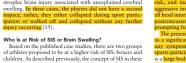
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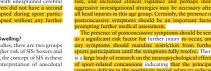
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reaction times and impaired speed of information process-ing (6). Prenature return of a concussed athleft personnably would lead to an increase in injury rates, consequent on the fact that the athlete's ability to respond appropriately to the demands and threats of the sport would be slowed—not an increase in mortality due to the putative SIS.

It also has been proposed that concussive head injury opens a temporary window of brain vulnerability because