'Subconcussive' is a dangerous misnomer: hits of greater magnitude than concussive impacts may not cause symptoms

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Concussion is a traumatic brain injury (TBI) defined by the presence of transient signs and symptoms related to alterations in brain function due to biomechanical force.^{1 2} However, not every such force results in acute signs or symptoms, and recent research seeks to better understand the sequelae of both forces and injuries that are subclinical. The term 'subconcussive' has emerged to refer to both subclinical head acceleration events (HAEs) and injuries (as defined by clinical, biomarker and/or neuroimaging changes). We believe that this term can be misleading in both instances and should be replaced.

When referring to impacts, the prefix 'sub' implies lower magnitude HAEs than those that cause a diagnosed concussion. However, sensor studies show that many HAEs are associated with greater head acceleration than impacts that result in a diagnosed concussion. We suggest replacing subconcussive with the more agnostic term 'non-concussive'.

When referring to injuries, the term subconcussive is a contradiction. If a concussion is a TBI, a subconcussive event implies no injury occurred. However, impacts that

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do not result in diagnosed concussions are sometimes associated with evidence of neurological injury, including functional changes that do not meet criteria for concussive symptoms, biomarker changes and structural changes evident on neuroimaging.³ There is increasing evidence that these injuries are associated with longterm sequelae.4 5 Researchers are misusing 'subconcussive' when referring to these injuries without overt clinical signs or symptoms. For the reasons detailed below, we suggest replacing subconcussive when referring to injury with 'subclinical TBI'.

HITS OF GREATER MAGNITUDE THAN **CONCUSSIVE HAES MAY NOT CAUSE SYMPTOMS**

With the arrival of football helmet accelerometers, researchers hoped to identify HAE magnitude thresholds that would characterise concussion. Unfortunately, there is no such threshold. A study of 319 college football players reported that peak linear and rotational accelerations were not correlated with symptom frequency, severity score or any symptom.⁶ In a study of 283 348 impacts from 185 college football players, Mihalik et al found that the positive predictive value of sensor data for identifying concussion at any threshold was less than 2%, despite conservatively adjusting for undiagnosed injuries.⁷

While concussions tend to be among the hardest impacts sustained, athletes experience HAEs of greater magnitude that do not appear to cause concussion symptoms. Campolettano et al studied 124 youth football players aged 9-14 and analysed the head acceleration experienced by the 15 diagnosed with concussion. They found that for only three (20%), the concussion occurred on the highest linear or rotational magnitude impact the athlete experienced during the study. On average, concussions occurred at the 93.9 percentile linear impact, and 85.2 percentile rotational impact.8 Similarly, Mihalik et al reported that over 4400 non-concussive impacts occurred with greater linear acceleration than for the median concussion. For each concussion diagnosed, there were approximately 340 non-concussive impacts of greater linear acceleration than the median concussive impact.7

Stemper et al studied 511 college football players who experienced 424059 head impacts. They sustained 4589 head impacts with greater linear and rotational acceleration than the mean accelerations for concussed athletes, and 249160 head impacts with greater linear and rotational acceleration than the lowest magnitude 5 concussive impact.9

copyright While the football helmet accelerometers are susceptible to measurement errors and peak linear and rotational acceleration may not best capture concussion risk, these studies highlight that 'subconcussive' impacts are frequently associated with greater head acceleration than concussive impacts. The term 'non-concussive' better captures this range of impacts and uses related to text and data mining reveals that a meaningful proportion of these impacts are 'high acceleration nonconcussive'. More recently, instrumented mouthguards have been developed to quantify head acceleration, and future studies using these devices may further substantiate this claim.¹⁰⁻¹²

HAES WITHOUT SYMPTOMS MAY **RESULT IN INJURY**

There is growing evidence that some non-concussive impacts sometimes cause subclinical, or silent, TBI, which may cause occult deficits and disease. Acutely, HAEs training without symptoms of concussion have been associated with increasing biomarkers of neuroinflammation, suggesting possible neurological injury.³ Imaging studies have demonstrated that repetitive head impacts in the absence of symptomatic concussion are associated with functional impairment and structural damage to the integrity of brain structures including white matter changes, cortical thinning and volume loss.³ Over time, athletes sustaining non-concussive HAEs may also be at higher og risk of concussion.9 13

In the long-term, cumulative exposure to non-concussive impacts may increase the risks of neurodegenerative disease including amyotrophic lateral sclerosis (ALS)⁵ and chronic traumatic encephalopathy (CTE).⁴ Duration of exposure to non-concussive HAEs has been described as being higher in professional football players with ALS compared with those without,⁵ and in predicting both CTE status and severity whereas concussion does not.

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Describing impacts to the head	
Describing injuries to the brain	
ptoms	
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TBI, traumatic brain injury.

If these HAEs have acute and long-term sequelae, they should be identified as separate from, but not necessarily less than, concussion, as the word 'subconcussive' may imply. The observation that some nonconcussive impacts can have clinical implications is unsurprising given the widely accepted evidence that subclinical injury can lead to neurological deficits in other disease processes, such as how some ischaemic events can lead to subclinical cerebrovascular disease that can accumulate and eventually manifest as dementia and cognitive decline.¹⁴ Introducing a 'subclinical' classification to TBI brings TBI in line with other neurological diseases.¹⁵

RECOMMENDATIONS

The term 'subconcussive' should be retired when referring to both impacts and injuries (table 1). We recommend using 'nonconcussive' to describe HAEs that do not result in a diagnosed concussion. Research-todate suggests that while some non-concussive HAEs are harmless, others may cause injury associated with changes in brain function, biomarkers and imaging. The absence of symptoms following an HAE does not indicate that no injury occurred, but rather that no injury occurred to a salient network; it is possible that damage occurred in an area that is not responsible for conscious processes. It is also possible that sensory obtundation has occurred in some individuals exposed to TBI, further weakening the link between TBI and symptoms. In addition, we anticipate there are differences in how individuals respond to the same head impact based on unique differences in brain anatomy, premorbid risk factors and comorbid conditions, which can lead to differences in subsequent injury risk. Moreover, given the wide range of HAE magnitudes and potential negative consequences associated with repetitive concussive and non-concussive HAEs, individual-based metrics considering an athlete's cumulative impact history are important in preventing subsequent injury and long-term sequelae. By changing the nomenclature, we hope to add specificity and clarity to the growing

conversation exploring non-concussive impacts on subclinical TBI and neurological outcomes.

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