The “other” concussion: spinal cord concussion

It was a breezy Sunday afternoon at a football game and the crowd falls silent as they watch the star running back lie motionless after a tackle. The medical team rushed over; the player is conscious and communicating, but unable to feel or move his arms and legs. Over the next fifteen minutes he gradually made a full recovery and decided to return to play.

The term concussion is now part of our general vocabulary. After years of research, we understand that a single concussion may have multiple short term effects - microscopic structural damage to brain cells, local chemical abnormalities that prevent proper brain functioning, and an increased susceptibility to repeat injury. There are also devastating, irreversible long term effects of repeated injury leading to motor and sensory deficits as well as neuropsychiatric and neurological problems, including dementia, Parkinson’s disease, depression, and suicide, respectively. Importantly, the understanding of the risks associated with brain concussion and the potential treatments have been improved significantly with the development of animal models. These models were able to describe the immediate and delayed effects of brain concussion and emphasized the dangers of repeated injury. Subsequent research defined a critical period of increased susceptibility to repeated injury that formed the foundation of medical guidelines governing return to play decisions from the amateur to professional level in contact sports.

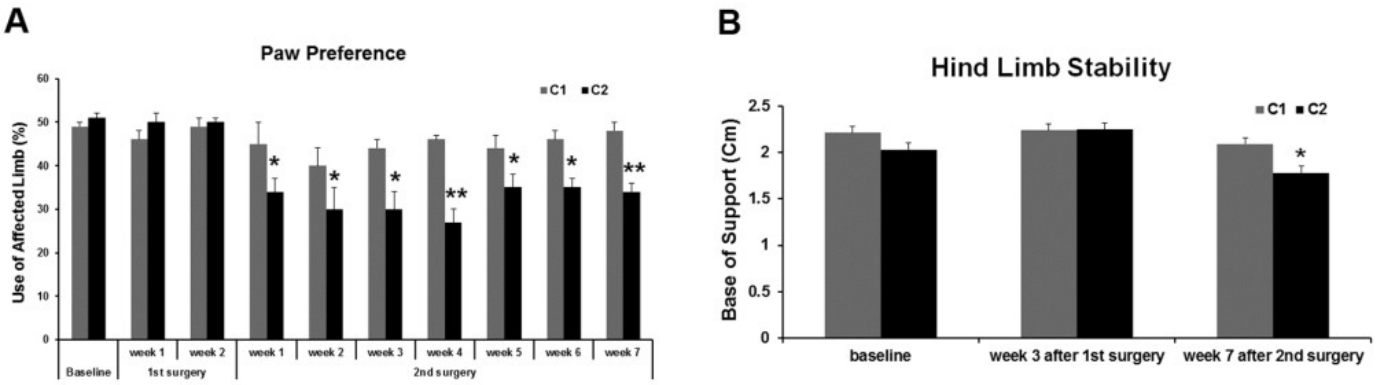

Fig. 1. Repeated spinal cord concussion demonstrates subtle but significant changes in motor function. Initial spinal cord concussion produced minimal reduction in overall motor function that rapidly recovered to normal levels, with no changes in other parameters of movement. Following repeated spinal cord concussion, overall locomotion once again recovered to normal levels, but there were permanent alterations in specific parameters, such as paw preference (A) and hindlimb stability, which occurred after the second concussion (B). C1: single spinal cord concussion. C2: repeated spinal cord concussion. Figures adapted from Jin et al., 2015, Exp Neurol 271:175-188.

Yet, the injury scenario described above refers not to the widely studied brain concussion, but to

the practically unrecognized “other concussion,” spinal cord concussion. Spinal cord concussion is a transient injury characterized by impaired sensory (numbness or tingling) and motor function (weakness or complete paralysis) of single or multiple limbs. Symptoms typically resolve within twenty-four hours, with no residual deficits. However, patients have reported multiple episodes of spinal cord concussion and it has been suggested that such repeated spinal cord concussions not only result in progressive neurological damage and increased time to recovery in the short term, but also long-term neurological damage, analogous to brain concussion. Nevertheless, concussion of the spinal cord remains poorly defined and understood, and this lack of understanding, coupled with infrequent reporting and a scarcity of information on the immediate and long-term consequences of single or repeated spinal cord concussion, has led to a lack of consensus within the field regarding medical clearance for return to play.

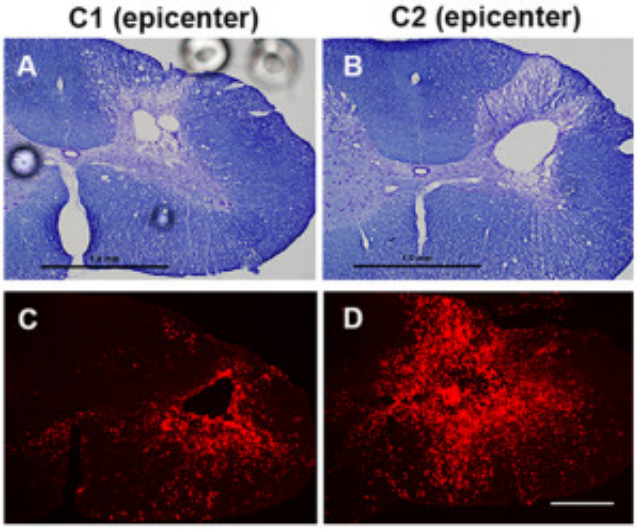

Fig. 2. Spinal cord concussion does not alter tissue architecture (A, B), but induces inflammatory cell influx (C, D). Panels A and B show minimal changes of spinal cord structure after single and repeated concussion. Panels C and D show that inflammatory cells accumulated at the injury center with more cells in repeated concussion rats (C2) than in single concussion rats (C1). Figures adapted from Jin et al., 2015, Exp Neurol 271:175-188.

To address this issue our group developed an animal spinal cord concussion injury model. Similar to the clinical scenario, our model demonstrated complete and rapid sensory and motor recovery following an initial spinal concussion. A second injury, incurred following full recovery, however, demonstrated significant changes in paw preference (Fig. 1A), as well as more subtle changes in fine motor function (Fig. 1B), despite maintenance of overall locomotion. These changes were associated with an increased inflammatory cell response, despite preservation of normal tissue structure (Fig. 2). Taken together, this suggests that the critical time period for progressive functional deficit extends beyond full locomotor recovery from an initial injury.

In conclusion, we have developed a model to study the consequences and risks of single and repeated spinal cord concussion, an underappreciated entity with significant medical implications. Our work demonstrated that a second spinal cord concussion, incurred following full recovery from an initial cord concussion, resulted in permanent functional deficits despite preserved overall locomotion and further highlight the need for additional studies to define the temporal parameters of spinal cord concussion.

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