Letter to the Editor

Mechanisms and pathophysiology in ‘stingers’ and brachial plexus injury

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We write to thank Windmueller and colleagues for their narrative review of brachial plexus injuries in contact athletes (1). The topic is of genuine clinical relevance, particularly given the identified frequency of “stingers” in collision sports and the sporadic but devastating occurrence of degenerative nerve injury and avulsion. We have treated cases ranging from brief recovering non-degenerative injuries to surgically managed five-root avulsions. The authors’ efforts in synthesising available literature highlight an important and often misunderstood area of neurological and sports-medicine practice.

We recognise that the approach and impact of a narrative review is limited and explicitly involves the authors’ selection judgment and biases. Accepting this gap in the literature we see value in a formal scoping review with formal PRISMA-ScR methodology and have registered such a project.

We agree on their point that the term “stingers”, or the potential direct synonym “burners”, encompasses a range of underlying pathologies, and join the authors in calling for improved awareness and research into risk factors, biomechanics, and long-term outcomes. However, we wish to raise several methodological and conceptual concerns which we believe are important for the academic record and for the clinical community.

We agree with the authors’ description of the principal mechanisms of brachial plexus injury: traction leading to strain of the brachial plexus (typically from lateral flexion of the cervical spine away from the depressed ipsilateral shoulder), compression within the costo-clavicular or scalene region, and direct blow to Erb’s point (1). These mechanisms usually produce transient neurological symptoms, while higher-energy collisions can cause more severe, axonal injury. However, we emphasise that similar clinical features can also result from injuries to one or more exiting cervical nerve roots, or even the spinal cord itself. Some cases may therefore represent central, rather than peripheral nerve injury. These injuries are often compressive rather than tractional, and the mechanisms can often be inferred from eyewitness accounts, player descriptions, or from video footage. Recognising this distinction is essential to avoid misclassifying central injuries as peripheral

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'stingers'.

Next, we express concern about the paper's attribution of pain and positive sensory phenomena to "neuropraxia* (sic)" (the correct term is neurapraxia, not neuropraxia) or conduction block (syn.). Spontaneous and evoked pain, paraesthesia, and dysaesthesia are cardinal signs of neuritis—mechanical, inflammatory, or ischaemic—and cannot be generated by a classical, non-degenerative neurapraxia/conduction block. A true neurapraxic lesion, as defined by Seddon (2), produces failure of action potential propagation without axonal injury or ectopic discharge. This is incapable of generating the positive sensory symptoms described in stingers. By contrast, the transient burning and radiating pain reported by athletes reflects neural irritation—a neuritis—induced by tensile strain, focal compression, or perineurial distortion or insult. Conflating these entities risks reinforcing a long-standing conceptual error, in which the presence of pain is mistakenly assumed to signify a benign neurapraxia, when in fact it suggests axonal impairment or microinjury.

This leads to a broader conceptual concern: the paper intermittently treats single-event conduction block (neurapraxia), persistent conduction block, axonotmesis, root-level avulsion, and traumatic or inflammatory neuritis as a single continuum and not as discrete clinical entities. While differentiation can be challenging in the early hours following impact, these injuries represent distinct biological entities with discrete prognoses, electrophysiological signatures, and timelines. The current literature already suffers from conflation of these pathologies; repeating these assumptions risks perpetuating diagnostic oversimplification. The field urgently needs greater emphasis on the distinction between transient physiological failure of conduction, mechanical neuritis, and structural axonal disruption, and collective exploration of mechanisms predisposing to injury after prior trauma.

We suggest that a pitch-side biomarker for axonopathy may be a useful tool for this in the future to guide assessment and rehabilitation. Experimental and clinical data from peripheral nerve research outside the sports-medicine setting indicate that repeated strain events may result in changes to the biomechanical properties of nerves. Fibrosis can stiffen the paraneurial and interfascicular connective tissues, causing adhesions, reducing nerve compliance and increasing vulnerability to subsequent mechanical loads (3,4). The possibility that prior stingers amplify susceptibility to later, more severe axonal injury deserves explicit discussion. This is particularly relevant in collision sports, where cumulative microtrauma is common.

Finally, another important omission concerns anatomical predisposition. There is discussion within the neurogenic thoracic outlet syndrome (nTOS) literature that congenital variants such as cervical ribs, elongated C7 transverse processes, fibrous bands, aberrant scalene insertions, and narrowed costoclavicular corridors substantially alter the mechanical environment of the brachial plexus (5-8). These variants are well-recognised contributors to symptomatic nTOS in adolescents and adults. It is plausible that similar variants predispose certain athletes to stingers or to more severe traction injuries under loads that would not injure others with more favourable anatomy. The review omits this, yet it likely explains why similar forces produce widely different clinical outcomes between players. We believe that future research should aim to determine whether these anatomic factors modulate risk in contact athletes and whether imaging or screening strategies might one day refine return-to-play or risk-mitigation protocols.

Taken together, these issues suggest that the review's mechanistic conclusions, particularly those concerning return-to-play, should be interpreted with caution. By relying on traditional terminology without clarifying underlying mechanisms, this paper risks inadvertently perpetuating misconceptions, particularly the association of pain with neurapraxia and the assumption that all transient neurological symptoms are benign. While we commend the authors for highlighting the clinical spectrum of brachial plexus injuries, a more nuanced framework is required—one that distinguishes neuritis from conduction block, recognises axonotmesis as a fundamentally different phenomenon, and accounts for cumulative strain effects and anatomical variation. We encourage continued dialogue and collaboration; clearer definitions, biologically grounded models, and methodological transparency will guide future research and clinical practice.

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