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Mechano-electrophysiological model for nerve tissue in TBI and SCI

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Traumatic brain injury (TBI) and spinal cord injury arise from complex mechanical loading scenarios such as fall accidents or impact events during sports among others. Such injuries alter the functional behaviour of the nervous system, resulting in a poor overall electrophysiological conduction [1]. The mechanisms responsible for this reduction in the conduction are poorly understood. Previous studies have focussed on spinal cord and white matter tissue, as well as on axons under tensile loading, relating the impaired electrophysiological conduction to axonal stretch and strain rate, while ignoring the effect of other loading modes. Additionally, these studies are mainly focussed on the cellular level and coupled with subcellular axonal electrophysiological models (usually based on the Hodgkin-Huxley theory) [2]. These approaches, although essential for the understanding of the problem, present a lack of applicability to real scenarios where scaling-up to the tissue level is required in a more general loading framework. To this end, this work presents a mechano-electrophysiological model that translates features from the mechanics of brain/spinal cord tissue to electrophysiological dysfunction, eventually cascading to cognitive deficits at the tissue level. A mechanical constitutive model is developed for axonal-based tissues in finite deformations and accounts for strain rate dependency, anisotropy due to axonal orientation, viscous effects and continuum mechanical damage within a thermodynamically consistent framework. In parallel, a FitzHugh-Nagumo based model that includes anisotropic conduction following fibre orientation is developed to describe the electrophysiological behaviour. Both mechanical and electrophysiological models are then coupled through energetics terms to define the resulting reduction of the conduction arising from mechanical deformation. The coupled model is calibrated and validated against experimental tests covering a wide range of strain rates for white matter and spinal cord [3,4]. Finally, the resulting electrophysiological dysfunction are analysed in the context of TBI.

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