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Chronic traumatic encephalopathy: Is a role for biomarkers premature?

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Chronic traumatic encephalopathy, previously known as dementia pugilistica, has been in the medical literature since the 1920's. It is characterized clinically by a diverse array of neuropsychiatric symptoms, and pathologically by variable degrees of phosphorylated tau accumulation in the brain. The evolving paradigm for the pathogenesis of CTE suggests that concussion or subconcussion initiates a cascade of pathologic events, encompassing neuroinflammation and possibly protein templating with trans-synaptic neurotoxicity. The end result is neurologic and neurobehavioral deterioration, often with self-harm. While these concepts warrant further investigation, the available evidence permits no conclusions as regards the pathogenesis of the reported findings. The role of concussion or subconcussion (if any), whether and how the condition progresses, the extent of phosphorylated tau in clinically normal athletes, the role of phosphorylated tau as a toxic species versus an inert disease response, and whether protein templating has any *in vivo* relevance, are all unclear. As such, any suggested role for biomarkers in diagnosis or otherwise assessing this putative entity is unclear.

Biography

Rudy J Castellani is Professor of Pathology and Director of Neuropathology at the University of Maryland in Baltimore. He has published extensively on the pathology and pathogenesis of neurodegenerative diseases and has received numerous awards for scholarship and teaching, including the 2010 Alzheimer medal and the 2011 Mark A. Smith award for excellence in neuroscience.

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