MILD TRAUMATIC BRAIN INJURY: ABSENCE OF PROOF IS NOT PROOF OF ABSENCE

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Technological advances in neuroimaging techniques have revealed that mild traumatic brain injury (MTBI) can result in subtle changes in the physiology of the brain.[1 2 3]

Researchers have clearly demonstrated that whiplash type, mechanical acceleration-deceleration injuries in primates produce observable brain damage.[4 5 6 7] Similar brain damage has been observed in humans. Oppenheimer examined the brains of individuals who sustained minor to severe head injuries who died within several days of causes unrelated to the head injury. He found evidence of microscopic brain damage in cases where the cerebral injury consisted of a concussion. Oppenheimer concluded:

> Following a head injury, diffuse microscopic lesions can be seen in a high proportion of human brains. ...They are believed to be mechanical in origin, and can be attributed to:
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> (1) surface sheering and contusions;
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> (2) stretching and tearing of small blood vessels;
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> (3) stretching and tearing of groups of nerve fibres;
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> (4) tearing of nerve fibres by a crossing vessel.
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> They are seen, not only after severe trauma, but also in cases of "concussion". Detailed studies of their sights and distribution could throw light on the mechanics of acceleration injuries of the brain.[8]

In a 1993 article "Mechanisms of mild traumatic brain injury" published in the J*ournal of Head Trauma Rehabilitation*, neurosurgeons from the Department of Neurosurgery at the University of Texas discussed the neuropathological mechanisms of diffuse axonal injury:

Axonal injury

Clinical studies suggest that diffuse axonal injury may be a factor in mild to moderate TBI in humans. These neuropathologic studies suggest a continuum of diffuse axonal injury severity in which the lesions are mechanical in origin and are caused by stretching and tearing of nerve fibers and small blood vessels. This postulated continuum of severity is also supported by neuropathologic studies in animals with experimental mild head injury. Experimental data suggest two viewpoints regarding the onset and development of axonal injury. It may be that axonal injury occurs at the moment of trauma by the shear or tensile forces physically disrupting the axons, leading to membrane retraction, extrusion of axoplasm and formation of large reactive swellings. On the other hand, it may be that a subtle and progressive axonal change after mild TBI eventually leads to swelling of axons and disruption of the neuraxis without initial tearing of the axons at the moment of trauma.

Substantial clinical and experimental evidence exists to support the theory that axons are consistently damaged in head injuries ranging from minor to severe. Until recently, investigators have focused primarily on the biomechanical aspects of axonal injury and have suggested that axons are almost preferentially vulnerable to mechanical forces generated with head injury. Gennarelli et al, in their early studies of experimental head injury in nonhuman primates, argued that diffuse axonal injury is the principal morphologic feature of TBI. Findings by Jane et al suggest that in some instances minor head injury or concussion can be associated with axonal injury. In animals sustaining an acceleration-deceleration non-impact injury, degenerating axons were noted in the inferior colliculus, pons, and dorsolateral medulla at 7 days after TBI. The finding of immediate mechanical axonal damage was based upon the identification of axon retraction balls, which are believed by most to form through the physical shearing or tearing of axons with subsequent retraction, extrusion of axoplasm, and formation of large reactive swellings.

Although mechanical forces are necessary to produce axonal injury, such injury may result from a biochemical cascade of pathophysiological events initiated by the mechanical events rather than formed at the moment of injury. Povlishock et al demonstrated that, as in the case of severe and moderate head injuries, mild head injury results in the genesis of reactive swellings within 12 to 24 hours of the traumatic episode. When comparably injured animals were examined before 12 hours posttrauma, however, no reactive swellings were found. To assess better the significance of such an apparent discrepancy, detailed light and electron microscopic studies were conducted to evaluate the intra-axonal anterograde transport of horseradish peroxidase over a 24-hour posttraumatic course.

> Through such an approach, it became apparent that the traumatic event did not tear or shear the axons to form retraction balls immediately but rather induced an initial, subtle form of axonal changes that then, over time, became progressively severe. Recent data suggest that axonal injury in humans is an evolutionary process.[9]

In cases of MTBI the results of neurodiagnostic testing are usually negative. This is because the standard tests such as computerized axial tomography (CT) and magnetic resonance imaging (MRI) depict brain structure and lack the resolution to visualize the damage which occurs in MTBI. In the last TBI Update I discussed the evidentiary role of positron emission tomography (PET) which reflects brain function by showing metabolic activity in different areas of the brain.

Another promising technological tool that may eventually prove useful in the assessment of MTBI is Quantitative Electroencephalogram (QEEG). Dr. Nathan D. Zasler has written a paper "QEEG and Mild Traumatic Brain Injury" which examines the current state of this diagnostic tool. Dr. Zasler is a physiatrist with extensive experience in TBI cases. He is internationally recognised as a leader in the field of neurorehabilitation and has published numerous articles on TBI. He is co-editor of the texts *Rehabilitation of Post-Concussive Disorders* published in 1992 and *Medical Rehabilitation of Traumatic Brain Injury* published in 1996. In an excellent review article "Mild Traumatic Brain Injury: Medical Assessment and Intervention", Dr. Zasler made the following observation:

> **Clinicians should remember that gross absence of proof is not necessarily proof of absence.** In unsophisticated hands there may be no evidence whatsoever that someone has had a significant injury, whereas in different hands and to other eyes the patient may indeed have objective examination findings clinically as well as neurodiagnostically. Awareness of current advances in neurodiagnosis, including neuropsychological assessment, and in rehabilitative treatment is of paramount importance in providing adequate care to patients with postconcussive symptomatology. Of critical importance also is a familiarity with the current literature on neurologic, rehabilitative, psychological, and neuropsychopharmacologic approaches to care of this special patient population. Educating the survivor of MTBI and significant others regarding symptomatology, treatment options, and prognosis is just as critical a role for the physician as performing the more generic neuromedical interventions. Care should be

> rendered in an interdisciplinary fashion and as early as possible to optimize patient/family understanding of the condition as well as to maximize neurologic and functional outcome.[10][emphasis added]

Dr. Zasler's paper on "QEEG and Mild Traumatic Brain Injury" is reproduced with his permission.

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