

Keynote Lecture

The Centripetal Theory of Concussion (CTC) revisited after 40 years and a proposed new Symptomcentric Concept of the Concussions

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Abstract This review asks the research question, “is the centripetal theory of concussion still valid today?” and works to answer the hypothesis that yes it is. To do so the methods include: 1) a review of the concept of CTC, 2) a review the pertinent literature, 3) applying the CTC to current biomechanics and pathophysiology and 4) proposing new concepts of concussion to deal with the changing nature of the contemporary definitions of concussion. The conclusion is that, although the concepts of the CTC remain valid after 40 years, the broadening of the definition of concussion requires that a wider perspective be taken regarding the term “concussion.” Currently, the term “cerebral” concussion for which the CTC was originally construed is being replaced by virtually any symptom arising after head motion whether it arises from the cerebrum or not. Thus, a Symptomcentric Concept of the Concussions is proposed whereby symptoms from various sites arise in response to a mechanical stimulation of the brain or other individual anatomic sources.

Keywords concussion, centripetal theory, biomechanics of head injury

I. INTRODUCTION AND REVIEW OF THE CENTRIPETAL THEORY OF CONCUSSION (CTC)

Presented in part at the first IRCOB Conference in 1973 [1] and published fully in 1974 [2], Ommaya and Gennarelli proposed the centripetal theory of concussion (CTC). This was proposed as:

- A hypothesis for cerebral concussion would then be defined as *a graded set of clinical syndromes following head injury wherein increasing severity of disturbance in level and content of consciousness is caused by mechanically induced strains affecting the brain in a centripetal sequence of disruptive effect on function and structure. The effects of this sequence always begin at the surfaces of the brain in mild cases and extend inwards to effect the diencephalic-mesencephalic core at the most severe levels of trauma.*
- It is suggested that rotational components of accelerative trauma to the head produce a graded centripetal progression of diffuse cortical-subcortical disconnection phenomena which is always maximal at the periphery and enhanced at sites of structural inhomogeneity.

Slightly restated, the CTC results from strain in the brain:

- The direction, type, rapidity and magnitude of head motions will determine the strains in the brain
- A particular set of input variables will produce a particular and unique “strain field/time profile”
- Certain “strain-field/time profiles” will result in clinically apparent symptoms (phenotypic presentation) and pathological alterations

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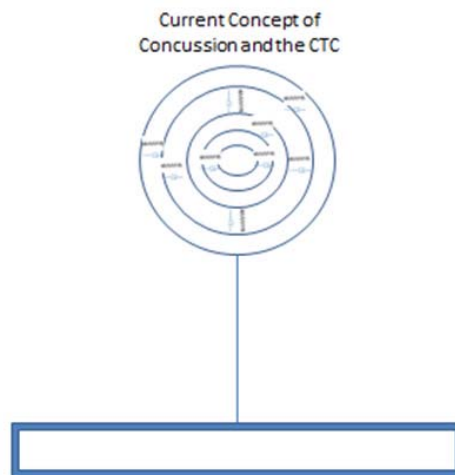


Fig. 1. Concentric circles of decreasing strain as depth increases, connected by various springs and dashpots.

II. LITERATURE REVIEW REGARDING THE CENTRIPETAL THEORY OF CONCUSSION

Evidence in favor of the CTC from a literature review shows that the CTC paper [2] is the third most cited paper on concussion since its publication in 1974, that it has been cited by 876 scientific publications to date, that it is one of four concussion papers cited more than 800 times and that it is one of 15 concussion papers cited more than 500 times [3]. In contrast, of all concussion papers, only 27% have more than 10 citations [3].

In the four decades since the CTC publication, it has had increasing visibility in the scientific literature, being cited 98 times from 1974-1984, 225 times from 1985-1994, 232 times from 1995-2004 and 321 times from 2005-2015.

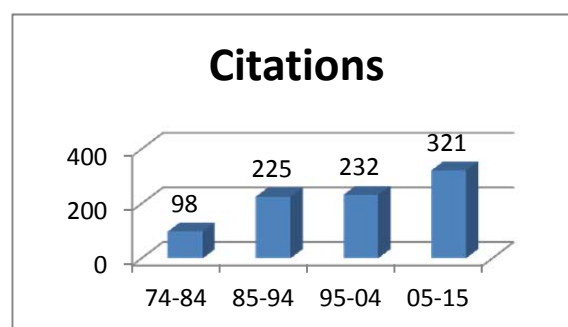


Fig. 2. Citations of the CTC paper [2] each decade since its publication in November 1974.

Almost all of these citations support the CTC hypothesis [e.g. 4].

Only a very few detractors of the CTC have been published:

- McCrory in a short piece in 2001 [5] opined that the absence of structural cortical pathology in concussion speaks against the validity of the CTC. He prefers brainstem disturbance and some cortical pathology as the mechanism. He clearly did not completely understand the concept of the CTC which does not exclusively involve (visible) damage (e.g. pathology) but rather a “disruptive effect.”
- Shaw in a book chapter in 2006 [6] reviewed the extant theories of causes of concussion including: 1) the Vascular theory where brief ischemia and decreased cerebral blood flow prevail, 2) the reticular theory where the brainstem ascending reticular activating system dysfunction produces LOC, 3) the CTC, 4) the Pontine Cholinergic System theory where activation of inhibitory systems in dorsal pons are causal, 5) the Convulsive theory where the symptoms of concussion are akin to those of seizures: e.g.

excitatory. He concluded that the CTC is “ambitious, ingenious but ultimately flawed” without further significant comment.

III. CURRENT BIOMECHANICS AND PATHOPHYSIOLOGY AND THE CTC

Contemporary head injury classification and the biomechanics of concussion use the term diffuse brain injuries for what the CTC called the “*graded set of clinical syndromes...of concussion.*” This terminology is reflected in the recent AIS, and ICD-10 and ICD-11 classification systems [7-9]. The diffuse brain injuries have been shown to correlate with their biomechanics [10] and with their clinical outcomes [11].

The factors that influence the strain pattern produced in the brain by various input conditions are numerous and include 1) the anatomy of neuronal soma, axons, synapses, dendrites and networks and the instantaneous centers of rotation that occur, 2) the homogeneity of axonal directions, 3) the numerous tissue density gradients, and 4) the physics of the input including the amplitude, type and duration of stress. So, it is understandable that the cerebral cortex apparently shows less structural damage than deeper structures because it is mostly soma and dendrites not axons, the axons present are multi-directional, the ultra-microscopic structural changes that may be present are difficult to demonstrate or simply that the cortex is more resistant to strain. These have been dealt with in our own presentations at IRCOBI which support the CTC [12, 13].

IV. CONCLUSION

So, despite 40 years of scrutiny, despite a few detractors, despite 40 years of newly developed TBI knowledge, the Centripetal Theory of Concussion (and diffuse brain injury) remains viable and soundly based in contemporary biomechanics and pathophysiology.

A new proposal: the symptomcentric concept of the concussions

However, despite the validity of the CTC, new concepts of concussion are necessary to deal with the changing nature of the contemporary definitions of concussion. No longer is “concussion” the same as “cerebral concussion” or “commotio cerebri” because many symptoms currently ascribed to “concussion” or its (more) confusing moniker mild traumatic brain injury (mTBI) are arguably not of cerebral or brain origin. These include headache, dizziness, seeing “stars”, tinnitus, fuzzy or blurred vision, fatigue, neck pain, photophobia, taste or smell disorders, sensitivity to noise, etc. Currently, the term “cerebral” concussion for which the CTC was originally construed is being replaced by virtually any symptom arising after head motion whether it arises from the cerebrum or not. Thus, a **Symptomcentric Concept of the Concussions** is proposed whereby symptoms from various sites arise in response to a mechanical stimulation of the brain or other individual anatomic sources.

It is therefore envisioned that mechanical input can lead to a response in various structures. These structures are thus “concussed” if the response (or symptoms) that results is of a temporary nature (more or less). Thus, the following structures which will be discussed can respond to mechanical energy in a concussive manner:

- **Brain Concussion:** posttraumatic symptoms arising from the brain such as loss of consciousness, confusion, or amnesia. Within the brain, there has been undue emphasis on the axon as “the” locus of mechanically-induced dysfunction. While this may be true, especially at the more severe end of the injury spectrum, it may not be the case is less severe injury. Thus, more broadly, “concussion” can occur and symptoms be generated, not only by dysfunction of the axon and other parts of the neuron (soma, mitochondria, dendrites, synaptic networks, etc.) but also by mechanically induced dysfunction of vascular (causing symptoms due to vasoconstriction or vasodilatation of arteries, vein, capillaries), oligodendrocytic (symptoms from demyelination or altered electrical conduction), astrocytic (symptoms from gliosis) or microglial (symptoms from inflammatory processes) components in single or multiple portions of the brain, not just the cerebrum.
- **Olfactory concussion** [14]: posttraumatic symptoms arising from the olfactory nerves, bulbs or tracts such as diminished or exaggerated smell.

- Retinal concussion [15]: posttraumatic symptoms arising from retinal motions or from traumatic alterations of the electroretinogram such as diminished, dim or “fuzzy” vision, photophobia or visual aberrations.
- Trigeminal concussion [16]: posttraumatic symptoms arising from stimulation or depression of the branches of the trigeminal nerve such as headache, facial pain or numbness.
- Vestibular concussion [17]: posttraumatic symptoms arising from semicircular canal dysfunction such as dizziness, balance problems, lightheadedness.
- Auditory Concussion [18]: posttraumatic symptoms arising from cochlear dysfunction such as hyper or hypoacusis, sensitivity to noise.
- Cervical concussion [19]: posttraumatic symptoms arising from the nerves, muscles, joints, ligaments or blood vessels in the neck such as neck pain, numbness/pain in posterior portion of head, lightheadedness.
- Spinal concussion [20]: posttraumatic symptoms arising from the cervical spinal cord such as tingling, numbness, weakness.
- Psychological Concussion [21]: posttraumatic symptoms arising from the influence of mechanical energy on one’s overall psychological state. This is a more abstract “injury”, the magnitude and expression (symptoms) of which depend on not only the magnitude of the mechanical input but also on the pre-existing personality “strength”.

It follows these concussions may occur in any combination and that although angular head motions have been described as casual for the CTC and classical cerebral concussion [2, 22, 23], this may or may not be the case for the other varieties of Symptomcentric Concussions. Investigation into the specific mechanical stimuli that produce these Symptomcentric Concussions would be welcomed.

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