

The Stereotactical Phenomena in Traumatic Brain Injury Biomechanics - diffuse axonal injury and brain concussion (1)

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ABSTRACT

Traumatic Brain Injury (TBI) is the consequence of the spatiotemporal pressure variations occurring inside the brain during head traumas. The spatial distribution of the pressure gradient (PG) is responsible for the cerebral lesions' localisation and the consequent neurological signs. Beside skull's deformation caused by the contact loading and determining skull vibrations and/or fractures, current biomechanical theories concern two inertial phenomena: the linear acceleration and the rotational head movements. The first theory explains the superficial brain lesions and is widely accepted. The second theory better explains the deep cerebral lesions and the concussion mechanism but is still controversial. The stereotactical approach here exposed mainly considers the approximately spherical shape of the interface skull-brain. The skull-brain relative movements, caused by the acceleration phenomena - linear or rotational - and by the skull vibrations, generate secondary pressure waves with approximately spherical wave fronts that concentrically propagate toward the deep cerebral structures. The wave front's spoke and its surface progressively decreases. According to the energy conservation law, the amplitude of the pressure waves progressively increases. Thus, the PG will be maximal in the geometrical centre of the implied skull vault segment. The stereotactical phenomena can explain common posttraumatic neurological signs and cerebral lesions and is compatible with previously reported experimental findings. Its complementarity with the other biomechanical theories could allow us to integrate the TBI biomechanics in a common concept in order to better understand the TBI pathophysiology and also related pathological entities like boxers' chronic encephalopathy or even Alzheimer's disease. Further experimental and especially human observational research on the TBI biomechanics is needed.

INTRODUCTION

Traumatic brain injury (TBI) is the main cause of death for patients less than 45 (1). TBI biomechanics explores the mechanical phenomena that cause the initial cranio-cerebral lesions and thus represents the starting point for the overall understanding of the TBI pathophysiology. TBI is the consequence of the spatiotemporal pressure variations occurring inside the brain during head traumas. The spatial distribution of the pressure gradient (PG) is responsible for the tissue strains (compression, tensile, shear), the cerebral lesions' localisation and the consequent neurological signs (2). Beside skull's deformation caused by the contact loading and determining skull vibrations and/or fractures, current biomechanical theories concern two inertial phenomena: the linear acceleration and the rotational head movements. The first theory explains the superficial brain lesions. The second theory seems better explain the deep cerebral lesions and the concussion mechanism but is still controversial (3). Here is exposed a new biomechanical approach that can explain the deep cerebral lesions and the common neurological signs observed after human head traumas.

Traumatic brain lesions

Focal and diffuse cerebral lesions are currently described. The focal lesions, also called cerebral contusions, are haemorrhagic and are visible on frequently performed radiological exams as the X-ray computed tomography. The focal lesions are often located in the superficial brain structures, close from the skull, but sometimes deep cerebral hematomas occur. The focal lesions always coexist with different degrees of diffuse cerebral lesions, also called "diffuse axonal injury" (DAI). The DAI is concentrated in the deep cerebral regions and is not visible on the radiological exams. Meanwhile, in accordance with its severity, the DAI is mainly responsible of various degrees of consciousness disturbances and further clinical outcome (2). The clinical entity corresponding to the rather pathological sense of the DAI term is the brain concussion.

CURRENT BIOMECHANICAL THEORIES

The linear acceleration theory was first evoked about one century ago. Relative movements and secondary impacts occur between the skull and the brain during a head impact. The pressure increases in the superficial cerebral structures below the impact zone, proportionally to the head linear acceleration (4). This theory explains the superficial cerebral lesions' occurrence. It cannot explain the preferential DAI localisation in the deep cerebral structures (8), neither the traumatic deep cerebral hematoma. It cannot explain neither why the loss of consciousness and the memory troubles are the most frequent clinic signs occurring after head trauma - despite the fact that the responsible cerebral structures are deeply located.

According to the rotational movements' theory evoked in 1943 by Holbourn (5), DAI and deep cerebral hematoma are caused by the tensile strains occurring between superficial and deep cerebral structures during the head circular movements. In a large series of experiments on primates, Thibault and Gennarelli particularly supported the role of the rotational movements in the DAI occurrence (6).

The consequences of the skull's vibrations are poorly understood. It is probable that the low-frequency skull vibrations (below 200 Hz) mainly cause deep cerebral lesions, while high-frequency vibrations have more consequences on the superficial cerebral structures (7).

In real life head trauma all these phenomena coexist. In the mean time, DAI and brain concussion also occurred in pure linear accelerated experimental head trauma (5) even if under the current approach, the linear acceleration theory cannot explain how deep cerebral structures can be injured while superficial cerebral structures are respected.

THE STEREOTACTICAL THEORY

The stereotactical approach considers the geometrical shape of the interface skull-brain, the close interactions between the two structures during their relative movements and the resultant pressure waves propagation.

The shape of the interface skull-brain is approximately spherical. The skull-brain relative movements, caused by the acceleration phenomena - linear or rotational - and by the skull vibrations, generate secondary pressure waves with an approximately spherical wave front. Because the brain tissue is isotropic on concentric plans, the wave propagation velocity toward the deep cerebral structures is spatially homogenous.

$$C = (E/r)^{0,5}$$

C = wave propagation velocity; E = resilience; r = density

The spherical shape of the wave front is thus conserved. Its spoke and its surface progressively decrease. Despite attenuation phenomenon and according to the energy conservation law, the amplitude of the pressure waves, and thus the pressure gradient, progressively increases toward the deep cerebral structures. It will be maximal in the geometrical centre of the implied skull vault segment (figure 1), particularly if no significant energy consumption process occurs in the superficial cerebral structures before. If such a superficial cerebral contusion occur, a pressure wave "shadow cone" is delimited towards the deep cerebral structures and thus the stereotactical summation phenomena are partly perturbed.



Figure 1: The stereotactical concept illustrated on a sagittal MRI view

In low or medium-energy impacts, the skull vibrations have a significant role by generating successive wave fronts. Cumulative effects related to the temporal summation phenomena thus add to the spatial (stereotactical) ones.

In high-energy impacts, the acceleration phenomena are predominant. Because of the skull fractures that often occur, the skull vibrations are perturbed and their stereotactical consequences reduced. In the mean time, the high acceleration effects diminish the relative importance of the skull vibrations' consequences.

DISCUSSION

The stereotactical phenomena explain common neurological signs

The stereotactical phenomena explain why the initial and reversible loss of consciousness (IRLC) is the most common posttraumatic neurological sign, even if the involved structure - the ascendant reticulate matter (ARM) - is placed in the deep cerebral regions. It can also explain why, after the IRLC, most patients don't have any focal neurological deficit (motor, sensitive or visual) related to the superficial cerebral structures' lesions or functional impairment. The functional recovery is faster for the neuronal circuits with less infrastructure lesions and if the superficial lesions were more important than the deep ones, the recovery of the focal deficits would be longer than the recovery of the consciousness.

The stereotactical phenomena also explain the high incidence of the memory disturbances after a head trauma as the result of the periventricular neuronal circuits' functional impairment or lesions.

At our best knowledge, this is the first theory to explain these clinical phenomena.

The isolated functional impairment of the ARM could also be explained by the fact that the consisting neuronal fibres are less resistant because nonmyelinated. This argument is not applicable to the myelinated neuronal fibres whose functional impairment or lesions generate the frequent posttraumatic memory disturbances.

The stereotactical phenomena explain the deep cerebral lesions

The stereotactical approach can also explain the preferential localisation of the DAI in corona radiata, corpus callosum, fornix and upper brainstem (8). These anatomical regions correspond to geometrical centres of different skull vault segments.

Deep traumatic cerebral hematomas can also be better understood as they occur close to the geometrical centre of the skull vault.

The stereotactical phenomena explain the importance of the contrecoup cerebral lesions

The stereotactical phenomena explain why the cerebral contrecoup lesions (CCL) are often more important than the direct lesions (DL). The bend spoke of the lateral skull segment is approximately equal to the bi-parietal skull diameter. According to the stereotactical theory the CCL are the consequence of the pressure waves' stereotactical summation toward the geometrical centre of the implied skull vault segment. Thus, up to a critical energy lateral impact, the CCL occur before the DL.

The stereotactical phenomena explain why the cerebral contusions (direct or contrecoup) are often located in the temporal and the frontal lobes. These cerebral regions correspond to small bend spoke segments of the skull and contain their geometrical centre - where maximal stereotactical summation occurs.

The stereotactical phenomena are supported by previously reported experimental data

In older experiments Gurdjian noted significant differences between the DAI localisation after head impacts on primates and dogs. The primates' brains had predominant DAI in the upper brainstem while the dogs' DAI was concentrated in the lower brainstem regions (9). These differences are unexplained by the current biomechanical theories, but they can be easily understood according to the stereotactical approach by the difference between the shape of the animals' skull vaults and their geometrical centre localisation. According to the stereotactical approach, the DAI is concentrated in the geometrical centre of the skull vault and, as the primates' skull vault is more incurved, their geometrical centre is placed higher than the dogs' one.

Other stereotactical phenomena

The radiosurgery uses the stereotactical principle in order to avoid massive irradiation of the superficial structures when a deep cerebral lesion (tumour, malformation) has to be irradiated. The total irradiation doze, aimed to destroy the deep lesion, is spatially fractionated in many convergent beams. Thus, the superficial structures receive only small fractions of the total irradiation doze and are preserved.

Sounds are also pressure waves. Microphones with parabolic reflective surfaces and the concert halls' architecture use the stereotactical principle in acoustic applications.

CONCLUSION

The stereotactical phenomena can explain common posttraumatic neurological signs and cerebral lesions. Its complementarity with the other biomechanical theories could allow us to integrate the TBI biomechanics in a common concept in order to better understand the TBI pathophysiology and also related pathological entities like boxers' chronic encephalopathy or even Alzheimer's disease (10). Further experimental and especially human observational research in TBI biomechanics is needed.

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First published : Neurochirurgia Iasi (ISSN 1454-5446) - Vol.1,Supl.,pp.197-201,1999
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