



Mini-Review

CURRENT CONCEPTS ON MORPHOGENESIS OF CRANIOCEREBRAL INJURIES

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ABSTRACT

Based on literature review the current concepts on morphogenesis and dynamics of CNS injuries are generalised. The data of the victims of road accidents are basically studied. The aim is clarifying the injury mechanisms and their comparison with the data on the car accidents. This research information is interpreted in the light of the accumulated practical experience of our team.

Key words: road accidents trauma, craniocerebral trauma, forensic neuropathology.

INTRODUCTION

Eleven years after 1885 when the first automobile was made, its first victim was declared. The first fatal car accident happened on 17.08.1896 in Crystal Palace, London, when Bridget Driscoll, 44 year-old woman, died instantly. The cause of death was brain injury. The car's speed was 4 miles/h (1). Since then millions of people have died on the road due to vehicular accidents and that number exceeds the victims of the I and II World Wars. Road accidents lead to plenty of researches on biomechanics of traumas, especially those of the central nervous system. The last fifty years of world scientific literature, especially in the field of neuropathology, have witnessed a lot of changes on the concepts of biomechanics of craniocerebral trauma (CCT).

Multidisciplinary researchers including neuropathologists, clinicians, bioengineers contributed to clarifying patho- and morphogenesis of brain injuries. Leading theories are: vibration theory of concussion, the liquor strike theory, theory of inertial intracranial dislocation of the brain, rotation theory, pressure gradient theory, cavitation and deformation theory.

Vibration theory (Petit, 1774) examines brain damages as consequences of vibrations

appearing at the very moment of the impact concentrating in the area of the contre-coup (2).

Vibration theory could explain the appearance of contre-coup damages in frontal and temporal areas only if the impact is from behind-occipital, but could not explain the damages of these areas of the brain in the absence of contusion in the occipital lobe if the impact is from the front. The liquor strike theory is formulated by Duret in 1878 based on experiments including supercharging fluid in the cranial cavity. Duret came to the conclusion that the injuries of the brain arise from the sudden increase of the liquor pressure during the head impact. This concept could not explain the absence of contre-coup foci in the occipital area hit from the front (3). The theory of inertial intracranial dislocation of the brain (Russel, 1932) is based on the fact that at the moment of impact, the skull movement as a solid system stops before the brain because of its rigidity and continues dislocating in the direction of the impact and after that quickly returns to initial position in the cranial cavity. The reverse impact of the brain to internal skull surface when it returns to its primary position, explains the appearance of contre-coup damages. According Russel's theory it is impossible to be explained why occipital and frontal impacts cause injuries in the same cortical areas (the bases and poles of the frontal and temporal lobes). Rotation theory was advanced by Holbourn in 1943 who postulated two main causes of brain injuries:

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the skull deformation (causing damages because of local brain distortion) and angular motion (generating shear strains and diffuse neural injury or injury to particular regions) (4). This theory was supported by experimental demonstration of movements of the brain surface at blows to the head (5) and in autopsy material (6). The authors of the pressure gradient theory (Kocher, 1880; Reger, 1884; Busch, 1875; Burns, 1889; Godgio, 1894; Selier and Müller, 1960; Unterharnscheidt, 1963) explain the mechanism of the brain injuries with hydrostatic and hydrodynamic principles (7). The pressure distribution after mechanical force on the brain has to be regular in all directions with equal strength. This theory is rejected above all because of the irregular distribution of the brain injuries, appearing after head trauma. On the basis of the cavitation theory also lie the hydrodynamic lows. According to his experiments Gross in 1958 came to the conclusion that the development of the cavitation, the contre-coup zone is connected with the negative pressure in this area, which arises as a result of the pushing out of the brain from the inner skull surface towards the direction of the impact and it causes focal cortical contusions. In the place of the applied force rises bone distortion, upon which the bone integrity does not disturb and it returns in the previous position. The reverse movement of the bone is very sudden and the brain could not catch up with it. Under these circumstances rises rarefaction, creating conditions for cavitation, which he called coup cavitation (8).

The researchers noted the significance of skull deformation (Bergmann, 1880; Ignatovskii, 1892) on appearance of brain injuries. A lot of authors have paid attention to the structure and thickness of the bones in the anterior and middle cranial fossa, connecting it with the fact that the most frequent injuries happen in the frontal and temporal areas (Ommaya, Hirsch and Flamm, 1966).

The current concepts of the mechanism and biomechanics of the non-missile skull and brain trauma are the result of generalization of the previous theories and extremely precise experiments (9). The brain injuries consider as dysfunction and structural failure as a result of mechanical load caused by the relative motions generated within the tissues (10, 11). They are specified according to their direction as tensile and shear strains and that strains are the most important factor for the creation of damages in live tissues. Despite agreement

that tissue strains are the ultimate direct cause of brain injury there has been a great deal of controversy regarding the mechanisms for the generation of these strains. The contact forces initiate two different mechanical effects: stress waves and skull deformation – contact phenomenon. Pure impulsive loading implies that effects of contact can be ignored, the sudden change of head motion (acceleration - deceleration) generates brain movements of inertia (12). The main characteristic parameters about acceleration-deceleration movements are: translation (linear), rotation, and bending-stretching at the cranio-spinal junction (13).

Translational cranial motion cause movement of the brain, absolute or relative to the skull bones and creates intracranial pressure changes (14). In the area opposite the contact, negative pressure may cause formation of cavitation bubbles, which at their collapse may possibly contribute to brain damages. In 1966 Lindgren made experiments on cadavers. Occipital impact on a cadaver skull produced initially a positive pressure area beneath the site of impact and a negative pressure area in the frontal region. Although these effects were mainly related to translational motion of the head, the pressure changes were possibly in part elicited by stress waves through the brain-effects of contact forces (14). Upon rotational head motion the brain lags behind the skull and inertial forces are exerted on the connection between the brain and skull-dura (bridging veins), and upon the brain tissues themselves. The strains elicited can cause rupture of the bridging veins, resulting in subdural haematomas and of the neural and vascular tissue of the brain parenchyma, resulting in widespread axonal damage and bleeding.

Martinez *et al.*, 1965, produced intracerebral haemorrhages by non-impact whiplash injuries in guinea pigs, hares and monkeys (14). Two years later Higgins and Shmall in 1967 presented a new head-accelerating device (HAD-II), by which reproducible angular accelerations were delivered to the head of monkeys (15). These experiments demonstrated that experimental concussion (15) and intracranial haemorrhages - subdural, subarachnoidal and in the superficial cortical layers were produced by angular acceleration (16).

There is an increasing tendency for clinicians and pathologists to classify brain damages as focal (cerebral contusions, intracranial haematoma and the different secondary types of brain damages – lesions, a high intracranial

pressure and shift and distortion of the brain) and diffuse (diffuse axonal injury, hypoxic brain damage, brain swelling and diffuse vascular injury) (17).

The contusions have a very characteristic distribution whatever the site of the original injury. They affect particularly the frontal poles, the orbital surfaces of the frontal lobes, the lateral and inferior surfaces of the temporal lobes and the cortex above and below the Sylvian fissures. The contusions of the parietal and occipital lobes and of the cerebellum are uncommon unless they are directly related to a fracture of the skull (13, 18). Contusions have been the subject of detailed and comprehensive studies for many years (18, 19, 20, 21, 22) and numerous types have been defined: fracture contusions – at the site of the fracture, coup contusions – bending bones generating strains in vascular and neuronal tissues, which is demonstrated in many experiments (23), contre-coup contusions – bones distortion caused by contact and inertial forces, herniation contusions and gliding contusions – a term introduced by Lindenberg and Freytag, 1960 (21), describing focal haemorrhages in the cortex and subjacent white matter at the superior margins of the cerebral hemispheres.

Strich in 1956 was the first to define clearly the occurrence of “diffuse degeneration of the cerebral white matter” in patients with severe post-traumatic dementia. (6). This type of brain damage resulting from head injury is now widely recognised, although it has been referred to by other authors under different names: shearing injuries (24, 25, 26); diffuse damages to white matter of immediate impact type (27), diffuse white matter shearing injury (28). Strich has always taken the view that diffuse damage to white matter is brought about by the shearing of nerve fibres at the moment of injury, but others have contended that it is secondary to hypoxic or ischaemic brain damage, cerebral oedema or secondary damage to the brainstem resulting from an intracranial expanding lesion (29). The clinico-pathological studies undertaken by Adams 1977, 1982b (27, 30) have endorsed Strich's views and have defined the time course of the structural abnormalities in white matter. Similar structural abnormalities have been produced in subhuman primates without there being any increase in intracranial pressure or episode of hypoxia. (31).

As a result of these experiments the pathogenesis of this type of brain damage now seems clear that the most appropriate term by

which to refer to it is diffuse axonal injury. This type of brain damage was present in varying degrees of severity in about 30% of the cases in the Glasgow database with brain trauma (13). There are three distinctive features in the pathology of diffuse axonal injury in its most severe form: focal lesions in the corpus calosum, focal lesions in the dorsolateral quadrant of the rostral brainstem adjacent to the superior cerebellar peduncles and diffuse damage to axons. Since the first two of these can often be identified macroscopically, it is usually possible to make the diagnosis of diffuse axonal injury at the time of dissection provided the brain has already been properly fixed, but the diffuse damages to axons can only be seen microscopically. Occasionally there are no macroscopic abnormalities, the diagnosis of diffuse axonal injury only being possible if appropriate histological studies are undertaken (Adams *et al.*, 1985b). The appearances of the individual lesions depend on the length of survival after injury. According to Blumbergs, 1995, the most vulnerable axons are those in corpus calosum, fornix and pons. He noted that the absence of vascular damages in patients with mild trauma was a sign of most expressive vulnerability of the axons in comparison with vascular tissues at traumatic damage (32). The clinico-pathologic study undertaken by Graham *et al* showed that DAI are strongly manifested in patients who did not have lucid period, absence of: skull fracture, superficial contusions, haemorrhages in basal ganglia and did not have increase intracranial pressure (33).

There are no contradictions between the latter theories in the new concept. In general, as a result of the head impact appear and act together the skull deformation, cavitation and rotation movement of the brain causing different types of damages.

Among future goals the development of models correlating the mechanical events and disturbances at the cellular and subcellular levels will be of immense importance. Such models may lead not only to improved tolerance criteria but, above all, to increased knowledge crucial to proper diagnosis and treatment of head injury patients.

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