

Brain Contusion Mechanopathogenesis: Arguments for Cortical Compression and Head Rotational Acceleration

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I. INTRODUCTION

Head injury (HI), as the fourth cause of death in the Western world, is a major concern in terms of prevention and therefore an important focus for research. Its incidence was found to lie between 235 and 540 per 100,000 in the USA and in the EU over the last decade [1-2]. Bicycle accident victims are prone to HI, with fatalities occurring in 69–93% of bicycle accidents [3-4]. Our multidisciplinary team (biomedical and engineering) aims at bicycle helmet improvement by establishing brain lesion-specific tolerance criteria (throughout age groups) for each of the pedal cyclist lesion: skull fracture, diffuse axonal injury (DAI), acute subdural hematoma (ASDH) and brain contusion (BC).

The current paper summarises our research findings on BC to bring forward elements of its possible mechanopathogenesis. BCs are defined as focal cortical lesions that may expand into the neighbouring white matter [4]. Being associated with ASDH, this type of lesion may represent a serious life-threat.

II. METHODS

Over the past 16 years, data on BC was produced in several projects at KU Leuven. First, a retrospective analysis of lesion types and accident circumstances in 86 head-injured cyclists was performed. Clinical and imaging data from neurosurgical patients in Leuven University Hospitals between 1990 and 2000 was collected, and accident data was obtained through questionnaires and available police reports [3-4]. Nineteen real-life bicycle accidents could then be reconstructed using MADYMO software. Combinations of mechanical parameters allowed the investigation of possible tolerance criteria for certain lesion types [3-5] [7]. Secondly, relative brain-skull motion (RBSM) was assessed in re-pressurised human cadavers, instrumented with intracerebral and skull-mounted accelerometers, by applying impacts of a varying intensity to the back of the head by means of a pendulum (20–40°) [4-5]. Thirdly, RBSM was assessed by magnetic resonance imaging (MRI) of 30 human volunteers in 4 different head positions (prone, supine, left and right lateral) [4] [8]. Finally, a modal analysis of the skull was performed by applying consecutive hammer impacts on dried or rehydrated, fresh or embalmed empty skulls in the most frequent locations during bicycle accident (frontal, temporal and parieto-occipital) [3-6]. These data were then used in finite element analyses of the skull in situations of impacts, vibration and deformations.

III. INITIAL FINDINGS

In our cyclist database, BCs were the second most frequent in bicycle-related HI (63%), and associated ASDH was seen in 2/3. Nearly 96% of BCs that could not be contributed to coup/contrecoup/fracture mechanisms occurred at the frontal and temporal lobes, with the inferior lobe surfaces being a predilection site in sagittal impacts and the lateral temporal lobe surface in lateral impacts [3-4]. In the accident reconstruction, a relation between peak rotational acceleration, impact pulse duration and the occurrence of BC in the frontal and temporal lobes was found [3-5] [7]. In the MRI study, regions with maximum motion amplitudes were identified at the inferolateral aspects of the frontal and temporal lobes, congruent with the predilection sites for contusions and with the above findings [4][8]. The results of the cadaver study indicated that in sagittal impacts, the major component of the motion at the base of the frontal lobe is in the craniocaudal direction and that the amplitude of this motion at least equalled the amplitude of the motion in anteroposterior direction at the vertex [4-5]. In the modal analysis, it was observed that the orbital roof, the temporal squama and the ala major of the sphenoid bone – skull regions underlying the frontal/temporal contusion predilection sites – at the lower natural frequencies vibrate with amplitudes that are higher (up to 2–3 mm) than the amplitudes in the rest of the skull [3-6].

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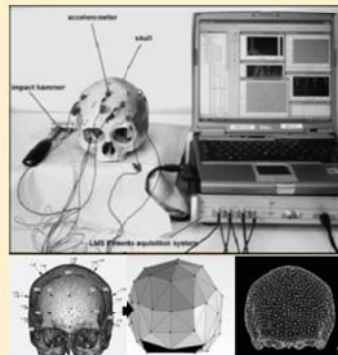
Epidemiology of HI in bicycle accidents

From Jan 1990 to Jun 2000 at UZ Leuven (Depreitere 2004)

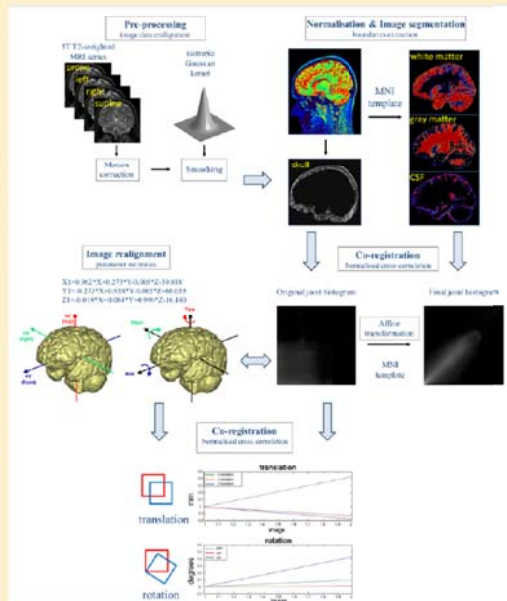
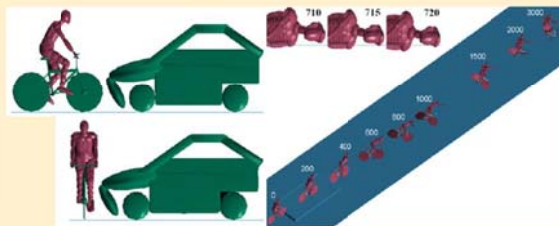
Injury	Total group (N=86)	No motor vehicle involved (N=42)	Collision w/ motor vehicle (N=44)
Skull fracture	74 (86.0%)	39(92.9%)	35(79.6%)
Extradural haematoma	34(39.5%)	17(40.5%)	17(38.6%)
Acute subdural haematoma	29(33.7%)	15(35.7%)	14(31.8%)
Contusion	63(73.2%)	32(76.2%)	31(70.5%)
Intracerebral haematoma (arising from contusion)	15 (17.4%)	11(26.2%)	4(9.1%)
Basal ganglia intracerebral haematoma	1(1.2%)	0(0%)	1(2.3%)
Gliding contusion	5(5.8%)	0(0%)	5(11.4%)
Diffuse axonal injury	11(12.8%)	3(7.1%)	8(18.2%)
Subarachnoid haemorrhage	45(52.3%)	21(50.0%)	24(54.5%)
Intraventricular haemorrhage	11(12.8%)	3(7.1%)	8(18.2%)
Brain swelling	66/80(82.5%)	28/38(73.7%)	38/42(90.5%)

Modal and finite element analysis on human skull

In situations of impact, vibration and deformation (Depreitere 2004, Van Lierde 2005, Delye 2010)

**Relative brain-skull motion by Magnetic Resonance Imaging**

With volunteers (Depreitere 2004, Monea 2013)

**Real-life bicycle accident reconstructions and simulations**
With MADYMO models (Verschuereen 2009)**IV. DISCUSSION & CONCLUSION**

Based on our results, we hypothesise that the majority of frontal and temporal contusions result from the forceful contact of the cortical surface against the skull interior, in other words, from compressive strains. The magnitude of such compressive strains is associated with the amplitude of the head rotational acceleration (RA). Prolonged RA provokes brain deformation and mainly produces DAI, whereas BCs likely result from short duration impact and RBSM [8]. RA can be reduced by the implementation of engineered anisotropic foams, offering a promising component to improve bicycle helmet design [4-5][7-8].

That compression rather than shear is responsible for contusion pathogenesis may be contra-intuitive based on the fact that the brain bulk modulus is higher than its shear modulus [9-12]. Most of our data have been obtained from cadaveric material or in a retrospective way. Complementary studies in a living pig model are now being performed in our group in order to elucidate tissue-level mechanics leading to BC. Their implementation into human finite element modelling should then result in a human BC tolerance criterion.

V. REFERENCES

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