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ORIGINAL

BRAIN INJURIES FROM FISTING. FRONT IMPACT MODEL

TRAUMAS CEREBRALES POR GOLPE DE PUÑO MODELO DE IMPACTO EN LA FRENTE

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ABSTRACT

Boxing and other combat sports are associated with repetitive head trauma related to damage to the central nervous system. This work aimed to model the effect of a punch to the forehead given by a heavyweight boxer. Methodology: the Finite

Element Method (FEM) was used. The research was based on simulating the effects of a dynamic impact and thus predicting, locating, and quantifying changes in the brain due to the blow. This simulation was validated by comparing medical research on brain injuries caused by impacts to the head. Results: The mathematical predictions showed significant brain effects: figures that exceed 100% risk. The MEF appears to be a practical, universal, inexpensive, and quick calculation tool, with important applications to detect evidence of brain trauma.

KEYWORDS: boxing, brain trauma, mathematical modeling, combat sports

RESUMEN

El boxeo y otros deportes de combate están asociados a traumas repetitivos en la cabeza, que pueden relacionarse con daños en el sistema nervioso central. El objetivo de este trabajo fue modelar el efecto de un golpe de puño en la frente, dado por un boxeador de peso pesado. Metodología: se utilizó el Método de Elementos Finitos (MEF). La investigación se basó en simular los efectos de un impacto dinámico y de esta forma predecir, localizar y cuantificar cambios en el cerebro debido al golpe. Para validar esta simulación, se comparó con investigaciones médicas sobre lesiones cerebrales, causadas por impactos en la cabeza. Resultados: Las predicciones matemáticas demostraron grandes efectos cerebrales: cifras que superan el 100% de riesgo. El MEF aparece pudiera ser una herramienta de cálculo práctica, universal, económica y rápida, con importantes aplicaciones para detectar evidencia de traumas cerebrales.

PALABRAS CLAVE: boxeo, trauma cerebral, modelación matemática, deportes de combate

INTRODUCTION

There is a large number of sports in which physical impacts inevitably occur, working from very early ages on the improvement of tactical, technical elements in order to reduce injuries or damage caused by them (Guillen, Copello, Gutiérrez & Guerra, 2017; Hurel, Guillen, Gutiérrez, Sanabria, Formoso & Rosero, 2020). However, many sports modalities, such as boxing, are associated with repetitive head trauma, which can be related to damage involving the Central Nervous System (CNS), both by single impact and repetitive trauma.

Among the brain damage in the practice of boxing, we can find acute damage, which can sometimes be related to death in the ring or a few days after the fight, and chronic damage, those of lesser impact, the latter are caused in the long term such as Alzheimer's disease, Parkinson's disease, pugilistic dementia, dysarthria, ataxia, among others.

Two leading causes of brain damage attributable to boxing, recognized by the World Boxing Association (WBA), are described. Immediate injuries as an acute and direct result of a severe impact and the second cause, reported by the cumulative effects of sustained exposure blows to the head (Fleming, 2006). The Chronic Brain Injury (CBI) scale is a scale that represents the long-term damage, rating it as the cumulative neurological consequences of repetitive brain trauma.

The scale contemplates the following variables: normal (CBI=O), low (CBI=1-2), moderate (CBI =3-4), and severe (CBI>4) [2]. The behavioral component of the CBI scale assesses the domains of psychopathology described in the Neuropsychiatric Inventory (Cummings et al., 1994).

Pugilistic Dementia (PD) is among the diseases that are usually found in the long term in boxers, equally they can occur in amateurs or professionals, as well as in athletes of other modalities (mixed martial arts) who suffer concussions(Martínez, Paredes I., Munarriz, Paredes, J.F.& Alén, 2017). It is also called chronic boxer's encephalopathy, boxer's traumatic encephalopathy, boxer's dementia, chronic traumatic brain injury associated with boxing (CTBI-B), and Punch syndrome. This disease is a variant of chronic traumatic encephalopathy; the symptoms and signs are similar to those of PD; it develops progressively over a long latency period, sometimes reaching decades, with an average onset time of 12 to 16 years, after the beginning of a boxing career. According to Atha et al., 1985; Corsellis, 1989; Jordan, 2000, damage in subjects with a history of repetitive encephalic trauma is preferentially located in the gray matter of the temporal region, where abnormal neurofibrils develop (the so-called Alzheimer's tangle) without neuritic (senile) plaques.

It is well known that athletes who receive frequent impacts often present short- and long-term damage (Cabeza, Guillen, Gutiérrez, Padrón, Vargas & Ortega (2019). However, it is necessary to determine the relationship between the blow produced and the possible variation or damage generated at the level of the brain, which is why it was necessary to conduct the present study whose objective was: to analyze, through a mathematical model, the possible brain variation produced by the effect of a first blow of a heavyweight fighter.

MATERIALS AND METHODS

An impact on the frontal region of a heavyweight boxer with a force of 4903 N was modeled. The human head model was composed of about 22,500 elements and 25,000 nodes. Hexahedron-type elements with 6 sides and 8 nodes were used. Three meshes were tested, each with a different number of elements. The selected mesh was the intermediate mesh, with a 4% difference in the number of elements compared to the mesh with the highest number of elements. Four different layers were considered: scalp, skull, cerebrospinal fluid (CSF), and brain. The first three

were modeled as layers of equal thickness), with the inner tissue being the brain mass.

Autodesk® (ALGOR) software was used. Since the brain is immersed in CSF, the model was constructed simulating the surrounding fluid (Figure 1); figure 2 shows the isolated brain. Almost all materials were considered to have isotropic and linear elastic behavior, except for the brain, which has visco-elastic properties.

The skull was copied from a genuine part. The interior of the brain was modeled with the geometry of the skull. This volume was then subdivided into the different macroscopic components: lobes, cerebellum, and hypothalamus; these pieces were reassembled inside the cranial vault.

After comparing different brain injury magnitude criteria, such as von Mises stresses, maximum linear acceleration, and maximum pressure head injury criterion, the von Mises stress (Meyer et al., 2010) equal to 0.048 N/mm² at 50% risk of injury and 0.080 N/mm² at 100% risk (48 000 N/m² and 80 000 N/m², respectively) was selected.

The model was based on previous publications (Ponce & Ponce, 2011; Ponce et al., 2011).

The direction of the loads was simulated perpendicular to the forehead. Table I lists the input data.

Table 1.
Adult head material data

Material	Density	Elasticity Module	Poisson Modulus
Units	Kg/ m ³	N/m ²	
Scalp	1200	16700000	0.42
Bone	1500	4500000000	0.2
Cerebrospinal fluid	1020	12000	0.46
Brain	1050	**	**

^{**} Viscoelastic materials have neither elastic modulus nor Poisson's modulus.

The input data for the primary materials are described in the works of authors such as: (Belingardi et al., 2005; Khalil&Hubbard, 1997; Miller &Chinzei, 1997; Roth et al., 2008).

The constitutive equation of the brain was based on a nonlinear stress-strain model developed from the strain energy, assuming isotropic tissue. It was initially formulated for finite element modeling (Khalil&Hubbard, 1997), then taken to experiments (Miller & Chinzei, 1997) and applied in specialized computer programs.

Currently, it can be included in commercial programs that can change the constitutive equations.

The equation was defined as:

$$G(t) = G(\infty) + ((G(0) - G(\infty)) \exp(-\beta t)(1)$$

where:

G (t) = Shear modulus

G (0) = Short-term shear modulus = 490 kPa

G (∞) = Long-term shear modulus= 167 kPa

 β = Constant of delay = 0,145 ms-1

t = time

This information was obtained from Belingardi et al. (2005).

Based on the fist velocity of 90 km/h or 25 m/s and a head displacement of 0.2 m, the simulated impact duration was 0.008 s.

The maximum force that could be delivered by a heavyweight boxer, according to The Virtual Human Brain on Line (2011), is 4903 N. Regarding the impact on the forehead of an adult: it was modeled with eight vectors of equal value, which were distributed in a small area, simulating two knuckles of a professional fighting athlete with full contact.

This version was based on the linear displacement of the head backward, in the direction of the applied force not rotating around the neck (which would be the product of a hit to the chin).

The estimated cushioning effect of head impact has been documented in tests on cadavers (Schneck & Bronzino, 2003): in automobile accidents, the skull's skin absorbs up to 70% of the impact energy.

Displacement of the attacked body. The absorption was calculated as conservation of the quantity of motion: mass of the arm by initial velocity = mass of the head and neck by final velocity.

Clearing the final velocity, the final kinetic energy is determined and compared with the initial kinetic energy. This comparison results in an approximate absorption of 20% of the energy of the blow. The calculation is based on the ballistic pendulum example, without including the bullet mass in the pendulum (as well as the fist stuck in the head).

Other undetermined effects include the hydraulic damping of the cerebrospinal fluid and the braking action of tendons and neck muscles.

If there is 70% + 20% absorption of the initial energy, only 10% passes to the brain. Then the mathematical predictions of stresses would have to be reduced to at least 10%. There would be a correction factor for attenuation equal to 0.1.

3D Model

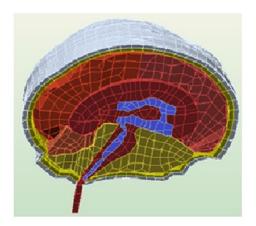


Figura 1. Configuración de elementos finitos de una cabeza. Las capas visibles de tejido de afuera hacia adentro son: cuero cabelludo, hueso craneal, líquido cefalorraquídeo y cerebro.

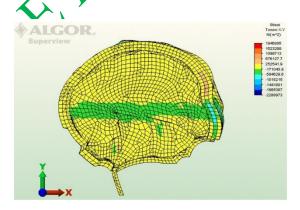


Figura 2. Sección longitudinal del cerebro. Esfuerzos cortantes internos en plano XY. Sin atenuación el máximo = - 171.043,9 N/m² (-0,17 N/mm²) en el cerebro y - 594.629,5 N / m² (-0,59 N/ mm²) en el FCR. Considerando la atenuación de 0,1 sería -17.104N/m² (0,017 N/mm²) en el cerebro y -59.463N/m² (-0,059 N/mm²) en el FCR.

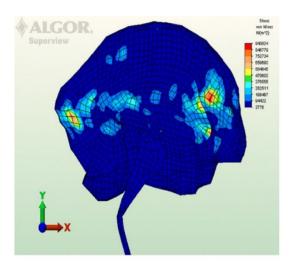




Figura 3. Sección longitudinal del cerebro. Tensiones de Von Mises en el interior del cerebro. Sin atenuación el máximo predicho = 940.824 N/m² (0,94 N/mm²). Considerando la atenuación de 0,1 sería 94.082N/m² (0,094 N/mm²) dentro del lóbulo frontal.

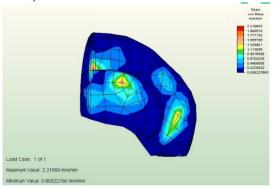


Figura 4. Deformación en el lóbulo frontal. Sin atenuación Valor máximo = 2,219 mm/mm. Con 0,1 de atenuación la deformación sería 0,2219 mm/mm.

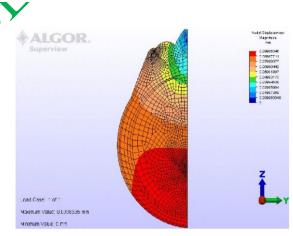


Figura 5. Desplazamiento nodal (mm) en 2D debido a un golpe concentrado en la frente.

RESULTS

Tables 2, 3, and 4 summarize the different aspects analyzed:

Table 2Brain stresses in the XY plane (0.048 N/mm2 with 50% risk of injury)

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Brain sector	Type of stress	Figure	Max. stress N/mm ²
Behind the stroke			
FCR	Shear	2	0.059
Brain	Shear	2	0.017
Frontal lobe	Compression	3	0.094
Occipital lobe	Compression	3	0.075

 Table 3

 Brain Deformation and Displacement

Brain Sector	Type of Movement	Figure		Max. Movement
Frontal Lobe	Deformation	4		0.2219 mm/mm
Occipital Lobe	Displacement	5		0.0998 mm

 Table 4

 Frontal lobe. Comparison of impact effects results with other authors.

Author	Type of Impact	Time ms	Force KN	Results N/mm ²
Belingardi et al. (2005)	Car crash	7.3	7.56	200
Yang (2011)	Car crash	.8	8	0.15
Ponce & Ponce, 2011; Ponce et al., 2011.	4	8	4.9	0.094

In Figure 2, a prediction of the horizontal beam of shear stresses, extending from the forehead to the occipital area, can be seen. Outside this area, the stresses are lower. The most straightforward interpretation is that the maximum compressive stresses occur in the XX axis when deforming the frontal bone. According to Möhr's circle, pure compression should generate XY shear stresses. This direction is parallel to the loading plane. Shear stresses could cut the axons of neurons, and their effect is more dangerous than that of compressive stresses. The highest shear stress would be 0.017N/mm².

In Figure 3, the longitudinal section of the brain, the von Mises stresses are indicated. Stress concentrations are observed in diffuse areas. There are large stresses in the frontal lobe and occipital lobe. The most relevant von Mises stresses (attenuated by 0.1) are: 0.094 N/mm2 in the frontal lobe; limbic area 0.037 N/mm2; occipital lobe 0.066 N/mm².

Figure 4 shows the deformation in the frontal lobe. With 0.1 attenuation the deformation would be 0.22 mm/mm. This deformation of a high-value can damage brain tissue.

Figure 5 shows the nodal displacement caused by a concentrated blow to the forehead. The observed effect is so significant that the largest displacements occur on the side opposite the impact. It is reasonable to think that the lesions also occur in the occipital lobe (posterior part of the brain) by a rebound effect. However, it should be mentioned that concerning the 2D analysis, the numerical results are not as powerful as in 3D, so only their qualitative value should be considered.

DISCUSSION

The stresses predicted in the results far exceed the allowable limits: for 100% risk, the von Mises criterion limit stresses are 0.080 N/mm2, but the maximum reaches 0.094 N/mm2 in the frontal lobe, including attenuation. The cerebrospinal fluid (CSF) stresses are not of significant importance since it is a fluid that is not part of the brain, and one of its functions is damping.

Increasing sources of information indicate a tendency for athletes who have suffered repeated head injuries to have an increased risk of degenerative diseases such as Alzheimer's or Parkinson's (Atha et al., 1985, Jordan, 2009; Jordan, 2000). Other effects of injury were described by McDonald (1957).

Because the brain has a gel-like texture and complex geometry, damage can be severe in sudden acceleration or deceleration (King et al., 2003). In this type of injury, the damage is not external but interpal (called closed injury). In the case of impact, the brain may rebound against the inside of the skull (opposite side of the blow) since the thickness of cerebrospinal fluid is limited and is not sufficient to absorb this event (Sempere, Rodríguez, Boyero & Egea, 2019).

Cerebral contusive injuries also occur on the opposite side of the direct impact, by impact against the skull walls (contrecoup).

It was indirectly compared with medical research on brain injuries caused by impacts to the head to evaluate this simulation: Corsellis (1989) described that of 11 cases analyzed from a total of 13, post mortem studies under the microscope showed that in the cerebellum, there was atrophy and glial fibrosis plus losses of Purkinje cells and in the substantia nigra there were noticeable lesions related to Parkinson's disease, which is consistent with Figure 3 where there are high stresses in the cerebellum.

In addition, this same author points out that in the thalamus and hypothalamus, under the microscope, it was found that there was severe gliosis (modification of the cells of the nervous tissue by a reaction to trauma).

According to Medicine Net (2020), repeated blows to the head decrease brain capacity: there are effects on frontal lobes related to reasoning, personality, and

judgment. Some post-traumatic disorders will affect the amygdala, the brain that controls emotions, and the prefrontal cortex, a decision-making area. Autopsies and brain dissection have confirmed these post-traumatic disorders.

The mathematical prediction is confirmed: in the frontal lobe, the maximum tension exceeds the von Mises criterion (0.080 N/mm²), and in the limbic area with attenuation, it reaches 0.037 N/mm².

There is also agreement with Cifu (2017), who demonstrates that repetitive blows to the head generate various neurodegenerative conditions, including that of the frontal lobe, which was detected by scanned images and postmortem diagnostics. Concerning predictions about the hindbrain (0.066 N/mm2 with attenuation), Blakemore (2003) indicates that abnormalities of the brainstem, which connects the brain to the spinal cord, are associated with cognitive disorders, sleep difficulties, hearing and balance problems. There is an agreement with this author. Dominic (2020) points out frontal lobe dysfunction, related to pugifistic dementia, in full agreement with the predictions of this work.

Neurologists have for years determined that there is a correlation between damage to the prefrontal between damage to the prefrontal cortex and psychopathic behavior (Raine, 2000). This cortex acts as an instrumental system that connects with the other systems through bundles of nerve fibers. The frontal lobe is the command center for executive functions. If there are lesions in this area, the ability to process information, solve problems, concentrate, memory, and learn is affected (Hardy & Khalil, 1994; Plassman et al., 2000). These findings from other authors are consistent with Figure 3, where there are more significant tensions in the frontal lobe and its connections with the posterior part and the rest of the brain. It was also compared with the results of medical research on brain injuries caused by head impacts (Strassmann & Harper, 1968; Blakemore & Jennet, 2001; Homeier, 2004; Spriggs, 2004; Coles, 2007; Stern & Baugh, 2013; Gandy et al., 2014; Olson, 2014; and Lim et al., 2019), agreeing with the predictions emanated by the FEM.

Finally, the results obtained were compared with those presented by other authors such as Belingardi, 2005; Yang, 2011; Nahúm, Adalberto, López, Rocha, 2017). As no publications of prediction by FEM related to pugilistic brain injuries were available, those available from protections in cyclists and automobile accidents, with impacts to the forehead, were used.

CONCLUSIONS

It is concluded that repeated high-impact trauma can cause alterations in the brain. It is found that for a 100% risk, the von Mises stresses are 0.080 N/mm², and the maximum mathematical prediction is 0.094 N/mm².

FEM can be used to simulate the effects produced by the impact of a heavyweight fighter's fist to the head and has the potential to predict the location and extent of changes in the brain due to an impact.

Finite element modeling would predict mechanical injuries from automobile accidents, bicycles, soccer, machine vibrations, pressure pulses, among others. This modeling would be done at a very low cost.

Safety equipment could be designed based on mathematical models, validated with fewer laboratory tests, and optimized design. These studies can be extrapolated to other injuries in different parts of the body under different loading conditions.

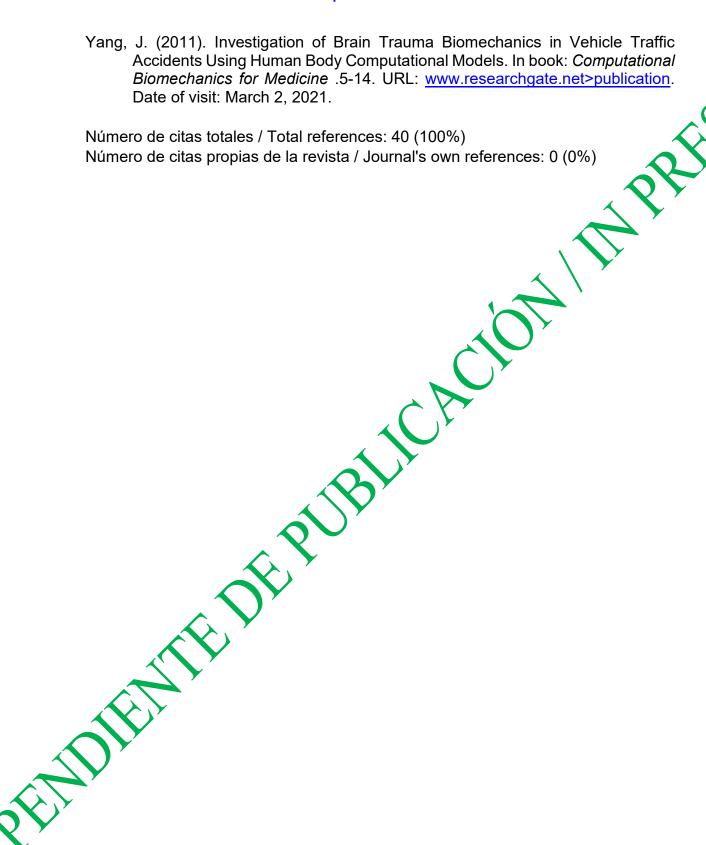
In this work, FEM has proven to be a reliable tool for calculation and an economical method, which provides results in a reasonable time, paramount in sports medicine, forensic medicine, and other applications. The research presented is a preliminary step before analyzing head impact using a more complex brain model. It should be mentioned that, although the data are promising, they need validation on real models before being used as an alternative method.

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Figures:

Figure 1. configuration of elements of a head. The visible layers of tissues from the outside are the scalp, cranial bone, cerebrospinal fluid, and brain.

Figure 2. longitudinal section of the brain. internal forces in XY plane. without attenuation the maximum: -171,043.9 N/m² (-0.17 N/m²) in the brain and -594,629.5 N/m² (-0.59 N/mm²) in the CSF. considering the attenuation of 0.1 serious -17,104N/m² (0.17N/m²) in the brain and -59,463N/m² (-0.059 N/m²) in the CSF.

Figure 3. Longitudinal section of the brain. Von Mises stresses inside the brain. Without attenuation the maximum predicted =904,824 N/m² (0.94 N/m²). considering an attenuation of 0.1 would be 94,082 N/m² (0.094 N/m²) inside the frontal lobe.

Figure 4. deformation in the frontal lobe, without attenuation maximum value= 2.219 mm/mm, with 0.1 attenuation the deformation would be 0.2219 mm/mm.

Figure 5. 2D nodal displacement (mm) due to a concentrated blow to the forehead.

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