Aims & Scope:
Traumatic brain injury (TBI) is one of the leading causes of death and disability worldwide. Our understanding of its pathobiology has substantially increased. Following TBI, the following occur, edema formation, brain swelling, increased intracranial pressure, changes in cerebral blood flow, hypoxia, neuroinflammation, oxidative stress, excitotoxicity, and apoptosis. Experimental animal models have been developed. However, the difficulty in mimicking human TBI explains why few neuroprotective strategies, drawn up on the basis of experimental studies, have translated into improved therapeutic strategies for TBI patients.

TBI applies powerful rotational and translational forces to the brain parenchyma, which results in a traumatic diffuse axonal injury (DAI) responsible for brain swelling and neuronal death. Following TBI, axonal degeneration has been identified as a progressive process that starts with disrupted axonal transport causing axonal swelling, followed by secondary axonal disconnection and Wallerian degeneration. These modifications in the axonal cytoskeleton interrupt the axoplasmic transport mechanisms, causing the gradual gathering of transport products so as to generate axonal swellings and modifications in neuronal homeostasis. Oxidative stress with consequent impairment of endogenous antioxidant defense mechanisms plays a significant role in the secondary events leading to neuronal death. Studies support the role of an altered axonal calcium homeostasis as a mechanism in the secondary damage of axon, and suggest that calcium channel blocker can alleviate the secondary damage, as well as other mechanisms implied in the secondary injury, and could be targeted as a candidate for therapeutic approaches. Increases in the defense mechanisms through the use of exogenous antioxidants may be neuroprotective, particularly if they are given within the neuroprotective time window. A promising potential therapeutic target for DAI is to directly address mitochondrial-related injury or to modulate energetic axonal energy failure.

The papers collected in this special issue aim at identifying the epidemiology, the main related risk factors and the possible potential therapeutic target or the ongoing related-therapeutic measures to treat TBI and the related adverse events.

Keywords: addiction, traumatic brain injury (TBI), pathology of brain injuries, reactive oxygen species (ROS), pharmacological therapy, surgical therapy, histological diagnosis, spinal cord injury, miRNA and prognosis, TBI sequela, TBI experimental models.

Subtopics:
The subtopics to be covered within this issue are listed below:

- Pathology of brain injuries;
- Traumatic brain injuries;
- Hypoxic-ischemic brain damage;
- Experimental model of brain injuries;
- Targeted therapy of brain injuries;
- Histological diagnosis of brain injuries;
- Measurement of ROS;
- Epigenetic of brain injuries;
- miRNA and prognosis of neural damages;
- Novel approach and proof of principle in therapy;
- Related quality of life after brain injuries.

Schedule:
- Manuscript submission deadline: June 30th, 2019
- Peer Review Due: July 31st, 2019
- Revision Due: August 31st, 2019
- Announcement of acceptance by the Guest Editors: September
- Final manuscripts due: September 30th, 2019

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