Brain edema

- Bone Graft Expansion in Cranioplasty Using a Split-Bone Technique
- Role of Magnetic Resonance Venography in the Evaluation of Cerebral Veins and Sinuses Occlusion
- Hypodensity Beyond the Ischemic Core: Penumbral Changes Detected With Relative Noncontrast Computed Tomography
- Fargesin Exerts Neuroprotective Effect Against Cerebral Ischemia/Reperfusion Injury in Rats via Alteration of NF-κB Signaling Pathway
- Advances and Integrations of Computer-Assisted Planning, Artificial Intelligence, and Predictive Modeling Tools for Laser Interstitial Thermal Therapy in Neurosurgical Oncology
- Single-Cell RNA Sequencing and Spatial Transcriptomics Reveal a Novel Mechanism of Oligodendrocyte-Neuron Interaction in Cognitive Decline After High-Altitude Cerebral Edema
- The Unlucky Variant: Artery of Percheron Infarction
- Screening for Heart Failure in Patients with Hypertension And/Or Diabetes Using Hand-Held Echocardiography: A Pilot Study

Brain edema, cerebral edema or cerebral œdema is an excess accumulation of fluid in the intracellular or extracellular spaces of the brain.

Types

At present, the following types of cerebral edema are differentiated:

The vasogenic edema resulting from an increased permeability of the endothelium of cerebral capillaries to albumin and other plasma proteins

The cytotoxic edema resulting from the exhaustion of the energy potential of cell membranes without damage to the barrier;

The hydrostatic cerebral edema resulting from disturbance of the autoregulation of cerebral blood circulation

The osmotic cerebral edema resulting from dilution of blood; and the interstitial cerebral edema resulting from acute hydrocephalus.

Simard et al. suggest a new theory suggesting that ischaemia-induced capillary dysfunction can be attributed to de novo synthesis of a specific ensemble of proteins that determine osmotic and hydraulic conductivity in Starling's equation, and whose expression is driven by a distinct transcriptional program [1)].

Etiology

Brain Edema Etiology.

Clinical Features

Certain changes in morphology are associated with cerebral edema: the brain becomes soft and smooth and overfills the cranial vault, gyri (ridges) become flattened, sulci (grooves) become narrowed, and ventricular cavities become compressed.

Symptoms include nausea, vomiting, blurred vision, faintness, and in severe cases, seizures and coma. If brain herniation occurs, respiratory symptoms or respiratory arrest can also occur due to compression of the respiratory centers in the pons and medulla oblongata.

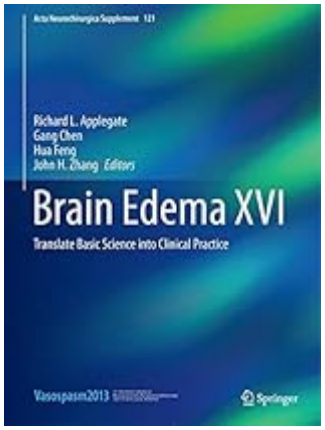
Treatment

see Brain edema treatment.

Outcome

Brain edema leading to an expansion of brain volume has a crucial impact on morbidity and mortality following traumatic brain injury (TBI) as it increases intracranial pressure, impairs cerebral perfusion pressure and oxygenation, and contributes to additional ischemic injuries.

Books



In this book, leading world authorities on brain edema and neurological disorders/injuries and experts in preconditioning join forces to discuss the latest progress in basic sciences, translational research, and clinical management strategies relating to these conditions. The range of topics covered is wide, including microglia, energy metabolism, trace metals and ion channels, vascular biology, cellular treatment, hemorrhagic stroke, novel technological advances, anesthesia and medical gases, pediatric brain edema, neuroimaging, behavioral assessment, clinical trials, peripheral to central signaling pathways, preconditioning translation, and animal models for preconditioning and brain edema research. The book comprises presentations from Brain Edema 2014, the joint meeting of the 16th International Conference on Brain Edema and Cellular Injury and the 3rd Symposium on Preconditioning for Neurological Disorders, held in Los Angeles on September 27–30, 2014.

[1)]

Simard JM, Kent TA, Chen M, Tarasov KV, Gerzanich V. Brain oedema in focal ischaemia: molecular

pathophysiology and theoretical implications. Lancet Neurol. 2007 Mar;6(3):258-68. Review. PubMed PMID: 17303532; PubMed Central PMCID: PMC2725365.

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