

# Managing Cerebral Edema



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Cerebral edema is challenging to manage because the brain is rigidly confined within the dura and calvarium. Clinical manifestations of cerebral edema vary in severity and presentation and may include: headache, nausea, vomiting, altered cognition, decreased level of consciousness, focal neurologic deficits, cranial nerve palsies, altered ventilatory patterns, cardiac dysrhythmias, coma and death.

Secondary injury occurs hours to days after the initial insult. Clinicians can lessen injury and prevent further cell loss by reducing intracranial pressure (ICP), optimizing cerebral perfusion and oxygen delivery, minimizing the brain's metabolic demands and avoiding significant disturbances in the ionic/osmotic balance between the brain and vascular compartment.

Traditionally, cerebral edema is classified as cytotoxic, vasogenic, or interstitial. Most brain injuries involve a combination of the three. Cytotoxic edema occurs when there is energy failure at the cellular level, classically after an ischemic stroke. Cellular injury/death occurs and the intracellular concentration of water increases; neurons, glia, and endothelial cells swell and cellular homeostasis is lost. Cytotoxic edema resists most current therapies. Vasogenic edema often is associated with neoplasia, inflammation, infection and trauma. The blood-brain

barrier breaks down, increasing vascular permeability and extracellular water. It potentially responds to steroids and osmotherapy. Impaired cerebral spinal fluid (CSF) absorption causes interstitial edema (hydrocephalus). Temporary or permanent CSF diversion is definitive treatment.

## Supportive Measures

Clinicians should use the ABCD's (Airway, Breathing, Circulation, and Disability) approach with neurologically compromised patients. Hypoxia and hypercapnia are potent cerebral vasodilators that may increase ICP and worsen outcomes. Priorities for these high-risk patients are endotracheal intubation and mechanical ventilation. Patients without elevated ICP should have their PaCO<sub>2</sub> maintained between 35 to 40 mmHg and PaO<sub>2</sub> at greater than or equal to 100 mmHg.

Hypotension and hypertension correlate with poor outcomes. Prolonged hypotension may worsen ischemic insult. Systolic and diastolic hypertension may propagate intracranial hemorrhage. Titrate blood pressure control and avoid dramatic swings. American Heart Association-published guidelines are available for patients with ischemic or hemorrhagic stroke and subarachnoid hemorrhage.

For most other conditions, clinicians should maintain the cerebral perfusion pressure (CPP) between 60 and 70 mmHg or the mean arterial pressure between 70 and 80 mmHg if an intracranial pressure monitor is not available.

Patients should be maintained in a euvolemic to slightly hypovolemic state. Clinicians should use normal saline,



Axial Head CT taken at the Foramen of Monroe demonstrating diffuse cerebral edema of the left hemisphere. Note the loss of gyral/sulcal definition and grey/white differentiation as well as partial effacement of the left frontal horn of the ventricle.

hypertonic saline, or colloid solutions with goal serum sodium between 140 and 145 meq/L. The serum potassium and magnesium levels should be kept at high normal levels (>4.0 mmol/L and > 2.5 mg/dL respectively) using a high acuity replacement protocol as myocardial irritability and resulting dysrhythmias may accompany some neurologic injuries. Avoid administering hypotonic solutions, dextrose containing solutions and/or free water.

Critical-care literature shows a strong correlation between hyperglycemia and poor outcomes in both ischemic stroke and traumatic brain injury, and improved outcomes with tight glucose control. An insulin drip is recommended to maintain the blood sugar between 80 and 110 mg/dL. Although hypoglycemia is a risk, the incidence appears to be small and without significant long-term sequelae.

All hospitalized patients with neurologic injury should undergo well-documented, easily reproducible, serial neurologic examinations. The frequency

of examination depends on the severity of the initial insult, the anticipated decline chance, the presence of other injuries, premonitory conditions and the overall stability of the condition.

The examination takes into account the patient's vital signs, level of consciousness, ability to produce coherent speech and follow commands, cranial nerve examination, and movement of the extremities. The two most common tools developed for this task are The National Institutes of Health Stroke Scale (NIHSS) and the Glasgow Coma Scale (GCS).

Significant changes in either exam should be relayed to the treatment team and physicians promptly. A systematic, protocol-driven approach to the patient with neurologic injury and elevated intracranial pressure will improve clinical outcomes and allow institutional quality control and process improvement.

For more information, view a full-length paper on [portal.fairview.org](http://portal.fairview.org) (call 612-672-6805 for a password) or contact Watts, [cwatts@neurosurgicalassocs.com](mailto:cwatts@neurosurgicalassocs.com).