

# ICU Monitoring

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*ICU monitoring for patients with acute stroke is often necessary, either secondary to the neurological compromise caused by the event due to the sheer size of the injury or due to the systematic complications. This article discusses the methods of monitoring, the potential systematic complications, the use of anticoagulation, blood pressure management, and methods of treating cerebral edema.*



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Guest Editors' Note. Early clot-removing treatment of acute stroke is only one of the advances in stroke care. Another is the increasing expertise in managing stroke patients. This expertise has derived in large part from the new discipline of Critical Care Neurology. A leader in this field is Dr. David Greer, Assistant Professor of Neurology at the Massachusetts General Hospital. In this article Dr. Greer comments on the art and science of critical care for stroke patients.

ICU Monitoring. Stroke patients often warrant ICU care. Decreased levels of arousal and concomitant poor airway protection may warrant early intubation and airway management. Stroke patients may require central venous catheterization or pulmonary arterial catheterization. Some may need arterial lines to closely monitor their blood pressure.

Cardiac monitoring is imperative in acute stroke. Monitoring may reveal a probable cause of stroke, such as intermittent atrial fibrillation. Also, stroke may produce secondary changes in the heart. Myocardial infarction may result from a stroke-induced surge of catecholamines. Arrhythmias are common, and may be related to stroke

location. The right temporal region gives predominantly parasympathetic input to the heart. The left brain, in general, supplies more sympathetic input. Thus, left insular strokes are often associated with bradycardia or AV block. Right temporal lobe strokes are often associated with ventricular tachycardia. One can also see dysrhythmias with hypothalamic and medullary lesions. As a general rule, most patients who have these types of events already have a damaged heart. Indeed, stroke is often a marker for unrecognized cardiac disease.

Typically, strokes produce negative phenomena (lack of function), but if they are associated with seizures, positive phenomena may occur. In the motor system, this may take the form of convulsions, but in the autonomic system, it could be manifest as inappropriate autonomic input to the heart, with resultant arrhythmias.

Nutrition, Fluids and Electrolytes. Stroke patients may need to be NPO, if the swallowing mechanism is compromised, but GI prophylaxis is important and early feeding should be undertaken whenever possible.

For hydration, we avoid hypotonic fluids. We use normal saline with potassium

and some glucose, at a relatively low rate. Excessive hypotonic fluids may worsen cerebral edema from stroke. Electrolytes must be watched closely. Low magnesium may lower the seizure threshold, so we keep magnesium greater than 2.0, especially with hemorrhagic stroke which are more prone to seizures.

Hyperglycemia causes multiple problems for stroke patients. It increases stroke size and worsens cerebral edema. After rt-PA, it increases the rate of hemorrhagic transformation. As a result, it impairs neurological outcome. We recommend tight glucose control, preferably with continuous insulin infusion and glucose monitoring on an hourly basis.

Anticoagulation. DVT prophylaxis is important for stroke patients, but concerns about intracerebral hemorrhage must be taken into account. Pneumatic compression stockings, TED hose and air boots can be recommended with little reservation, but anticoagulants are more controversial. We typically wait 48 hours after intracerebral hemorrhage and then start prophylaxis with low molecular weight heparin.

Data supporting heparin use in acute ischemic stroke is lacking, but a useful role for heparin has not been disproven. There may be subsets of patients, such as those with large vessel disease, who benefit from early anticoagulation. Fluctuating lacunar syndromes are problematic. Hypertensive therapy, glycoprotein IIb/IIIa inhibitors and heparin have been tried, but there is no proven benefit. The RAPID trial (in Europe) is currently evaluating unfractionated heparin versus aspirin in acute, non-lacunar strokes.

For patients who have large ischemic strokes or intracerebral hemorrhage, but who need unfractionated heparin to prevent

thromboembolism (atrial fibrillation, mechanical heart valves, known LV thrombus, prothrombotic states), we typically wait a week to begin anticoagulants. The risk of early, recurrent embolization from atrial fibrillation is not as high as once thought.

Patients with cerebral venous thrombosis benefit from anticoagulation, even if they have a hemorrhagic component. Here, the problem is one of outflow obstruction, and we recommend anticoagulating these patients early on.

Blood Pressure. Blood pressure is an important consideration in stroke patients. For hemorrhagic strokes it should be controlled early on. For ischemic strokes in general we avoid acute lowering blood pressure, as doing so may compromise vital collateral circulation. When patients with ischemic stroke have received thrombolytics, we keep their systolic less than 185 and their diastolic less than 110, according to standard guidelines. We use intravenous labetalol or nicardipine. If the patient has renal insufficiency fenoldopam may be preferable. Nitroprusside can quickly reduce blood pressure, but it causes cerebral vasodilatation and increased ICP. This is problematic if cerebral edema is already an issue.

There may be a role for inducing hypertension in acute ischemic stroke. The rationale is to provide increased collateral flow to penumbral tissue. A small pilot study at our institution showed clinical improvement in 7 out of 13 patients when the systolic blood pressure was increased by 20 percent. A nationwide NIH-sponsored trial is now looking at induced hypertension in acute ischemic stroke.

Cerebral Edema. Cerebral edema may be life-threatening with cerebellar infarction.

These patients may be candidates for decompressive craniectomy. Patients with cerebellar stroke may also deteriorate from acute hydrocephalus, and ventriculostomy placement can be life saving.

Life-threatening cerebral edema can also be seen in patients with massive cerebral hemispheric infarction. Again, decompressive craniectomy can be life saving. Two prospective but non-randomized clinical studies showed that mortality was significantly reduced in the surgical group. Patient selection is clearly important, but criteria are not well defined. The functional outcome is less likely to improve in elderly patients, and the side of the lesion (dominant or non-dominant hemisphere) is sometimes taken into consideration, as well.

Randomized studies are underway, but it appears that the timing of surgery is crucial. If one waits for clinical deterioration, it may be too late. Risk factors for malignant cerebral edema include early depression in level of consciousness, early nausea and vomiting, hypertension, congestive heart failure, leucocytosis, and younger age (patients with have less cerebral atrophy have less room to swell). ICP monitors are poor predictors of whether patients will deteriorate, as they are location dependent.

Decompressive craniectomy is the most definitive (and most invasive) management for life-threatening cerebral edema. Less invasive management includes include elevation of the head of the bed, fluid restriction, and avoidance of fever.

Hyperventilation lowers ICP, but the effect is short-lived (minutes to hours), and prolonged hyperventilation may worsen cerebral ischemia.

Osmotic therapy can be useful. Mannitol draws water into the intravascular component from the extracellular and

interstitial spaces. It may have favorable rheological effects that increase blood flow in the microcirculation. Unfortunately, water tends to be extracted better from normal brain tissue than infarcted brain tissue with Mannitol. It may leak into the infarcted hemisphere and paradoxically worsen the tissue shift. It can be nephrotoxic, crystallizing in the renal tubules. There are no randomized controlled trials looking at Mannitol for space occupying cerebral infarction.

Hypertonic saline has a high tonicity and it is actively excluded from an intact blood-brain barrier. It decreases edema in affected and unaffected hemispheres in rat models. It causes hemodilution, natriuresis and improved pulmonary gas exchange. It shrinks endothelial cells and red blood cells, which improves blood flow and oxygenation in the microcirculation. It can be given as a continuous infusion, or can be given as bolus, or as both. We typically use 3% saline and titrate based on the sodium levels, aiming for a level between 150 and 160. It causes a transient volume expansion. Adverse effects include electrolyte abnormalities, CHF, bleeding and phlebitis. There is concern about inducing central pontine myelinolysis if sodium is increased too quickly. There are no randomized trials evaluating functional outcome with hypotonic saline for massive hemispheric cerebral infarction.

Other measures to control cerebral edema include furosemide and barbiturates. Steroids have not proven beneficial in ischemic infarction or hemorrhagic stroke in numerous studies. We recommend against steroids for either situation.

*Neuroprotection.* Medications have thus far been disappointing as neuro-

protectants, but hypothermia seems promising. It has been shown to be neuroprotective following cardiac arrest. It decreases cerebral metabolic rate, stabilizes lipoprotein cell membranes, preserves the blood-brain barrier, reduces the inflammatory response and reduces cytotoxic glutamate release. Early treatment may decrease infarct volume and control elevated intracranial pressure.

There are various means of inducing hypothermia. These include ice bags, cooling fans and cooling blankets, external vests and cooling pads, and intravascular techniques.

Complications of hypothermia may include increased infection rate, increased bleeding rate, cardiac dysrhythmias, and hypotension. Rebound elevations in ICP can be seen with rapid re-warming. Shivering is also a problem, as it generates heat. To avoid shivering, patients may need to be paralyzed. Medications such as Demerol, Wellbutrin and Propofol may help to suppress shivering.

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