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This was a large observational study carried out in 374 hospitals that participated in a resuscitation registry between 2000 and 2009. The dataset included nearly 85,000 patients who suffered in-hospital cardiac arrest and were followed to discharge. Analysis revealed that survival to discharge improved from 13.7% in 2000, to 22.3% in 2009 – both in patients suffering ventricular fibrillation or tachycardia, and in patients suffering pulseless electrical activity. Immediate survival and survival in the post-resuscitative period both improved. Rates of significant neurological injury decreased from 32.9% to 28.1%.

This study employed a fairly robust data collection method. The risk adjustment variables seemed reasonable, but the strength of the resulting model is not provided. It is unlikely that this model is highly effective in modeling all the known (and unknown) factors that could influence mortality in such a diverse patient population.

The use of historical control groups introduces confounding by many factors that may have changed in the nine years of this study. These may include improvements in patient care, such as an increased awareness of the importance of non-interruption of CPR, and the advent of post-arrest hypothermia therapy. They also may include changes in the patient population. During the last decade, many hospitals have put a great deal of effort into improving end-of-life decision-making. At our hospital, we are currently less likely to perform ACLS on a patient with a terminal disease than we were in 2000 – this alone could theoretically increase ACLS survival without any change in resuscitative care.

Whatever the cause, the study shows an encouraging trend. One factor that might have contributed is participation in the quality improvement registry. This can be beneficial, even when the associated guidelines are not – a result of the Hawthorne effect that occurs when extra attention is focused on a specific problem.

Dr. Garcia-Orr made an excellent point in regards to the results of this study: Overall, only 12% of patients survived to discharge without significant neurological disability. This can be important result for families to consider during discussion of code status in critically ill patients.


The authors performed a systematic review of randomized and observational studies on the use of bolus etomidate in septic patients, and performed a meta-
analysis focusing on mortality and the surrogate outcome of abnormal cosyntropin stimulation response. I’m going to focus discussion on the clinical outcome of mortality, which was described in five studies including 865 patients. Only one study individually showed a significant association between bolus etomidate and mortality, but the combined data resulted in a pooled relative risk of 1.2, that barely achieved significance (95%CI 1.02 – 1.42).

I believe everyone at our conference has used etomidate as an induction agent for intubation, and unanimously feel that it is a highly effective drug for that critical juncture in patient care. Alternatives such as ketamine 2mg/kg, or propofol 1.5-3mg/kg are also highly effective. Although this study raised awareness of the potential for temporary adrenal dysfunction after the use of bolus etomidate, experience has shown that meta-analyses often yield misleading conclusions. I don’t think any of us were convinced that etomidate increases mortality, or that we should abandon the favorable qualities of etomidate induction based on this study.


We used this article to discuss global management of intracranial hypertension. Surrogate goals of therapy include maintenance of intracranial pressure (ICP) < 20 mm Hg, and cerebral perfusion pressure > 60 mmHg (CPP = mean arterial pressure minus ICP). The article provides a detailed review of osmolar therapy that can be used as part of a therapeutic regimen to achieve these goals. In patients suffering intracranial hypertension, 3% or 23% saline can be administered as a bolus – the volume can be calculated by determining the dose of sodium necessary to raise the patient’s serum sodium to 150 mmol/L, assuming a volume of distribution of sodium of 0.6 times total body weight (the same formula we are familiar with using in the management of hypo or hypernatremia). Alternately, 0.5 grams/Kg of 20% mannitol can be administered. In either case, repeated doses can be given to achieve a measured or calculated serum osmolarity of 300-320 mOsm/L. Other measures may be more helpful if intracranial hypertension persists despite achieving targeted hyperosmolality. We have found mild hypothermia (32-34° C.) and pentobarbital (250-500 mg intravenous boluses) to be effective in lowering and maintaining ICP. Vasopressor agents are often necessary to maintain CPP. Note that a patient with an ICP of 20 mm Hg will require a mean arterial pressure of 80 mm Hg to maintain a CPP > 60 mmHg.

In our experience, preventative measures are critically important in patients at risk for intracranial hypertension. Once the CSF space contracts in response to cerebral edema, the compliance of the cranial vault approaches zero, and even minor changes can cause life-threatening increases in ICP. Triggering events fall into several categories: 1) those that can cause cerebral vascular dilation (hypoxemia, hypercarbia), 2) those that reduce venous outflow (flat or Trendelenburg head position), 3) those that trigger deleterious neurological reflexes (airway suctioning, intubation), 4) medications that directly increase ICP.
(succinylcholine, ketamine), and 5) interventions that suddenly reduce osmolarity (infusion of hypo-osmolar IV fluids, intermittent hemodialysis). We have frequently observed life-threatening increases in ICP that occur within seconds of simply placing a patient in Trendelenburg for central line placement. It’s difficult for an individual clinician to keep track of all the aspects of neuroprotective care without the use of a locally-supported protocol.

Robert A. Raschke, MD
Associate Editor