POST-RESUSCITATION CARE

The Task Force reviewed evidence regarding hypothermia for pediatric patients who remain comatose following resuscitation from cardiac arrest. There is clear benefit for adult patients who remain comatose after VF arrest, but there is little evidence regarding effectiveness for infants (ie, beyond the neonatal period) and young children who most commonly have asphyxial arrest.

Some patients with sudden death without an obvious cause have a genetic abnormality of myocardial ion channels (ie, a channelopathy), which presumably leads to a fatal arrhythmia. Because this is an inherited abnormality, family members might be affected, but special tests are required for the detection of this inherited genetic defect.

Hypothermia[Peds-010A,Peds-010B]

Consensus on Science

There are no randomized pediatric studies on induced therapeutic hypothermia following cardiac arrest.

Two prospective randomized LOE 5 studies of adults with VF arrest[585,586] and 2 prospective randomized LOE 5 studies of newborns with birth asphyxia[587,588] showed that therapeutic hypothermia (32° to 34°C) up to 72 hours after resuscitation has an acceptable safety profile and may be associated with better long-term neurologic outcome.

One LOE 2 observational study[589] neither supports nor refutes the use of therapeutic hypothermia after resuscitation from pediatric cardiac arrest. However, patients in this study were not randomized, and the cooled patients were much sicker and younger than those not cooled.

Treatment Recommendations

Therapeutic hypothermia (to 32°C to 34°C) may be beneficial for adolescents who remain comatose following resuscitation from sudden witnessed out-of-hospital VF cardiac arrest. Therapeutic hypothermia (to 32°C to 34°C) may be considered for infants and children who remain comatose following resuscitation from cardiac arrest.

Knowledge Gaps

Does therapeutic hypothermia improve outcome following pediatric cardiac arrest? Is there a difference in effectiveness for VF arrest versus asphyxial arrest? What is the optimal protocol for cooling after pediatric cardiac arrest (timing, duration, goal temperature, rate of rewarming)?

Vasoactive Drugs[Peds-024A,Peds-024B]

Consensus on Science

There are no studies evaluating the role of vasoactive medications after ROSC in children. Evidence from 2 LOE 3 studies in children,[590,591] 2 LOE 5 studies in adults,[592,593] and 2 LOE 5 animal studies[594,595] documented that myocardial dysfunction and vascular instability are common following resuscitation from cardiac arrest.

Evidence from 6 LOE 5 animal studies[594,-,599] documented hemodynamic improvement when vasoactive medications (dobutamine, milrinone, levosimendan) were given in the post-cardiac arrest

period. Evidence from 1 large LOE 5 pediatric[444] and 4 LOE 5 adult[600,-,603] studies of patients with low cardiac output or at risk for low cardiac output following cardiac surgery documented consistent improvement in hemodynamics when vasoactive medications were administered.

Treatment Recommendations

It is reasonable to administer vasoactive medications to infants and children with documented or suspected cardiovascular dysfunction after cardiac arrest. These vasoactive medications should be selected and titrated to improve myocardial function and/or organ perfusion while trying to limit adverse effects.

Knowledge Gaps

What is the optimal vasoactive drug regimen for postarrest myocardial dysfunction in infants and children?

Glucose[Peds-016]

Consensus on Science

There is insufficient evidence to support or refute any specific glucose management strategy in infants and children following cardiac arrest. Although there is an association of hyperglycemia and hypoglycemia with poor outcome following ROSC after cardiac arrest, there are no studies that show causation and no studies that show that the treatment of either hyperglycemia or hypoglycemia following ROSC improves outcome.

Two studies of adult survivors of cardiac arrest, including 1 LOE 5 prospective observational study[604] and 1 LOE 5 randomized controlled trial comparing tight with moderate glucose control[605] observed no survival benefit with tight glucose control. Two studies of tight glucose control in adult surgical ICU patients, including 1 LOE 1 prospective randomized controlled trial[606] and 1 LOE 1 meta-analysis[607] observed reduced mortality with tight glucose control. Two LOE 5 meta-analyses comparing tight with moderate glucose control in adult ICU patients[608,609] and 1 LOE 5 randomized controlled trial comparing tight with moderate glucose control in adult medical ICU patients[610] observed no differences in survival. Three LOE 5 studies of tight glucose control in adult ICU patients, including 1 randomized controlled trial in cardiac surgical patients,[611] 1 multicenter randomized controlled trial in medical and surgical ICU patients,[612] and 1 cohort-controlled study of medical and surgical ICU patients[613] demonstrated increased mortality with tight glucose control.

One LOE 5 randomized controlled trial of critically ill children[614] observed an improvement in inflammatory biochemical markers and reduced ICU length of stay with tight glucose control. One study of tight glucose control of critically ill neonates[615] was terminated early for reasons of futility. Significant rates of hypoglycemia are widely reported with the use of tight glucose control without explicit methodology or continuous glucose monitoring in critically ill neonates,[615] children,[614] and adults.[607,608,612]

Evidence from LOE 5 animal studies of neonatal cerebral ischemia[616] and critically ill adults[617,618] suggest that hypoglycemia combined with hypoxia and ischemia is harmful and associated with higher mortality. Evidence from 3 LOE 5 animal studies[619,-,621] showed that prolonged hyperglycemia after resuscitation is harmful to the brain. One LOE 5 animal study[622] showed that glucose infusion

with associated hyperglycemia after resuscitation worsened outcome, whereas another LOE 5 animal study[623] showed that moderate hyperglycemia managed with insulin improved neurologic outcome.

Treatment Recommendations

It is appropriate to monitor blood glucose levels and avoid hypoglycemia as well as sustained hyperglycemia following cardiac arrest. There is insufficient evidence to recommend specific strategies to manage hyperglycemia in infants and children with ROSC following cardiac arrest. If hyperglycemia is treated following ROSC in children, blood glucose concentrations should be carefully monitored to reduce episodes of hypoglycemia.

Knowledge Gaps

Does the use of "tight" glucose control improve outcome following pediatric cardiac arrest?

Channelopathy[Peds-048A,Peds-048B]

Consensus on Science

In 4 LOE 4 studies[624,-,627] 14% to 35% of young patients with sudden, unexpected death had no abnormalities found at autopsy.

In 7 LOE 3 studies[628,-,634] mutations causing channelopathies occurred in 2% to 10% of infants with sudden infant death syndrome noted as the cause of death. In 1 LOE 3[635] and 2 LOE 4[636,637] studies 14% to 20% of young adults with sudden, unexpected death had no abnormalities on autopsy but had genetic mutations causing channelopathies. In 4 LOE 4 studies,[638,-,641] using clinical and laboratory (electrocardiographic, molecular-genetic screening) investigations, 22% to 53% of first- and second-degree relatives of patients with sudden, unexplained death had inherited, arrhythmogenic disease.

Treatment Recommendations

When sudden unexplained cardiac arrest occurs in children and young adults, a complete past medical and family history (including a history of syncopal episodes, seizures, unexplained accidents/drownings, or sudden death) should be obtained and any available previous ECGs should be reviewed. All infants, children, and young adults with sudden, unexpected death should, if possible, have an unrestricted, complete autopsy, preferably performed by pathologists with training and expertise in cardiovascular pathology. Consideration should be given to preservation and genetic analysis of tissue to determine the presence of a channelopathy. It is recommended that families of patients whose cause of death is not found on autopsy be referred to a healthcare provider or center with expertise in cardiac rhythm disturbances.