Therapeutic Hypothermia Following Cardiac Arrest

Cassandra Patrick

Otterbein University, cassandra.patrick@otterbein.edu

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Therapeutic Hypothermia Following Cardiac Arrest

Casey Patrick, RN, BSN, CCRN
Otterbein University, Westerville, Ohio

Pathophysiology

The pathophysiology of brain injury following cardiac arrest is extremely complex. Due to the high metabolic demand, the brain is very susceptible to damage from deprivation of blood supply (Burt & Greer, 2010). Hypothermia is induced by the brain’s inability to recover from ischemic insults, which leads to death. The pathophysiology of brain injury following cardiac arrest and subsequent hypothermia-injured brain, as described by Burt and Greer (2010), involves:

- **Intracranial hypertension**
- **Hypoperfusion**
- **Inflammmation**
- **Ischemia-Reperfusion injury**
- **Hypoxic-Ischemic injury**
- **Excitotoxicity**
- **Mitochondrial damage**
- **Neuroprotection**
- **Sepsis**
- **Hypothermia**
- **Temperature regulation**
- **Hypotension and profuse diuresis**

Therapeutic Hypothermia (TH) is the only intervention shown to improve neurological outcomes following cardiac arrest (Lundby, Dym, & Hinsen, 2013). As described by Lundby, Dym, and Hinsen (2013), hypothermia is subdivided into four stages: (1) mild hypothermia (32°C to 34°C), which is estimated to occur out of hospital cardiac arrest (OHCA) patients survived and over 90% having good neurological outcomes at long term follow-up. Therapeutic hypothermia has results that can greatly impact the outcome of such a devastating pathophysiological injury.

**Eligibility Criteria**

Table 3 describes the eligibility criteria for therapeutic hypothermia (Malhotra et al., 2013).

<table>
<thead>
<tr>
<th><strong>Eligibility Criteria</strong></th>
<th><strong>Inclusion Criteria</strong></th>
<th><strong>Exclusion Criteria</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Initial rhythm of ventricular fibrillation (VF) or pulseless ventricular tachycardia (VT)</strong></td>
<td>- Adipose tissue</td>
<td>- Allergy to intravenous catheters</td>
</tr>
<tr>
<td><strong>Cardiac arrest related to trauma</strong></td>
<td>- Major head injury</td>
<td>- Head injury with severe depression or skull fracture</td>
</tr>
<tr>
<td><strong>Evidence of neurological response to commands after resuscitation</strong></td>
<td>- Thermal instability</td>
<td>- Seizure activity</td>
</tr>
<tr>
<td><strong>Hypothermia (32°C to 34°C)</strong></td>
<td>- Severe metabolic acidosis</td>
<td>- Contraindication to deep hypothermia</td>
</tr>
</tbody>
</table>

**Complications**

Table 4 briefly lists the possible adverse effects of hypothermia treatment as described by Malhotra et al. (2013).

<table>
<thead>
<tr>
<th><strong>Complications</strong></th>
<th><strong>Adverse Effects</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypertension</td>
<td>- Blood pressure (SBP &gt; 90 mmHg) despite one vasopressor for &gt;30 minutes</td>
</tr>
<tr>
<td>Hypothermia</td>
<td>- Severe metabolic acidosis</td>
</tr>
<tr>
<td>Hypothermia</td>
<td>- Severe hypotension and profuse diuresis</td>
</tr>
</tbody>
</table>