How Cool is Cool?

Evidence-based practice for whole body hypothermia in neonates suffering from Hypoxic Ischemic Events.

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HIE Facts

HIE is a primary cause of devastating neurological deficits in children.

Systemic asphyxia occurrence rate is 2-4:1000 live births.

Crisis of cerebral blood flow/oxygenation in the setting of systemic asphyxia leads to injurious destruction of neurons that occurs in two phases.

20%-50% of infants with HIE die in the neonatal period.

As many as 25% of survivors have severe morbidity: epilepsy, cerebral palsy, with/without mental retardation and learning disabilities.
HIE Facts

Shankaran (2005)

- Further breaks down statistics to those with moderate HIE have 10% risk of death, 30% risk of disability.

- Those with severe HIE have a 60% mortality rate, and nearly all have disabling neurological dysfunction.
Events Preceding HIE

- Significant Obstetric Events
  - Uterine rupture
  - Placental Abruption
  - Cord prolapse or prolonged compression
  - Impaired placental gas exchange
  - Prolonged birth resuscitation
  - Twin-Twin transfusion syndrome
  - Evidence of other organ dysfunction (liver, kidneys)
Initiating Therapy

- On-site medical provider recognizes the birth events and clinical presentation during and immediately following resuscitation as possible early HIE.

- On-site provider contacts regional center to confirm inclusion criteria is met and receiving provider initiates transport sequence.
TRIAGE OF PATIENTS TRANSFERRED FOR COOLING FOR HIE

Time is vital for infants with possible HIE. The longer cooling is delayed, the less benefit that can be expected from cooling. Cooling must start by 6h of life, but sooner is better.

The objective of this triage plan is to streamline the identification of subjects who are eligible for cooling, and to minimize the delay between diagnosis and the start of cooling (either active or passive).

The practitioner taking the transport call should complete the triage checklist with the referring physician.

We recommend that,
1. All infants who meet clinical ("A") criteria be urgently transferred to UCDMC
2. If "A" and "B" criteria are meet, the baby is eligible for cooling. Passive cooling should be started and the referring hospital and continued during transport. Active cooling will be started at UCDMC.
3. If "B" criteria are not meet the baby may still be eligible for cooling if the aEEG is abnormal. The baby should not start cooling but should be referred urgently for an aEEG.

INFANT CRITERIA
   Gestational age ≥36wks
   Age ≤ 6h

A. CLINICAL CRITERIA
   Apgar ≤ 5 at 10mins of birth
   or Need for ongoing resuscitation (ETT or BMV) at 10mins of birth *
   or Umbilical cord (A or V) pH < 7.00 within 60mins of birth
   or Umbilical cord (A or V) BE > 16 within 60mins of birth

* Qualifies if reason for ongoing resuscitation or assisted ventilation is apnea or poor respiratory effort, not if the reason is a primary lung disease.
Triage Checklist

NAME: ____________________________
MRN: ____________________________
Date and time of Birth: _________ h _____ / ____ / _____

Referring Physician: ____________________________
Referring Hospital: ____________________________

Is infant ≥ 36 wk and age < 6 h?  
Yes ☐  No ☐

If "Yes" proceed to "A", if not = baby NOT eligible for therapeutic hypothermia

A. CLINICAL CRITERIA

Need 1 “yes” to proceed
Apgar score @ 10 minute < 5?  
Yes ☐  No ☐
Needed assisted ventilation (ETT or BMV) 
at 10 minutes for reduced respiratory effort?  
Yes ☐  No ☐
Blood gas pH < 7.00 within 60 minutes of birth?  
Yes ☐  No ☐
Blood gas BE < -12 within 60 minutes of birth?  
Yes ☐  No ☐

If 1 or more “Yes” proceed to “B”, if not = baby is NOT eligible for therapeutic hypothermia

B. NEUROLOGICAL CRITERIA

Need 1 “yes” to be eligible for cooling
Altered state of consciousness (lethargy, stupor, coma)?  
Yes ☐  No ☐
Hyperalert?  
Yes ☐  No ☐
Hypotonia?  
Yes ☐  No ☐
Abnormal reflexes (Moro, asymmetric tonic neck reflex)?  
Yes ☐  No ☐
Abnormal papillary reflexes (constricted or dilated)  
Yes ☐  No ☐
Absent/weak suck?  
Yes ☐  No ☐
Clinical seizures?  
Yes ☐  No ☐

If 1 or more “Yes” baby is eligible for therapeutic hypothermia, so start passive cooling and arrange urgent transfer to UCDMC for active cooling

If not, baby is NOT eligible for therapeutic hypothermia YET, but may be qualify if aEEG is abnormal, so arrange urgent transfer to UCDMC for further evaluation, do not start passive cooling
FLOWSHEET FOR ELIGIBILITY FOR COOLING FOR HIE

Gestation ≥ 36wks
Age ≤ 6h

YES

10min Apgar < 5, OR
Ventilation at 10min of age *, OR
pH < 7.00 within 60mins, OR
BE < -12 within 60min

NO

NEED 1 TO ADVANCE

YES

Neurological Examination
- Stupor, lethargy, or coma OR
- Hyperalert OR
- Hypotonic OR
- Abnormal reflexes OR
  (Moro, ATNR, suck, pupillary)
- Weak or absent suck OR
- Seizures (clinical or EEG)

NO

VENTILATED FOR RESPIRATORY
DEPRESSION NOT FOR LUNG DISEASE

NEED 1 TO ADVANCE

YES

QUALIFIES FOR ACTIVE COOLING
Start Passive cooling
- Radiant Warmer off
- Target Temp 33-34°C
- Rectal temp Q15min

NO

DOES NOT QUALIFY FOR ACTIVE COOLING UNLESS aEEG IS ABNORMAL
Consider transfer for aEEG

YES

URGENT TRANSFER TO UCDMC
FOR ACTIVE COOLING

NO

CONSIDER URGENT TRANSFER FOR ASSESSMENT AND aEEG

* ventilated for respiratory depression not for lung disease
HIE Initial Supportive Management

Goals of Referral Center:

- Support all systems
- Restore homeostasis
- Begin passive cooling
Passive Cooling

- Hypothermia should be started ASAP, preferably by 2 hrs of age and certainly before 6 hrs of age.
- Involves removing external heat sources
- Take rectal temperature Q 5 min, inserting thermometer 2-3 cm into the rectum using a flexible thermometer.
- Target temperature is between 33°C and 34°C. Once target temp is reached, an external heat source may be used at lowest setting and rectal temp is checked Q 15 min.
Cooling During Transport

- Passive Cooling continues during transport.

- External heat source (usually the transport incubator) is on manual setting.

- In summer months, when outside temp is very high, passive cooling efforts may be inadequate. Cooling packs may be covered with thin blanket or cloth diaper and placed on baby’s back, arms, legs or head. Start with one cool pack and add additional one or two packs at 15 min intervals if needed to achieve target temp.
Seizure Management

- HIE is the most common etiology of neonatal seizures
- If seizures are absent, prophylactic anticonvulsants provide no beneficial impact on morbidity or mortality, per Cochrane Review.
- Aggressive IV anticonvulsant therapy using Phenobarbital or Fosphenytoin in the setting of clinical or electrographic seizures may provide benefit, but can also damage immature brains.
- Topiramate and Levetiracetam are being studied for use in this population as they are antiepileptic and neuroprotective.
Management of Sepsis with HIE

- Clinical presentation of severe HIE can be comparable to septic shock. Initial investigations are aimed to diagnose and treat both entities.

- Ampicillin and Cefotaxime are the preferred antimicrobials to provide empirical therapy. Cefotaxime has excellent blood-brain barrier penetration and may contribute to neuroprotection while protecting against nephrotoxicity.
Glucose Management

- Lower glucose stores correlates with higher Sarnat scores

- In HIE, glycogen stores are rapidly depleted by anaerobic glycolysis. As a result, glucose production in the liver becomes inadequate to meet cerebral metabolic demands.

- Hyperinsulinemia often accompanies HIE, complicating glucose management by increasing the use of glucose by peripheral tissues instead of the brain.

- Target glucose should be at least 40 mg/dl, and preferably >60 mg/dl.
Cardiovascular Management

- Hypotension can compound cerebral ischemia associated with HIE.
- Goal MAP 40-60 mmHg
- Treat with appropriate blood products if infant is experiencing coagulopathy and with evidence of perinatal events such as placental abruption, severe anemia, and cord accident.
- Closely monitor fluid balance, as too much volume could lead to hypertension—a contributing factor in the event of intracerebral hemorrhage.
Respiratory Management

- Monitor ventilatory status with frequent blood gas analysis with target PaO2 close to 50-100 mmHg, and PaCO2 between 40-55 mmHg.

- Hypocapnia causes hemoglobin to bind more tightly with oxygen, reducing release of oxygen to tissue, leading to increased cerebral hypoxia.

- Hyperoxia increases free radicals, contributing to apoptotic neuronal cell death.
<table>
<thead>
<tr>
<th>STAGE **</th>
<th>Stage 1</th>
<th>Stage 2</th>
<th>Stage 3</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Level of Consciousness</strong></td>
<td>Hyperalert</td>
<td>Lethargic or obtunded</td>
<td>Stupor or coma</td>
</tr>
<tr>
<td><strong>Activity</strong></td>
<td>Normal</td>
<td>Decreased</td>
<td>Absent</td>
</tr>
</tbody>
</table>

**Neuromuscular Control**

<table>
<thead>
<tr>
<th>Muscle Tone</th>
<th>Normal</th>
<th>Mild hypotonia</th>
<th>Flaccid</th>
</tr>
</thead>
<tbody>
<tr>
<td>Posture</td>
<td>Mild distal flexion</td>
<td>Strong distal flexion</td>
<td>Intermittent decerebration (extension)</td>
</tr>
<tr>
<td>Stretch Reflexes</td>
<td>Overactive</td>
<td>Overactive</td>
<td>Decreased or absent</td>
</tr>
</tbody>
</table>

**Complex / Primitive Reflexes**

<table>
<thead>
<tr>
<th>Suck</th>
<th>Weak</th>
<th>Weak or absent</th>
<th>Absent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Moro (startle)</td>
<td>Strong; low threshold</td>
<td>Weak; incomplete; high threshold</td>
<td>Absent</td>
</tr>
<tr>
<td>Tonic Neck</td>
<td>Slight</td>
<td>Strong</td>
<td>Absent</td>
</tr>
</tbody>
</table>

**Autonomic Function**

<table>
<thead>
<tr>
<th>Pupils</th>
<th>Mydriasis</th>
<th>Miosis</th>
<th>Variable; often unequal; poor light reflex; fixed, dilated</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart Rate</td>
<td>Tachycardia</td>
<td>Bradycardia</td>
<td>Variable</td>
</tr>
</tbody>
</table>

**Seizures**

| None | Common; focal or multifocal | Uncommon (excluding decerebration) |


** STAGE 0 = Normal**
Sarnat Scoring

- Stage 1, Mild HIE
  - Occurs within 24 hrs
  - Hyperalert, not sleeping,
  - Hyperreflexic, irritable, normal tone, exaggerated Moro, weak suck
  - Activation of Sympathetic nervous system
  - EEG: no evidence of seizures, normal awake
  - Outcome at 6-12 mos: Good, if did not advance to stage 2 and 3.
Sarnat Scoring

- Stage 2, Moderate HIE
  - Duration: 2-14 days
  - Lethargic
  - Hypotonia of trunk and extremities with myoclonus, strong distal limb flexion
  - Hyperreflexic, with weak brainstem reflexes; Moro and Suck
  - Parasympathetic activation
  - EEG tracing: low voltage, with multifocal seizures occurring later.
  - Outcome 6-12 mos: Good, if EEG and exam are normal by 5 days.
Sarnat Scoring

- Stage 3, Severe HIE
  - Duration: Hours to weeks
  - Coma, Flaccid tone of extremities and trunk with occ decerebrate posture
  - Diminished or absent brainstem reflexes
  - Diminished tendon reflexes
  - No response to stimuli, absent myoclonus
  - Weak or absent spontaneous respirations
  - Severely abnormal EEG
  - Outcome at age 6-12 mos: Poor
Case study

- 41-wk EGA baby, uneventful pregnancy

- Long labor with pitocin augmentation, epidural anesthesia. Sending hospital is 2+ hrs from regional ctr.

- Sudden loss of fetal heartrate leads to emergent C/S (accomplished in 20 min) There is complete placental abruption.

- Baby is transferred, receiving passive cooling. Baby is lethargic/flaccid.

- Initial aEEG tracing is discontinuous. After 4 hrs, tracing is c/w status epilepticus. Baby is given a Phenobarb loading dose.
EXAM
General: Intubated, off sedation
Mental status: Eyes closed, occasional fidgeting movements
Cranial nerves: Pupils are equal, round, and reactive to light. EOM intact with passive head turn. Weak suck reflex.
Sensory exam: No movements to noxious stimuli.
Motor exam: Hypotonic. At rest, arms hang down at sides. Palmar and plantar grasp absent. Moro is absent.

Hypoxic Ischemic Encephalopathy Scoring

<table>
<thead>
<tr>
<th>DAY</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tone</td>
<td>2</td>
<td>3</td>
<td>3</td>
<td>3</td>
<td>3</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>LOC</td>
<td>2</td>
<td>3</td>
<td>2</td>
<td>3</td>
<td>3</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>Seizures</td>
<td>2</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Posture</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Moro</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>-</td>
<td>2</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Grasp</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Suck</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Respiration</td>
<td>3</td>
<td>3</td>
<td>3</td>
<td>3</td>
<td>3</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>Fontanelle</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td><strong>HIE SCORE</strong></td>
<td>14</td>
<td>16</td>
<td>15</td>
<td>14+</td>
<td>15</td>
<td>14</td>
<td>12</td>
</tr>
</tbody>
</table>

**HIE Chart:**

<table>
<thead>
<tr>
<th>Tone</th>
<th>Normal</th>
<th>Hypertonic</th>
<th>Hypotonic</th>
<th>Flaccid</th>
</tr>
</thead>
<tbody>
<tr>
<td>LOC</td>
<td>Normal</td>
<td>Hyperalert</td>
<td>Lethargic</td>
<td>Coma</td>
</tr>
<tr>
<td>Seizures</td>
<td>None</td>
<td>Infrequent (&lt;3)</td>
<td>Frequent (&gt;3)</td>
<td>Decerebrate</td>
</tr>
<tr>
<td>Posture</td>
<td>Normal</td>
<td>Fisting / cycling</td>
<td>Strong distal flexion</td>
<td>Absent</td>
</tr>
<tr>
<td>Moro</td>
<td>Normal</td>
<td>Partial</td>
<td>Absent</td>
<td>Absent</td>
</tr>
<tr>
<td>Grasp</td>
<td>Normal</td>
<td>Poor</td>
<td>Hyperventilation</td>
<td>IPPV (ventilator)</td>
</tr>
<tr>
<td>Suck</td>
<td>Normal</td>
<td>Poor</td>
<td>Brief apnea</td>
<td>Tense</td>
</tr>
<tr>
<td>Respirations</td>
<td>Normal</td>
<td>Full not tense</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fontanelle</td>
<td>Normal</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Prognosis:** Determined by highest score in first 7 days of life

<table>
<thead>
<tr>
<th>Severe</th>
<th>Died</th>
<th>Survived</th>
<th>Convulsions</th>
<th>Devel Delay</th>
<th>Cerebral Palsy</th>
</tr>
</thead>
<tbody>
<tr>
<td>1-10</td>
<td>2%</td>
<td>98%</td>
<td>7.1%</td>
<td>7.7%</td>
<td>2.9%</td>
</tr>
<tr>
<td>11-14</td>
<td>44%</td>
<td>56%</td>
<td>63.6%</td>
<td>63.6%</td>
<td>36.4%</td>
</tr>
<tr>
<td>15+</td>
<td>93.8%</td>
<td>6.3%</td>
<td>100%</td>
<td>100%</td>
<td>100%</td>
</tr>
</tbody>
</table>

HIE Pathophysiology

- Initial Phase
  - Starts at time of insult.
  - ATP and Phosphocreatine are rapidly depleted
  - Depolarization of cells spreads causes cytotoxic edema and free radical formation
  - Imbalance of ions causes calcium to rush into cells, activating destructive enzymes
  - If circulation/oxygenation are restored, the metabolic dysfunction recovers in 30-60 min.
HIE Pathophysiology

- Latent Phase
  - Occurs from time of resolution of the initial phase until 6 hrs after asphyxia event.
HIE Pathophysiology

- Second phase of energy failure
  - Starts from 6-15 hours post asphyxia event—may last up to 72 hrs after initial injury event
  - Reperfusion injury stage: inflammatory response that leads to cell damage or death.
- Seizures
- Clinical neurological exam deteriorates
- The degree of deterioration in this phase is closely associated with the degree of disability post recovery and survivability
Neuronal Cell Death and Apoptosis

Necrotic cell death is due to acute injury, due either to trauma or hypoxia.

Apoptotic cell death is programmed cell death and occurs as healthy cells become aged or damaged by microbial or chemical substances.
Apoptosis

Cell death due to apoptosis is the focus of cooling treatment for HIE.

The premise is to reduce the "ripple" effect of collateral cell damage/death.

A simplified explanation is that cooling reduces tissue metabolic rate and slows the rate of cell destruction.
Admission

Baby is placed on pre-cooled cooling blanket in open radiant warmer that is turned off.

VS Q 1hr for 4 hr, or until stable, then Q 2hr. If UAC or PAL is present, BP is continuous, otherwise Q 1hr

Daily wt and daily head circumference

Foley is placed, PIV placed (if no UVC), OG to gravity

IV fluids: D 10W @ ~ 65 ml/kg/day

Central access lines or PAL will have NS with Heparin TKO
Admission

Meds:
- Morphine infusion is initiated at 10-20 mcg/kg/h, plus PRN dosing 0.05 mg/kg Q 4hr for shivering or pain.

Labs:
- CBC w/diff, PTT/INR, D-dimer, fibrinogen
- CMP, direct bilirubin, GGT, ammonia, lactate
- Troponin-1, whole blood glucose, blood culture
- Urinalysis, blood gas, ionized calcium
Admission

Rectal temp probe is placed as soon as possible for continuous monitoring. The cooling blanket’s set point is 33.5°C.

Apply ECG electrodes for continuous monitoring.

Apply aEEG electrodes. (We use needle electrodes, the other option is hydrogel electrodes.) Shaving may be necessary to reduce impedance with sticky electrodes—and is sometimes controversial.
aEEG (amplitude EEG)

Provides a compressed view of the variation in amplitude of the EEG waveforms.

1. Background Activity (depicts overall brain function)
2. Interburst intervals (depicts level of brain development)
3. Periodic variation in background activity due to sleep/wake cycles.
4. Seizure activity
aEEG

- Background activity—
  - Note the lower edge of the tracing and compare to numbers on left of monitor screen. The desired level of lower voltage should be \( \geq 5 \). The upper level of the tracing consistent boundary should be near 10.
  - The overall background pattern varies with gestational age. Term infants typically have a wide background tracing. The more immature the infant, the more narrow is the background tracing.
aEEG—Interburst Interval

Between the spikes of amplitude (bursts), there are low amplitude waves, showing that the brain has suppressed cortical activity. The younger the infant, the greater the suppression.

Suppression is measured in percent and ranges from 0-100. Babies who are less than term CGA usually have 50-60% suppression.
aEEG—Sleep/Wake Pattern

- The background pattern should be examined to show variation in baseline and amplitude that is rhythmic, indicating cycling of sleep.
- Good sleep/wake variation is a good indication of normal EEG in the newborn.
- As the brain matures, more quiet sleep (lower baseline, but still above 5 microvolt and a wider waveform)
aEEG--Seizures

- Infantile seizures are usually difficult to discern clinically, but can be confirmed on an aEEG.

- As seizures are treated, the clinical manifestations may disappear, but the electrical seizure activity persists and is evident on the aEEG monitor.

- Seizure activity is marked by an abrupt upward shift in the baseline.

- The seizure duration can be estimated by using the time marker at the bottom of the monitor screen.
aEEG

Abnormal findings:

1. No sleep/wake cycles

2. Upper and lower bandwidth are found outside of acceptable margins (≥10 for upper, ≥5 for lower)

3. Increased or reduced variability

4. Minimal appearance of electrical activity
aEEG

While aEEG is very useful in identifying abnormal background patterns and documents the onset and duration of seizures, it has not been proven to be a reliable indicator of the stage of encephalopathy, or in predicting death and disability in infants with HIE.
Sample aEEG—abnormal/discontinuous
Seizures

Seizure events that are only detected on the EEG monitor are common in infants and small children.

As many as 46% of critically ill children have seizures. 70% of these children have only non-convulsive seizures.
Seizures

Severe seizures are more difficult to treat.

Neonatal seizures are commonly refractory to first and second line anticonvulsants.
Discerning Seizures on aEEG

- Typically, both the upper and lower margins of the tracing are elevated from their previous baseline.
- Duration of true seizures last at least 10 seconds.
- Placement of aEEG electrodes interprets signals from the most common areas of brain injury in HIE, but it does not “see” the entire brain.
- Signals from injured areas that are relatively far away from the electrode are not easily perceived on the aEEG.
Discerning Seizures on the aEEG

- Commonly, seizures will have a rhythmic signal output and the tracing will show one type of wave pattern.

- Seizures that are close to 10 seconds duration or those that produce a low amplitude tracing are also difficult to recognize on the aEEG.
Discerning Clinical Seizures

- Newborns are very different from children and adults in the way they present clinically with seizures.

- Often there are no overt clinical signs of seizure—these seizures are detected electrographically only.
Clinical Seizure Signs

- Apnea, desaturation episodes
- State change
- Sucking movements or tongue fasciculations
- Lip smacking
- Nystagmus or diverted eye gaze
- Myoclonic jerking that is not suppressible
- Posturing
Adverse Affects of Cooling

- Subcutaneous fat necrosis—presents as reddened, firm indurated area of skin with previous contact with cooling blanket.

- Blood is shunted away from skin and can lead to inflammation of granulation tissue and necrosis of fat.

- Care measures:
  - Frequent skin inspection
  - Frequent repositioning—avoid facial contact with cooling blanket
  - Protect skin from prolonged exposure to moisture. Place one blanket between baby and cooling blanket
  - Subcutaneous fat necrosis often resolves without lasting effects

- Frequent repositioning—avoid facial contact with cooling blanket
- Protect skin from prolonged exposure to moisture. Place one blanket between baby and cooling blanket
- Subcutaneous fat necrosis often resolves without lasting effects
Re-Warming

- Before re-warming starts, laboratory tests are done to measure coagulation stability, CBC, electrolytes with liver panel, ammonia, lactate, blood gas, glucose, Troponin-1, magnesium.

- We have adjusted our protocol to repeat only the lab tests that had previous abnormal results in 12 and 24 hr intervals during the re-warming phase.
Re-Warming

- Baby remains on aEEG
- Skin integrity is checked Q 2hr
- Pain assessment is continuous and baby is monitored for shivering.
- Blanketrol maximum gradient is set at 8°C
- Temperature is slowly increased over 4-12 hrs until infant temp is normalized.
Follow Up

• Important to follow HIE babies post discharge

• Many states have a system in place to follow developmentally fragile babies and children

• California has regional centers that monitor children at risk for developmental delay, and High Risk Infant follow up clinics that provide neurodevelopmental testing, teaching of family/caregivers and community resource referral.
UCDMC HIE Outcomes

- Previous 2-yr period
- 31 babies with follow up documentation beginning at 9 months post birth and continuing to 36 months
- Babies are tested and evaluated using the Bayley Score of Infant Development, or Mullen Scales of Early Learning.
UCDMC Outcomes

- Demographic variance:
- Ethnicity:
  - 10 Caucasian
  - 9 Hispanic
  - 5 Asian
  - 4 Other
  - 1 Indian
  - 1 Hmong
  - 1 undeclared
UCDMC Outcome

- Demographic variance:

- Education level of mother:
  - 3 post Graduate
  - 3 BA
  - 8 some college
  - 6 HS
  - 2 <HS
  - 9 unknown
UCDMC Outcome Data

- Of 31 babies, 19 were vaginal births, 12 C/S
- Maternal Age range: 18-37
- There were 2 deaths prior to 2 weeks of age.
- 11 babies showed definitive signs of neurodevelopmental impairment.
  - Four of these babies were demonstrating global developmental delay with spastic quadriparesis,
  - one child required speech therapy for mild expressive language delay,
  - one had speech delay,
  - one received PT until was able to walk.
- 15 babies were testing within normal range for age.
UCDMC Outcome Data

- Of the 31 babies captured in our outcome documentation, 15 experienced seizures during their aEEG monitoring phase.

- 10 babies, during the initial phase of aEEG monitoring, were demonstrating abnormal background pattern.

- 15 babies (including the above 10) showed severely abnormal aEEG tracing.

- 15 babies showed clinical signs of seizures. (Some overlap with above 15).

- 5 babies had seizures that were intractable.
MRI results at 7 days

- Of 31 babies:
  - 4 babies with severe HIE
  - 4 with moderate HIE
  - 3 with mild/moderate HIE
  - 6 with mild HIE
  - 11 were normal
  - (3 no data)
Follow Up Goals

- Detect neurological impairment or confirm normalcy
- Promote early intervention
- Inform parents/caregivers
- Discern changes in outcomes
Other long-term concerns

- Visual impairment—due to damage to the primary visual cortex. Ophthalmology exams can discern optic atrophy or optic nerve hypoplasia
- Sensorineural hearing loss
- Epilepsy
- Microcephaly
Four-month Observations

- Head growth
- Oral/motor function, feeding and swallowing, adequate nutrition
- Tone evaluation of face and mouth, salivary control
- Vocalization attempts
- Visual awareness and motor development
Six-month observations

- Assess balance
- Normalizing muscle tone
- Primitive reflexes should be subsiding
12-24 month observations

- Reliable assessment of oral-motor impairment can be made at this age
- Bayley scales are able to be implemented—measuring cognitive, language, motor and adaptive behavioral scales.
- Early learning programs can be started
3-5 year observations

- Speech/Language assessment is performed.
- Referral to early education program is encouraged if delays are identified.
- Examination of gross and fine motor skills
- Cognitive and adaptive behaviors are evaluated
- Motor skill speed and visual attention
“Take-Home”

- Total prevention or cure of HIE is not currently attainable.
- Cooling is one piece of the complex collaborative approach to care and treatment of HIE.
- Cooling seems to have most positive impact on mild-moderate or moderately severe HIE infants.
- Severely brain-injured infants’ outcomes are not significantly changed by current interventions.
- Mild HIE infants recover with no or minor sequelae with or without cooling therapy.