Diagnostic Imaging
Fetal & Neonatal
Brain Injury
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Head & Neck
Imaging Modalities
- Plain Film / Computer Radiography (PF/CR)
- Ultrasonography (US)
- Computed Tomography (CT)
- Magnetic Resonance Imaging (MRI)
- Nuclear Medicine (NM)
- Angiography
- Myelography
- Interventional

Spine & Cord
Imaging Modalities
- Plain Film / Computer Radiography (PF/CR)
- Ultrasonography (US)
- Computed Tomography (CT)
- Magnetic Resonance Imaging (MRI)
- Nuclear Medicine (NM)
- Angiography
- Myelography
- Interventional
Practice Parameter: Role of Neuroimaging

- Preterm vs. Term Newborn.
- Pattern of Injury – Sensitivity and Specificity.
- Timing Parameters.
- Diagnosis for Immediate Management.
- Prognostic Predictors for Neurodevelopmental Outcome.
- Guide / Assess Preventive, Protective, or Rehabilitative Interventions.


Neuroimaging Modalities

- **US** - Ultrasonography, including Doppler.
- **CT** - Computed Tomography, including multidetector CT (MDCT).
- **MRI** - Magnetic Resonance Imaging.
- **DWI** - Diffusion weighted imaging.
- **DTI** - Diffusion tensor imaging.
- **MRS** - MR Spectroscopy.
- **fMRI** - Functional MRI.


Guidelines by Modality

**US**

- Accessible, portable, fast, real time, multiplanar
- Less expensive, relatively noninvasive (nonionizing radiation)
- No sedation/anesthesia/contrast media
- Available window
- Acoustic reflectance
- Operator / Interpreter dependant
Guidelines by Modality

**US**
- Fetal Screening - MRI
- Neonatal Screening (e.g. premature) - MRI
- Bedside Screening
- Cystic v. Solid
- Hemodynamics (i.e. Doppler – Galen VM)
- ECMO
- Real-time Invasive procedure Guidance
- Tethered Cord (e.g. dimple) - MRI
- Head & Neck Mass (Thyroid!) - NM

Normal Term infant US

* Warning - Limited Sensitivity / Specificity

Guidelines by Modality

**CT**
- Ionizing radiation (ALARA – Image Gently)
- MultiDetector CT (MDCT)
- Ultra-fast axial acquisition
- Isotropic multiplanar reformatting
- 3D Reconstruction
- Soft tissue, bone, vascular algorithms
- Dynamic imaging (e.g. angiography)
- Portable CT
Guidelines by Modality
MDCT - Brain

- Trauma
- Acute neurologic symptoms/signs
- Term Encephalopathy
- Increased Intracranial Pressure
- Macrocephaly
- Shunted hydrocephalus
- Infection
- Post-operative
- Headache
- Rule out mass pre-lumbar puncture
- Craniofacial Anomalies (e.g. Synostosis)
- Calcification
- Vascular contrast enhancement (CTA, CTV)
- Ventricular / Cisternal contrast enhancement

CT* of term neonate (c), 2 month infant (d), & 2 year child (e) show progress of maturation including myelination.

Guidelines by Modality
Magnetic Resonance Imaging - MRI

- Relatively Noninvasive
- Multiple Techniques/Parameters (T1, T2, FLAIR, GRE, DWI, MRA, MRS, DTI, MTC, PMRI, Gadolinium, etc.)
- MultiChannel Technology
- CranioSpinal Imaging
- Fast, Ultrafast, Dynamic Imaging
- Molecular Imaging
- Image-Guided Intervention & Therapy
- MRI Safety Issues – 1.5 T, 3.0 T.
- Sedation & Anesthesia
- Sensitivity / Specificity
- Expense

*Radiation Warning - ALARA - Image Gently
Guidelines by Modality

MRI – Brain

- Term Encephalopathy
- Developmental Delay (Static vs. Progressive)
- Unexplained Seizures (e.g. focal)
- Unexplained Neuroendocrine Disorder
- Unexplained Hydrocephalus
- Unexplained Encephalopathy
- Neoplastic Processes (diagnosis, treatment planning, response, treatment effects)
- Inflammatory Processes (infectious, post-infectious)
- Dysgenesis
- Neurocutaneous Syndrome
- Intractable / Refractory Epilepsy
- Vascular Disease, Hemorrhage, Sequelae of Trauma

MRI (T1) of term neonate (f) & 1 year Infant (g) show progress in brain growth with myelination of the corpus callosum.

- Warnings? - Access, Safety, Expense
Evidence-Based Medicine
Quality of Evidence (QOE)

Diagnosis or Prognosis

- **Class I**: Prospective study, broad case spectrum v. controls; gold standard; blinded evaluation; tests for diagnostic accuracy.
- **Class II**: Prospective study, narrow case spectrum; Retrospective study broad case spectrum; gold standard; controls; blinded evaluation; tests for diagnostic accuracy.
- **Class III**: Retrospective study; narrow case / control spectrum; blinded evaluation;
- **Class IV**: Non-blinded evaluation; expert opinion alone; descriptive case series; no controls.

Crosskerry P. The importance of cognitive errors in diagnosis and strategies to minimize them. Acad Med 2003;78:775-780.


Practice Parameter: Neuroimaging of the Neonate (Evidence-based).

- Quality of evidence (Classes I-IV).
- Diagnostic & Prognostic Ratings (A,B,C,D).
- Neuroimaging Strategies (US, CT, MRI).
- Diagnosis in at-risk newborn for management.
- Detect lesions associated with long-term neurodevelopmental disability.
- Very low birth weight (VLBW) preterm neonates.
- Encephalopathic term neonates.

Practice Parameter: Neuroimaging
VLBW Preterm

- Routine screening US, for whom and when.
- Need for follow-up MRI.
- US - long-term outcome predictor accuracy.
- Risk factors for cerebral palsy (CP):
  1. Grade III-IV Germinial Matrix / Intraventricular Hemorrhage.
  2. Periventricular leukomalacia (PVL).
  3. Ventriculomegaly (VG).


Recommendations: Neuroimaging
Very Low Birth Weight Preterm

- Routine screening US in all infants < 30 wk. GA at 7-14 days of age; repeated at 36-40 wk. PMA.
- Allows detection of early lesions (e.g. IVH) which influence clinical care (level B) and later lesions (e.g. PVL, VG) as predictors of long-term neurodevelopmental outcome (level A).
- Insufficient evidence for routine MRI follow-up of abnormal US (level C) regarding outcome.
- Revision? - MRI (with DTI) at near-term, especially if negative or nonpredictive US*.

[*Test prospectively with multicenter studies.]

VLBW Preterm - Predictors of "CP":
Grade III-IV GMH/IVH (a,b), PVL (c), VG (d)

Grade IV GMH/IVH with periventricular hemorrhagic infarction (a) & post-hemorrhagic hydrocephalus with porencephaly (b).

US - Evolving Cystic PVL
Normal (a), Edema (b), Cysts (c)
[Cystic Phase 2-6 wks.]
Preterm Brain Injury (Extreme, VLBW)
Predictors of “CP”

Higher Grade (III-IV) Germinial Matrix / Intraventricular Hemorrhage.
Post-Hemorrhagic Hydrocephalus.
Ventriculomegaly.

White Matter Injury of Prematurity:
1. Focal / Multifocal Cystic (Periventricular Leukomalacia - PVL).
2. Focal / Multifocal Noncystic (Gliosis).
3. Diffuse White Matter Gliosis.


Neuroimaging Practice Parameter
VLBW Preterm - Revision?

- Routine screening US in all infants < 30 wk. GA at 7-14 days of age; repeated at 36-40 wk. PMA.
- Allows detection of early lesions (e.g. IVH) which influence clinical care (level B) and later in clinical care (level B) and later.
- Insufficient evidence for routine US in all infants < 30 wk. GA at 7-14 days of age; repeated at 36-40 wk. PMA.
- Diffuse white matter gliosis.

Largest extreme preterm cohort with near-term MRI and serial US, compared with early (1-4 weeks postnatal age) and late (34-42 weeks PMA) US in 480 infants (mean 25.9 weeks EGA). All had late US and MRI within 2 weeks of each other. Independent MRI and US central readers were masked to the clinical and other neuroimaging findings.

- Results: 306 (89.5%) of 342 infants with normal late US had normal or mild white matter abnormalities (WMA) on MRI; 36 (10.5%) of them had moderate/severe WMA. All 18 infants with late US findings of moderate/severe ventriculomegaly (VMG) had MRI findings of moderate/severe WMA, moderate/severe VMG, or cystic lesions. 76% of 46 infants with grade 3 or 4 hemorrhage on early US had moderate/severe WMA on MRI. On MRI, cortical grey matter abnormalities were present in 79 (16.5%) and cerebral grey matter abnormalities were present in 9 (1.9%). Pediatric fosa lesions were seen on US in 1.6% (maximal views in only 50%).

- Conclusions: Largest extreme preterm cohort with near-term MRI and serial US. The presence of moderate/severe WMA on brain MRI, similar to previous reports. Cerebellar abnormalities were detected more frequently by MRI than by US. Neurodevelopmental outcomes at 18-22 months and school age will assess the relative and combined values of MRI and US as outcome predictors.
Brain MRI & Cranial US of Extremely Preterm Infants: The Neuroimaging and Neurodevelopmental Outcomes (NEURO) cohort

SR Hintz, PD Barnes, LA Wrage, D Bulas, TL Slovis, A Das, N Finer, H Cheng, RD Higgins, for the SUPPORT Subcommittee and the NICHD Neonatal Research Network

Summary

- In the NICHD Neonatal Research Network NEURO extremely preterm cohort (<28 week EGA):
  - The rate of moderate-severe white matter abnormalities by near-term brain MRI was consistent with previous published studies.
  - Cerebellar lesions were not uncommonly seen by brain MRI, but infrequently detected by CUS.

Sneak Preview - Summary

- Near term brain MRI findings, including cerebellar lesions and increasing severity of WMA, were associated with neurodevelopmental outcomes.
- Brain MRI findings were independently associated with adverse outcomes, independent of adverse early CUS and late CUS and other factors.
- Late CUS remained independently associated with death or NDI in full models; predictive accuracy was marginally improved with addition of MRI.
Practice Parameter: Neuroimaging
Term Encephalopathy

- Which modalities provide clinically important diagnostic information.
- Which modalities provide prognostic information.

Recommendations: Neuroimaging
Term Encephalopathy

- Nonenhanced CT for birth trauma, low hematocrit, or coagulopathy, to rule out hemorrhage (level B).
- If CT noncontributory, conventional MRI between days 2 and 8 for pattern of injury for both diagnosis and prognosis (level A).
- DWI may allow earlier detection of injury (level C).
- In addition to conventional MRI (level A), DWI (level C) or H1-MRS may provide prognostic information for outcome.
- Revision: DTI: microstructural injury?


Term Trauma: CT - Hemorrhage
Term Trauma: CT - Depressed Skull Fracture

Term Encephalopathy
Differential Diagnosis

- Hypoxia-Ischemia (HIE)
- Infection (including FIMS?)
- Trauma (including uterine hyperstimulation?)
- Hypoglycemia
- Hypothyroidism
- Metabolic Disorders (including C.D.E.Copper deficiencies)
- Coagulopathy
- Arterial Thrombosis (embolic, thrombotic, stenotic)
- Venous Thrombosis (e.g., thrombophilia)
- Galenic AV Malformation
- Multifactorial?
- Prior remote prepartum injury?
- HIE with negative/nonspecific imaging?

Term HIE: US - Normal (a), Edema (b), Doppler (c)
Term HIE: CT - Normal (a) vs. Edema (b)

Low Radiation Dose CT

Term Hypoxic-Ischemic Encephalopathy (HIE):
Predictors of “CP”

- **Doppler US**: Resistive indices (RI) < 0.5 – 0.6.
- **CT**: low attenuation basal ganglia / thalami.
- **MRI**: Characteristic T1 and T2 abnormalities, especially in basal ganglia / thalami.
- **DWI**: Restricted diffusion.
- **H1 MRS**: elevated lactate, decreased NAA.

Term HIE: Pattern of Injury

- **Factors**: severity, duration, GA.
- **Profound**: basal ganglia, thalami, midbrain, vermis, hippocampi, pericerebral cortex.
- **Partial prolonged**: watershed / borderzone (cortical / subcortical > periventricular).
- **Combined**: profound + partial prolonged (Total Asphyxia).
- **Reperfusion.

- **White matter injury - old vs. new?
- **Sequela**: atrophy, cystic encephalomalacia, ulegyria, gliosis, mineralization.
Term HIE: CT - Normal (a) vs. Edema (b)

Term Combined HIE - Total Asphyxia

Neonatal Encephalopathy and Cerebral Palsy
ACOG/AAP 2003 (Updated 2013?)

<table>
<thead>
<tr>
<th>Modality</th>
<th>Finding</th>
<th>Imaging</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fetal MRI</td>
<td>Decreased brain size</td>
<td>Decreased intracranial pressure</td>
</tr>
<tr>
<td>Diffusion MRI</td>
<td>Reduced diffusion</td>
<td>Reduced diffusion</td>
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<tr>
<td>T1-weighted MRI</td>
<td>Increased signal in subcortical areas in basal ganglia</td>
<td>Increased signal in subcortical areas in basal ganglia</td>
</tr>
<tr>
<td>T2-weighted MRI</td>
<td>Decreased signal in subcortical areas in basal ganglia</td>
<td>Decreased signal in subcortical areas in basal ganglia</td>
</tr>
<tr>
<td>CT scan</td>
<td>Decreased density in subcortical areas in basal ganglia</td>
<td>Decreased density in subcortical areas in basal ganglia</td>
</tr>
<tr>
<td>Ultrasonography</td>
<td>Increased echogenicity in subcortical areas in basal ganglia</td>
<td>Increased echogenicity in subcortical areas in basal ganglia</td>
</tr>
</tbody>
</table>

*Table 5-2. Summary of Imaging Findings in Relation to the Time of Birth Injury*

Note: This table is not applicable and may not be relevant given the context. There is a need for clearer, more precise labeling or possibly including more detailed information.
Term HIE: US - Normal (a), Edema (b), Doppler (c)

Edema 1-7 d (peak 2-4d); atrophy > 14d

Term HIE - CT Evolution
Normal (a), Edema (b), Necrosis (c)

Low Radiation Dose CT

Term HIE - CT Timing
Edema 1-7 d (peak 2-4d); atrophy > 14d.
**Term HIE - MRI Timing**

- **DWI**: < 24h - 2w; T1 high: 24h - 2w; T1 low: 2-4d to 1m; T2 low: 6-7d to 1m; atrophy > 14d.
- **MRS**: < 24h - 7d; T2 high: 2-4d to 1m; T2 low: 6-7d to 1m; atrophy > 14d.


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**Future Challenges**

- Minimize Imaging Time
- Control Data: Normal Ranges / Regional Variations / Timing
- Prenatal Imaging
- MRI, NIRS, SPECT, PET
- Molecular Imaging (e.g. Annexin V)
- Multicenter - Prospective Studies (Class I, II).
- Clinical Trials: Neuroprotective Strategies (e.g. hypothermia).
- Controversies (e.g. Uterine Hyperstimulation, FIRS)
- Practice Parameter Modification.

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**Advanced MRI Techniques**:
(a) MR-compatible Incubator, (b) DTI Tractography, (c) fMRI, (d) Spectroscopy.

MRI in a Trial of Therapeutic Hypothermia for Term Hypoxia-Ischemia


Methods: Brain MRI findings were obtained by 44 weeks PMA in 136 infants with moderate-severe HIE who were randomized to cooling (33.5 degrees C for 72 hours). There were 73 in the hypothermia group and 63 in the control group. All MRIs were reviewed by a central reader masked to the clinical findings, groupings, and outcomes. The MRI findings were systematically scored according to pattern and extent of injury. Brain injury scores were correlated with death or disability at 18 months.

Conclusions: Fewer areas of brain injury on MRI were observed following whole-body cooling. The brain injury score is a marker of death or disability at 18 months following hypothermia for term HIE.

MRI in Term Infants with Hypoxic-Ischemic Encephalopathy (HIE) Treated by Hypothermia


Brain MRI findings were obtained by 44 weeks PMA in 136 infants with moderate-to-severe HIE. Fewer areas of brain injury on MRI were observed following whole-body cooling. The brain injury score is a marker of death or disability at 18 months following hypothermia for term HIE.

Conclusions

- Trend for more normal MRIs among cooled infants (52%) compared to control infants (35%).
- Trend for fewer deaths & less severe disability among cooled infants (22/73 - 30%) compared to control infants (30/63 - 48%).
- NICHD pattern of brain injury is a marker of death or moderate-to-severe disability (18-24 months) for term HIE (cooled or not).
- NICHD pattern of brain injury is a marker of death or moderate-to-severe disability (18-24 months) for term HIE (cooled or not).
Hypothermia Summary Score (SS) = 0
(normal)
Sag T1 (a), Ax T1 (b), Ax T2(c)

Hypothermia SS = 1A
(minimal cerebral lesions only)
Sag T1 punctate high intensities (a - arrows),
Ax T2 punctate low intensities (b - arrows)

Hypothermia SS = 1B
(more extensive cerebral lesions without other involvement);
Ax FLAIR (a), Cor FLAIR (b) bilateral high intensities (arrows)
Hypothermia SS = 2A
(basal ganglia, thalamic, internal capsule lesions only)
Ax T1 high intensities (a - arrows),
Ax T2 high/low intensities (b - arrows)

Hypothermia SS = 2B
(basal ganglia, thalamic, internal capsule and cerebral lesions)
Ax T1 high intensities (a, b - arrows),
Ax T2 diffuse white matter high intensities (c - arrows)

Hypothermia SS = 3
(cerebral hemispheric devastation)
Ax FLAIR low intensities (a - arrows),
Ax T2 high intensities (b - arrows)
Term Encephalopathy
Differential Diagnosis

- Hypoxia-Ischemia
- Infection (including FRS?)
- Trauma (including uterine hyperstimulation?)
- Hypoglycemia
- Hypothyroidism
- Metabolic Disorders (C, D, E, Copper deficiencies)
- Coagulopathy
- Arterial Spoke (embolic, thrombotic, stenotic)
- Venous Thrombosis (e.g. thrombolic)
- Gynecic AV Malformation
- Multifactorial?
- Prior remote prepartum injury?
- HIE with negative/nonspecific imaging?

Maternal-Fetal-Neonatal Vitamin D Deficiency

- Ziegler EE, Hollis BW. Vitamin D deficiency and skeletal outcomes in infants and young children. Am J Clin Nutr 2008;88:1525S-153S.

Congenital-Neonatal Rickets vs. Abuse

Maternal Vitamin D deficiency

- congenital rickets
- decreased fetal muscle loading
- uterine dysfunction
  - Pitocin induction
  - prolonged labor
  - increase extrinsic forces to fetus
- increased bone fragility

**BIRTH FRACTURES**

Radiographic Findings

**Congenital Rickets**

**Skull**

3m infant with alleged NAI; also, "history" consistent with congenital rickets. Chest film (a) shows bilateral recent and old, healing rib fractures (pseudofractures? rachitic rosary? - arrows). Knee films before (b) & after (c) vitamin D supplementation show "healing" CML (arrows)?

9w infant with Subdural & Retinal Hemorrhage
Alleged Child Abuse
Vitamin D Deficiency & Traumatic labor and delivery.

Mimic - BECC + Hemorrhage
5m infant with the Triad and alleged NAI; also, macrocephaly from birth, recent seizure but “no” trauma. CT (a) and T2* MRI (b) show large extracerebral collections with smaller recent hemorrhages (arrows). CT 3 months post-drainage (c) shows rehemorrhage (arrows). Diagnosis: BECC or chronic SDHG with rehemorrhage?

Child Abuse - The Triad
Differential Diagnosis
- Trauma (AI vs. NAI)
- Hypoxia / Ischemia / Reperfusion
- Birth Injury
- Vitamin Hemorrhage
- Apnea / Choking / Respiratory Arrest
- Infection & Post-infections (e.g. vaccine)
- Status Epilepticus
- Hematologic (Coagulopathies)
- Vitamin Deficiency (C, D, K, Ca)
- Metabolic Disorders (e.g. Galactosemia)
- Vascular / Connective Tissue Diseases (e.g. OI)
- ECMO
- Congenital Heart Disease
- Cervical Spinal Cord Injury
- Multifactorial / Synergistic (including CPR)
- No Body Knows

### Evidence Base SDH - Rebleed
#### Birth Factors

### Evidence Base
#### Benign Extracerebral Collections (B ECC)
- SDH - Rebleed
- Evidence for Evidence for SDH with Rebleed
- - Nonaccidental injury (NAI) and relationship to obstetric and neonatal risk factors. Radiology 2007; 242:535-41 (26%).
**Term Encephalopathy**

**Differential Diagnosis**

- Hypoxia-Ischemia
- Infection (including FIE?)
- Trauma (including uterine hyperstimulation?)
- Hypoglycemia
- Hypothyroidism
- Metabolic Disorders (C,D,E,Copper deficiencies)
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- Arterial Stroke (embolic, thrombotic, stenotic)
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- Multifactorial?
- Prior remote prepartum injury?
- HIE with negative / nonspecific imaging?

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**Term Encephalopathy**

**Congenital Rubella** on CT (a) and T2 MRI (b) including calcifications (short arrows) and leukoencephalitis (long arrows) with white matter low densities and T2 high intensities.
Fetal Inflammatory Response Syndrome (FIRS) 

Preterm  Term  2 yr.

High rate maternal infection preterm & term - White Matter (42%), BG/Thal (12%), Cort / Subcort (9%), Foc Infarcts (7%).

Hypoglycemia in the subacute phase (arrows - a,b,c) and chronic phase (d,e).

Billirubin Encephalopathy & Kernicterus with globus pallidus and subthalamic involvement.
Sulfite Oxidase Deficiency

Maple Syrup Urine Disease

Term Trauma: CT - Hemorrhage
Timing of Hemorrhage
CT Density Evolution of Hemorrhage
- Hyperacute: Isodense <3 hours
- Acute: Hypodense Few hours → 7–10 days
- Subacute: Isodense 2–3 weeks
- Chronic: Hypodense >3 weeks

Timing of Hemorrhage
MRI Signal Evolution of Hemorrhage.
- Hyperacute: <12–24 h T1↑ ↔ T2↑
- Acute: 1–3 days T1↑ ↔ T2↓
- Early subacute: 2–3 days → 1–2 weeks T1↑ T2↓
- Late subacute: 1–2 weeks → 1–2 months T1↑ T2↑
- Chronic: Few weeks → months/years T1↑ T2↓
- Chronic: Few weeks → months/years T1↑ (CSF) T2↑

Term Trauma: CT - Depressed Skull Fracture

Vezina G. Assessment of the nature and age of subdural collections in nonaccidental head injury with CT and MRI. Pediatr Radiol. Online 21 March 2009
Venous Thrombosis (Thrombophilias)


- The Canadian Pediatric Ischemic Stroke Registry.
- Incidence of the disorder: 0.67 case per 100,000 children per year.
- Neonates were most commonly affected.
- Fifty-eight percent of the children had seizures, 76 percent had diffuse neurologic signs, and 42 percent had focal neurologic signs.
- Risk factors included head and neck disorders (in 29 percent), acute systemic illnesses (in 54 percent), chronic systemic diseases (in 36 percent), and prothrombotic states (in 41 percent).
- Venous infarcts occurred in 41 percent of the children.
- Neurologic deficits were present in 38 percent of the children, and 8 percent died; half the deaths were due to sinovenous thrombosis.
- Predictors of adverse neurologic outcomes were seizures at presentation and venous infarcts.
- Sinovenous thrombosis in children affects primarily neonates and results in neurologic impairment or death in approximately half the cases. The occurrence of venous infarcts or seizures portends a poor outcome.

Diagnostic Imaging
Fetal & Neonatal Brain Injury

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TORCH Infections
Congenital CMV

Congenital Toxoplasmosis with hydrocephalus (H), porencephaly (P), and calcifications (arrows).

Term Encephalopathy

TORCH Infections
Congenital Toxoplasmosis with hydrocephalus (H), porencephaly (P), and calcifications (arrows).
HSV2 Encephalitis on CT. Subacute phase (a) including hemorrhages (arrows); Chronic phase (b) with encephalomalacia, subdural collections (s), and calcifications (arrows).
Citrobacter Meningitis with contrast-enhancing abscess.

Term Encephalopathy

Citrobacter Meningitis on CT (a,b) with contrast-enhancing abscesses (arrows), ventriculitis, and hydrocephalus.
Methylmalonic Acidemia

Vein of Galen Vascular Malformation.

Post-Parameter: VLBW Preterm Brain Injury
White Matter Injury of Prematurity

- Macrostructural vs. Microstructural Injury.
- Focal / Multifocal (e.g., PVL) vs. Diffuse White Matter Injury (FIRS).
- Immature oligodendroglial and subplate neuronal injury.
- Oxidative stress, excitotoxicity, inflammation, genetic factors.
- Necrosis-apoptosis continuum.

Post-Parameter: VLBW Preterm
Brain Injury – US vs. MRI

- **US**: structural and temporal resolution issues.
- **MRI > US**: macrostructural resolving power

**MRI > US**: neurodevelopmental outcome

Post-Parameter: VLBW Preterm
Brain Injury – MRI > US

Noncystic PVL with foci of gliosis (arrows - a,b) + mineralization or hemorrhages (arrows - c).
Brain Injury – MRI > US

Diffuse PVL (arrows) at near-term (a) & later infancy (b, c).

Diffusion Weighted Imaging (DWI)
Diffusion Tensor Imaging (DTI)

- Molecular diffusion of water within tissues.
- Apparent Diffusion Coefficient (ADC): Quantitation of diffusion in all directions (isotropy).
- ADC: CSF > immature WM > myelinated WM > GM.
- Fractional Anisotropy (FA): Quantitation of Directionality of Diffusion.
- FA: mature WM > immature WM > GM > CSF
- Global vs. Regional variations.


Diffusion Weighted Imaging (DWI)
Restricted Diffusion

Regions of interest (ROI)

Genu
Internal Capsule anterior
posterior
Splenium

DTI – Mechanism for Decreased FA
- Delayed myelination or demyelination.
- Oligodendroglial injury.
- Axonal injury.
- Apoptotic neuraxonal degeneration.
- Lack / Loss of connectivity (synaptogenesis).
Whole-Body Hypothermia for Neonates with Hypoxic-Ischemic Encephalopathy

Case 1 Not Cooled Case 2 Not Cooled

Case 3 Cooled Case 4 Cooled

Moderate hypothermia for neonatal asphyxial Encephalopathy

In animal experiments, induced hypothermia ameliorates the effects of perinatal asphyxia on the brain. The effects on human neonates, however, remain uncertain.

Of two randomised trials, only one showed significant benefit. Now, a multicentre trial has shown that moderate hypothermia improves neurological outcomes in survivors of perinatal asphyxia.

A total of 325 term infants with perinatal asphyxia were randomised at 42 centres in the UK, Hungary, Sweden, Israel and Finland to intensive care with or without hypothermia in the first 72 h. Infants in the hypothermia group were nursed on a thermostatically controlled cooling blanket, and the target rectal temperature was 33–34°C.

Among the 163 infants in the hypothermia group, 42 died, and 32 survived with severe neurodevelopmental disability. Among the 162 infants in the control group, 44 died, and 42 survived with severe disability. Neither of these differences was significant. However, there was a significant 57% increase in probability of survival without neurological abnormality in the hypothermia group. Hypothermia was associated with a significant 33% reduction in risk of cerebral palsy among survivors and significantly improved cognitive, developmental and gross motor function scores at the age of 18 months.

Hypothermia did not reduce the combined outcome of death or severe disability, but it did improve neurological outcomes in survivors.

Role: Central MRI reader/Neuroimaging consultant

This project investigates the value of brain magnetic imaging (MRI) in predicting neurodevelopmental outcome in term infants with hypoxic-ischemic encephalopathy enrolled in a whole body hypothermia trial.