

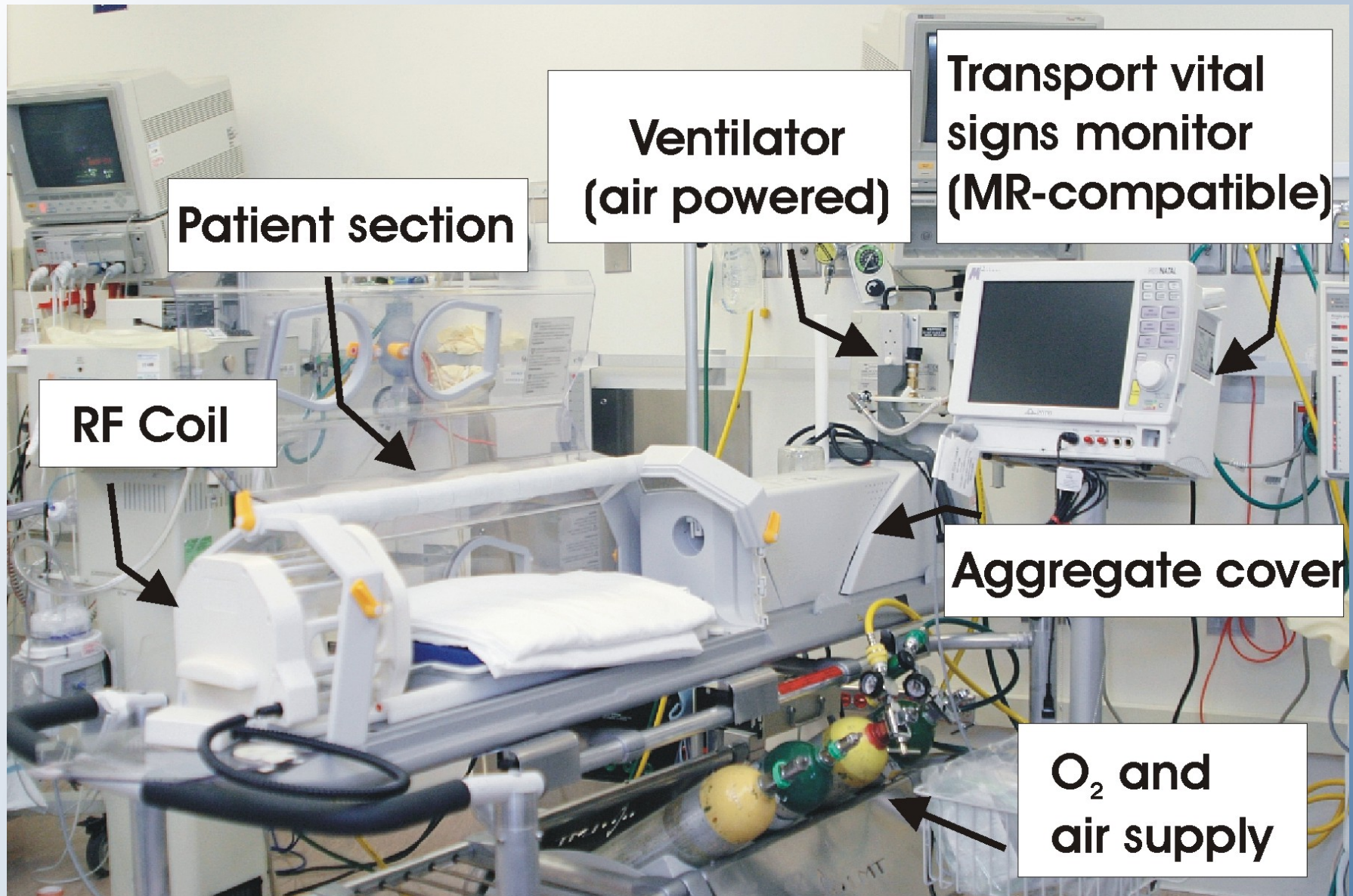
MR IMAGING OF THE TERM INFANT

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Childrens Hospital of Pittsburgh of UPMC
Associate Professor



Presentation Outline

- Technology
- HIE- cooling
- Diffusion imaging /MR spectroscopy
- Perfusion imaging
- Metabolic Disorders
- Infection
- Vascular lesions/tumors
- Malformations



Bluml et al., Radiology 2004 May; 231(2):594-601

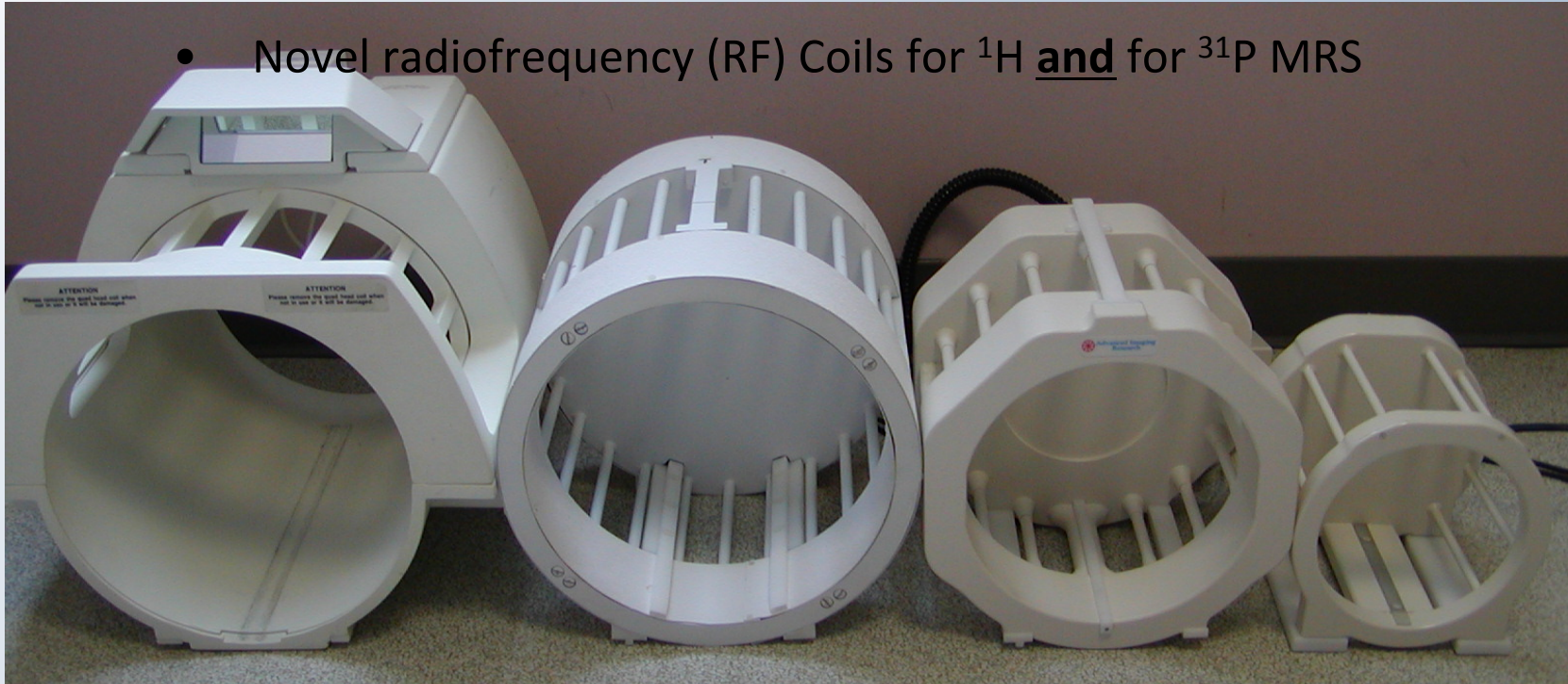
Future Trend- Hammersmith Neonatal Group, Imperial Group were the first!



http://www.european-hospital.com/en/article/523-Neonatal_MRI_of_premature_babies.html

MRI scanner in the NICU

- Novel radiofrequency (RF) Coils for ^1H and for ^{31}P MRS



Standard head coil for MRI and ^1H MRS¹

Dual-tuned coil for MRI, ^1H MRS, and ^{31}P MRS²

Dual-tuned coil for MRI, ^1H MRS, and ^{31}P MRS optimized for children²

Newborn head coil for MRI and ^1H MRS with MR-compatible incubator²

¹General Electric, ²Advanced Imaging Research Inc. (AIRI)

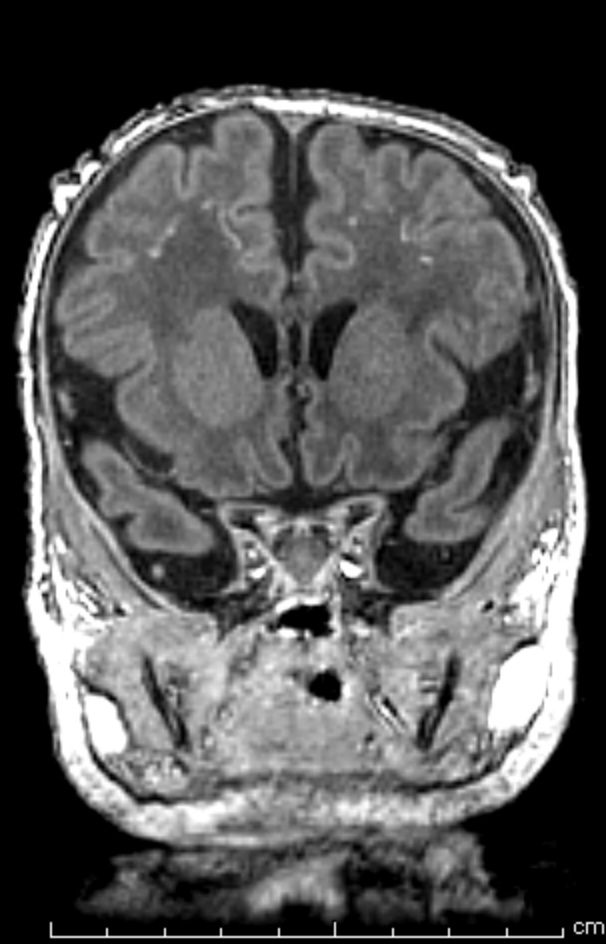
00732

5:1



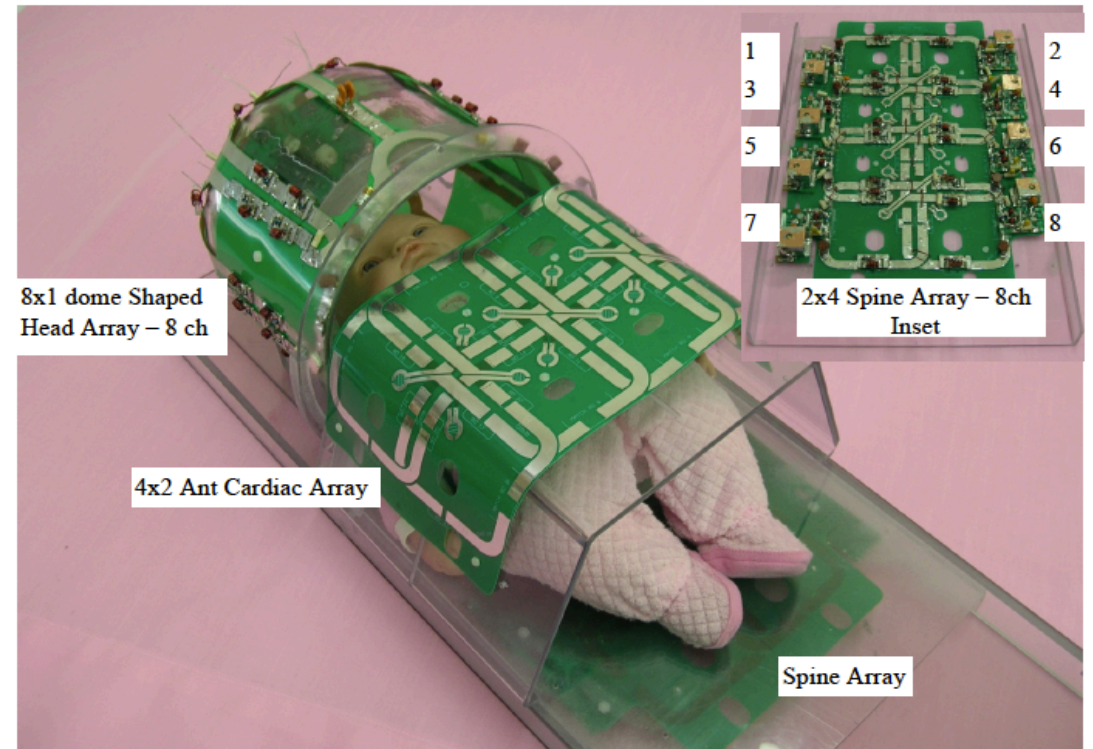
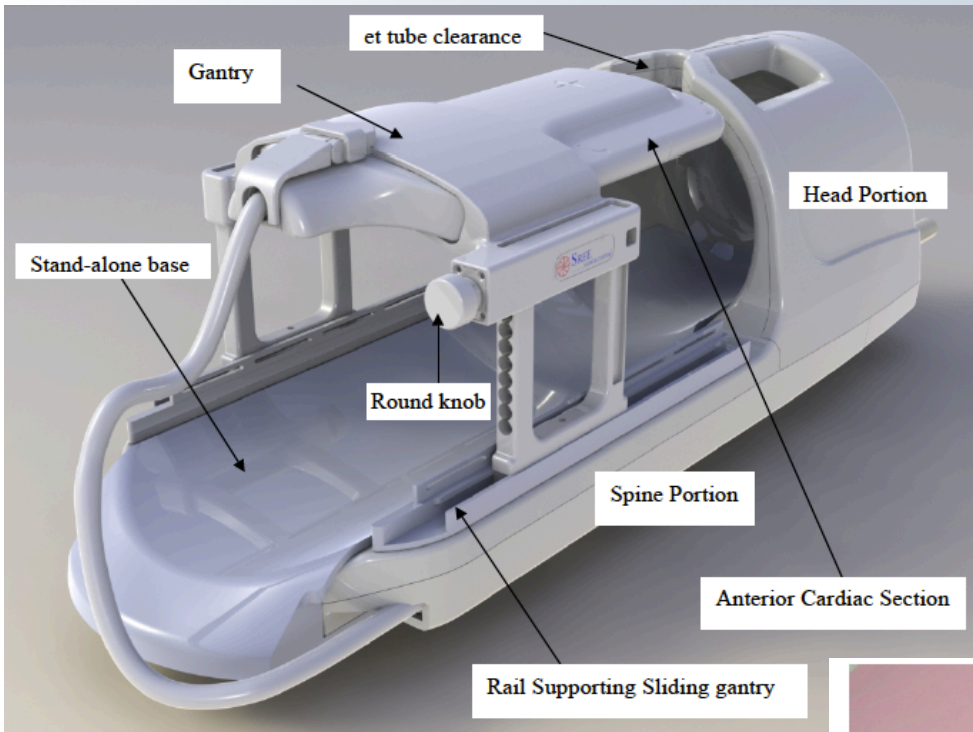
Page: 10 of 23

IM: 10 SE: 5 Page: 61 of 104



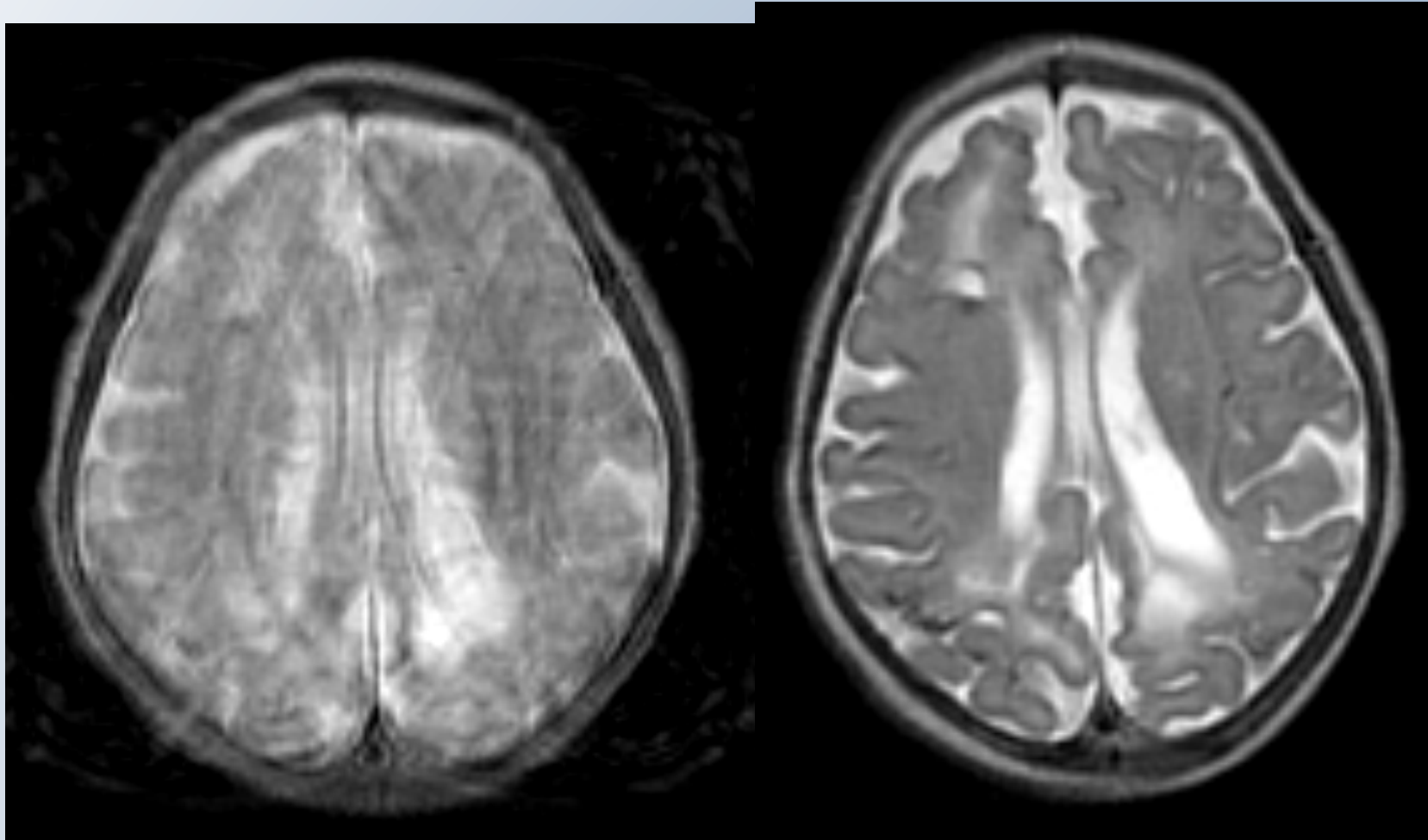
IM: 61 SE: 3

Dr. Ravi Srinivasin Advanced Imaging



PROPELLER IMAGING

Motion Correction Sequence



Clinical Trials to Evaluate Cooling

- Cool Cap Trial (International)
 - Lancet 2005; 365:663-70
- NICHD Whole Body Cooling Trial (US)
 - NEJM 2005; 353:1574-84
- TOBY Trial (UK/Europe/Israel)
- ICE Trial (Australia)



Effect of Hypothermia on cMR

TOBY TRIAL

- Images available from 131 infants
- HYP- decreased lesions in basal ganglia/ thalamus; white matter; PLIC
- The predictive value of MRI for subsequent neurological impairment is not affected by HYP

Effect of Hypothermia on cMR

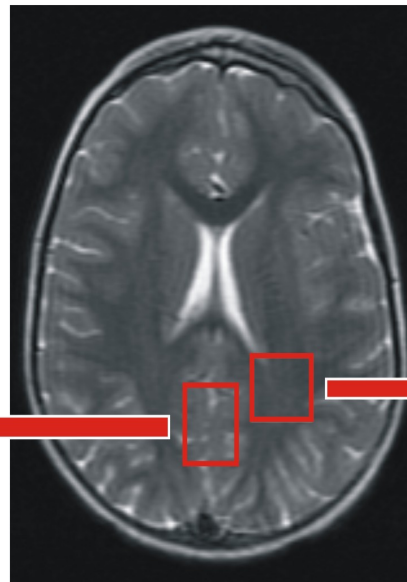
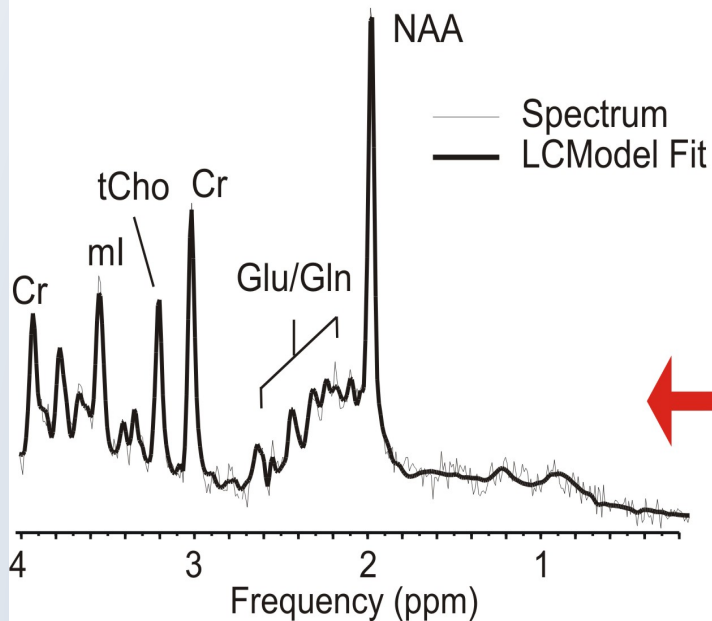
NICHD study

- 151 infants with MRI
- Three scales: Rutherford;Barkovich;NICHD
- Fewer watershed infarcts, less abnormalities in the ALIC and PLIC
- The predictive value of the injury was not altered by hypothermia
- The reduction of areas of brain injury in both groups shows whole body cooling is neuroprotective
- S. Shankaran et al, 2012

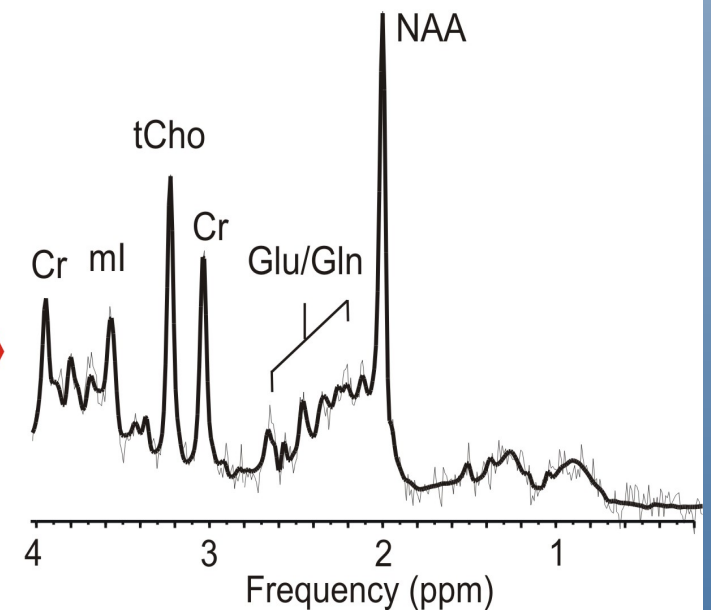
MRI and proton (^1H) MRS

- **MRI** uses the signal from hydrogen protons of water to reconstruct an image
- **^1H MRS** uses the signal from the protons of chemicals to generate a spectrum

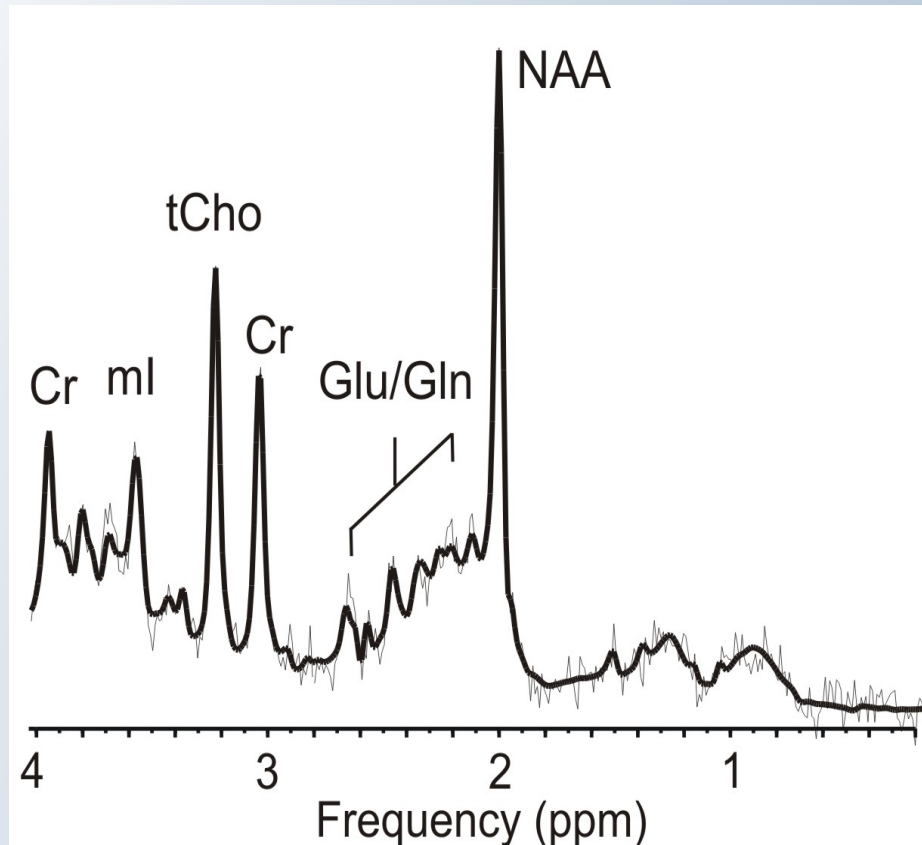
Occipital Grey Matter



Parietal White Matter

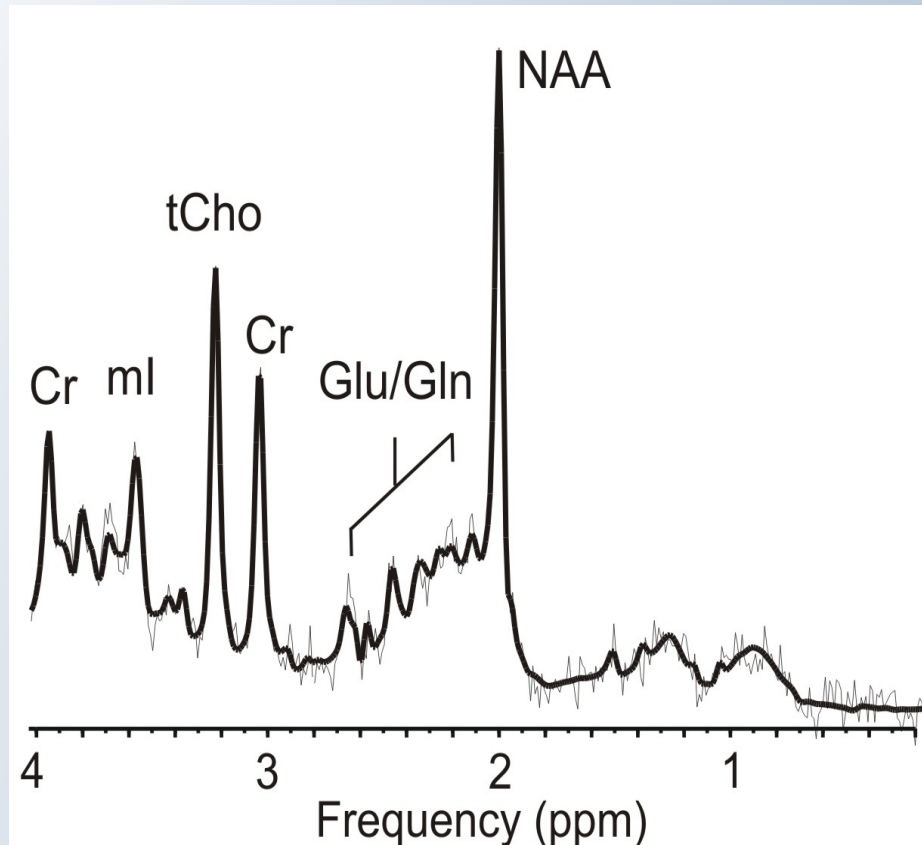


“Normal” proton (^1H) MRS



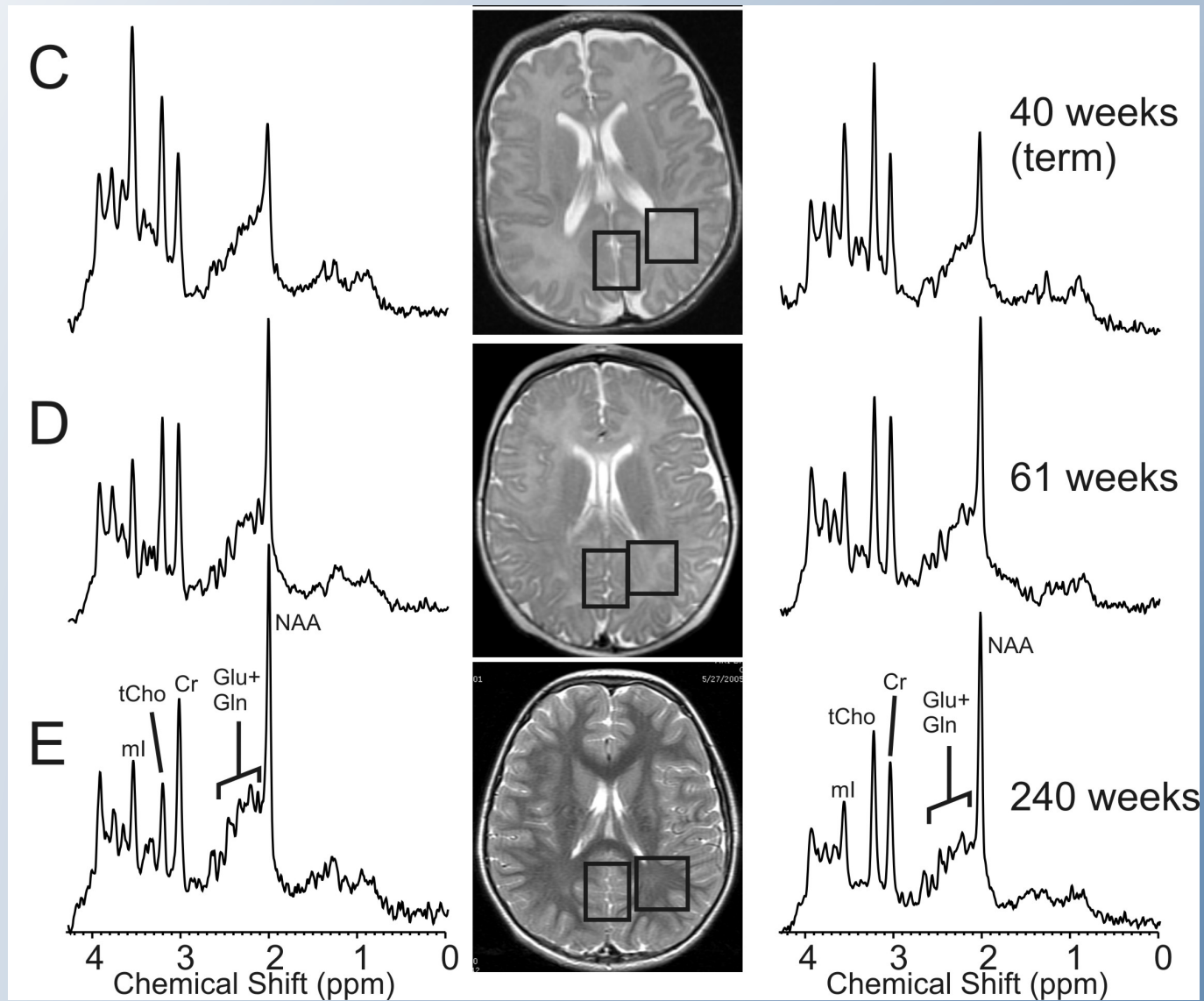
- Position on x-axis relative to a standard (measured in ppm = parts per million) identifies chemicals
- Peak height is proportional to concentration
- MRS is inherently quantitative (mmol/kg tissue)

“Normal” proton (^1H) MRS

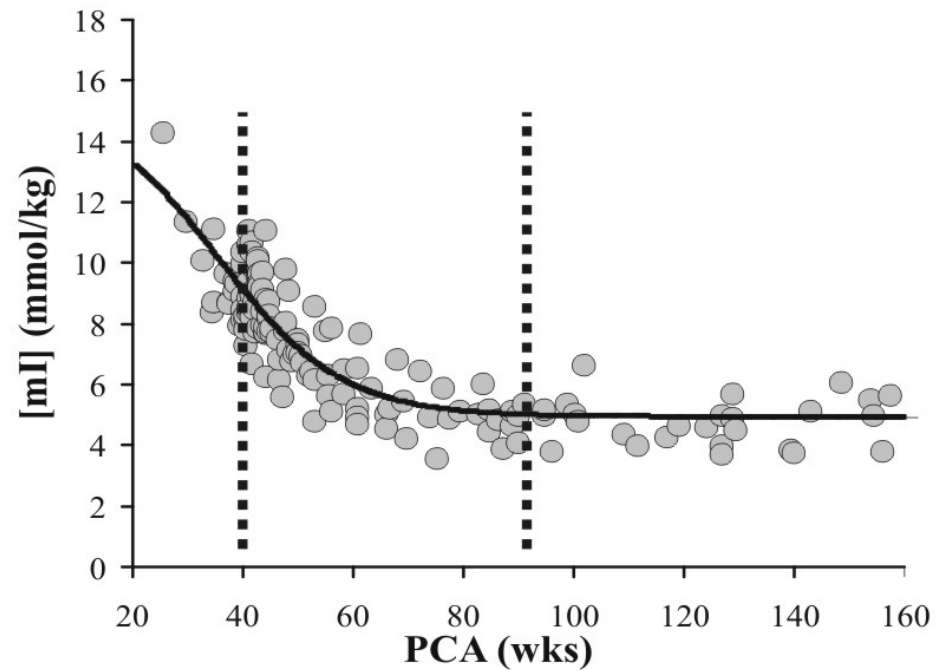
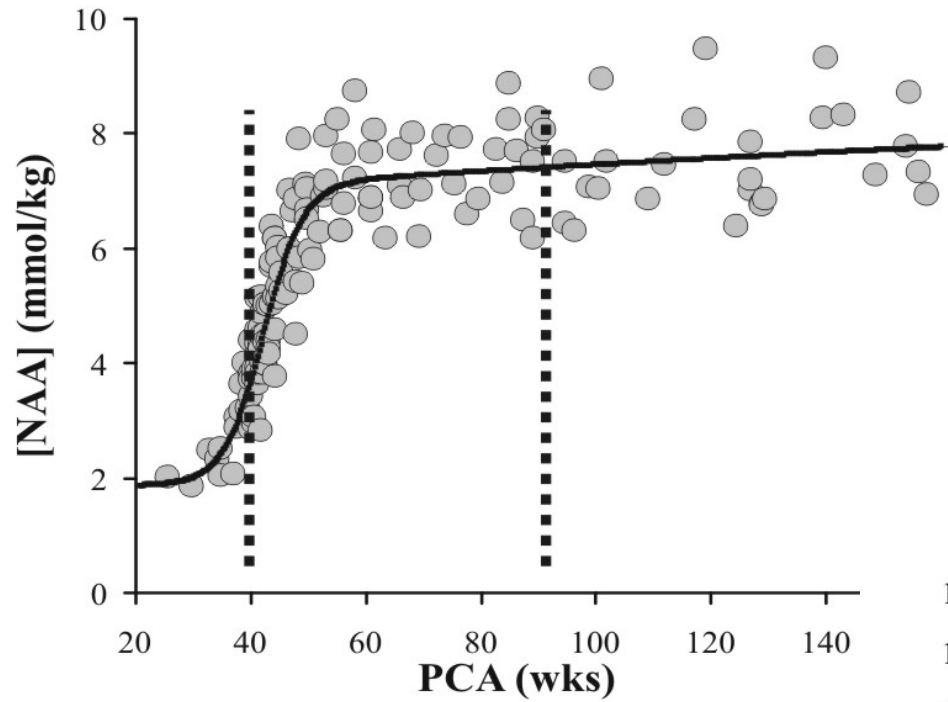


- **NAA (N-acetyl-aspartate):** Marker for mature neurons and axons.
- **Cr (creatine):** Free creatine and phosphocreatine. Replenishes ATP via $\text{PCr} + \text{ADP} \rightarrow \text{Cr} + \text{ATP}$
- **tCho (choline containing metabolites):** Group of choline containing metabolites involved in synthesis and breakdown of membrane phospholipids (phosphatidylcholine = lecithine)
- **ml (myo-inositol):** Sugar-like molecule, osmolyte, astrocyte marker, involved in membrane metabolism, detoxification
- **Glu (glutamate):** Neurotransmitter, high in neurons
- **Gln (glutamine):** Glutamate detoxification, osmolyte, astrocyte marker

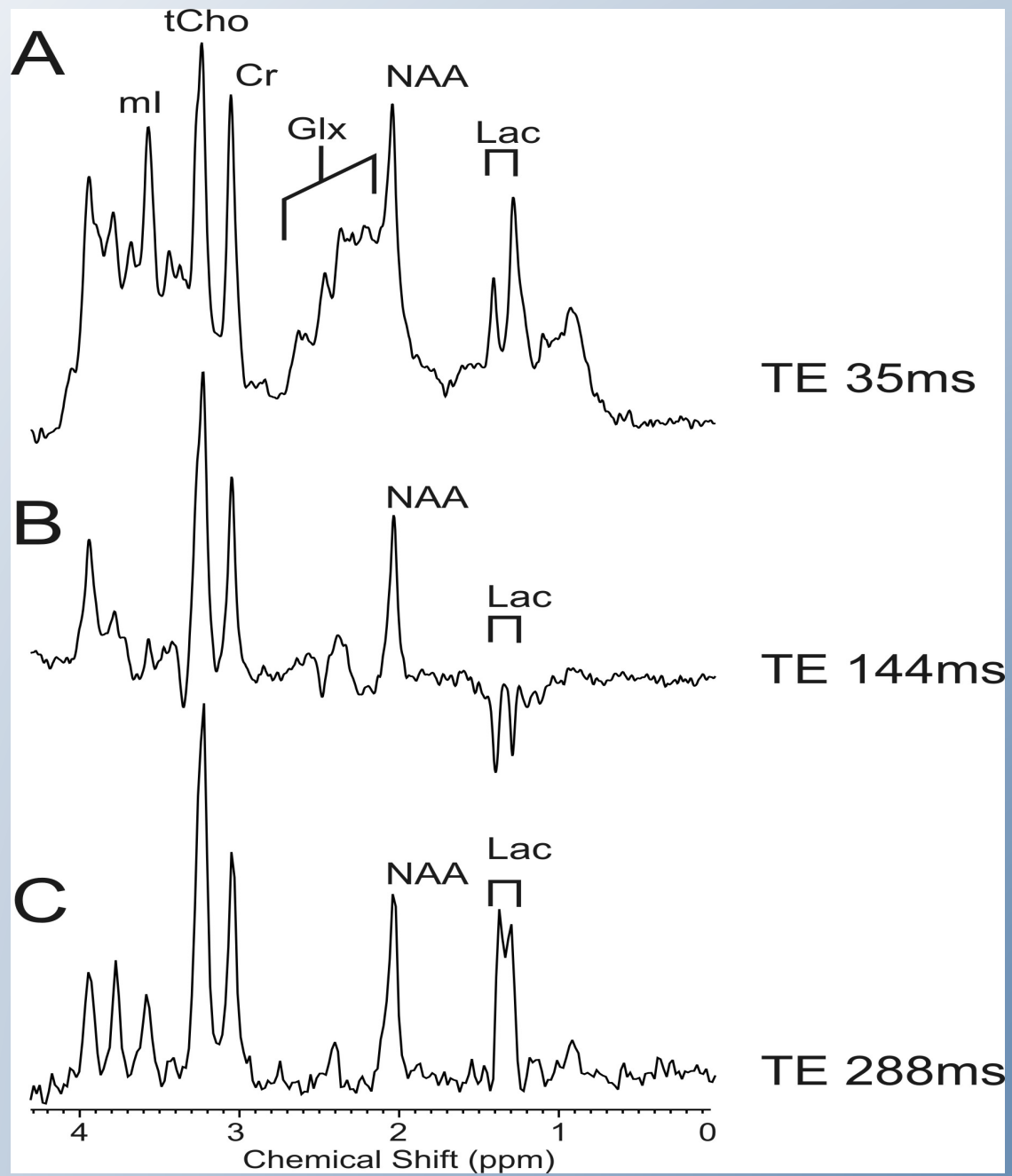
Normal age-dependent changes



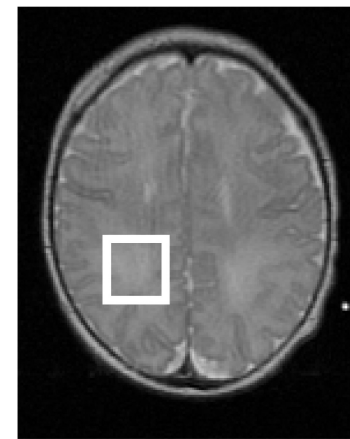
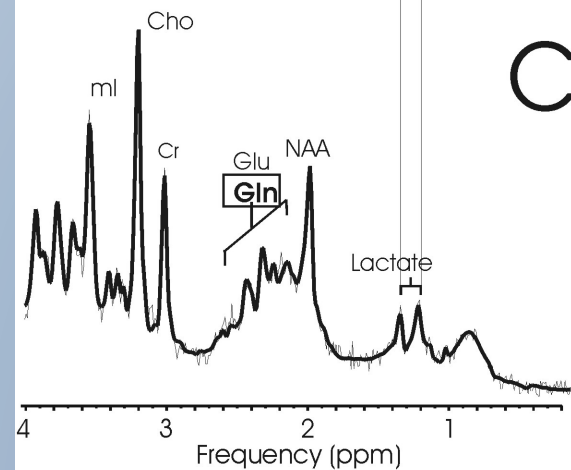
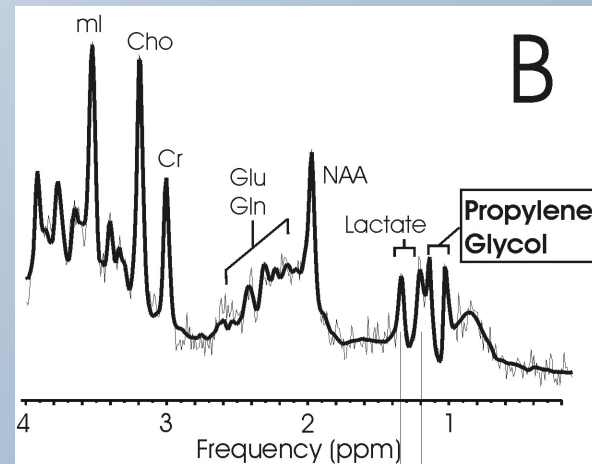
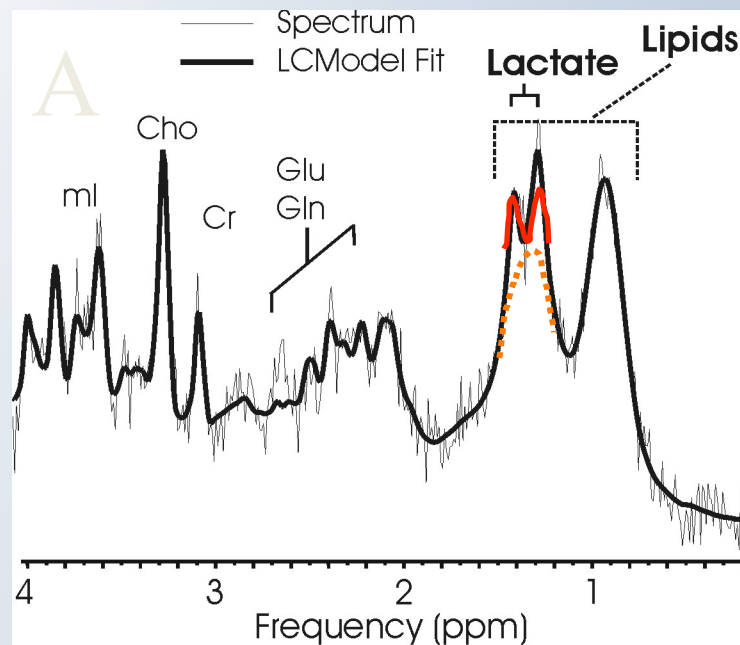
Normal age-dependent changes



Bluml et al. Cerebral Cortex,
2012

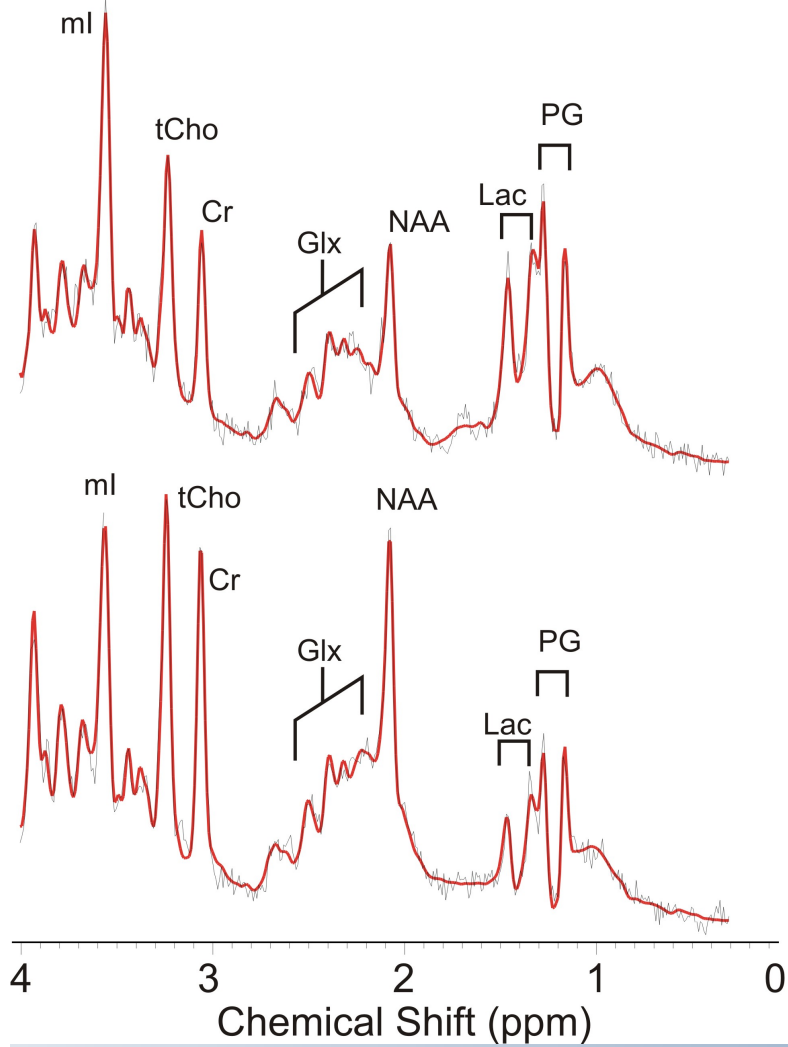


Differentiation of lactate, lipids, and propylene glycol

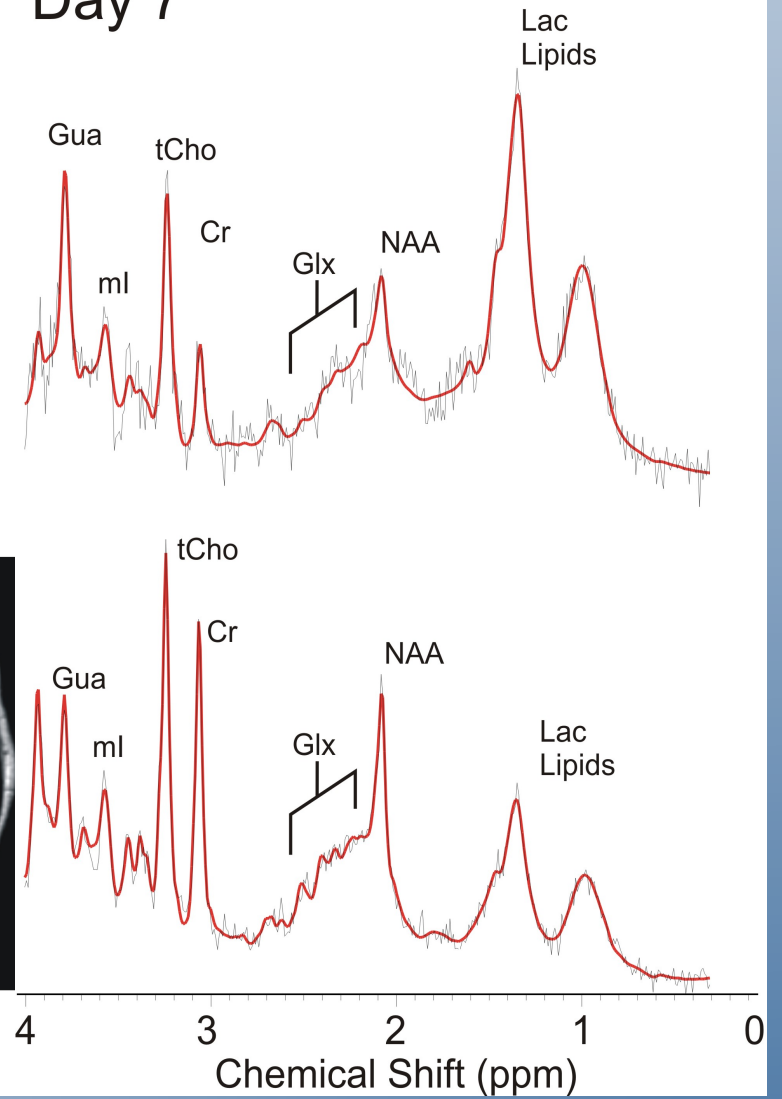
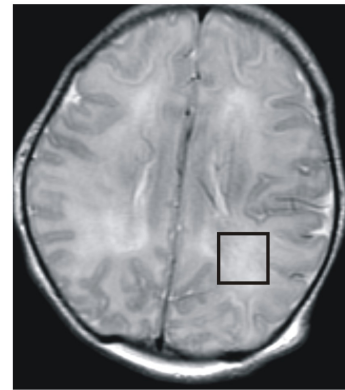


- Lac: Characteristic doublet of narrow lines at 1.33 ppm
- PG: Characteristic doublet of narrow at 1.2 ppm
- Lipids (macromolecules): Broad peaks at 0.9 and 1.3 ppm

Day 2



Day 7



Cerebral Magnetic Resonance Biomarkers in Neonatal Encephalopathy: A Meta-analysis



WHAT'S KNOWN ON THIS SUBJECT: MRI of the brain is often used for prediction of long-term neurodevelopmental outcome after NE; however, there is no consensus with regard to which particular MR biomarker and at which age are most accurate for predicting adverse outcome.



WHAT THIS STUDY ADDS: Meta-analysis of the published data suggests that deep gray matter Lac/NAA and Lac/Cr are the most accurate quantitative MR biomarker for prediction of neurodevelopmental outcome after NE. This biomarker may be useful as a surrogate end point and as a bridging biomarker.

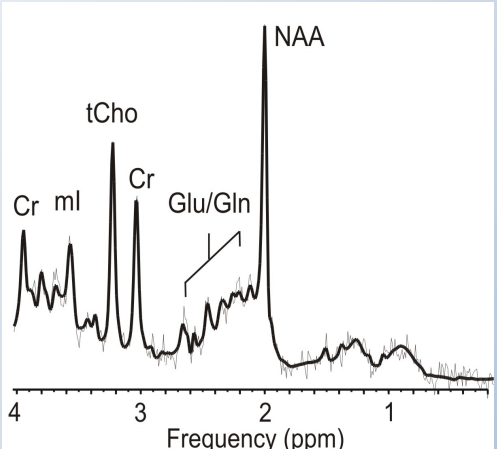
AUTHORS: Sudhin Thayyll, MBBS, MD, DCH, MRCPCH,^a Manigandan Chandrasekaran, MBBS, DCH,^a Andrew Taylor, MD,^b Alan Bainbridge, PhD,^c Ernest B. Cady, FInstP, BSc,^c W. K. Kling Chong, MD,^d Shahed Murad, PhD,^{e,f} Rumana Z. Omar, PhD,^g and Nicola J. Robertson, PhD^g

^aUniversity College London Institute of Women's Health, London, UK; ^bCentre for Cardiovascular Imaging, Great Ormond Street Hospital for Children, London, England; ^cDepartment of Medical Physics and Bioengineering, University College London Hospitals NHS Trust, London, England; ^dDepartment of Pediatric Neuroradiology, Great Ormond Street Hospital for Children, London, England; ^eDepartment of Statistical Science, University College London, Biomedical Research Unit, London, England; ^fBiostatistics Group, UCLH/UCL Biomedical Research Unit, University College London, London, England

Fully Automated Processing and Documentation of SV-MRS - MRS Database

1

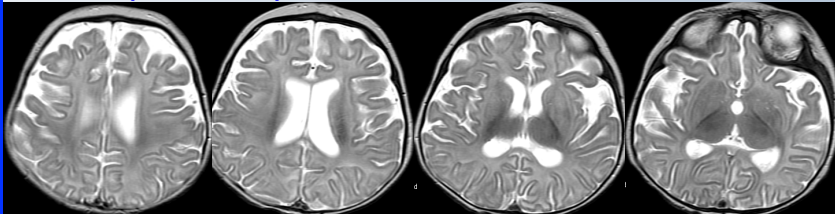
Spectrum +
H₂O +
T2-fit



Cr ml tCho Cr Glu/Gln NAA

Frequency (ppm)

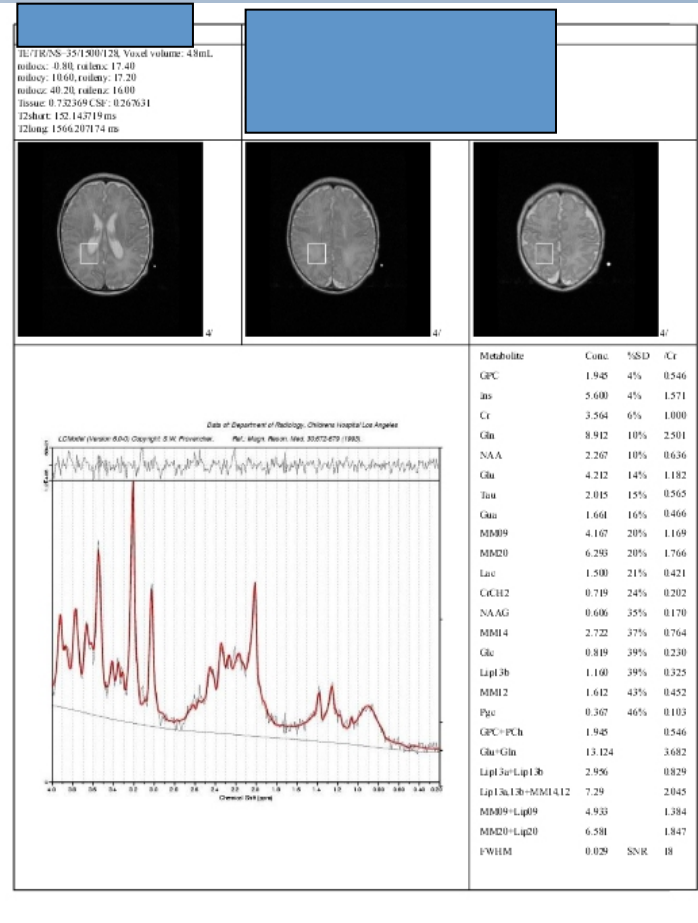
MRI (Dicom)



2

Processing Workstation

- “Query/retrieve”
- LCModel, MatLab, Perl
- “Raw results”



Metabolite	Conc.	%SD	Cr
GPC	1.945	4%	0.546
Ins	5.600	4%	1.571
Cr	3.564	6%	1.000
Glu	8.912	10%	2.501
NAA	2.267	10%	0.636
Gln	4.212	14%	1.182
Tau	2.015	15%	0.565
Gua	1.661	16%	0.466
MD09	4.167	20%	1.169
MD20	6.293	20%	1.766
Lac	1.500	21%	0.421
CrCh2	0.719	24%	0.202
NAAG	0.606	35%	0.170
MD14	2.722	37%	0.764
Glc	0.819	39%	0.230
Lip13b	1.160	39%	0.325
MD12	1.612	43%	0.452
Psy	0.367	46%	0.103
GPC+PCh	1.945		0.546
Gln+Gln	13.124		3.682
Lip13a+Lip13b	2.956		0.829
Lip13a+Lip13b+MD14+12	7.29		2.045
MD09+Lip09	4.933		1.384
MD20+Lip20	6.531		1.847
FWHM	0.029	SNR	18

3

• MRS Database

Additional Information:
(ROI, patient history, pathology, etc.)

MULTI-SITE HYPOTHERMIA ECMO STUDY

Table: There were no apparent differences when the quality of spectra obtained at multiple sites was compared with spectra acquired at CHLA.

		FWHM ^a	S/N ^b	Cr ^c	Cr SD ^d	Cho ^c	Cho SD ^d
multi-site (n=13)	mean	0.033	0.31	5.00	4%	2.22	4%
	stdev	0.009	0.10	1.66	2%	0.50	1%
CHLA (n=15)	mean	0.031	0.34	5.22	4%	2.35	3%
	stdev	0.007	0.07	0.55	0%	0.24	1%

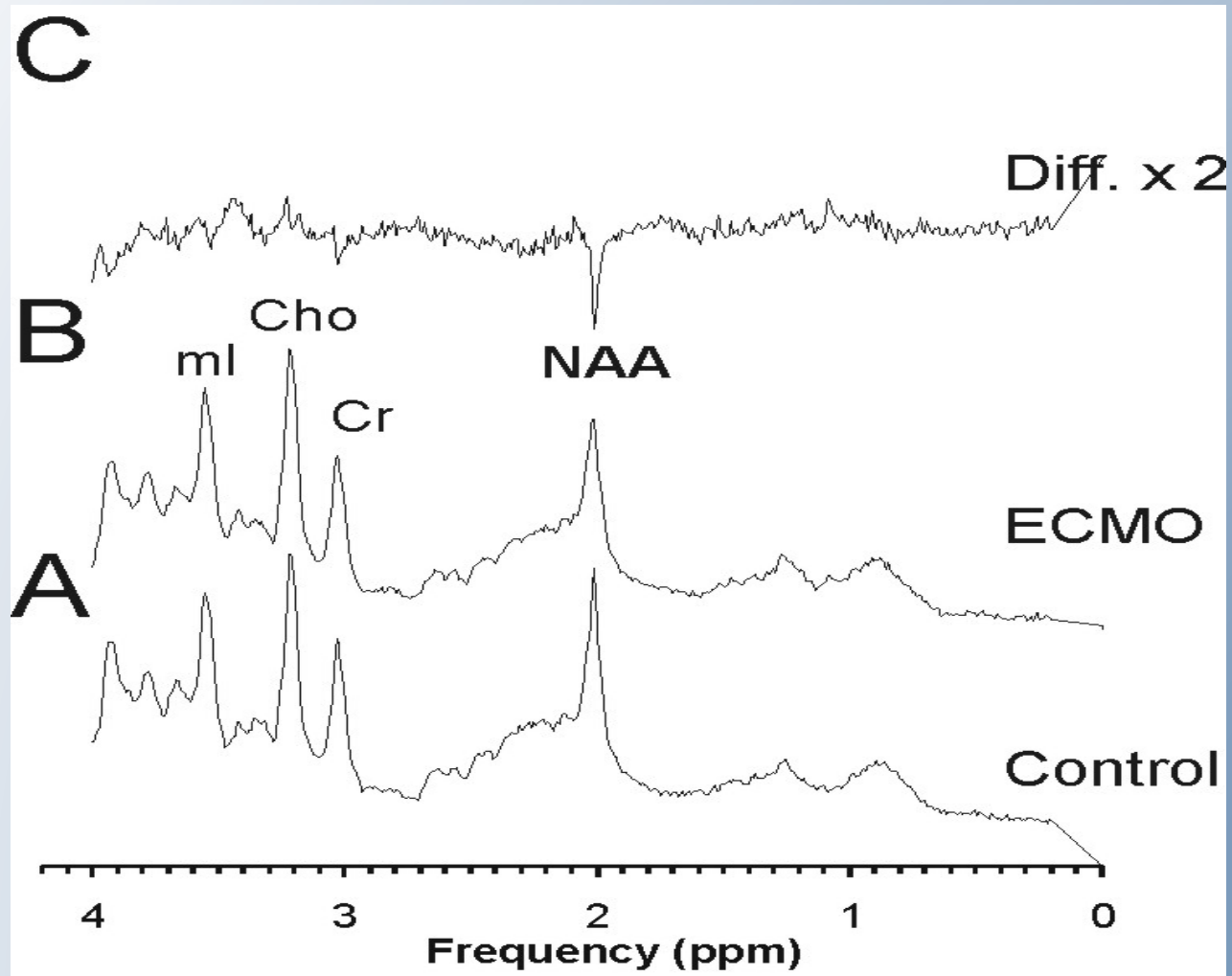
^aFWHM = full width of half maximum of the unsuppressed water peak, an objective and quantitative measurement of the field homogeneity within the the selected region of interest for MRS

^bS/N = signal-to-noise-ratio, determined from signal amplitude of the water peak and the random noise signal of spectra

^cabsolute concentrations of creatine (Cr) and choline (Cho)

^dSD = standard deviation, these are the Cramer-Rao lower bounds (CRLB). CRLB are objective indicators for the reliability of a measurement taking into consideration the noise level of the MR signal.

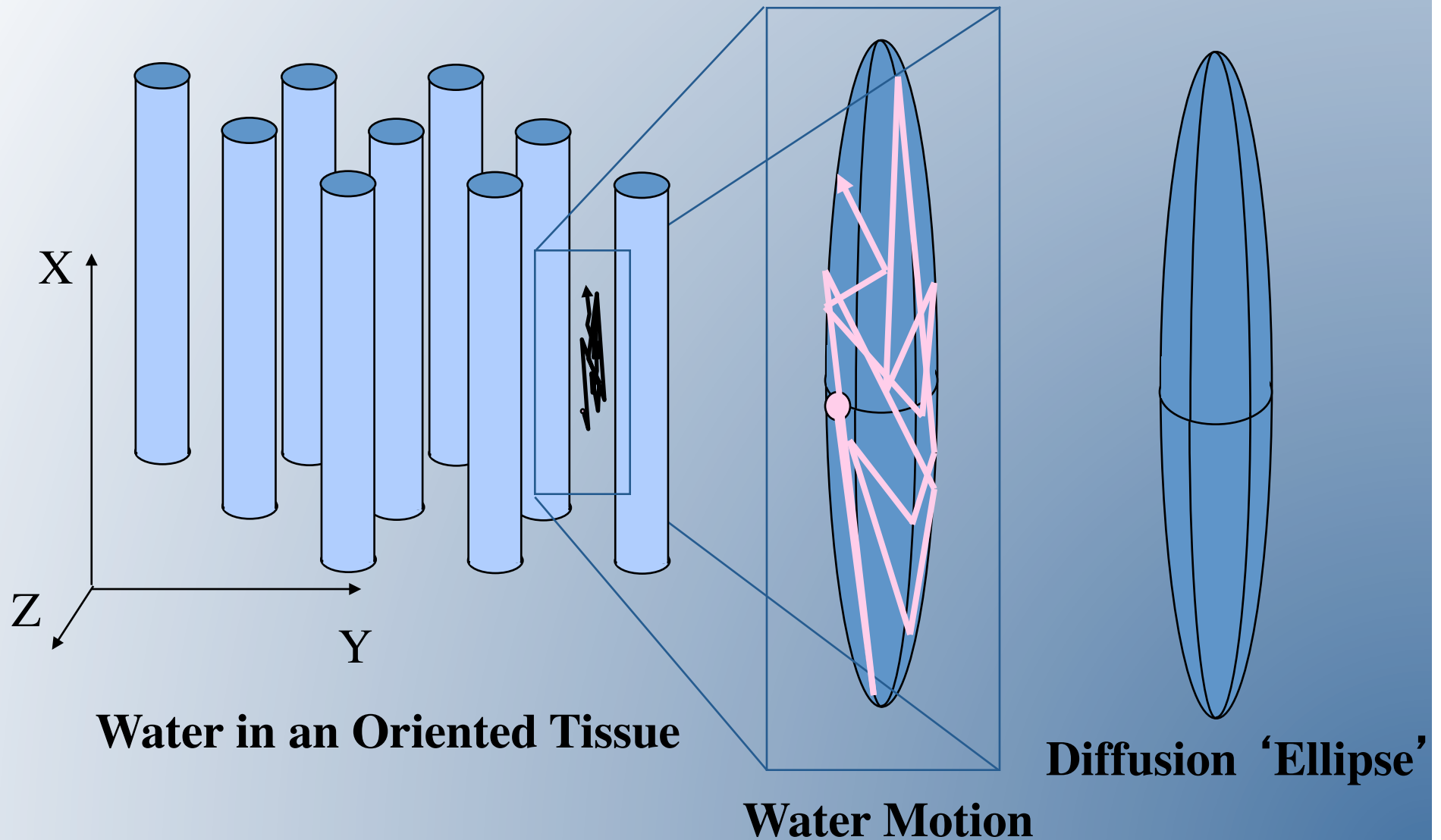
MRS- ECMO RELATED BRAIN INJURY



DIFFUSION TENSOR IMAGING

- By manipulating magnetic field gradients MR images can be sensitized to diffusion- the random thermally driven motion of water molecules in tissue
- Water motion: isotropic or anisotropic

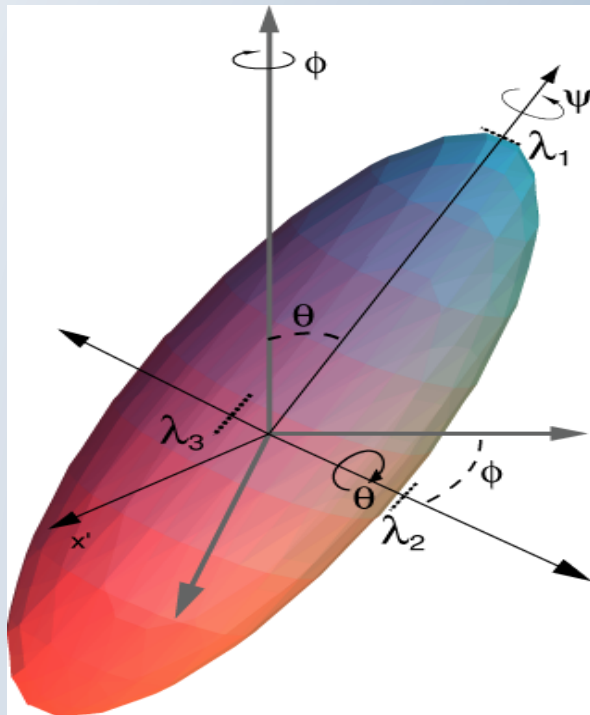
Diffusion in 3-D: White Matter



Eigen-system Analysis of Diffusion Tensor

$$\vec{\mathbf{D}} = \begin{pmatrix} D_{xx} & D_{xy} & D_{xz} \\ D_{xy} & D_{yy} & D_{yz} \\ D_{xz} & D_{yz} & D_{zz} \end{pmatrix} = \begin{pmatrix} V_{1x} & V_{1y} & V_{1z} \\ V_{2x} & V_{2y} & V_{2z} \\ V_{3x} & V_{3y} & V_{3z} \end{pmatrix} \cdot \begin{pmatrix} \lambda_1 & & \\ & \lambda_2 & \\ & & \lambda_3 \end{pmatrix} \cdot \begin{pmatrix} V_{1x} & V_{2x} & V_{3x} \\ V_{1y} & V_{2y} & V_{3y} \\ V_{1z} & V_{2z} & V_{3z} \end{pmatrix}$$

$$\lambda_1 \geq \lambda_2 \geq \lambda_3$$



- Major Eigen-Value :

$$\lambda_1$$

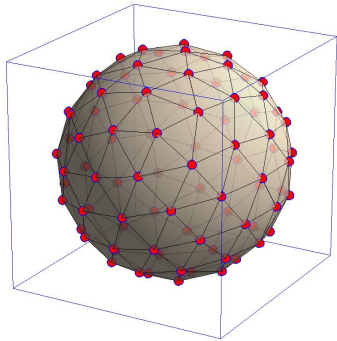
- Major Eigen-Vector:

$$\begin{bmatrix} V_{1x} & V_{1y} & V_{1z} \end{bmatrix}^T$$

$$FA = \sqrt{\frac{3 \cdot \sum_{i=1,2,3} (\lambda_i - \bar{\lambda})^2}{2 \cdot \sum_{i=1,2,3} \lambda_i^2}}$$

$$\bar{D} = \frac{\lambda_1 + \lambda_2 + \lambda_3}{3}$$

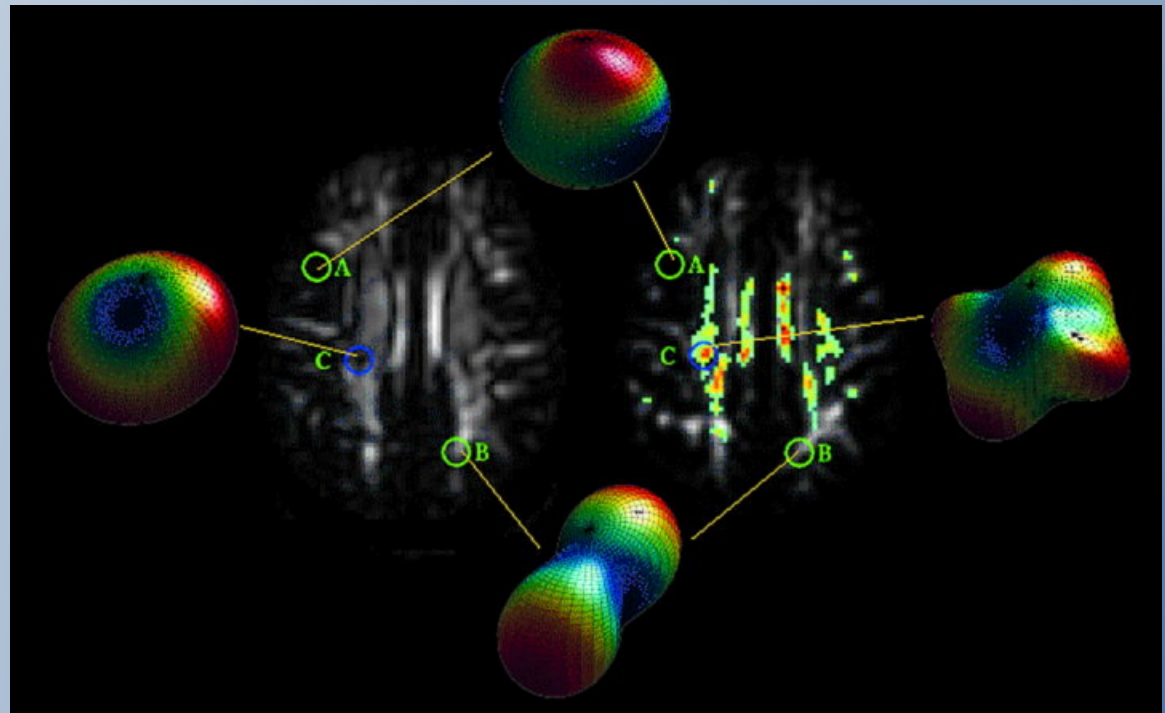
High Angular Resolution Diffusion (HARDI)



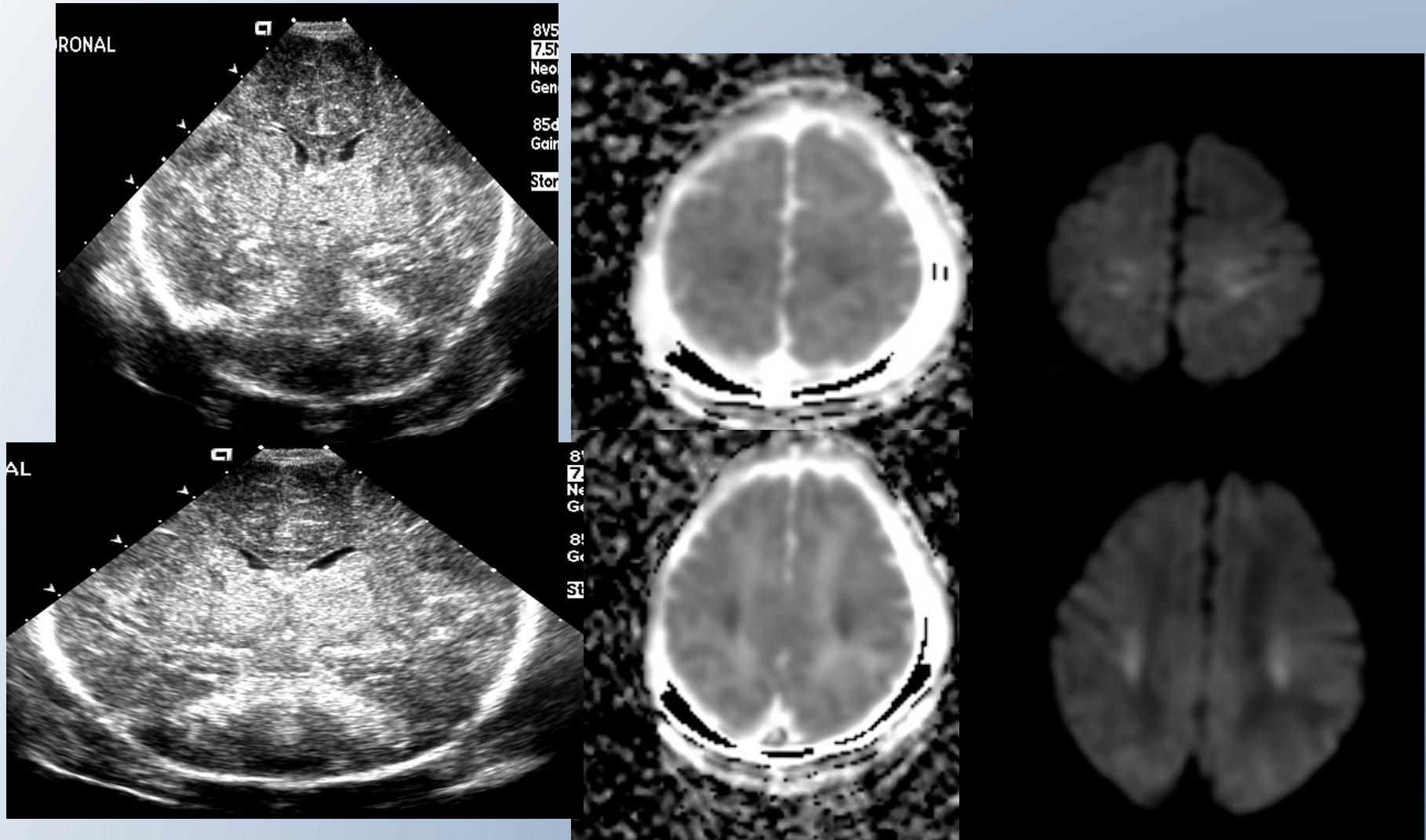
HARDI Encoding

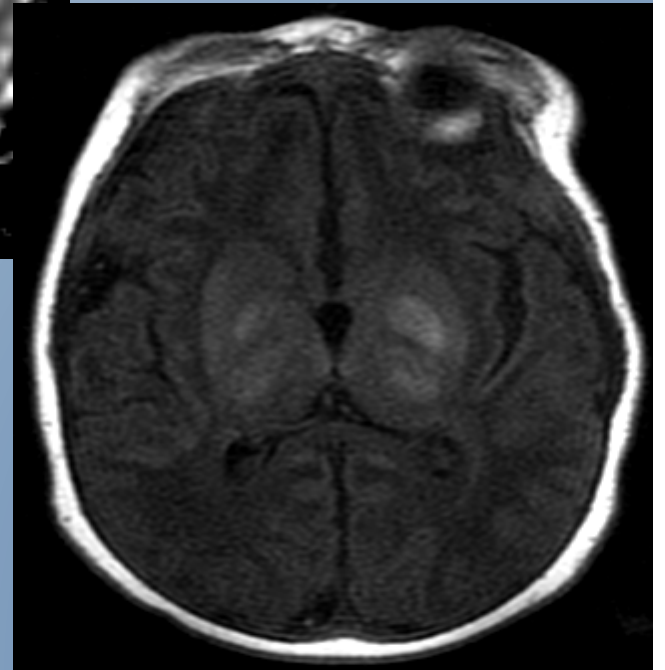
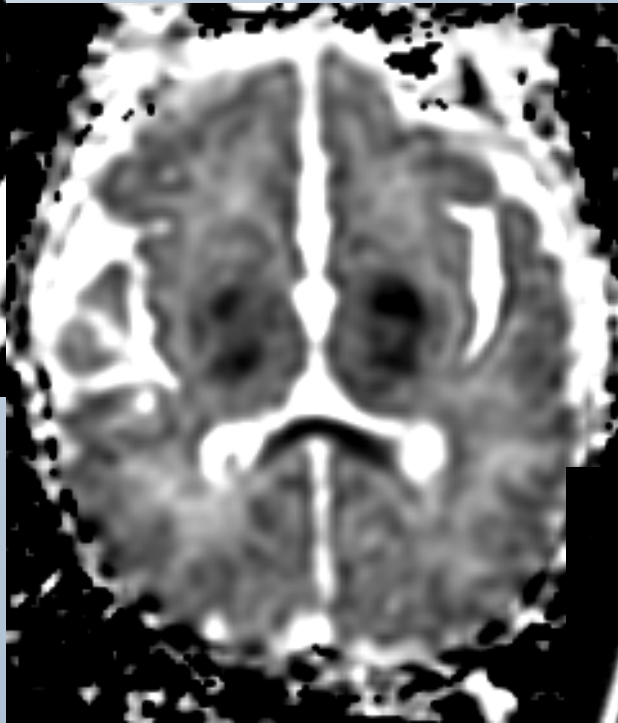
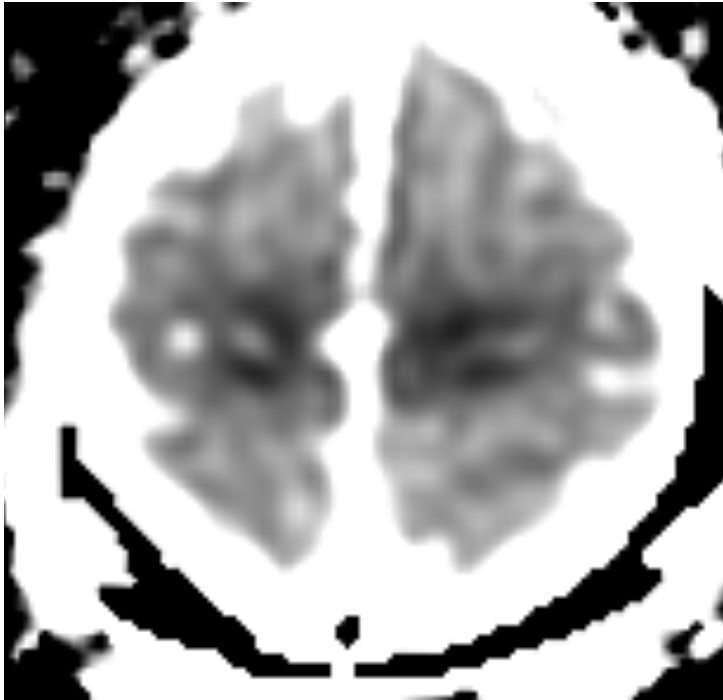
- Identify complex white matter structures
- Improve diffusion MRI based tractography
- Increase number of directions

Frank, MRM 2002

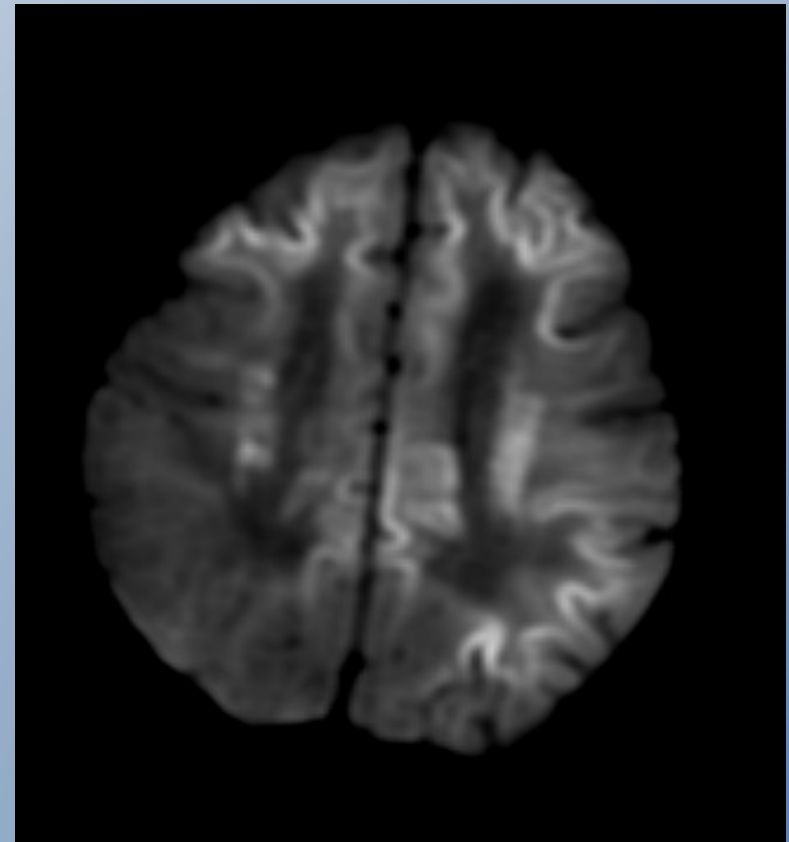
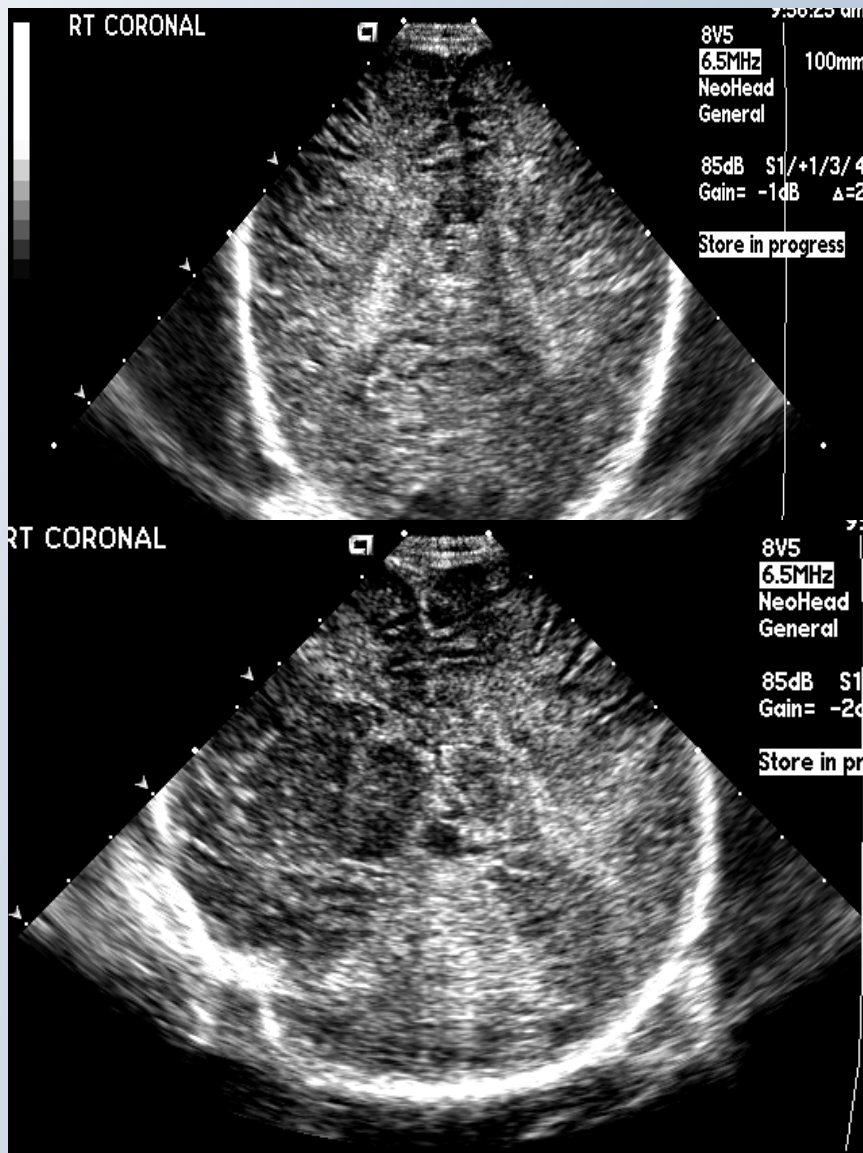


Central Pattern of Injury





Peripheral Pattern of Injury



RT CORONAL

R □

8:20:20 am

10V4-S

HC7.5MHz 110mm

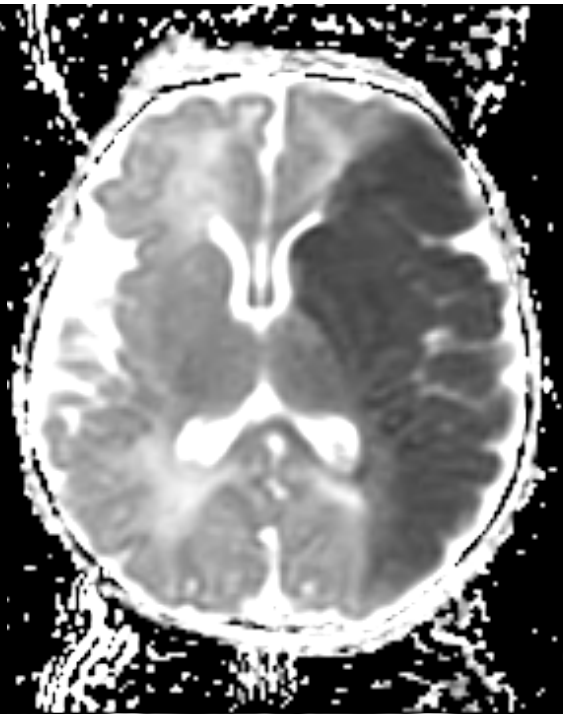
NeoHead

General /V

77dB S1/+1/2/4

Gain= 6dB Δ=2

Store in progress



RT CORONAL

R □

8:18:15 am

10V4-S

HC7.5MHz 100mm

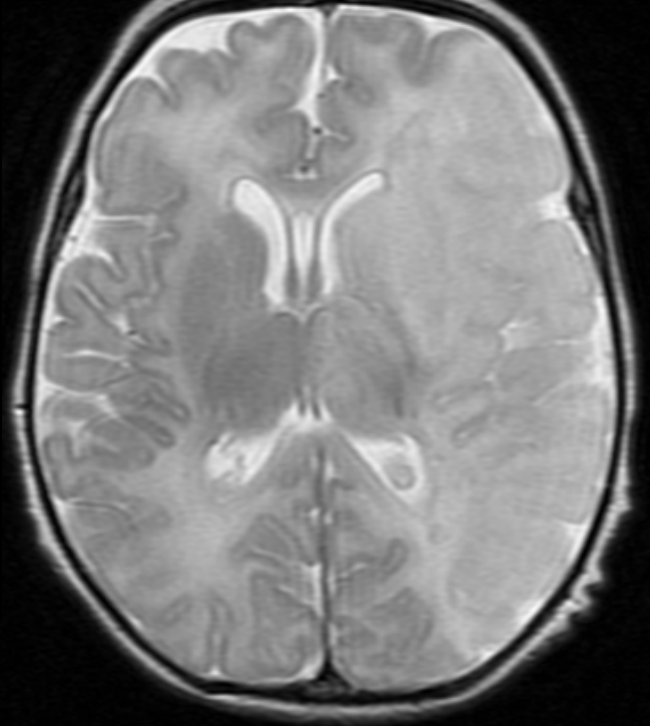
NeoHead

General /V

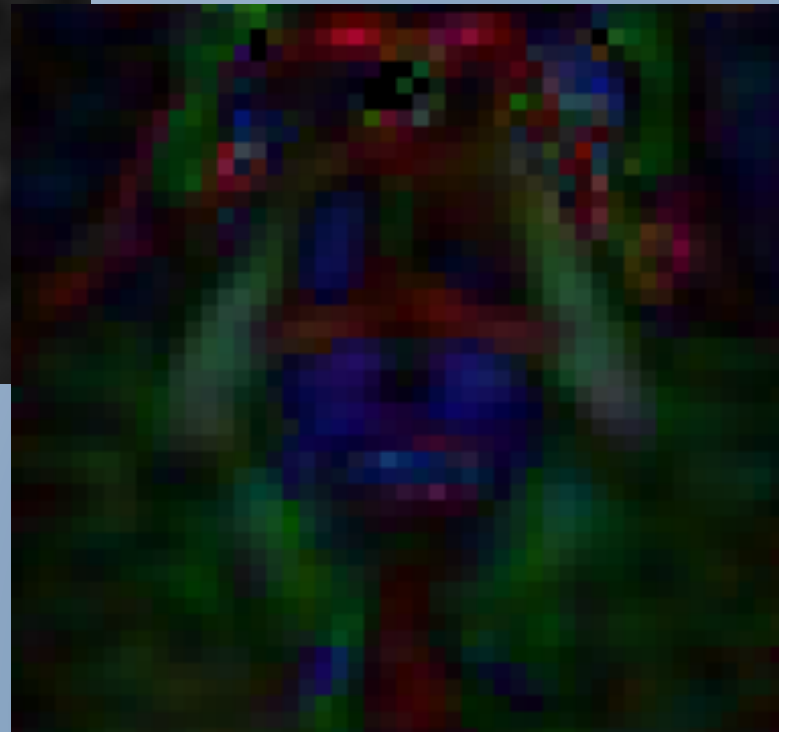
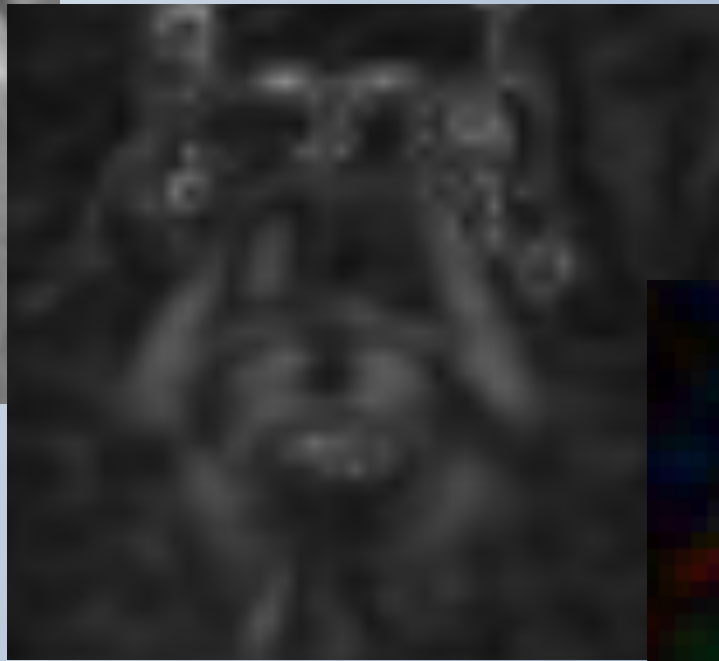
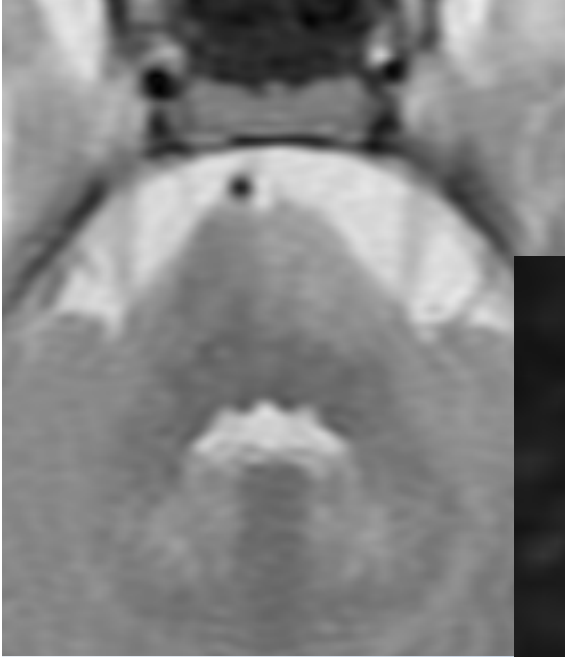
77dB S1/+1/2/4

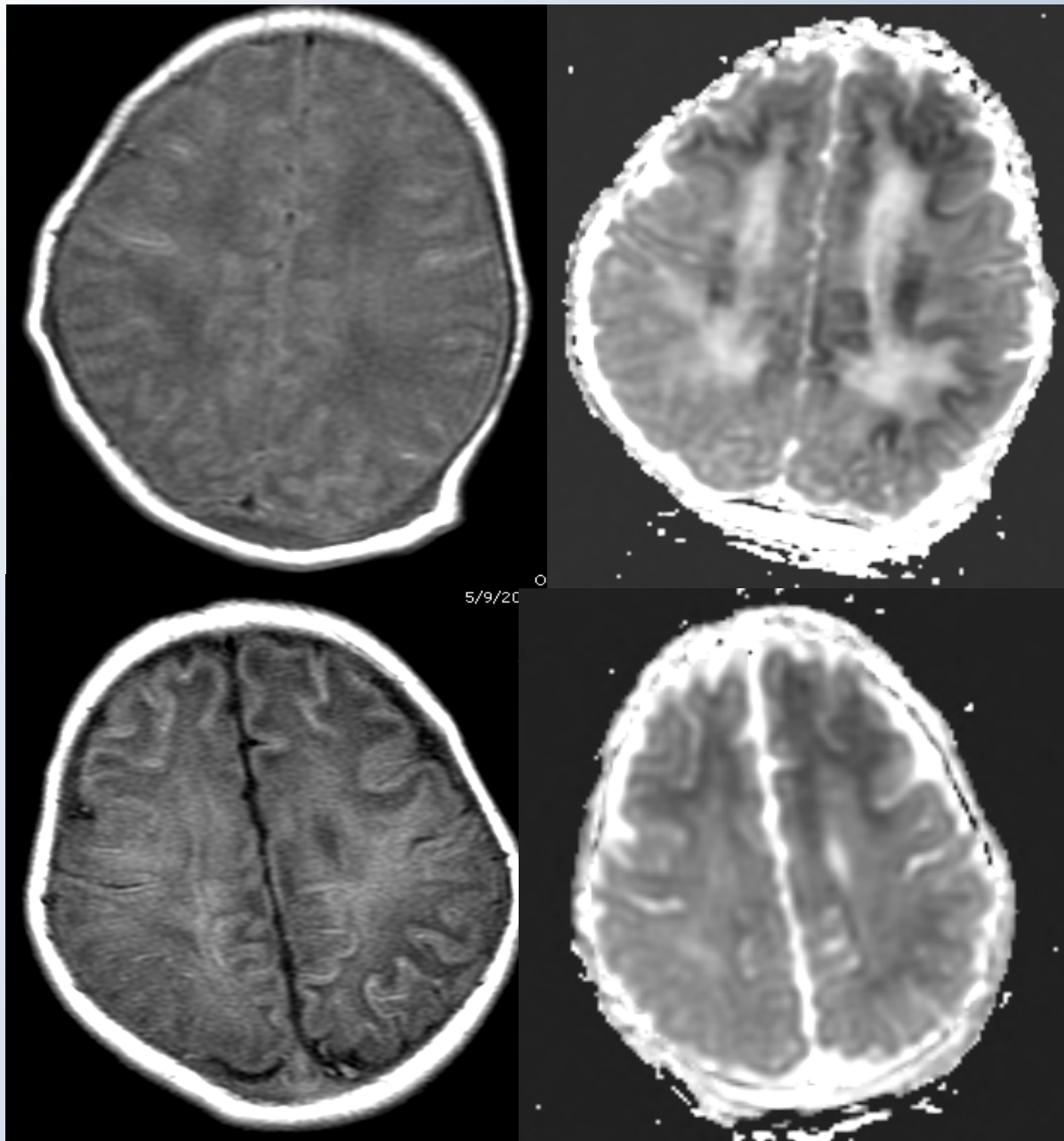
Gain= 0dB Δ=2

Store in progress



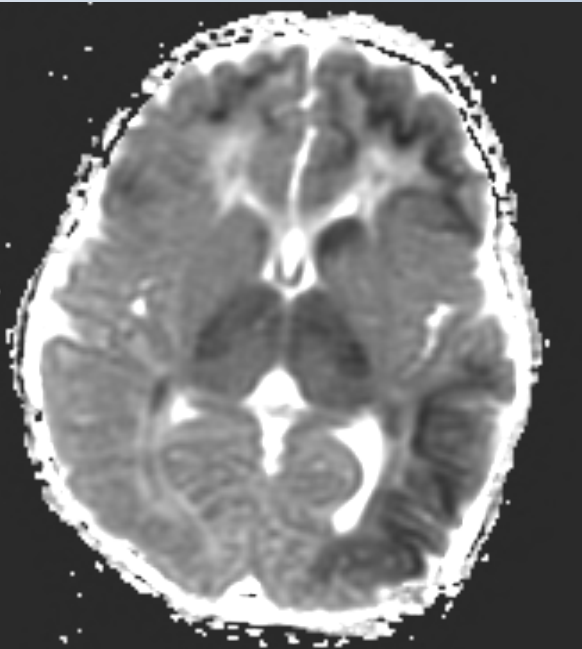
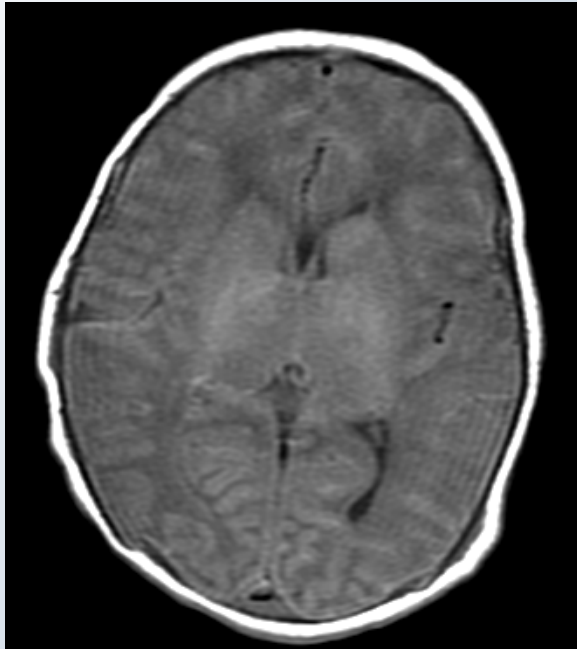
Acute Wallerian Degeneration



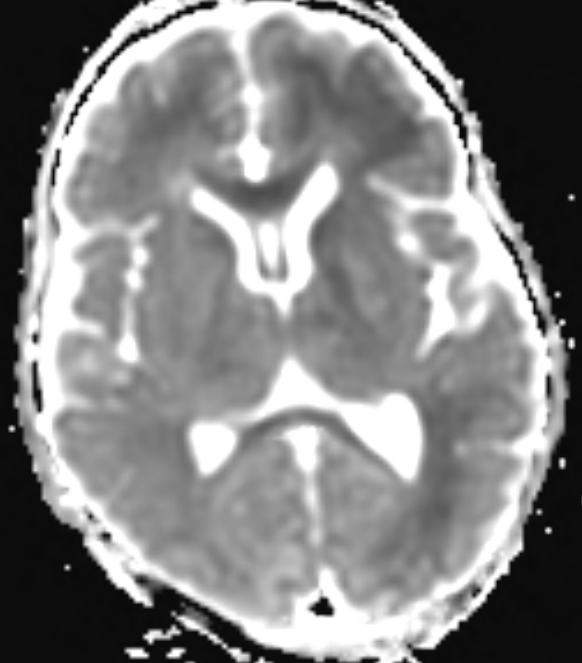


2 Days

7 Days



2 Days



7 Days

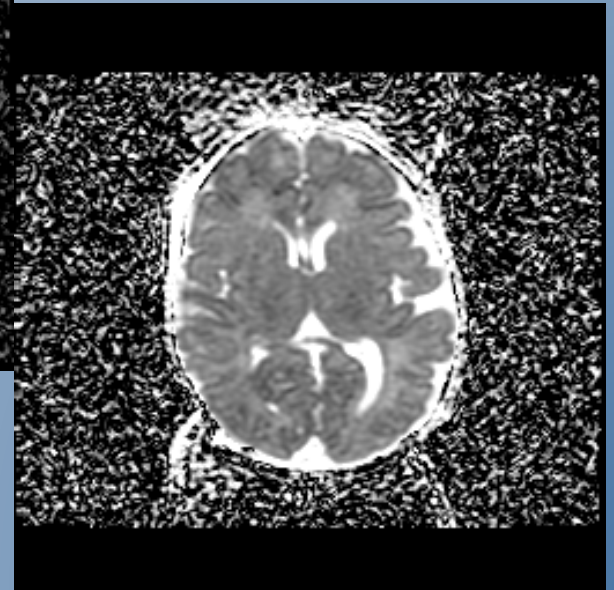
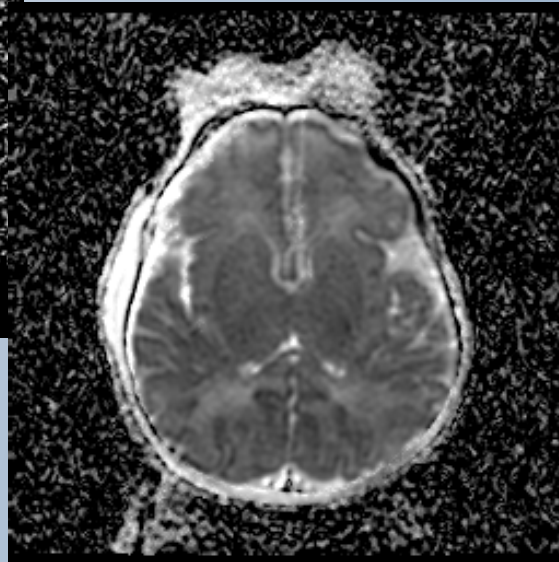
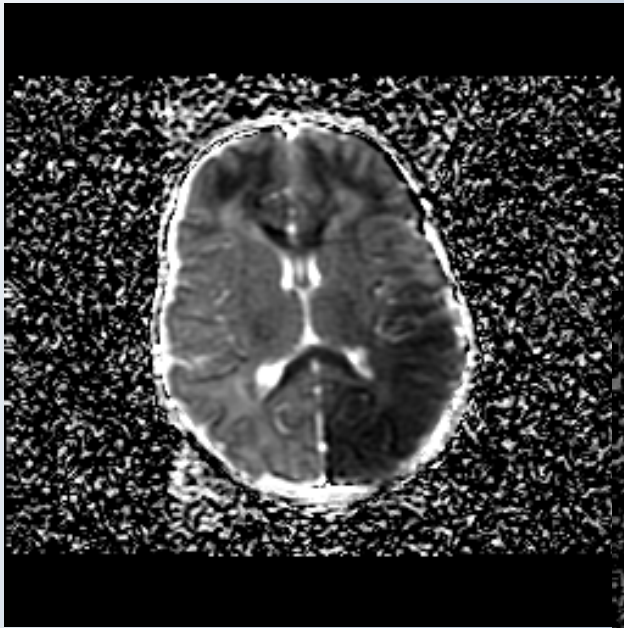
Diffuse Pattern of Hypoxic-Ischemic Injury-CENTRAL

- Central pattern: dorsal brainstem tegmentum, the posterior putamen, the lateral thalamus, the cortical spinal tract and peri-rolandic cortex
- Profound hypoxia/hypotension
- Poor neurological prognosis
- May be fatal depending on brainstem involvement

Diffuse Pattern of Hypoxic-Ischemic Injury-PERIPHERAL

- Periperal pattern: parasagittal cortex in the borderzone/watershed regions
- Mild to moderate hypoxia/hypotension
- Better prognosis compared to central
- Both central and peripheral patterns can occur together

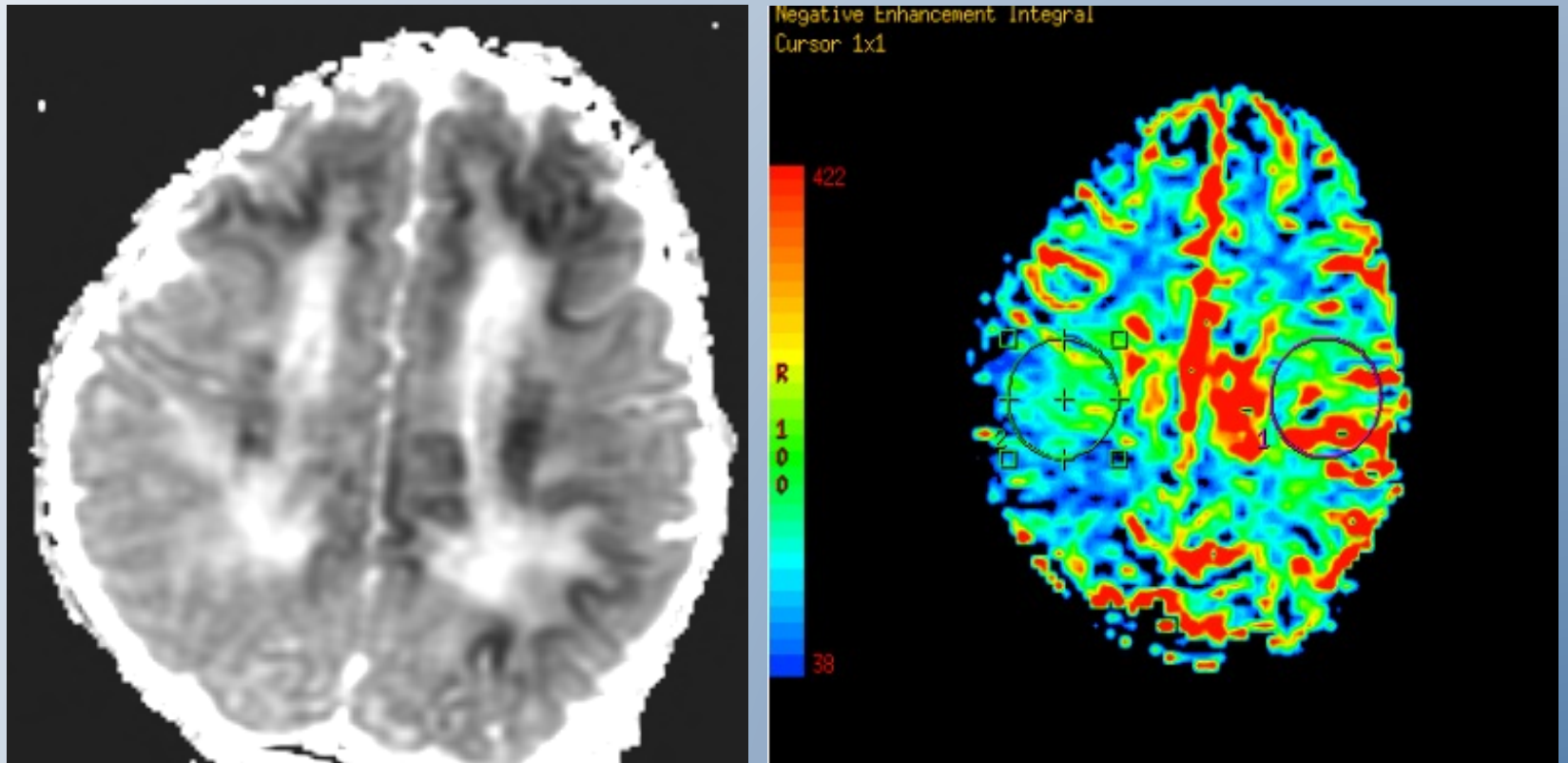
How does cooling effect microstructure and metabolism?



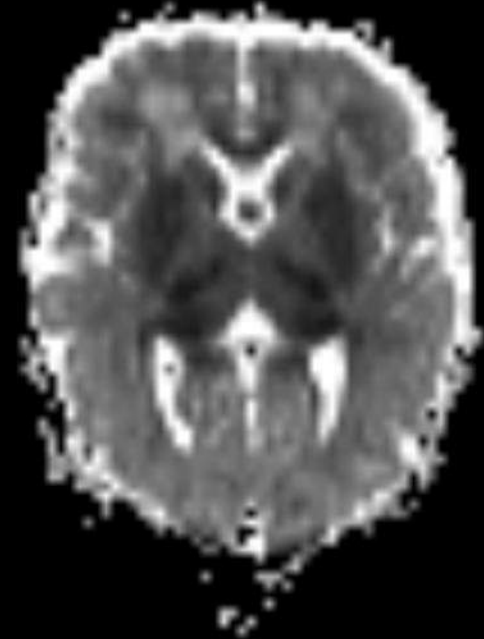
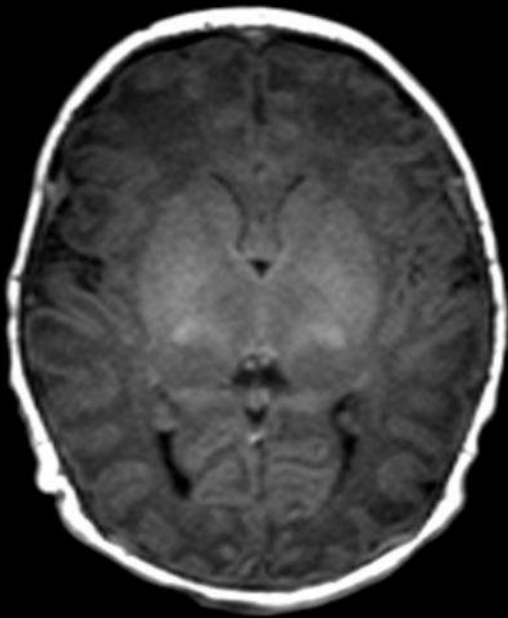
Hypothermia-MRS/DTI

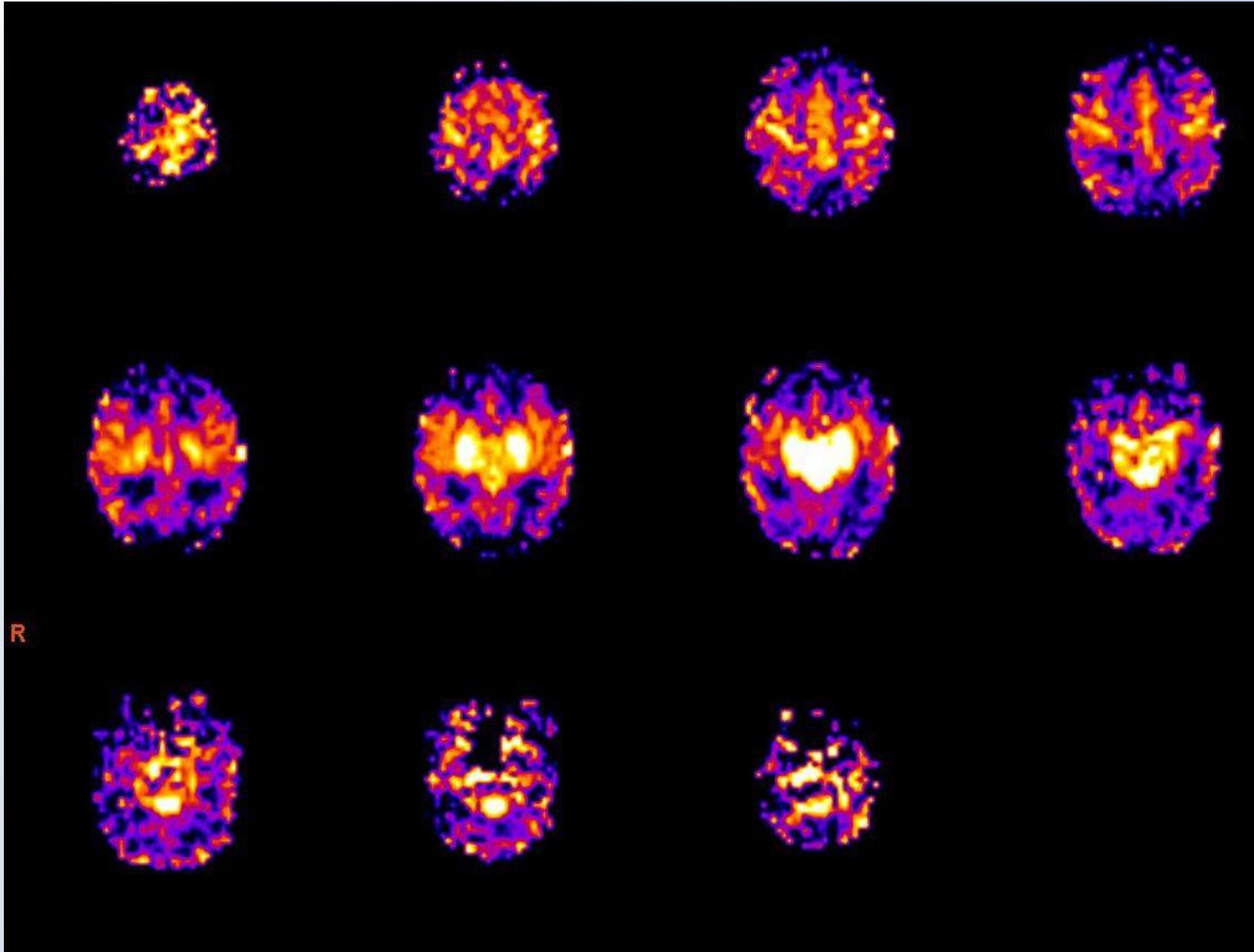
- Slower recovery of ADC to pseudo-normalization
- Hypothermia may extend the window in which HIE lesions are visible on DWI sequences
- Reduction in lactate levels

Hyper-perfusion in HIE



2 day old - History of umbilical cord prolapse and emergent C-section





Courtesy of Dr. A. Vassough , CHOP

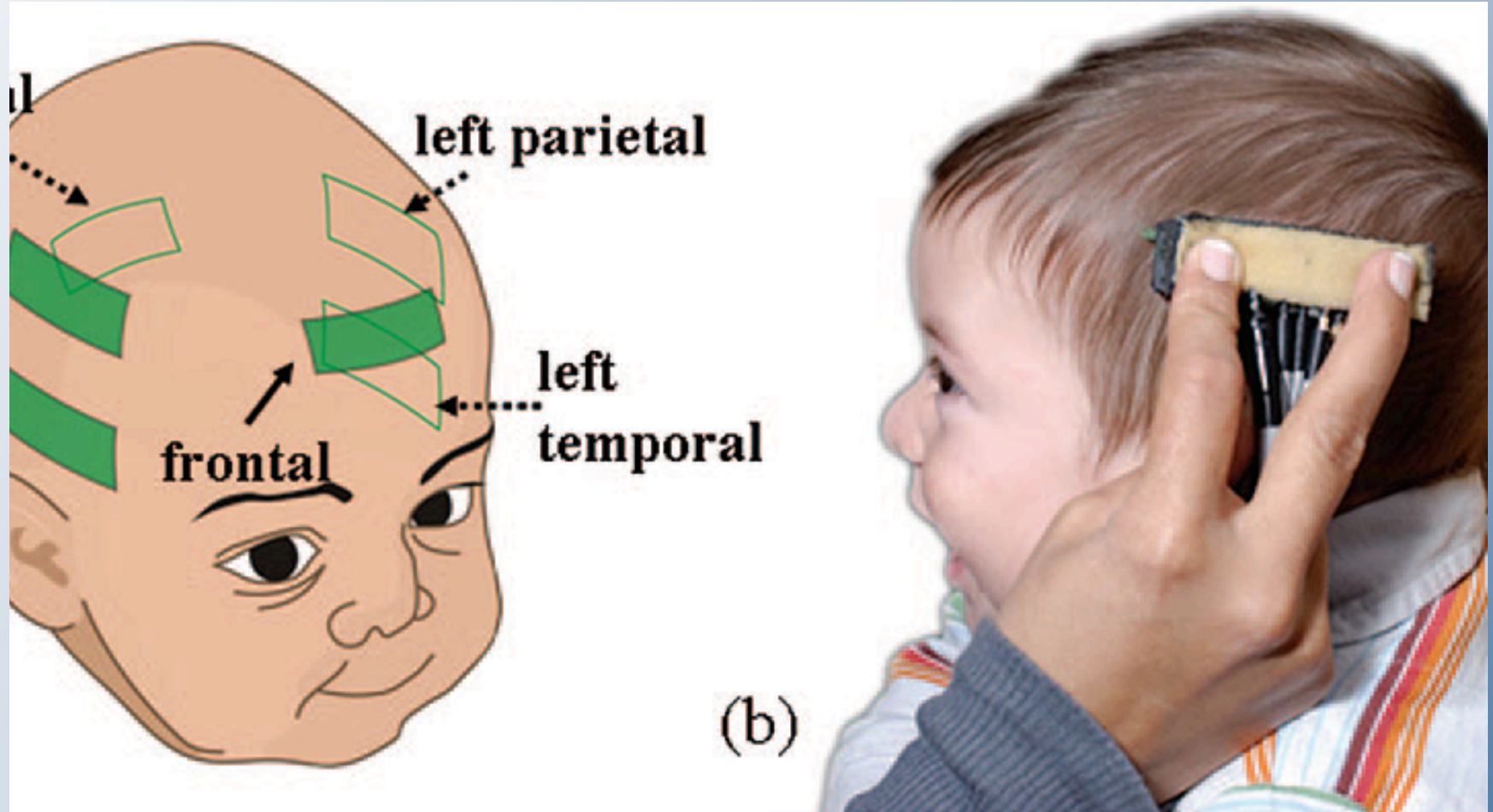
Hyper-perfusion?

- Decreased ADC- associated with increased cerebral blood flow
- Hyperfusion/perfusion- contributing to injury or secondary phenomenon to the development of injury
- biomarker of outcome predicting?
- Treatment effect- hypothermia?

NIRS- Near-infrared spectroscopy

- Light to measure blood volume and oxygenation-frequency domain
- Low quantitative accuracy to measure absolute cerebral hemoglobin
- Grant et al- increased CBV and relative cerebral oxygen consumption in HIE patients compared to control (not cerebral tissue oxygenation)- JCBFM 2009
- Increased perfusion-increased neuronal activity?

NIRS- Frequency Domain Continuous wave

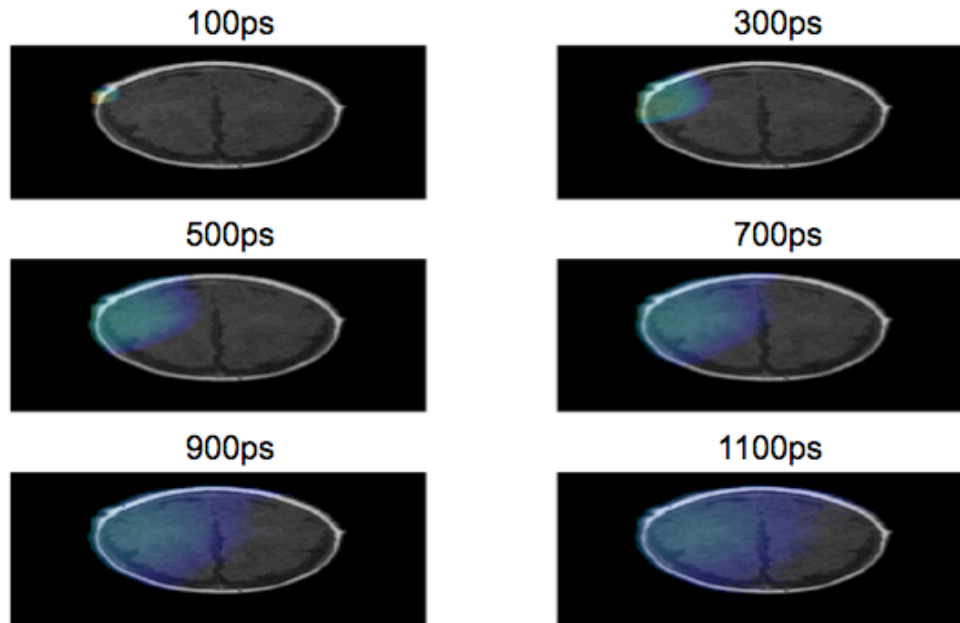


Issue with FD- NIRS

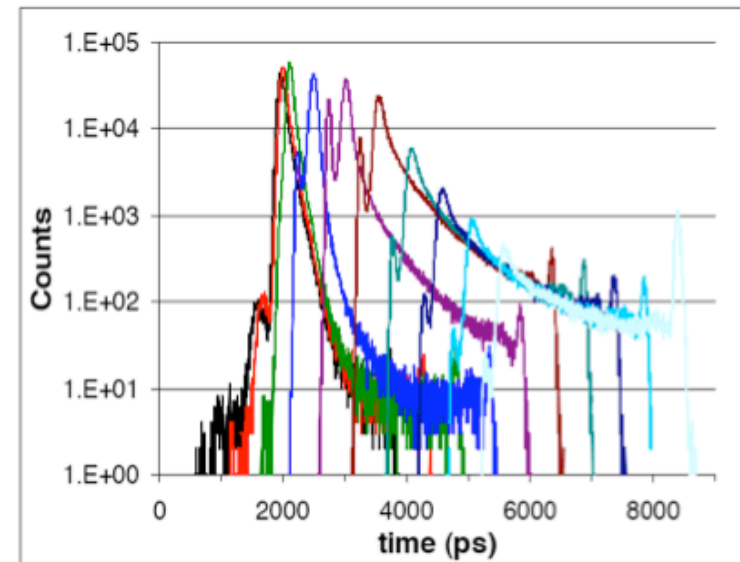
- Only able to measure changes in optical absorption of tissue – rely on look-up tables for correction factors for quantitative results
- Consensus from clinical trials is that FD-NIRS may not have quantitative accuracy for on-demand measurements

Development of Time Domain NIRS

A) Light migration in neonate head



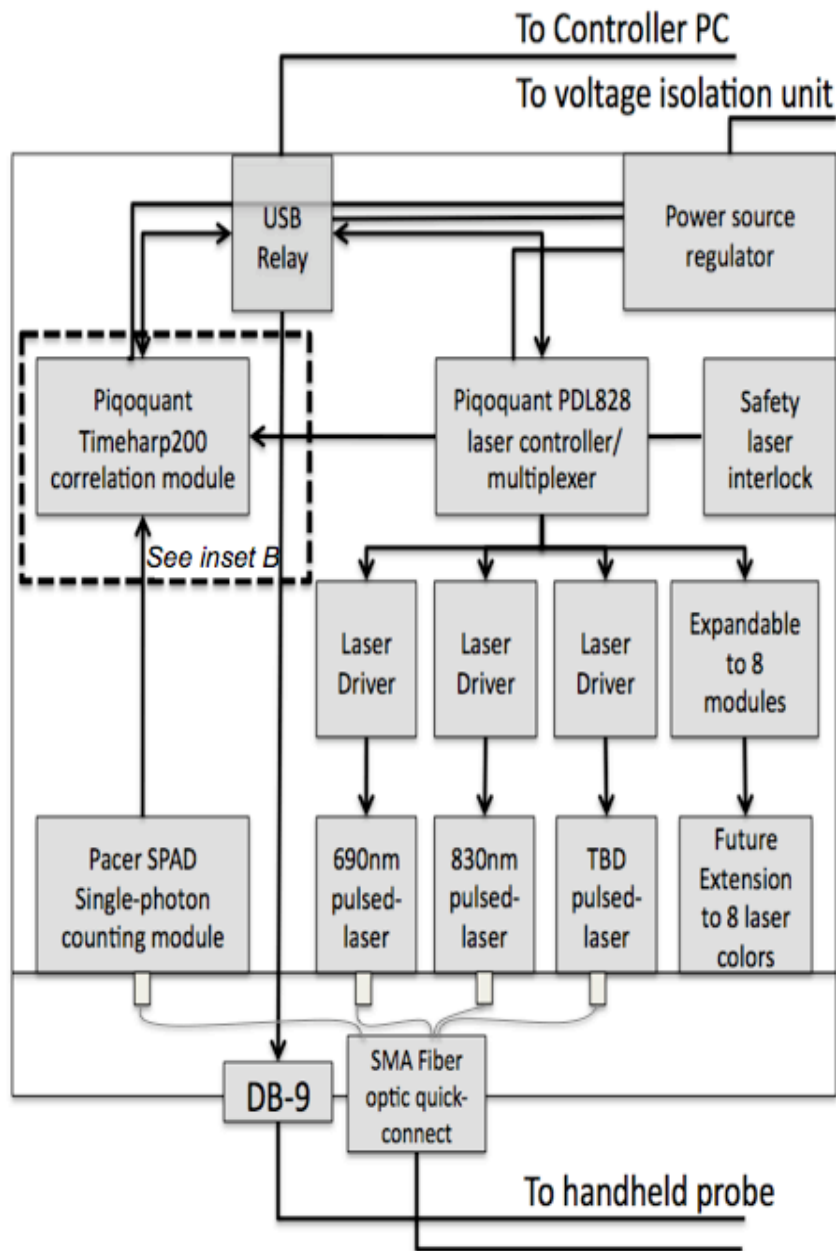
B) Time-of-flight NIRS signals



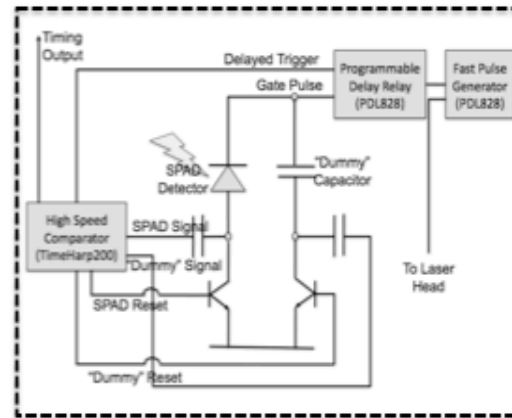
2nd generation NIRS pulsed-laser light source allow time of flight information about light transmission – directly measure optical pathway information- more accurate baseline measurements

Ted Huppert et al. University of Pittsburgh

A) Schematic of TD-NIRS System



B) Detail of Gate-delay circuit



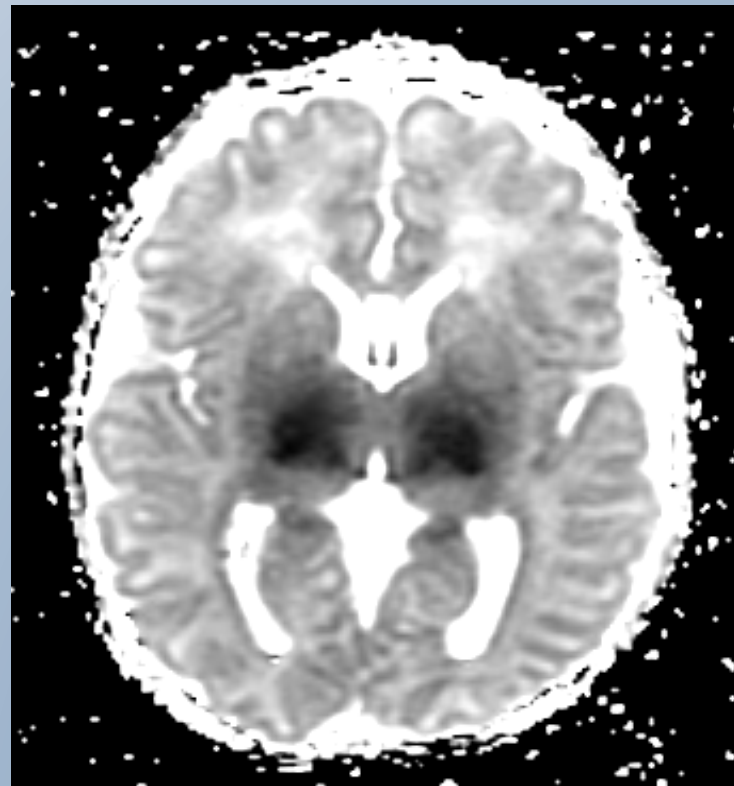
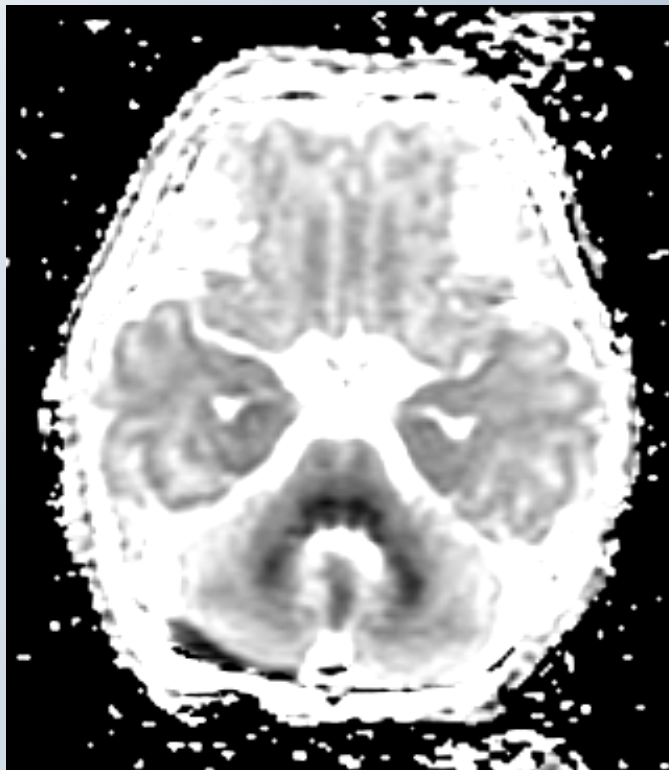
C) CAD model of probe and cart

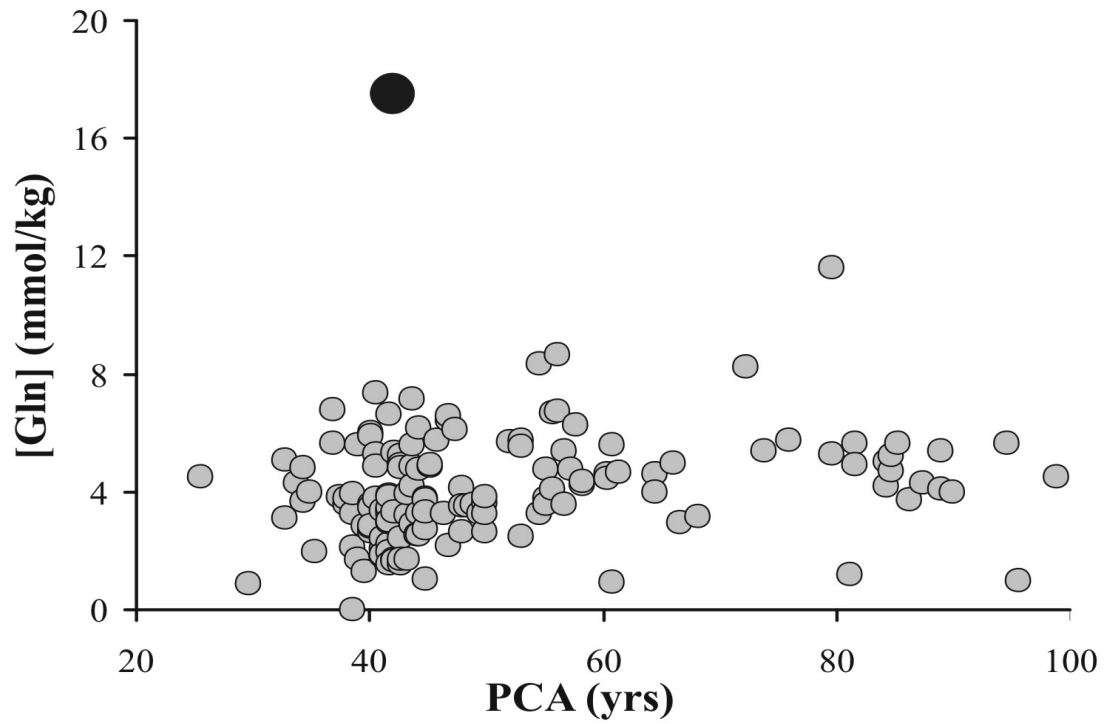
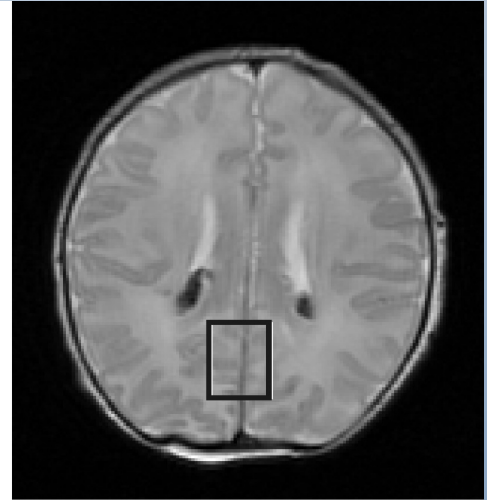
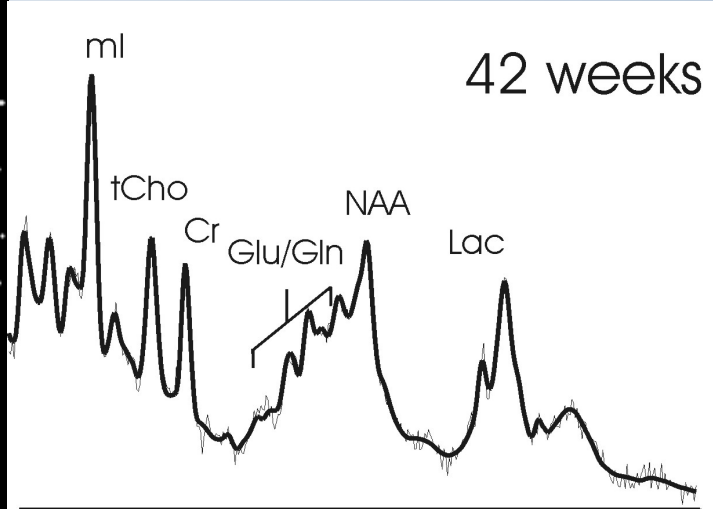
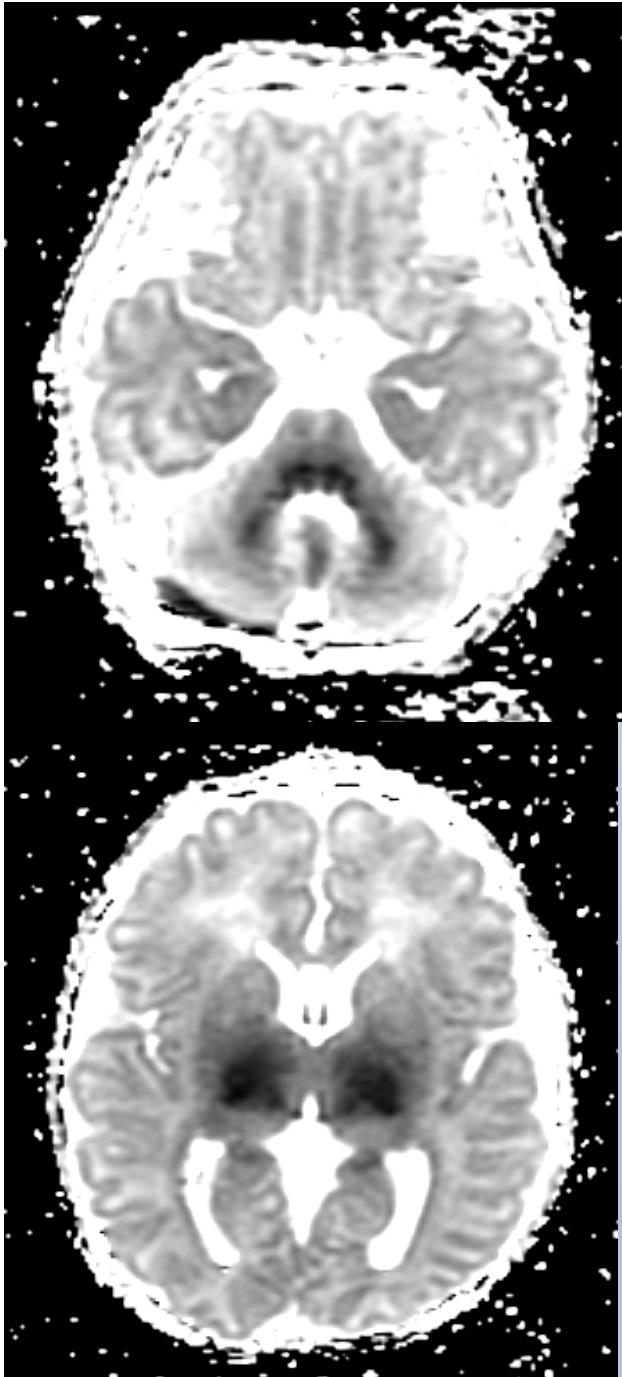


Metabolic Disease Can “Mimick” Patterns of Perinatal Hypoxic- Ischemic brain Injury

- Maple syrup urine disease
- Urea cycle disorders
- Organic aciduria
- Isolated sulfite oxidase deficiency
- Mitochondrial disorders
- Peroxisomal disorders

Mean Diffusivity/ADC:Mimic Central Hypoxic-ischemic Injury





Classification of Hydrocephalus

Entity involved	Parameter	Subtypes
Patient	Onset	Congenital/acquired Fetal/neonatal/infantile/child/adult/geriatric Acute/subacute/chronic
	Causes	Primary/secondary/idiopathic
	Underlying lesions	Dysgenetic/posthemorrhagic/post-SAH//post-IVH/postmeningitic/post-traumatic/ with brain tumor/spinal cord tumor/brain abscess/arachnoid cyst/cysticercosis, etc.
	Symptomatology	Macrocephalic/normocephalic/microcephalic Occult/symptomatic/overt Coma/stupr/dementia Hydrocephalus/parkinsonism complex, etc.
Hydrocephalus	Pathophysiology: -CSF circulation	Communicating/noncommunicating Nonobstructive/obstructive External/internal/interstitial Isolated compartments: UH/IFV/IRV/ICCD/DCH/DLFV, etc.
	-ICP dynamics	High/normal
	-Chronology	Slowness progressive/progressive/long-standing/arrested
Treatment	Postshunt	Shunt-dependent/shunt-independent slit-like ventricle/slit ventricle syndrome, etc.

SAH, subarachnoid hemorrhage; *IVH*, Intraventricular hemorrhage; *UH*, unilateral hydrocephalus; *IFV*, isolated fourth ventricle; *IRV*, isolated rombencephalic ventricle; *ICCD*, isolated central canal dilatation; *DCH*, double-compartment hydrocephalus; *DLFV*, disp

Representative subgroups in the classification of hydrocephalus

Congenital hydrocephalus

Simple hydrocephalus

(hydrocephalus with congenital change limited in the CSF pathway)

- Atresia of foramen of Monro
- Aqueductal stenosis
- Maldevelopment of arachnoid granulation
- Others

Dysgenetic hydrocephalus

(hydrocephalus associated with CNS dysgenesis)

- Hydranencephaly
- Holoprosencephaly
- Dandy-Walker syndrome
- Dysraphism
- Chiari malformation
- Syringobulbia-myelia
- Lissencephaly
- Arachnoid cyst, etc.
- Others

Secondary congenital hydrocephalus

(hydrocephalus secondary to underlying lesion in the fetal period)

- Tumor
- Posthemorrhagic
- Postinfectious
- Others

Acquired hydrocephalus

Acquired hydrocephalus

(hydrocephalus secondary to underlying lesion in the postnatal period)

- Tumor
- Posthemorrhagic
- Postinfectious
- Post-traumatic
- Others

Postshunt hydrocephalus

(hydrocephalus after shunt placement)

- Shunt-dependent
- Unilateral hydrocephalus
- Isolated fourth ventricle
- Isolated rhombencephalic ventricle
- Isolated central canal dilatation

Adapted from [Oi S, 2003], Volpe, JJ, *Neurology of the Newborn*, 4th Ed., 2001

Genetics of Hydrocephalus (malformation)

- X-linked hydrocephalus
- Dandy-Walker malformation
- Holoprosencephaly
- Neural Tubes Defects
- Tuberous Sclerosis
- Agenesis of Corpus Callosum
- Interhemispheric Cysts
- Migration Abnormality

Neonatal Hydrocephalus (other causes)

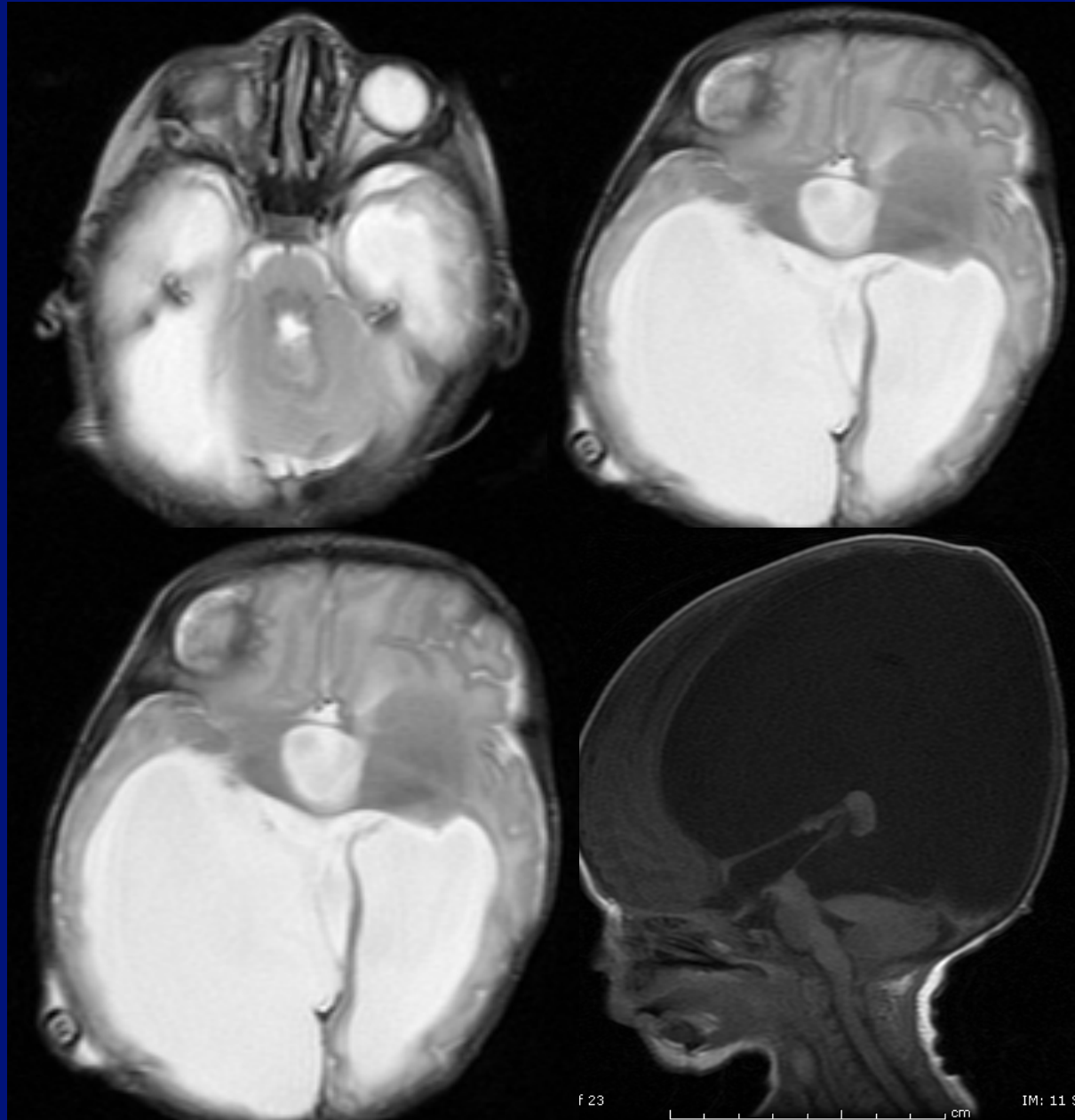
- Hemorrhage
- Infection
- Tumors

NEONATAL CONGENITAL MALFORMATIONS

X-linked Hydrocephalus

- Aqueductal Stenosis
- 1:30,000 male births
- Chromosome Xq28
- L1 CAM gene (neural cell surface adhesion molecule)
- CRASH syndrome (corpus callosal hypoplasia, mental retardation, adducted thumbs, spastic paraplegia and hydrocephalus)

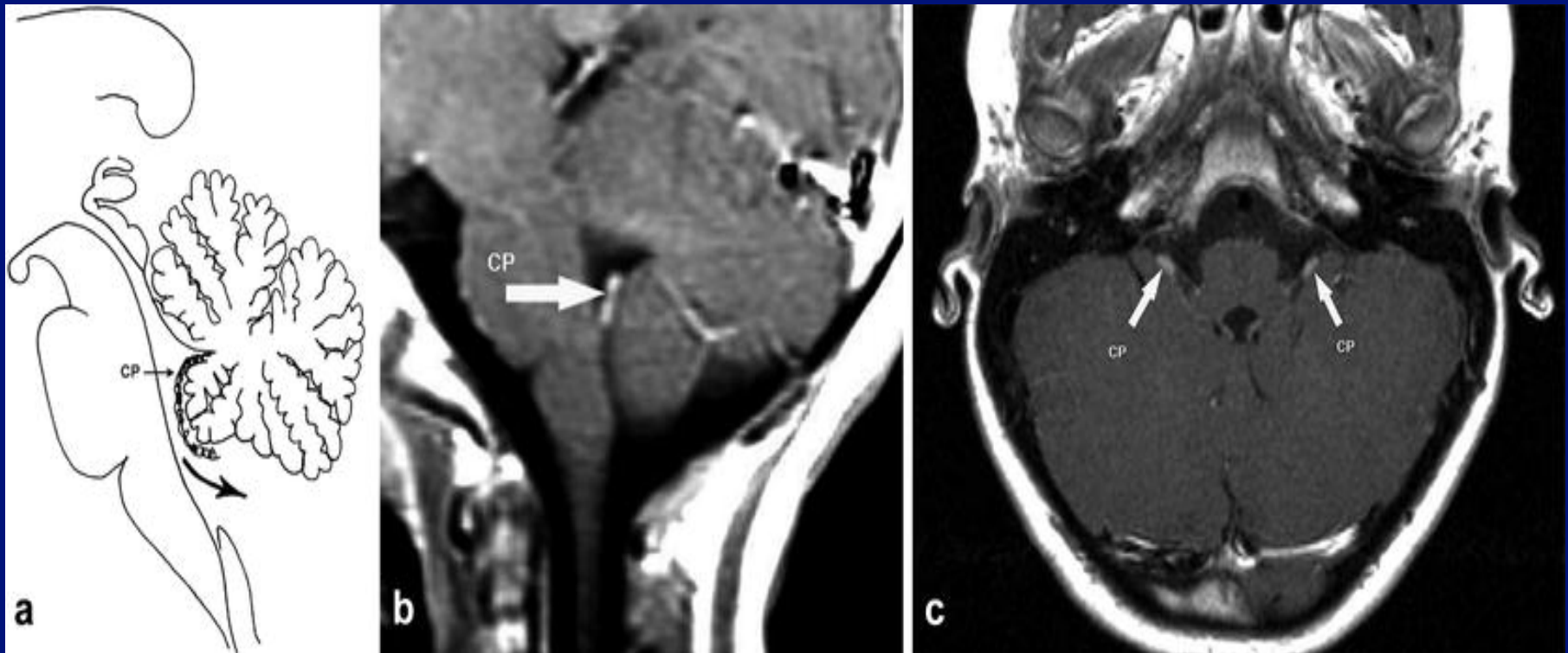
Aqueductal Stenosis



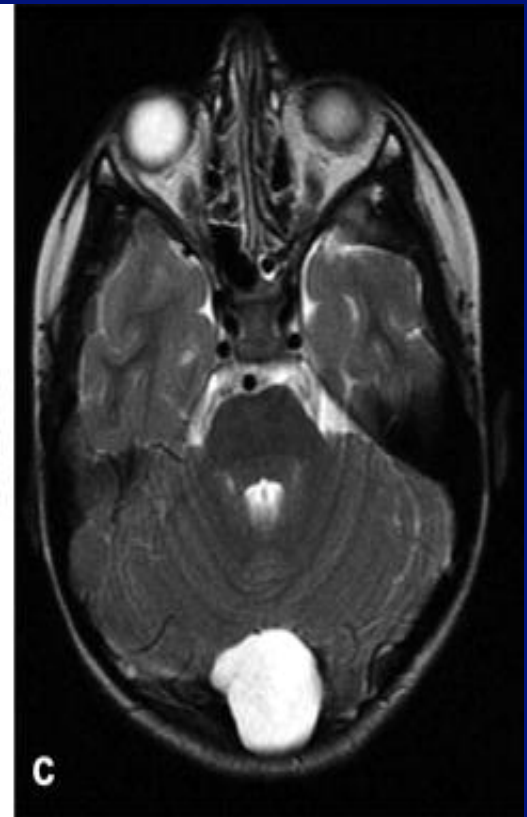
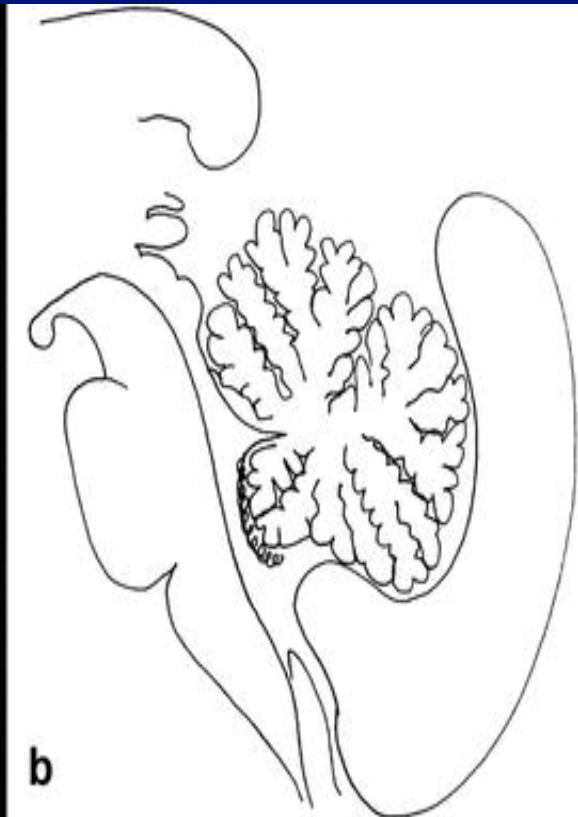
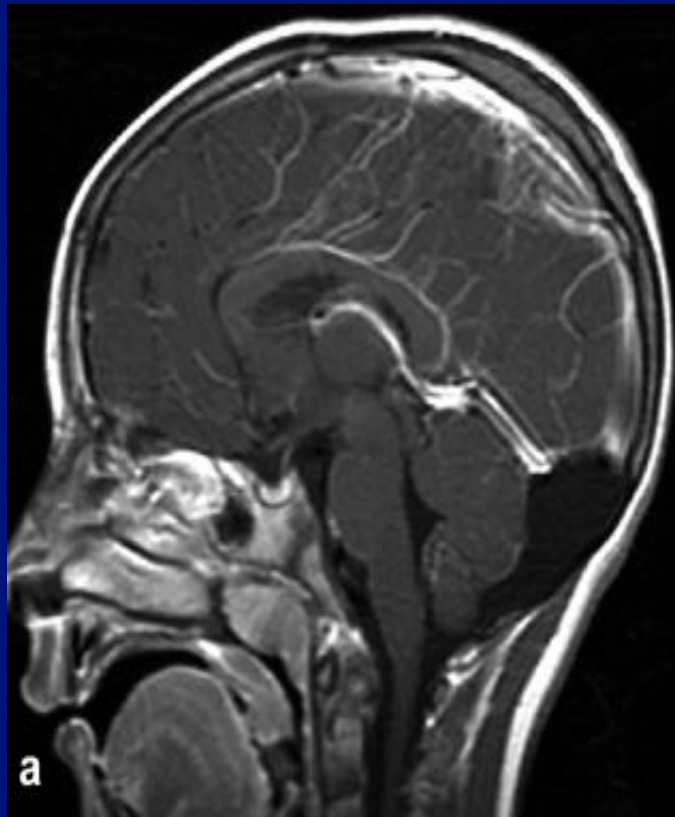
Posterior Fossa Cyst Differential: Role of Choroid Plexus

- Retrocerebellar arachnoid cyst
- Blake's pouch
- Dandy-Walker variant
- Dandy-Walker malformation (syndrome)

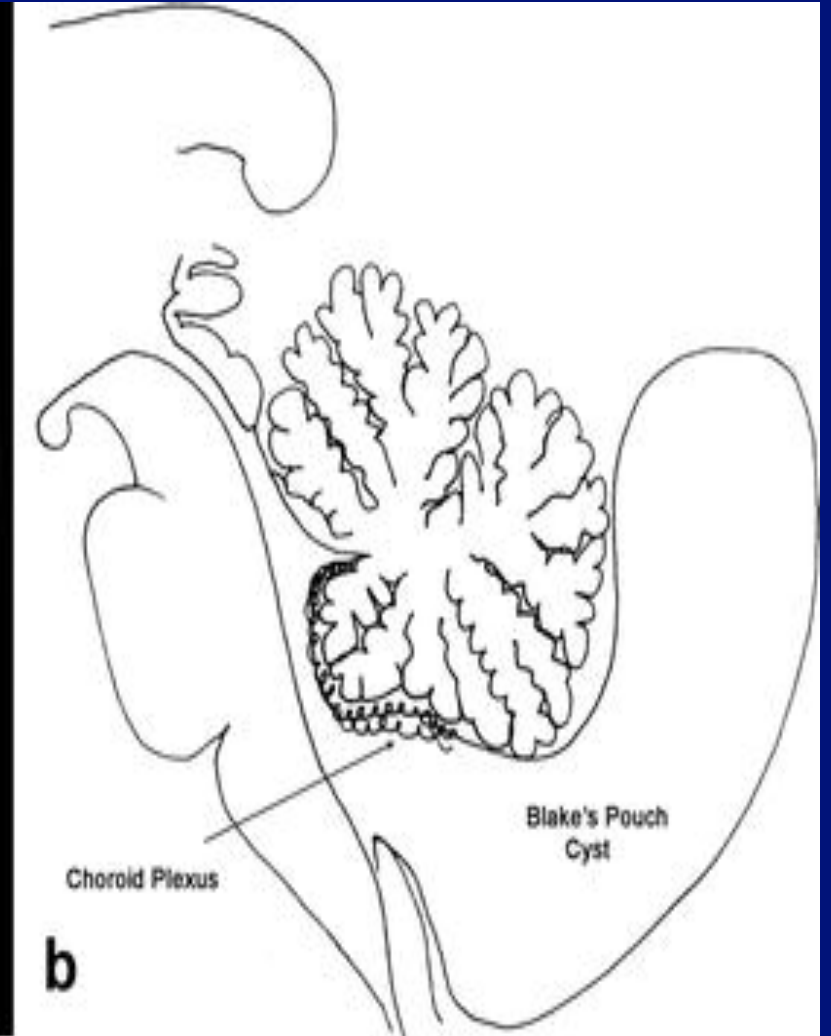
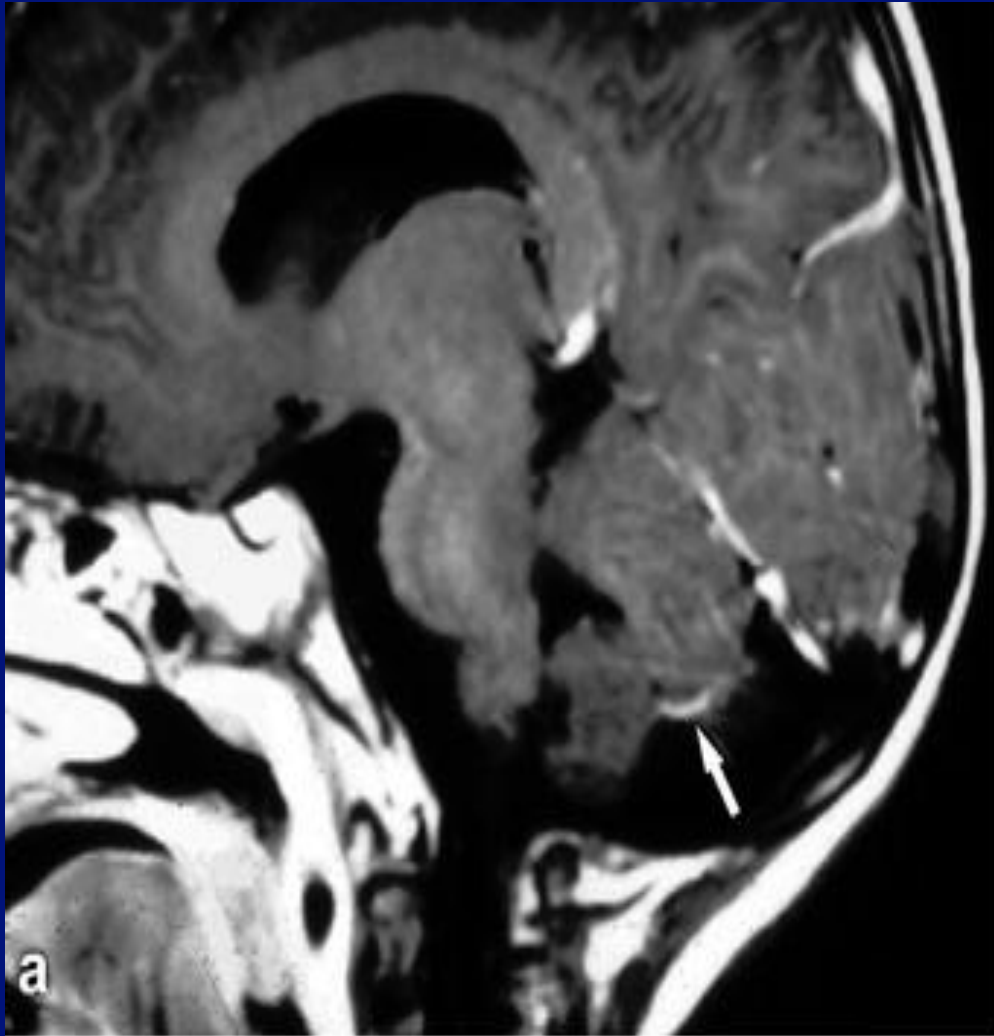
Normal Position of Choroid Plexus



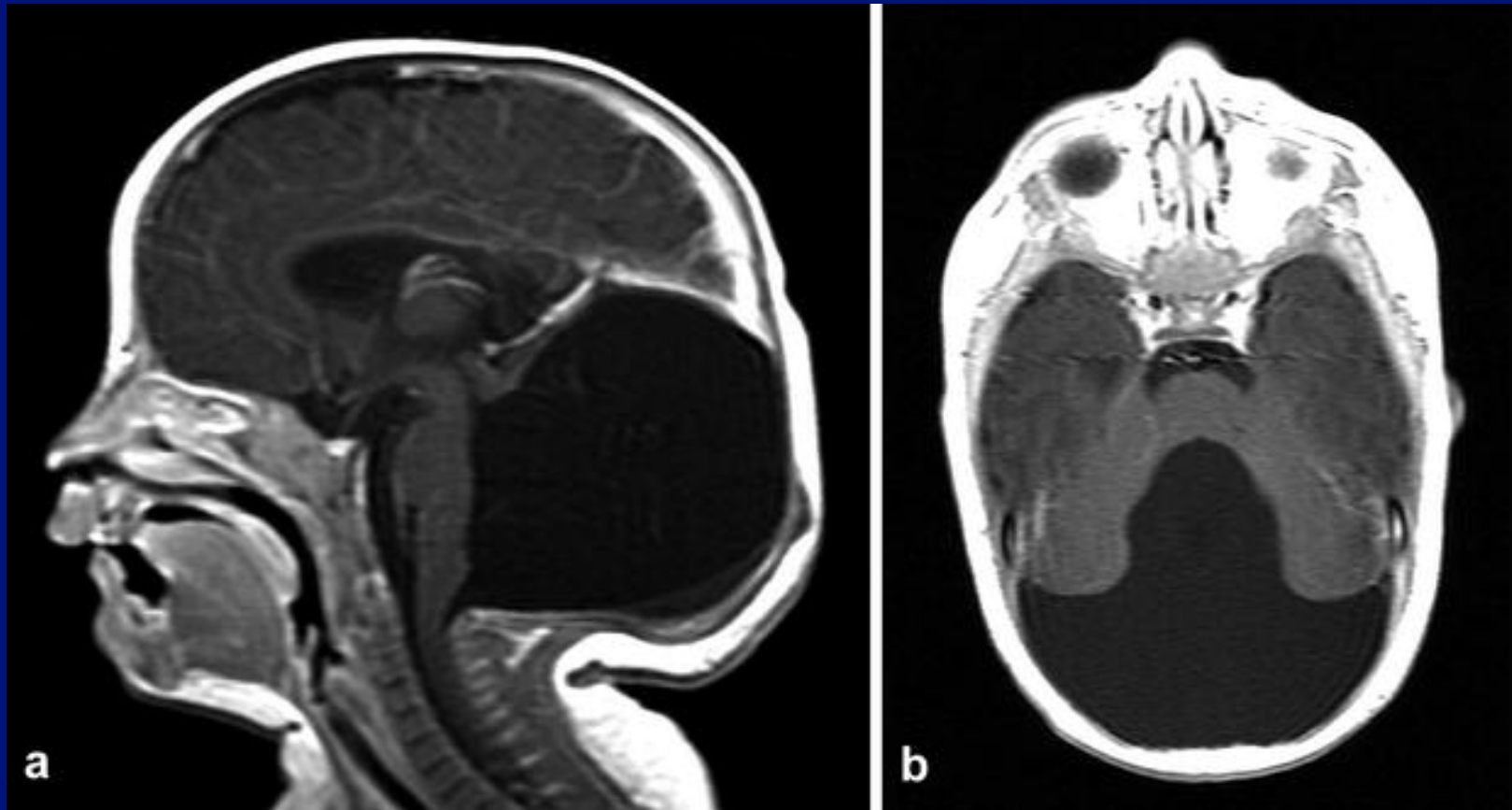
Retrocerebellar Arachnoid Cyst



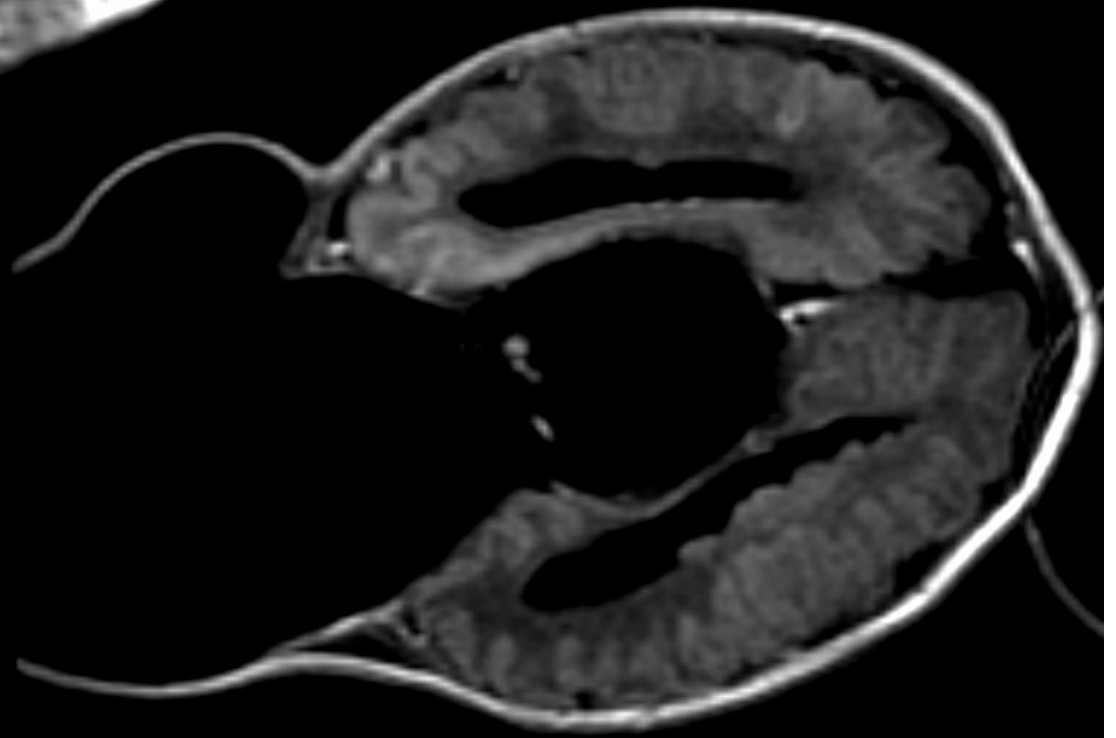
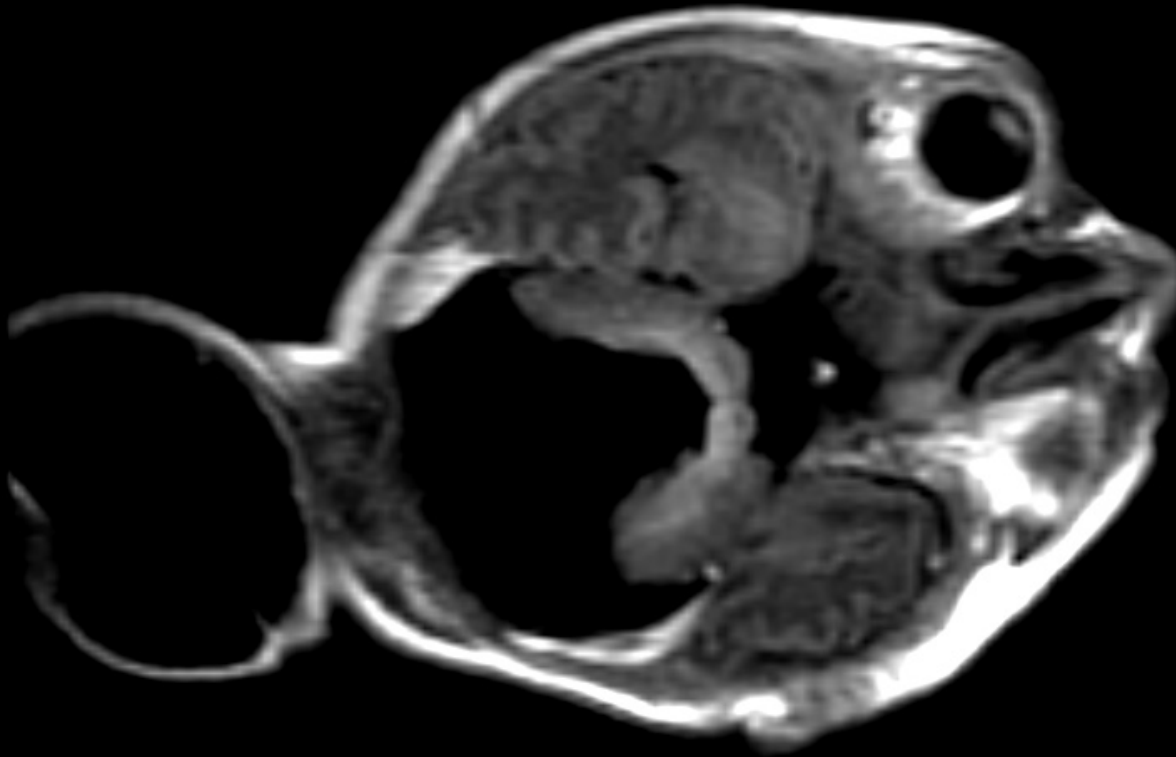
Blake's Pouch



Dandy-Walker Malformation



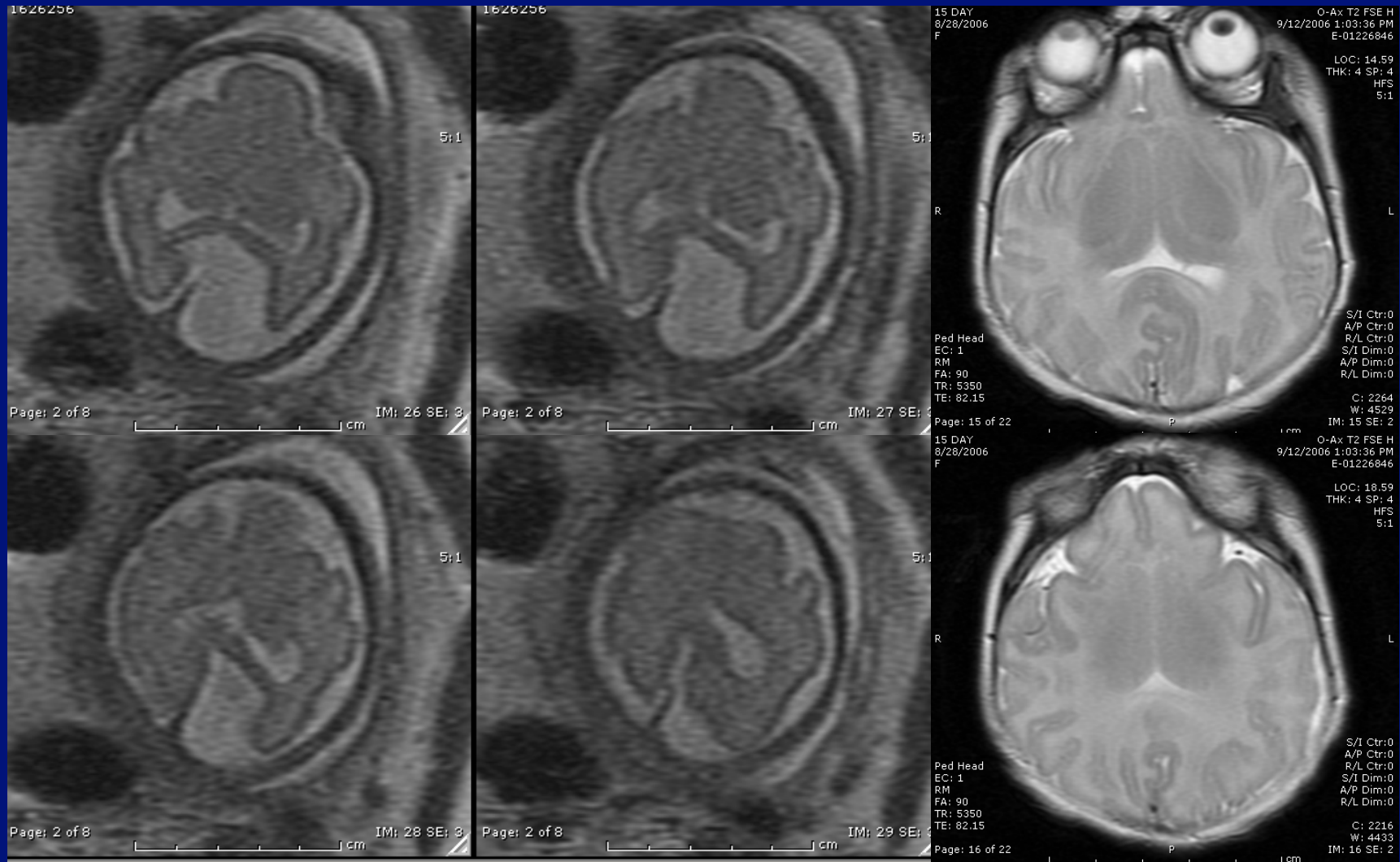
Associated
Abnormalities with
Dandy-Walker
Malformation

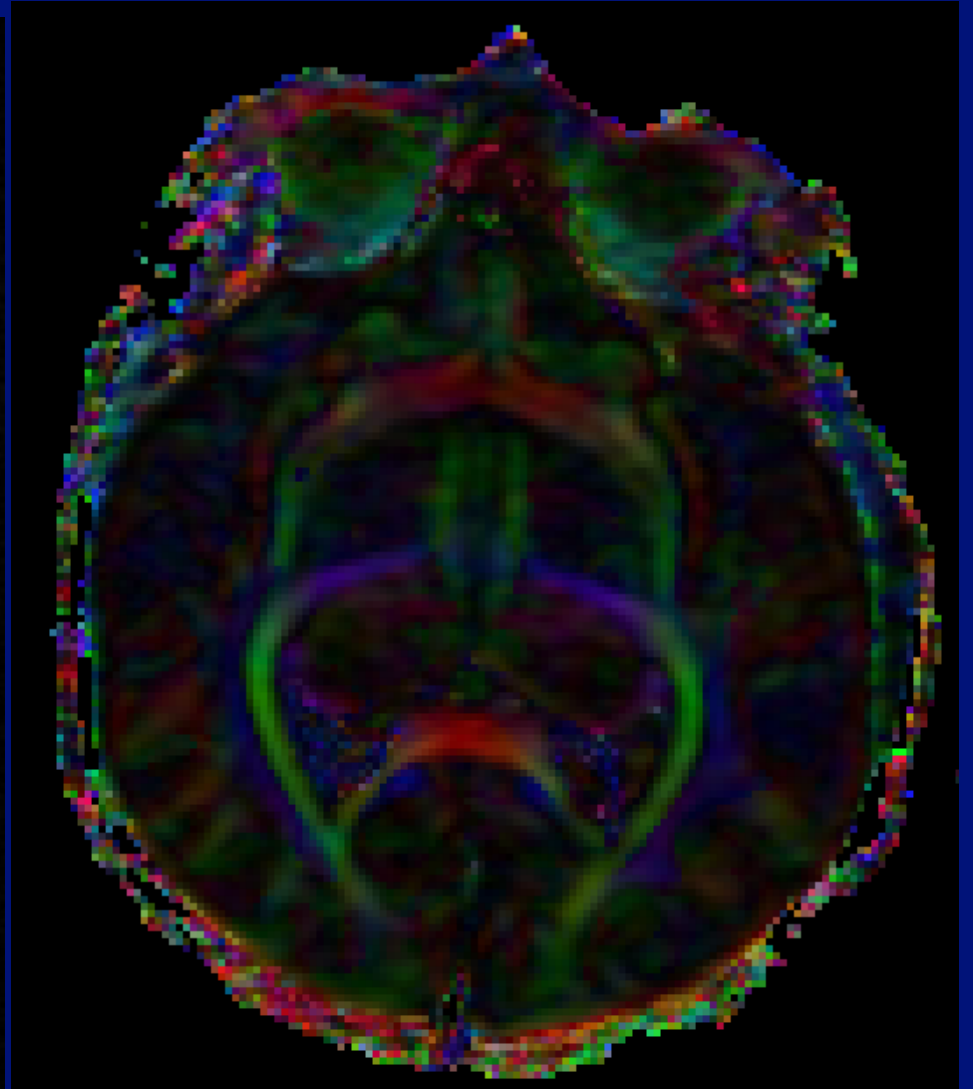


Holoprosencephaly

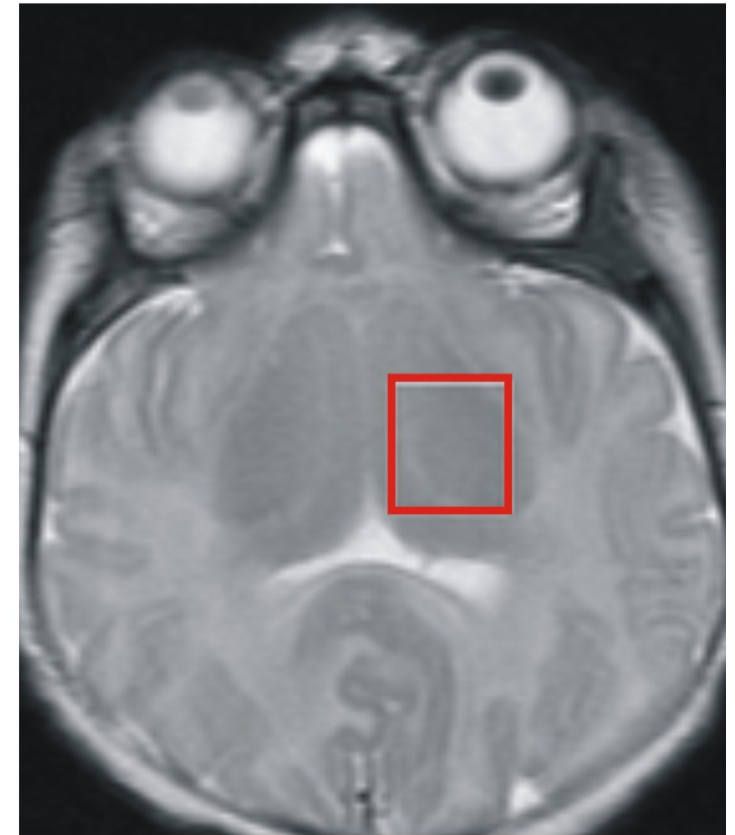
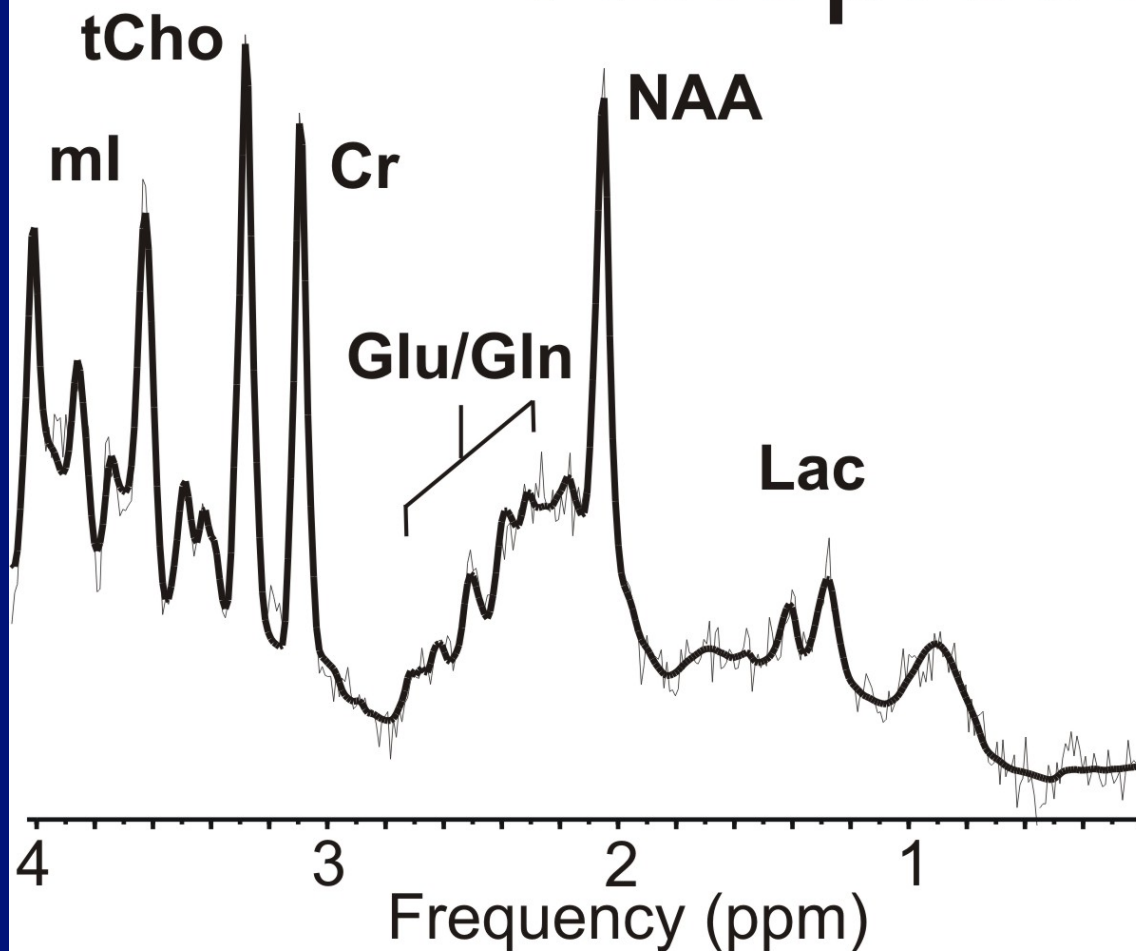
- **Incomplete cleavage of the forebrain into two hemispheres**
- **Alobar, semi-lobar, lobar**
- **Wide spectrum of associated clinical abnormalities**
- **Sonic hedgehog (Shh) gene**

Semi-Lobar Holoprosencephaly





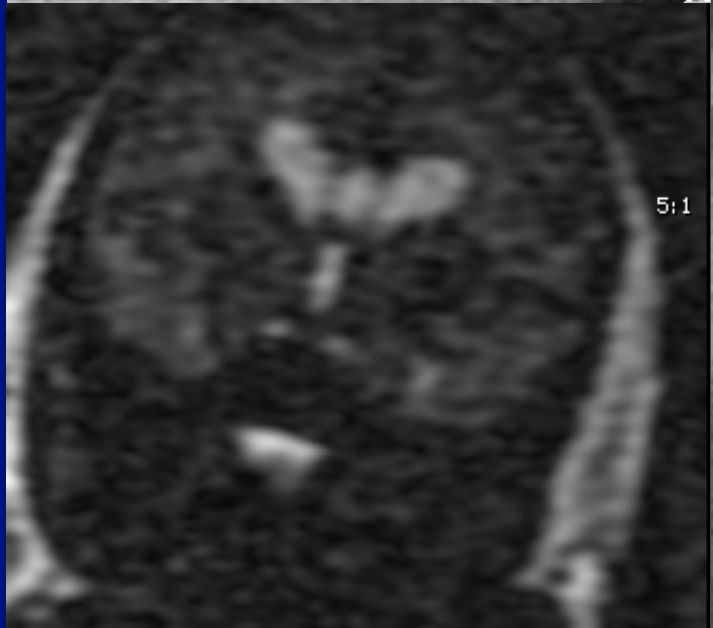
Holoprosencephaly



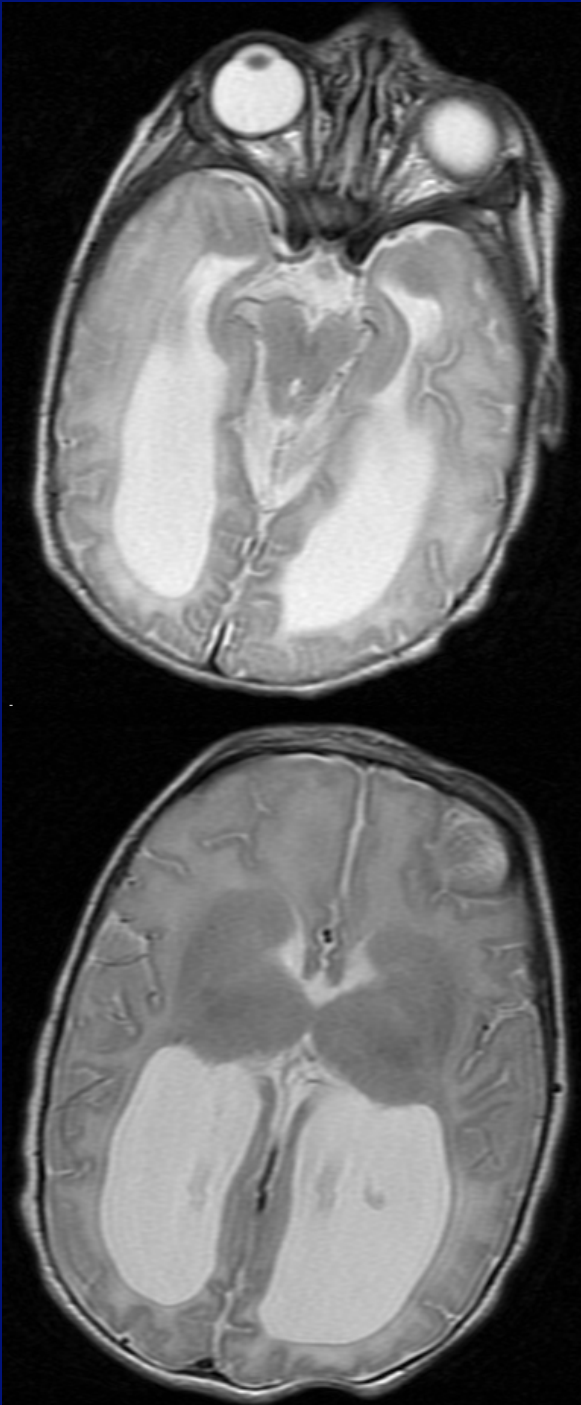
Neural Tube Defects

- 1:1000 live birth (common)
- Failure of anterior neuropore closure (anencephaly); failure of posterior neuropore closure (myelomeningocele); encephaloceles
- Gene mutations, chromosomal abnormalities, environmental factors
- Prevented by folic acid

Chiari II Malformation: Prenatal Imaging

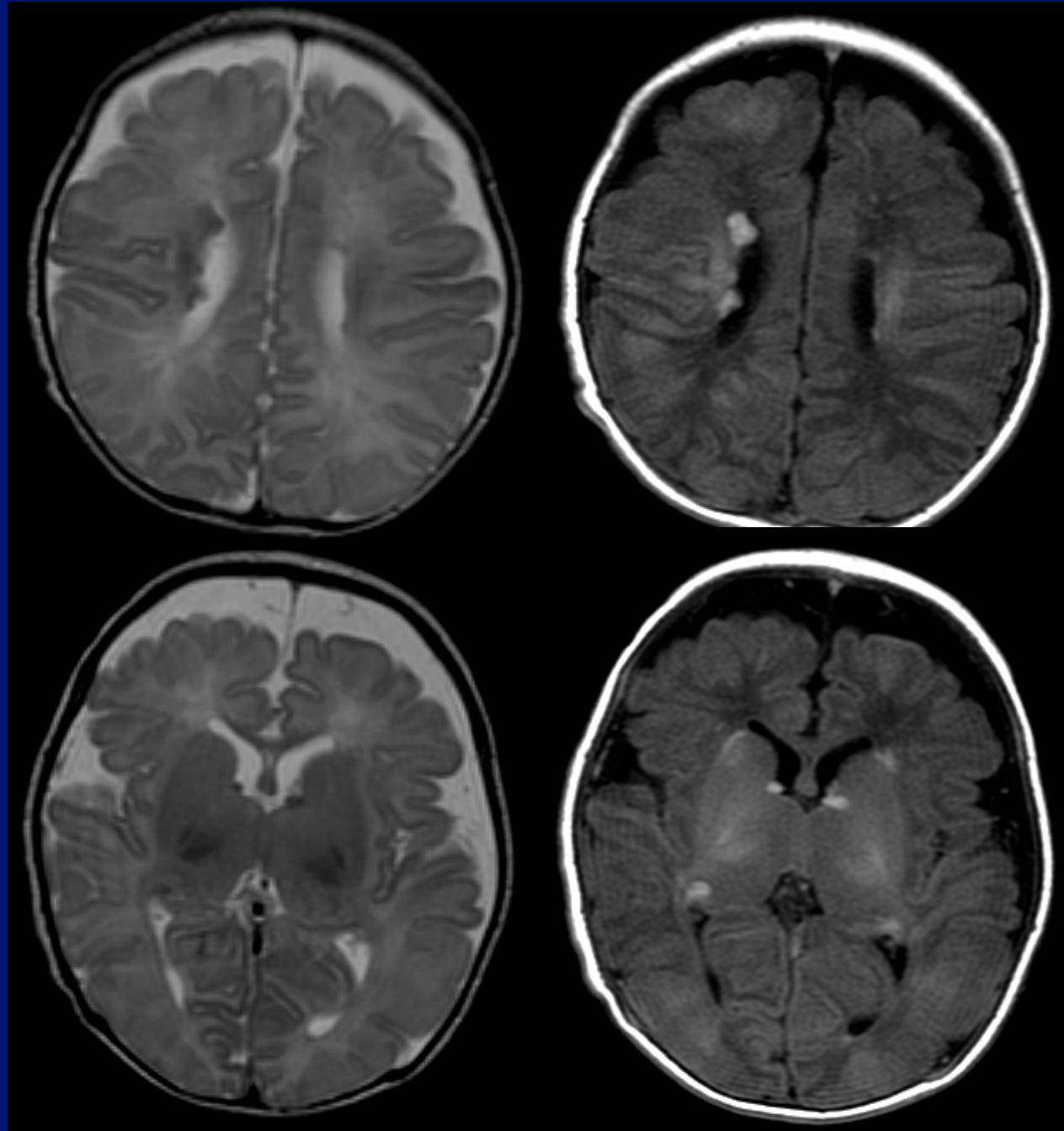


Chiari II Malformation: Postnatal Imaging



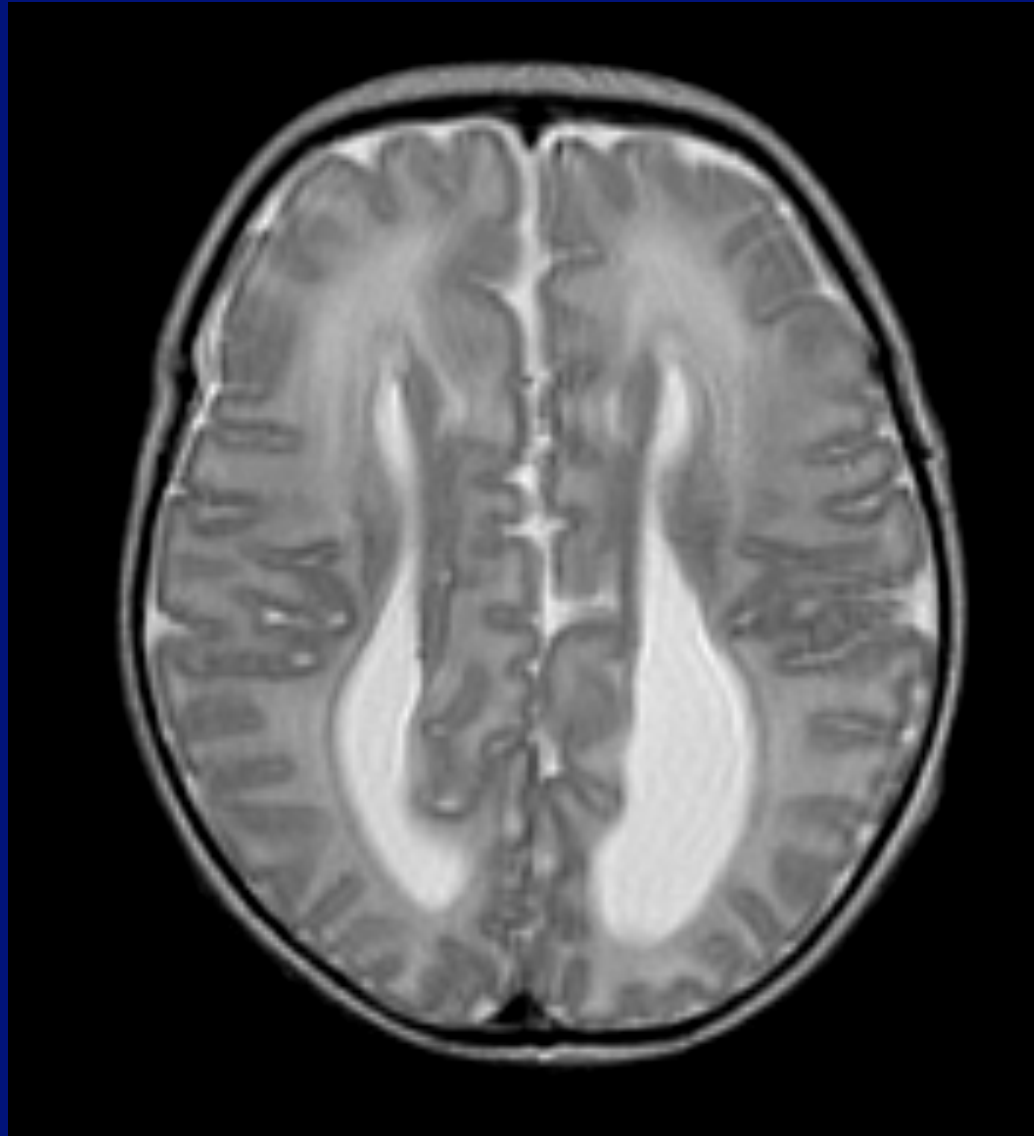
Neonatal Tuberous Sclerosis

- Autosomal Dominant syndrome: multi-system involvement including: brain eye, skin, kidneys, heart
- Hamartomas of all three germ layers: cortical tuber, subependymal nodules and subependymal giant cell astrocytomas
- TSC₁ and TSC₂ gene

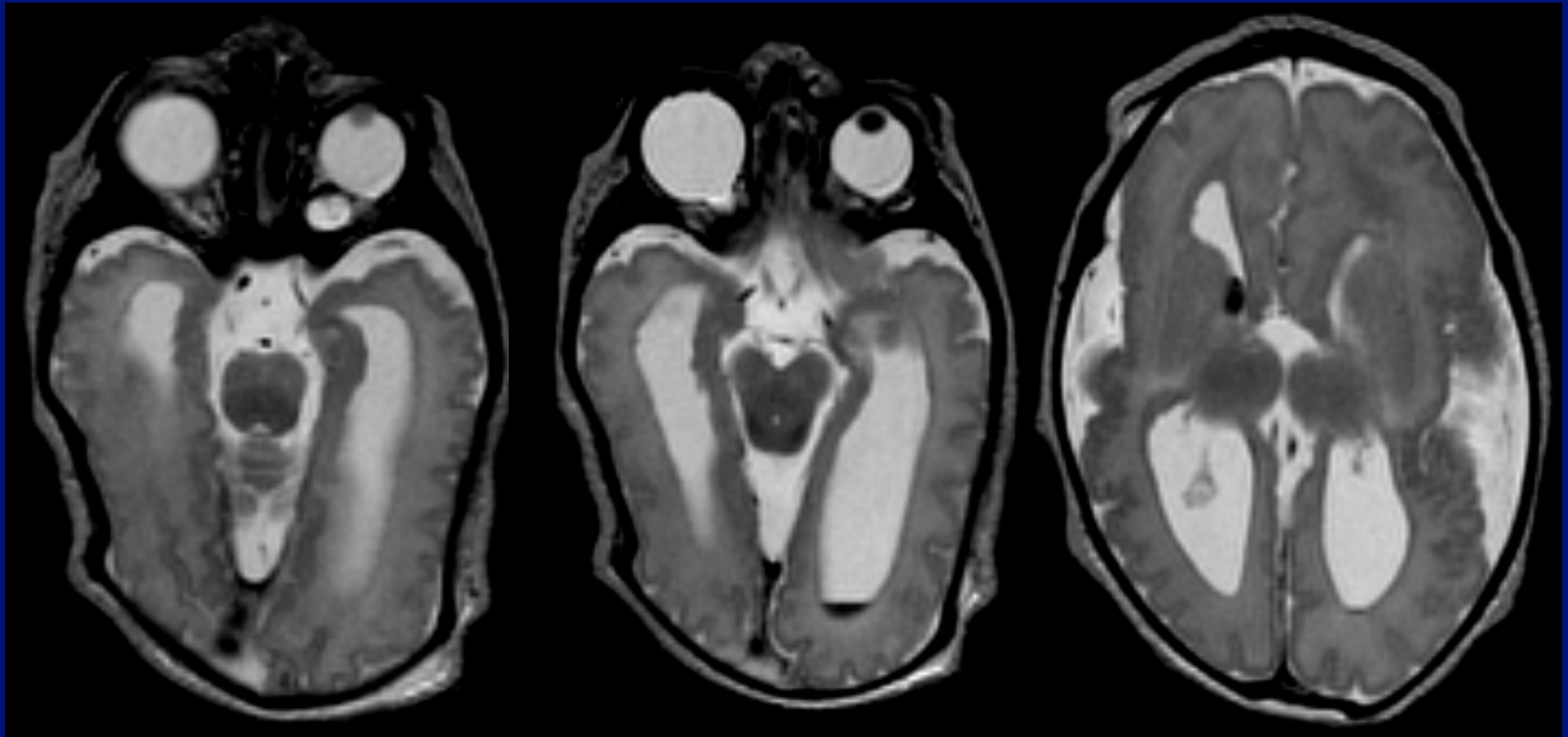


AGENESIS OF THE CORPUS CALLOSUM

Agnesis of the Corpus Callosum: Isolated



Agnesis of the Corpus Callosum: Associated with Other Abnormalities (syndromic)



Aicardi Syndrome

- Rare genetic disorder characterized by infantile spasm (jerking) and mental retardation
- Less than 500 cases in the world
- X-chromosome or random sporadic mutation
- Polymicrogyria, agenesis of CC, coloboma, heterotopia

Neonatal Hydrocephalus (other causes)

- Hemorrhage
- Infection
- Tumors

NEONATAL HEMORRHAGE

Neuropathology of Subdural Hemorrhage

SOURCE OF BLEEDING	LOCATION OF HEMATOMA
TENTORIAL LACERATION Straight sinus, vein of Galen, transverse sinus, and infratentorial veins	Infratentorial (posterior fossa), supratentorial
OCCIPITAL OSTEODIASTASIS Occipital sinus	Infratentorial (posterior fossa)
FALX LACERATION Inferior sagittal sinus	Longitudinal cerebral fissure
SUPERFICIAL CEREBRAL VEINS	Surface of cerebral convexity

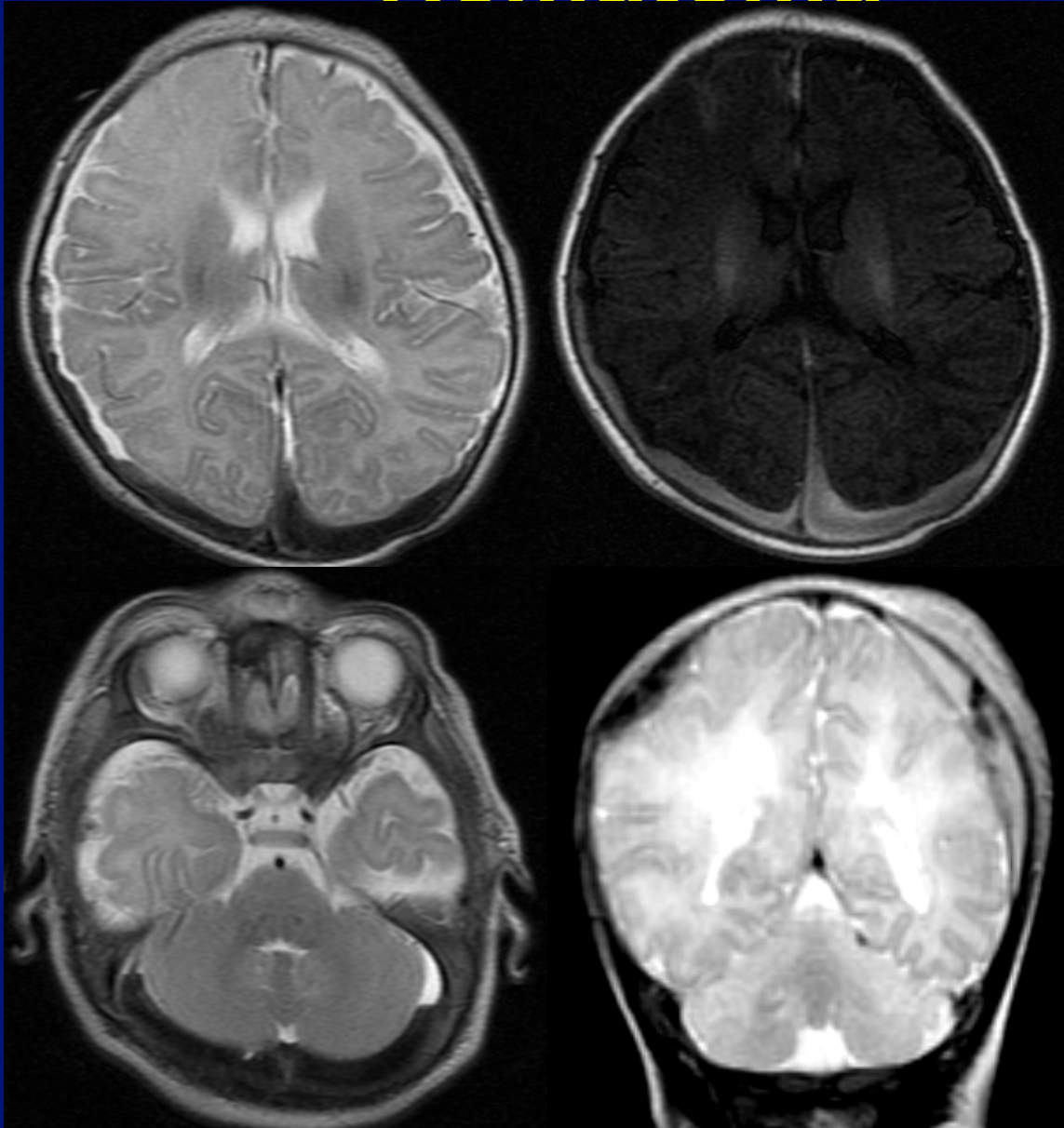
Adapted from Volpe, JJ, *Neurology of the Newborn*, 4th Ed., 2001

Pathogenesis of Neonatal Subdural Hemorrhage

AT RISK	PREDISPOSING FACTORS
Mother	Primiparous Older multiparous Small birth canal
Infant	Large full term Premature
Labor	Precipitous Prolonged
Delivery	Breech extraction Foot, face, brow presentation Difficult forceps or vacuum extraction Difficult rotation

Adapted from Volpe, JJ, *Neurology of the Newborn*, 4th Ed., 2001

Subdural vs Epidural Hematoma



Miscellaneous Examples of Neonatal Intracranial Hemorrhage

TRAUMA

Epidural hemorrhage
Intracranial hemorrhage

HEMORRHAGIC INFRACTION

Embolus
Venous thrombosis
Arterial thrombosis

COAGULATION DISTURBANCE

Thrombocytopenia
Deficiency of coagulation factors

VASCULAR DEFECT

Aneurysm
Arteriovenous malformation
Coarctation of the aorta

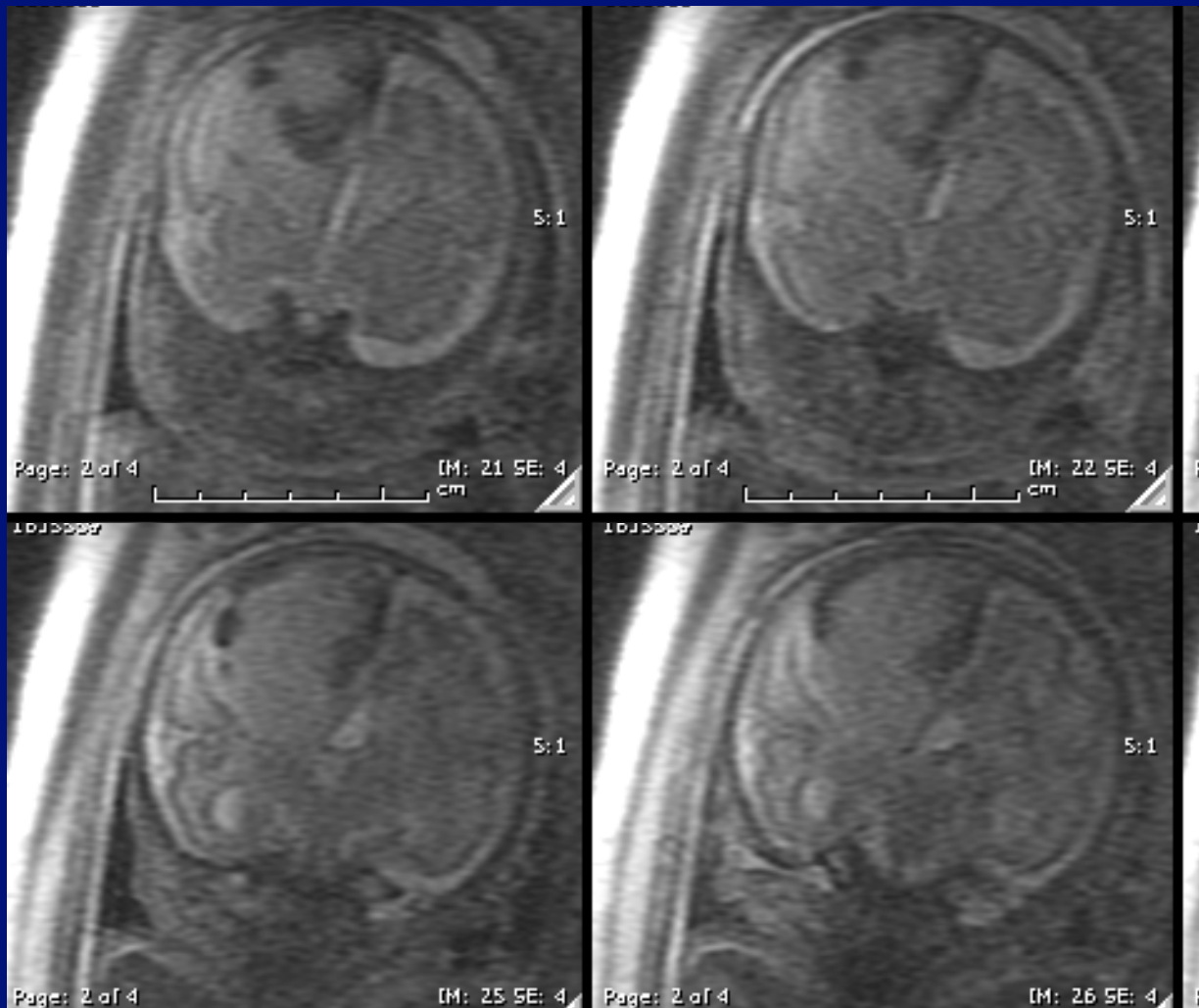
CEREBRAL TUMOR

UNKNOWN CAUSE

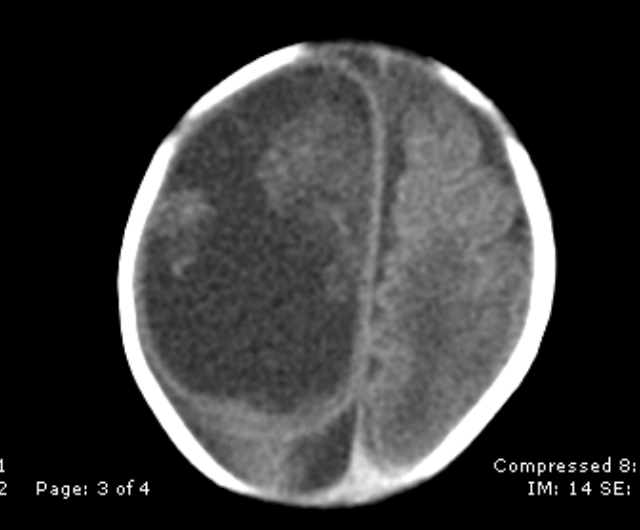
EXTRACORPOREAL MEMBRANE OXYGENATION

Adapted from Volpe, JJ, *Neurology of the Newborn*, 4th Ed., 2001

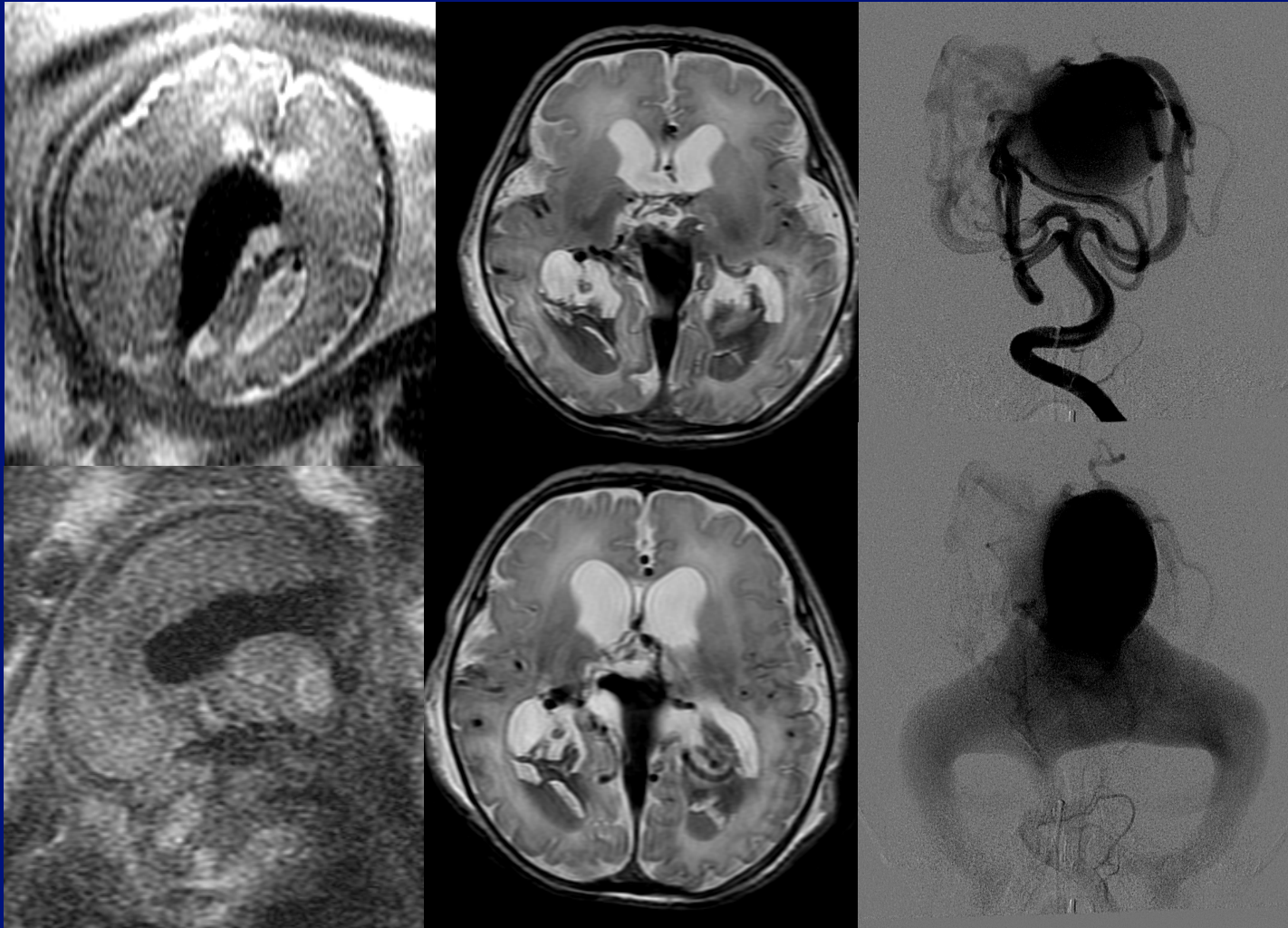
Alloimmune Thrombocytopenia



Alloimmune Thrombocytopenia



Vein of Galen AV Fistula

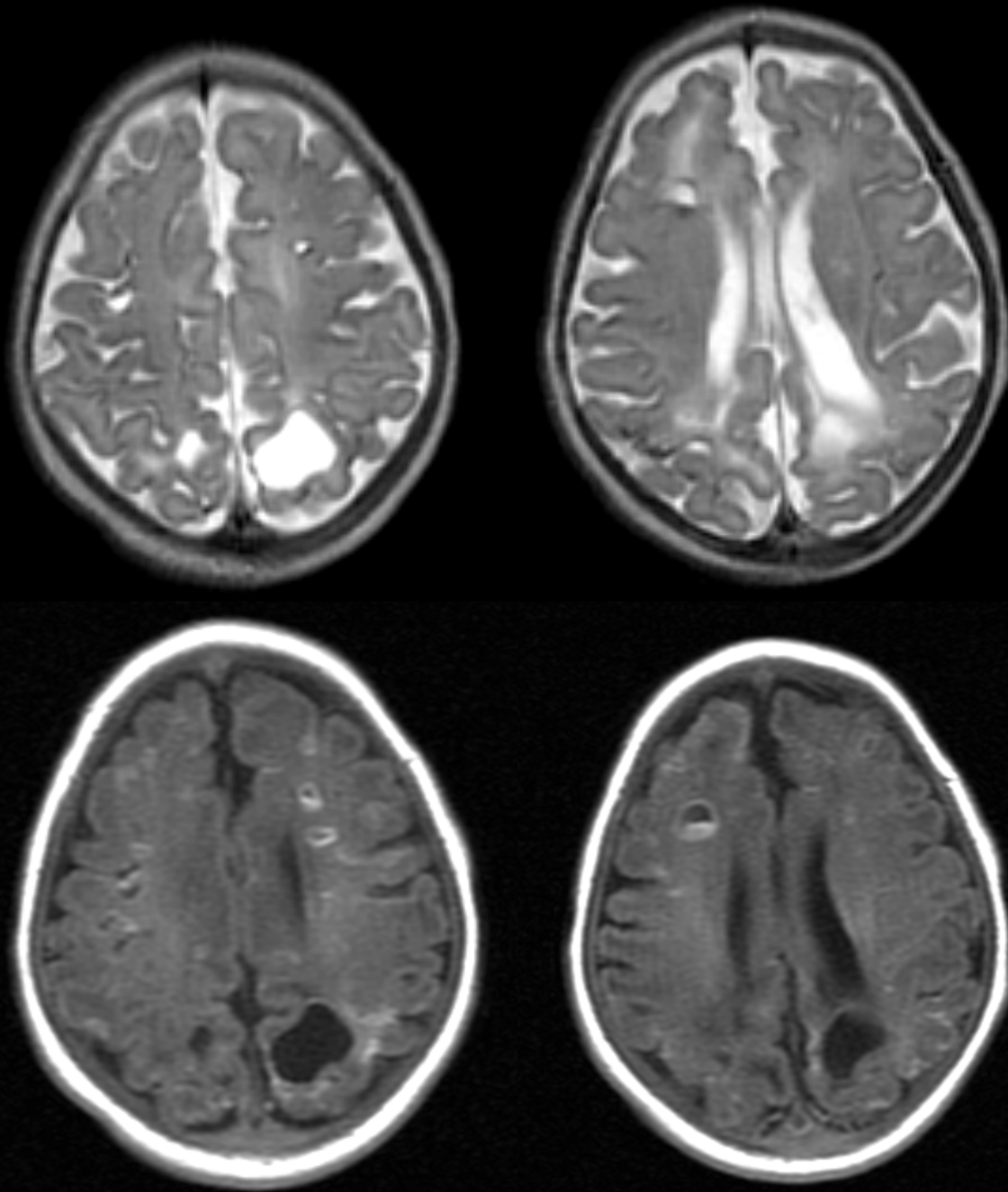


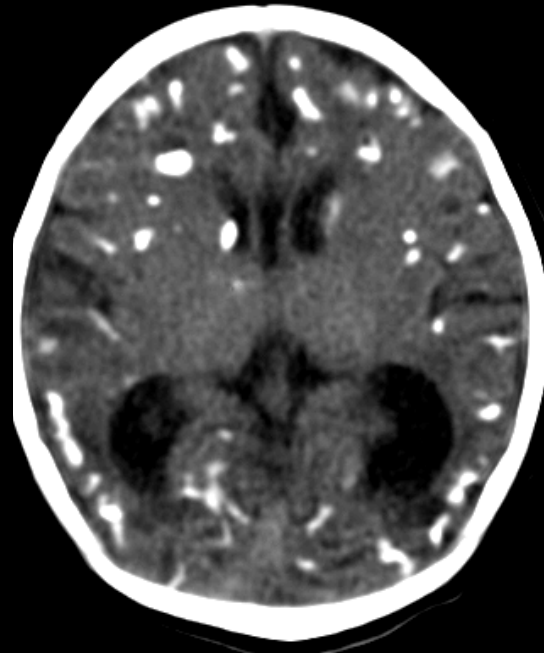
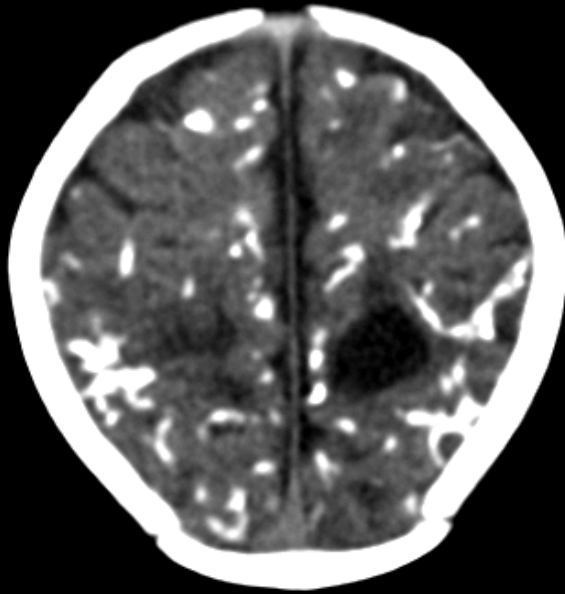
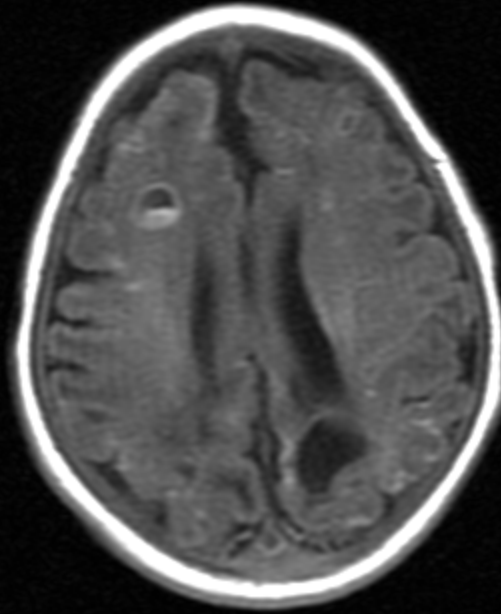
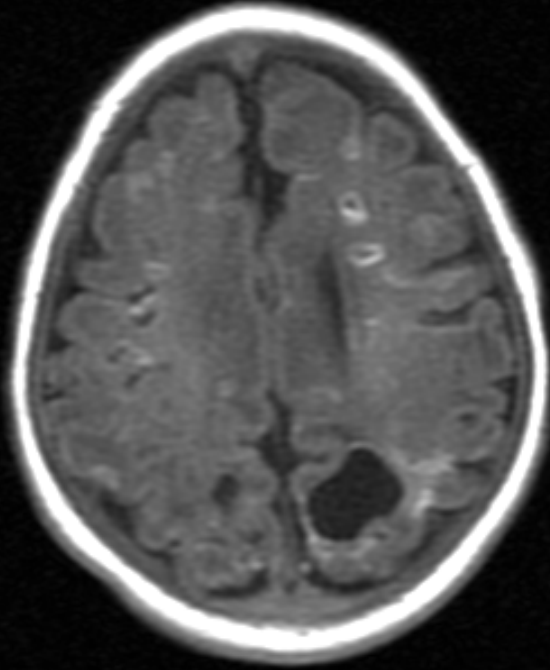
NEONATAL INFECTION

Central Nervous System Involvement by the TORCH Group

ORGANISM	MAJOR ROUTE OF INFECTION
Cytomegalovirus	Transplacental
Herpes simplex	Ascending and/or parturitional
Rubella	Transplacental
Toxoplasmosis	Transplacental
Syphilis	Transplacental
Human immunodeficiency virus	Transplacental/parturitional

Volpe, JJ, *Neurology of the Newborn*, 4th Ed., 2001





Compress

Compress

Bacterial Etiology of Neonatal Meningitis

BACTERIAL ETIOLOGY	PERCENT OF TOTAL
Group B <i>Streptococcus</i>	49
Other streptococci and staphylococci (includes groups D and E beta-hemolytic and alpha-hemolytic streptococci, <i>Streptococcus pneumoniae</i> , <i>Staphylococcus epidermidis</i> , and <i>Staphylococcus aureus</i>)	7
Escherichia Coli	20
Other gram-negative enterics (include <i>Pseudomonas aeruginosa</i> , <i>Klebsiella</i> and <i>Enterobacter</i> species, <i>Procteus</i> species, <i>Citrobacter</i> species, <i>Serratia marcescens</i> , and so forth)	10
Listeria monocytogenes	7
Other (includes <i>Haemophilus influenzae</i> , <i>Salmonella</i> species, <i>Flavobacterium meningosepticum</i> , and so forth)	7

Adapted from Volpe, JJ, *Neurology of the Newborn*, 4th Ed., 2001

Neonatal Bacterial Meningitis

ACUTE

Arachnoiditis

Ventriculitis--choroid plexitis

Vasculitis

Cerebral edema

Infarction

Associated encephalopathy (cortical neuronal necrosis,
periventricular leukomalacia)

CHRONIC

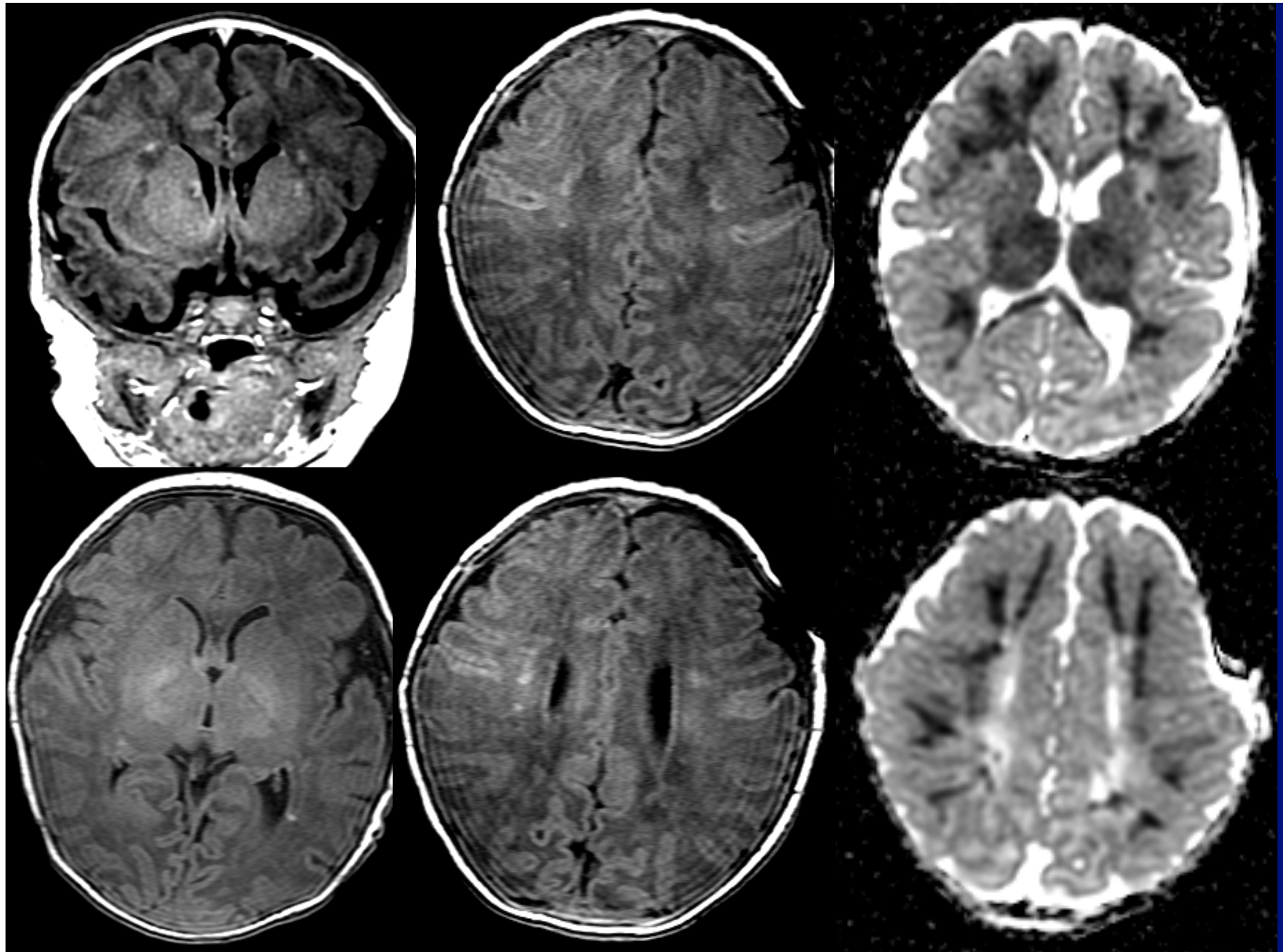
Hydrocephalus

Multicystic encephalomalacia--porencephaly

Cerebral cortical and white matter atrophy

Cerebral cortical developmental (organizational) defects (?)

Adapted from Volpe, JJ, *Neurology of the Newborn*, 4th Ed., 2001



Human Parechovirus Causes Encephalitis with White Matter Injury in Neonates

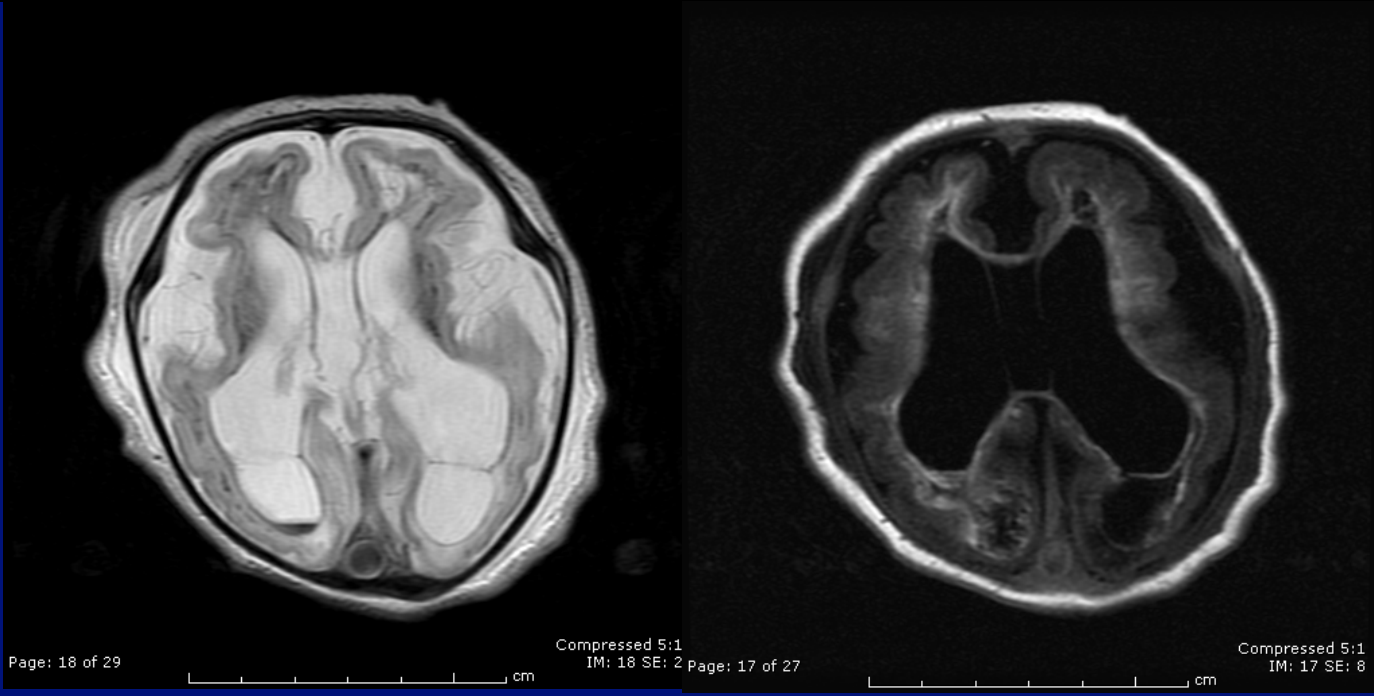
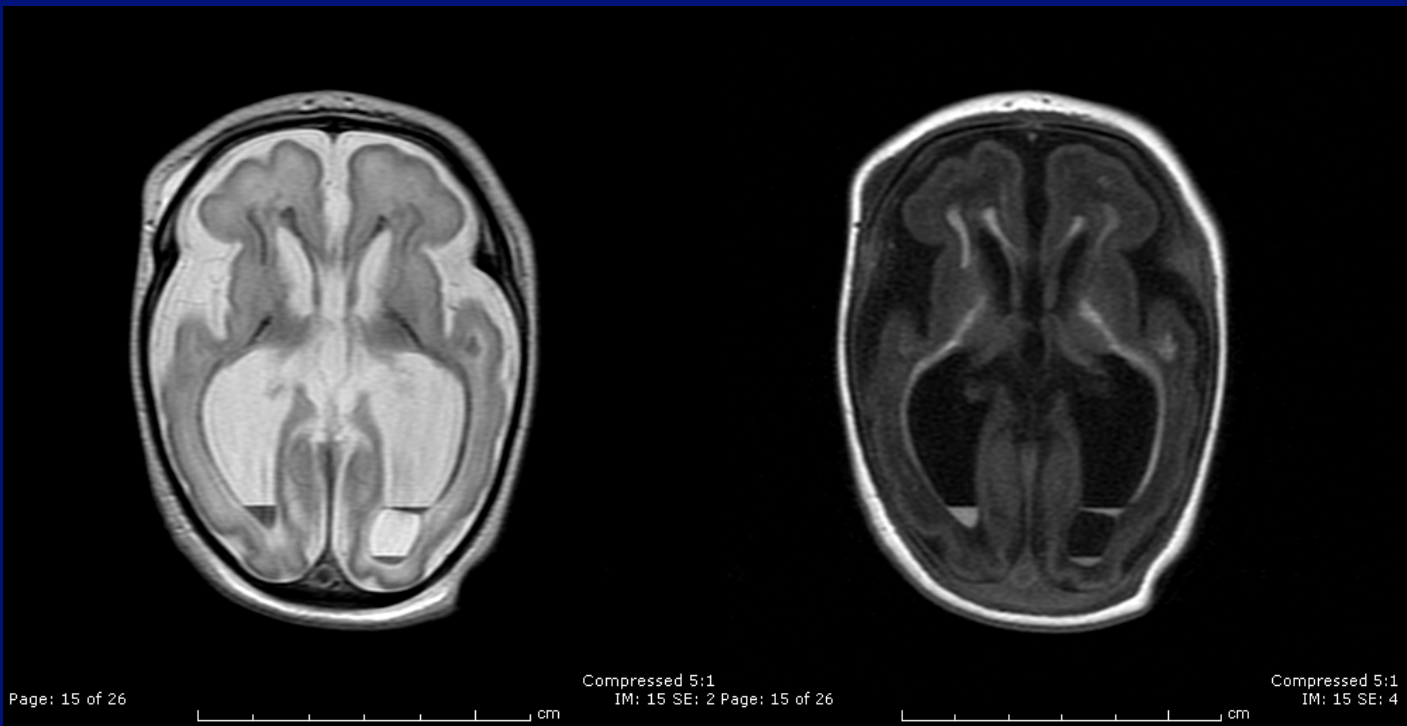
Malgorzata A. Verboon-Maciolek, MD, PhD,¹ Floris Groenendaal, MD, PhD,¹ Cecil D. Hahn, MD, MPH,² Jonathan Hellmann, MBBCh,³ Anton M. van Loon, PhD,⁴ Guy Boivin, MD, PhD,⁵ and Linda S. de Vries, MD, PhD¹

Objective: To assess the role of human parechoviruses (HPeVs) as a cause of neonatal cerebral infection and to report neuroimaging findings of newborn infants with encephalitis caused by HPeVs.

Methods: Clinical presentation, cranial ultrasonography, magnetic resonance imaging (MRI) findings, and neurodevelopmental outcome of 10 infants admitted to a neonatal intensive care unit and diagnosed with encephalitis caused by HPeVs are reported.

Results: Nine of 10 infants, with a gestational age of 29 to 41 weeks, presented at 36 to 41 weeks postmenstrual age with clinical seizures. Seven had a fever and six had a rash. Clinical presentation was similar to that of infants with enterovirus infection. Cranial ultrasonography showed increased echogenicity in the periventricular white matter in all infants. Neonatal MRI confirmed white matter changes in nine infants, which changed to gliosis on later MRI. Outcome was variable with cerebral palsy in one, a suspect outcome at 18 months in one, learning disabilities at 7 years of age in one, epilepsy in one, and normal neurodevelopmental outcome in five children. Follow-up of one infant was only 9 months.

Interpretation: HPeVs should be added to the list of neurotropic viruses that may cause severe central nervous system infection in the neonatal period. White matter injury can be visualized with cranial ultrasonography, but more detailed information is obtained with MRI and especially diffusion-weighted imaging. Because clinical presentation of HPeV encephalitis is similar to that of enterovirus, real-time polymerase chain reaction for both viruses should be performed in atypical presentation of neonatal seizures. *Ann Neurol* 2008;64:266–273



NEONATAL BRAIN TUMORS

Neonatal Brain Tumors: Histological Types

	PERCENT OF TUMORS WITH PRESENTATION AT BIRTH	PERCENT OF TUMORS WITH PRESENTATION IN FIRST 2 MONTHS
Teratoma	48	26
Neuroepithelial	37	65
Other	17	9

Adapted from Volpe, JJ, *Neurology of the Newborn*, 4th Ed., 2001

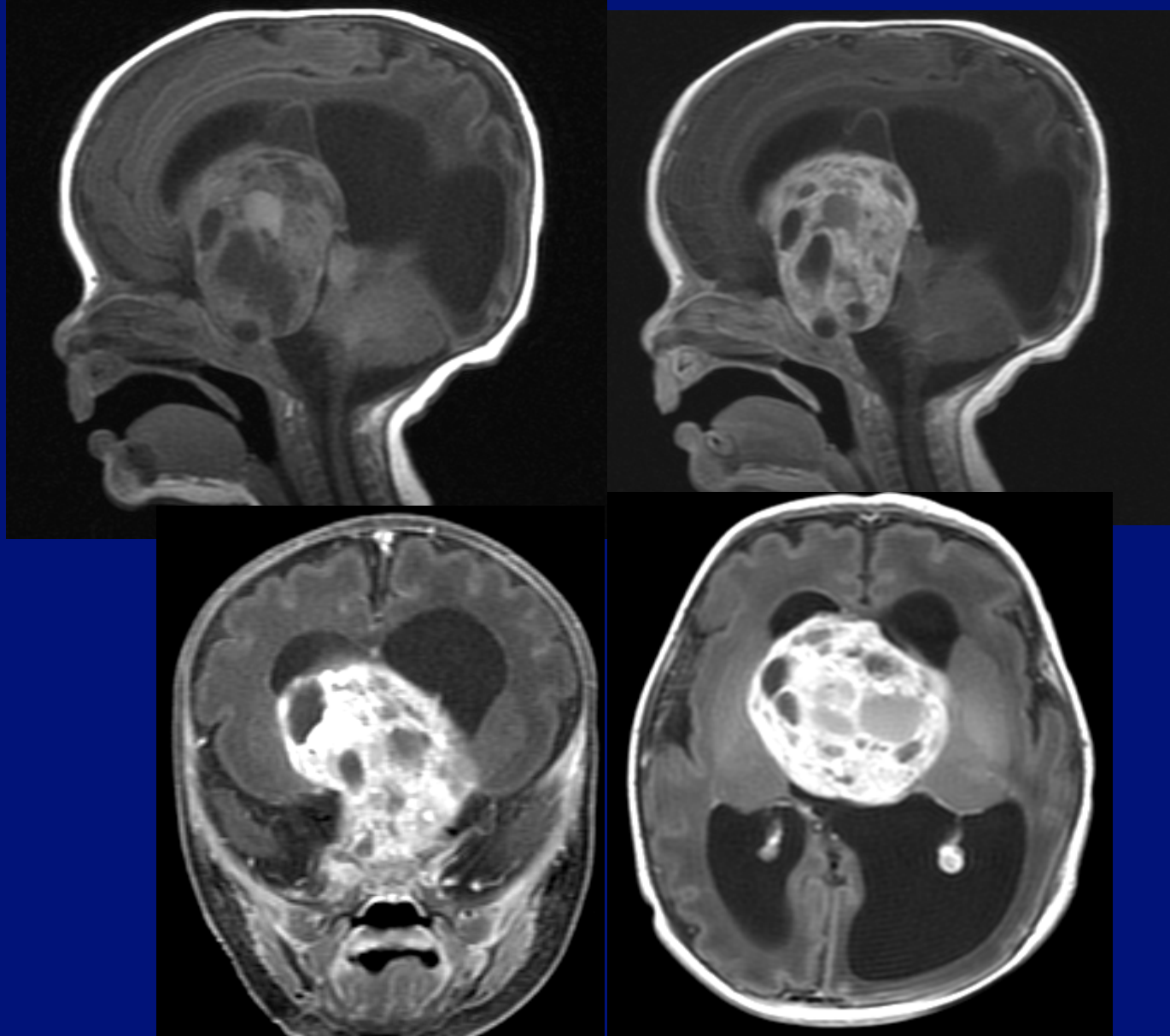
Types of Nonteratomatous Neonatal brain Tumors

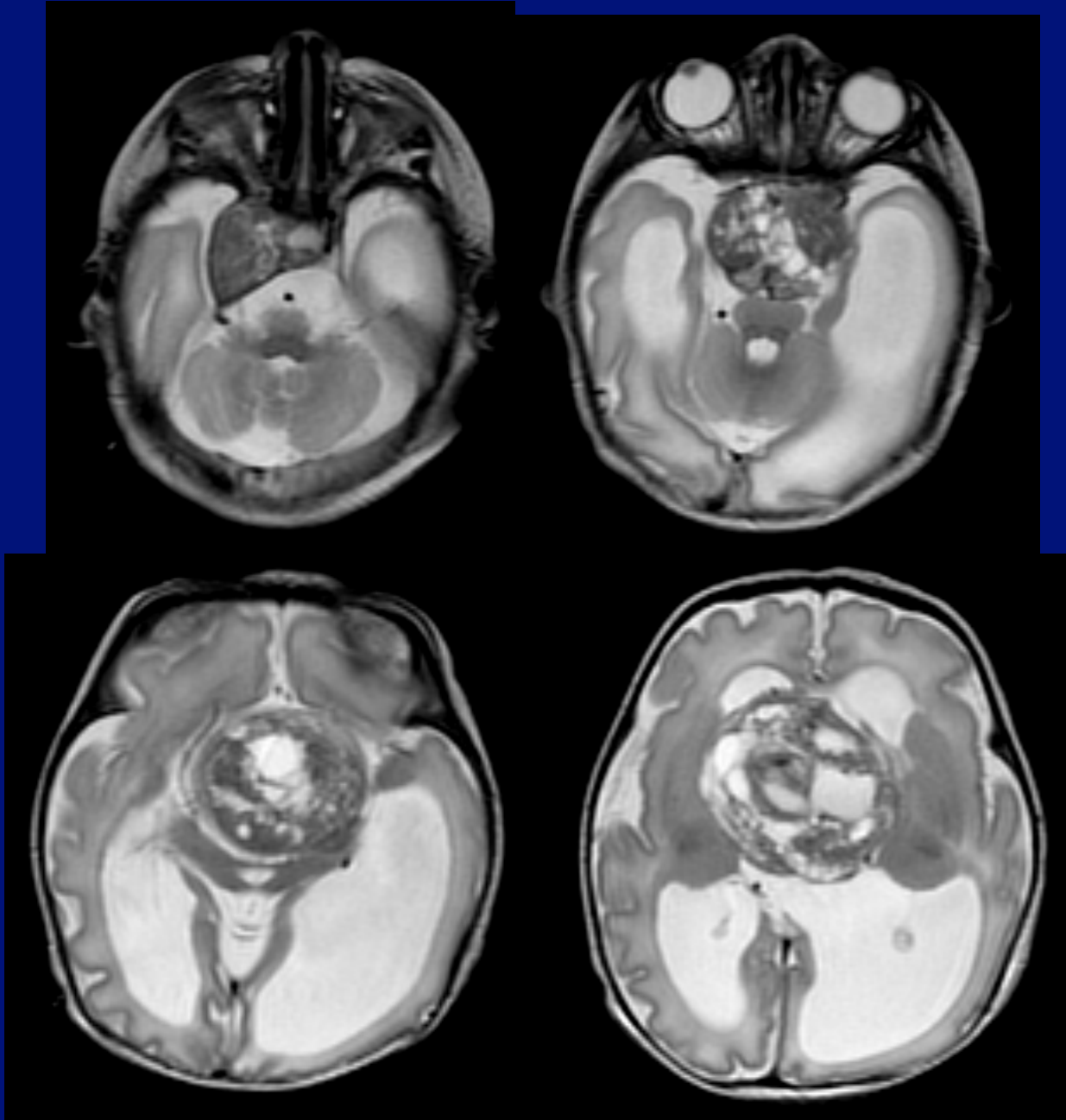
	PERCENT OF NONTERATOMA TUMORS
NEUROEPITHELIAL	
Medulloblastoma	18
Astrocytoma	15
Choroid plexus papilloma (and carcinoma)	13
Ependymoma, ependymoblastoma	11
Miscellaneous neuroepithelial	24
OTHER (MESENCHYMAL)	
Craniopharyngioma	7
Miscellaneous	12

Data from Wakai S, Araj T, Magai M: Surg Neurol 21:597-609, 1984

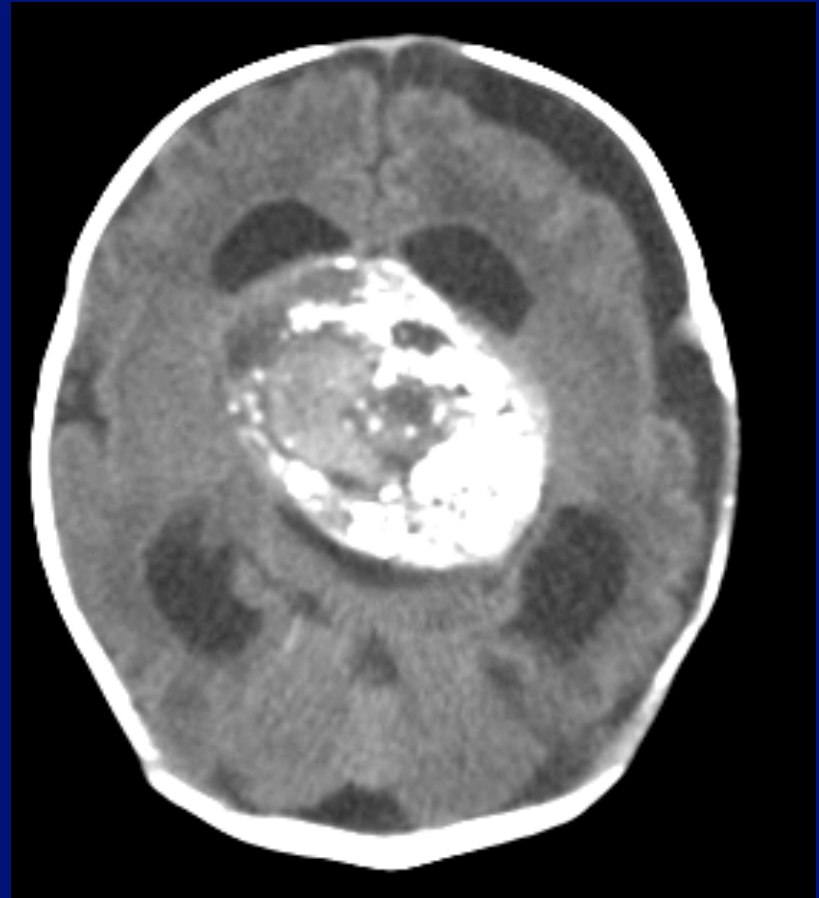
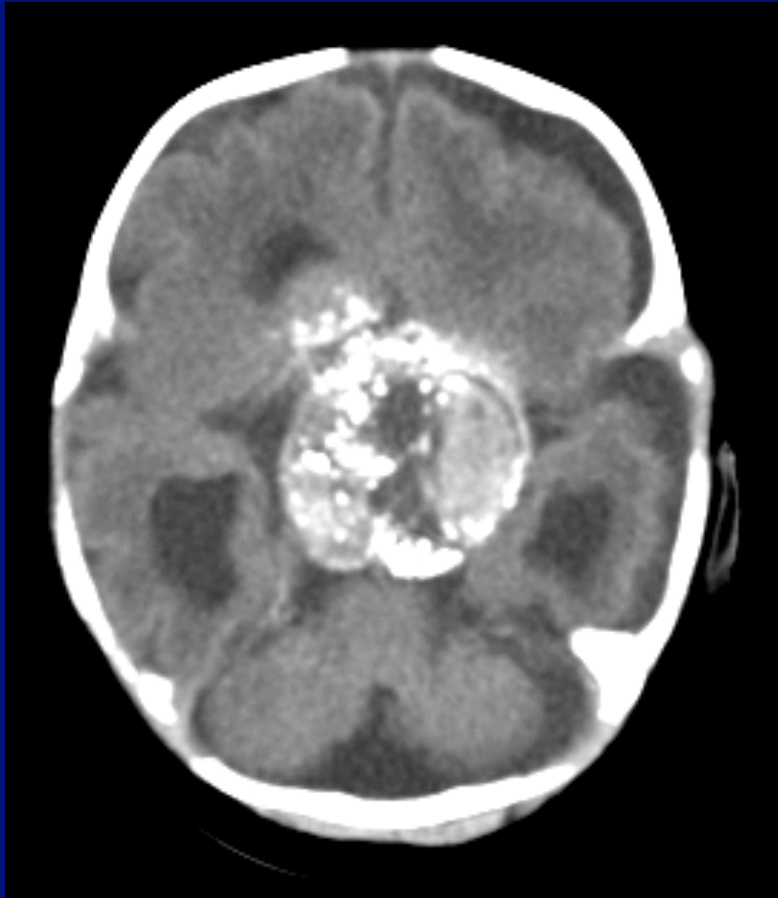
Adapted from Volpe, JJ, *Neurology of the Newborn*, 4th Ed., 2001

Suprasellar Mass

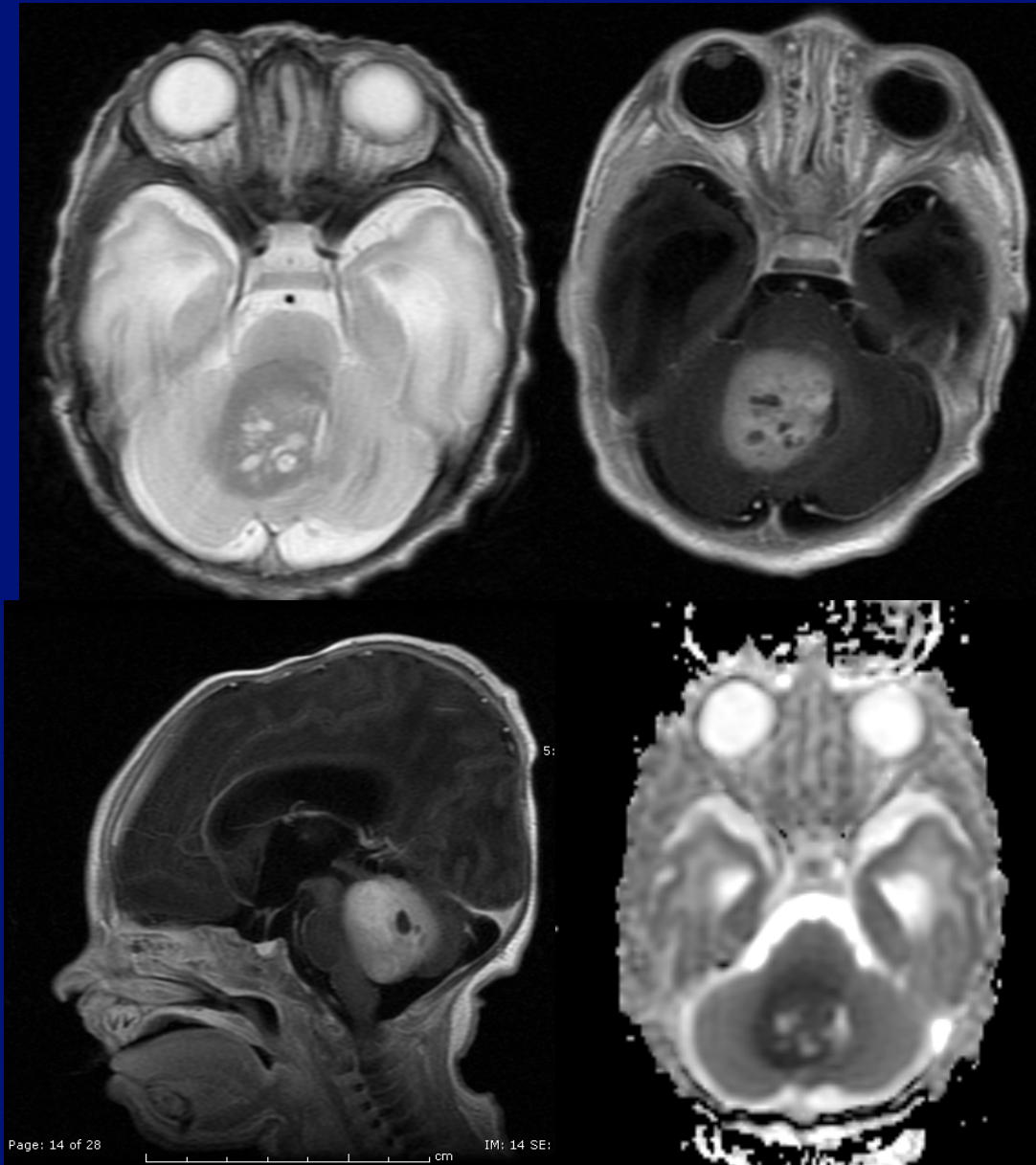




Congenital Craniopharyngioma (Differential: Teratoma)



Posterior Fossa Tumor: ADC Map



Posterior Fossa Tumor: MRS PNET (teratoid features)

