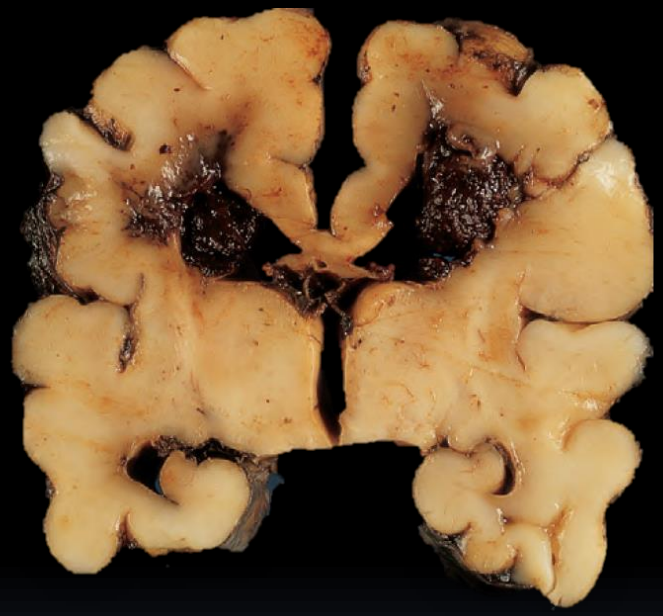
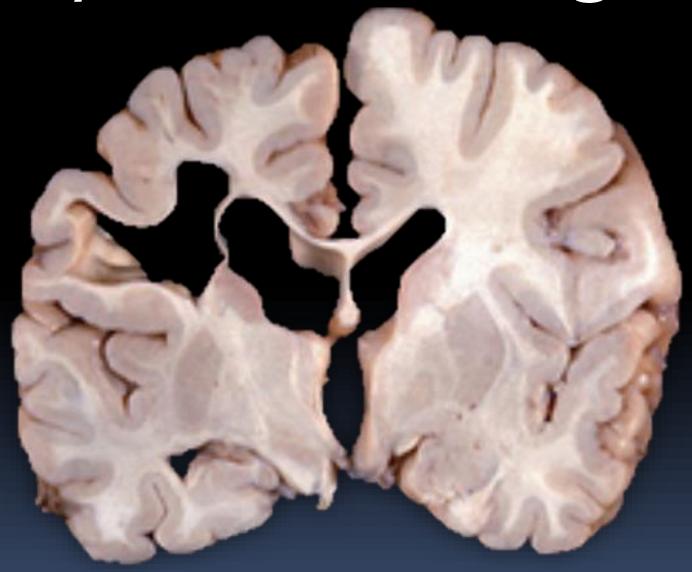
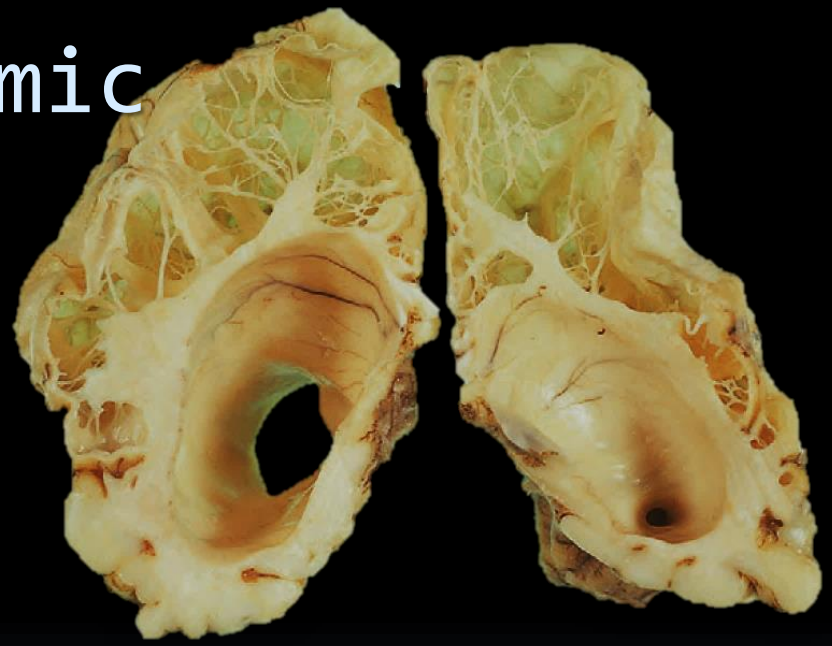
Panda SA
The Paediatric Neurology and Development Association of Southern Africa

Acquired Fetal Brain Lesions- Hemorrhagic/Ischemic







Tally Lerman-Sagie

Fetal Neurology ClinicCenter, Wolfson Medical Center, Holon, Israel

Perinatal Stroke

- Perinatal stroke is defined as a focal vascular brain injury that occurs from the **fetal period to 28 days of postnatal age**
- The overall incidence is 1 in 1,000 live births
- The most focused lifetime risk for stroke occurs near birth
- Modern MRI techniques have high accuracy in diagnosing the different types

Perinatal stroke: mechanisms, management, and outcomes of early cerebrovascular brain injury

Dunbar, et al.
Lancet Child Adolesc Health, 2018

Origin:

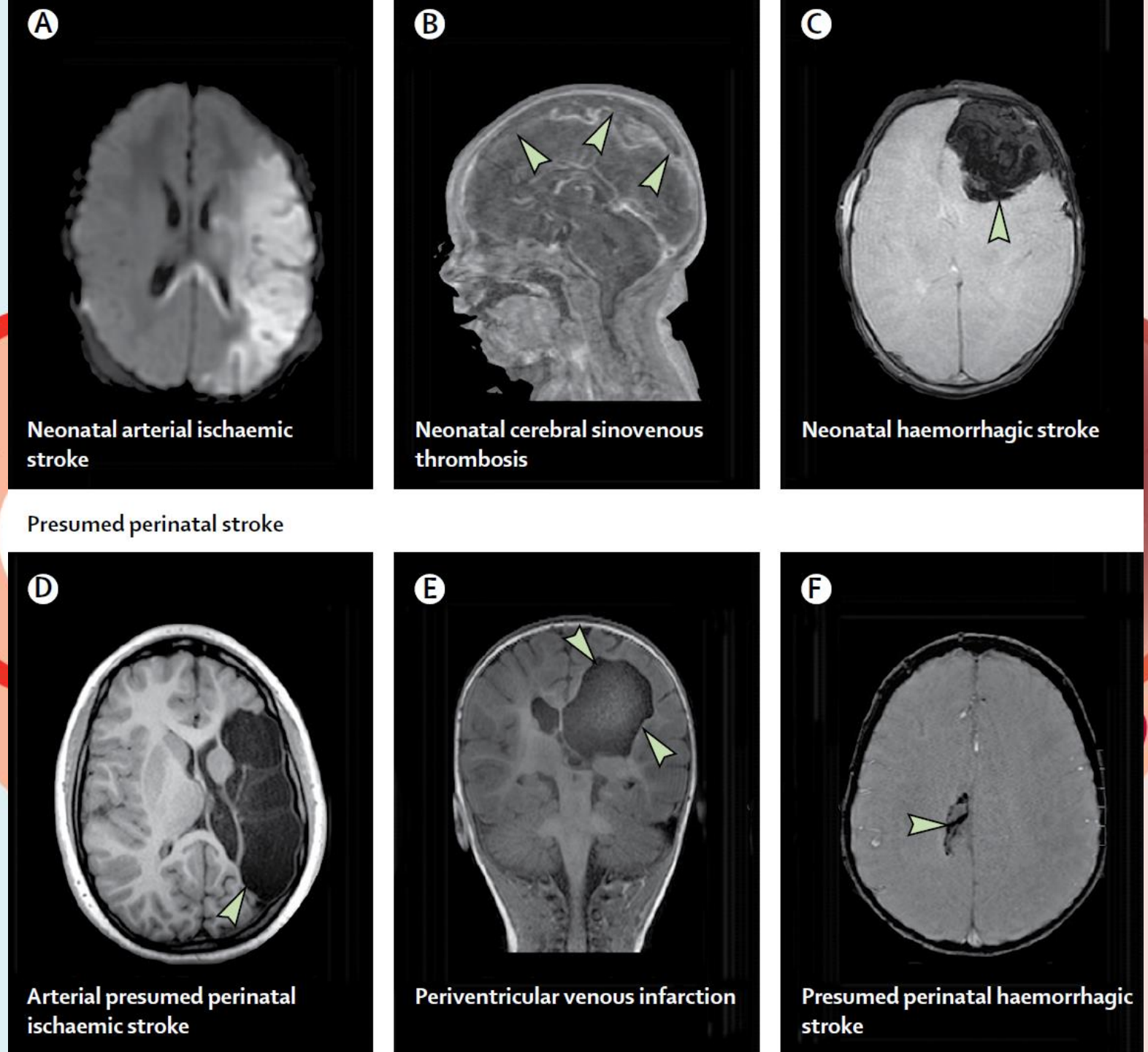
- arterial
- venous

Mechanism:

- ischemic
- hemorrhagic
- both

Presenting symptoms:

- acute
- late (presumed stroke)



DEVELOPMENTAL MEDICINE & CHILD NEUROLOGY ORIGINAL ARTICLE

Risk factors for perinatal arterial ischaemic stroke: a large case–control study

ANNA-LISA SORG[1] | RÜDIGERVONKRIES[1] | MATHIAS KLEMME[2] | LUCIA GERSTL[3] | RAPHAEL WEINBERGER[1] | ANDREAS BEYERLEIN[4] | NICHOLAS LACK[5] | URSULA FELDERHOFF-MÜSER[6] | MARK DZIETKO[6]

- Pre-eclampsia
- Oligohydramnios
- Intrapartum fever
- Birth asphyxia
- SGA
- Male sex
- Preterm birth (<37wk)
- Chorioamnionitis
  - Oligohydramnios
- Nulliparity
- Caesarean section
- Vaginal-operative delivery
- Low umbilical artery pH (<7.1)
- Low 5-minute-apgar score (<7)
- Multiple pregnancies
- Hypoxia
- Intubation/mask ventilation
- Hypoglycaemia

Presumed Arterial Perinatal Ischemic Stroke

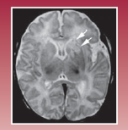
- Same as neonatal arterial stroke, differing only in presentation timing
- Affected newborns lack symptoms
- Diagnosed in infancy or early childhood, with asymmetric motor development, focal epilepsy or other focal neurologic deficits
- Represent up to 50% of all perinatal arterial ischemic strokes
- Chronic arterial infarction may appear as cystic encephalomalacia
- Most lesions affect the MCA territory

Periventricular Venous Infarction

- A germinal matrix (GM) hemorrhage leads to compression of medullary veins and secondary venous infarction of the periventricular white matter (analogous to periventricular hemorrhagic infarction in preterm newborns)
- The GM hemorrhage occurs at mid-gestation (unlikely after 32 weeks)
- May disrupt the descending corticospinal tracts leading to hemiparesis
- MRI reveals well-defined white matter lesions and residual hemosiderin depositions

Presumed Perinatal Hemorrhagic Stroke

- No symptoms until after the newborn period
- Presumed to occur during the perinatal period caused by an initial hemorrhage
- Rare diagnosis
- MRI reveals parenchymal lesions and residual hemosiderin depositions



Pediatric Neuroimaging

FIFTH EDITION

CHAPTER 4

Brain and Spine Injuries in Infancy and Childhood

ERIN SIMON SCHWARTZ AND A. JAMES BARKOVICH

"…all of the types of injury described in prematurely born infants can develop in fetuses of the same postconceptional age."

TABLE 4-4 Brain Injury in Premature Neonates

Type of Injury	Location	Pathology
Germinal matrix hemorrhage	Walls of lateral ventricles (may extend into ventricle and cause hydrocephalus) Cerebellar cortex	Hemorrhage secondary to rupture of thin walled capillaries in germinal matrix
White matter injury	Deep cerebral white matter (may be multifocal or diffuse)	Focal/multifocal necrosis, which forms astroglial scar. May cavitate.
Venous infarction	Cerebral basal ganglia, deep and periventricular white matter	Infarction often hemorrhages. May liquefy, leading to porencephaly
Cerebellar atrophy	Cerebellar vermis and hemispheres	Unknown

Peri- and intraventricular hemorrhage

Periventricular leukomalacia – PVL

PVHI

Cerebellar infarction/hemorrhage

unilateral cerebellar hypoplasia

CHAPTER

4

Brain and Spine Injuries in Infancy and Childhood

ERIN SIMON SCHWARTZ AND A. JAMES BARKOVICH

Brain insults differ radiologically and pathologically depending upon:

- Cause
- Maturity of the brain
- Severity of the insult

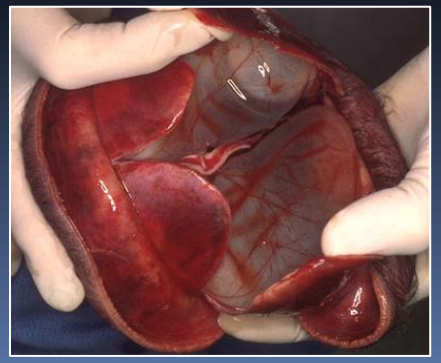
- **The mature brain** reacts to injury by significant astrocytic proliferation, resulting in a soft brain lesion **(encephalomalacia)** consisting of astroglial cells and an irregular surrounding wall composed primarily of reactive astrocytes.

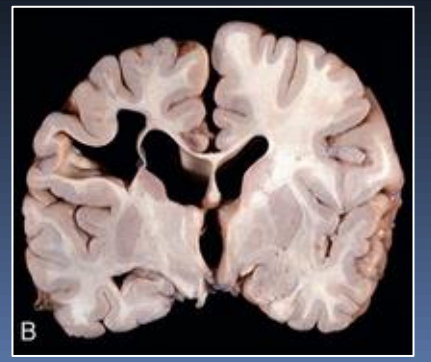
The neonatal and infant brains fall somewhere in between

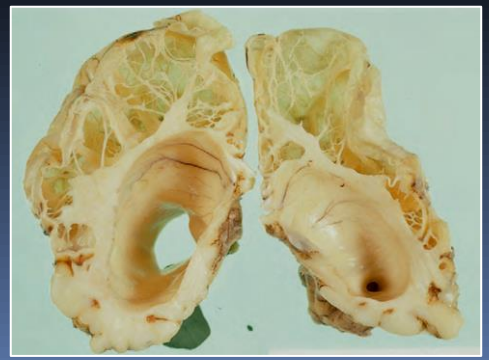
- **The fetal brain** has limited capacity for astrocytic reaction → necrotic tissue is completely reabsorbed **(liquefaction necrosis)** resulting is a smooth-walled, fluid-filled cavity **(porencephalic cyst)**.

Specific end-stage patterns of tissue reaction to severe / global injury:

- Hydranencephaly
- Porencephaly
- Multicystic encephalomalacia







Germinal matrix and intraventricular hemorrhages (GMH-IVH)

Severity of germinal matrix and intraventricular hemorrhage on cranial ultrasonography

Grade	Description on parasagittal view
I	Germinal matrix hemorrhage (GMH) only or germinal matrix hemorrhage plus intraventricular hemorrhage less than 10% of ventricular area
II	GMH and intraventricular hemorrhage; 10 to 50% of ventricular area
III	GMH and intraventricular hemorrhage involving more than 50% of ventricular area; lateral ventricles are usually distended
IV	Hemorrhagic infarction in periventricular white matter ipsilateral to intraventricular hemorrhage (also called periventricular hemorrhagic infarction)

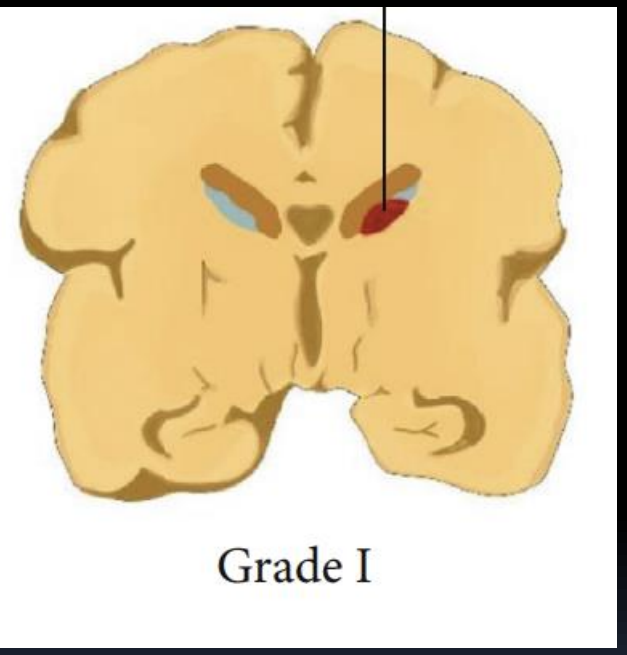
GMH pathogenesis:

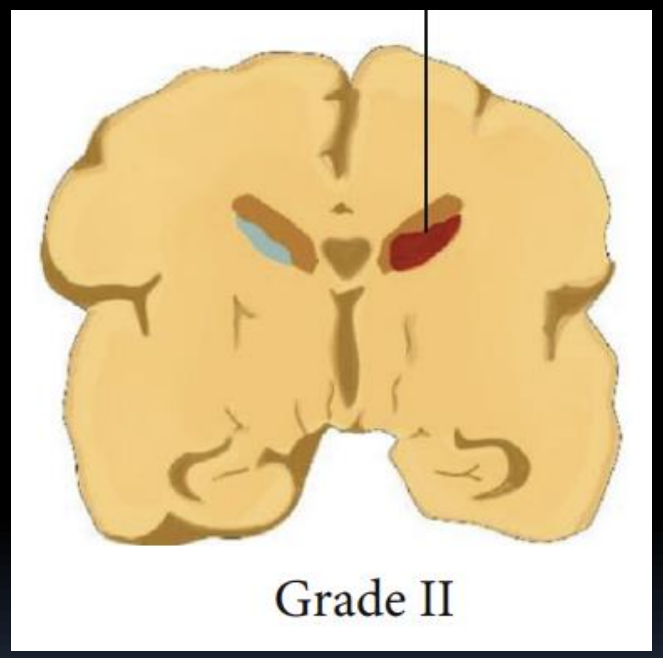
- Intrinsic fragility of the GM vasculature
- Disturbance in the cerebral blood flow

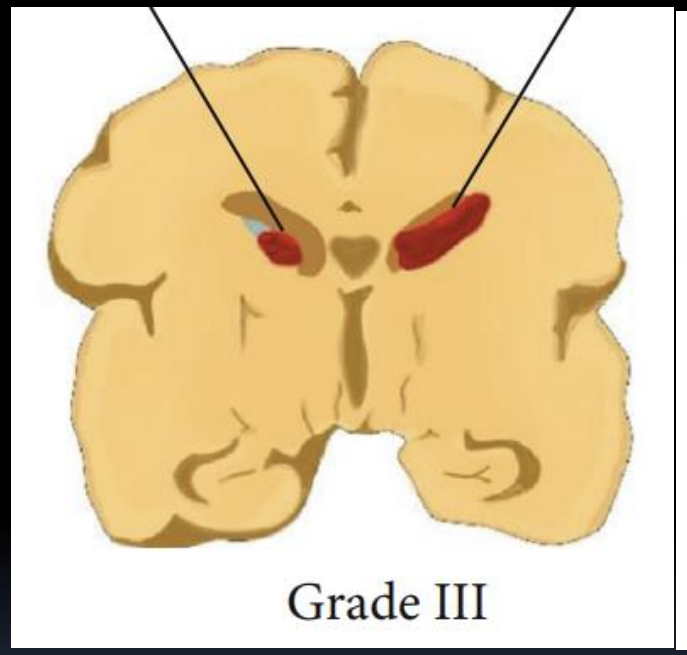
Linda de Vries, 2019, UpToDate

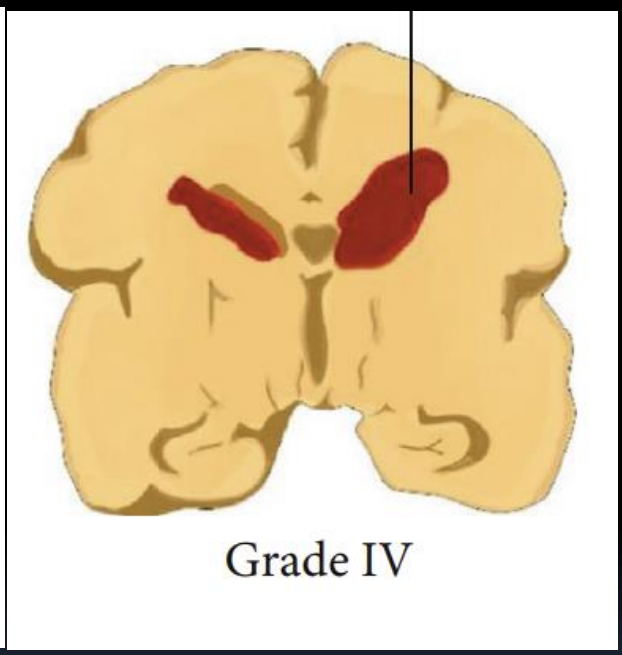
IVH Grading

Papile´s Grading (1978)



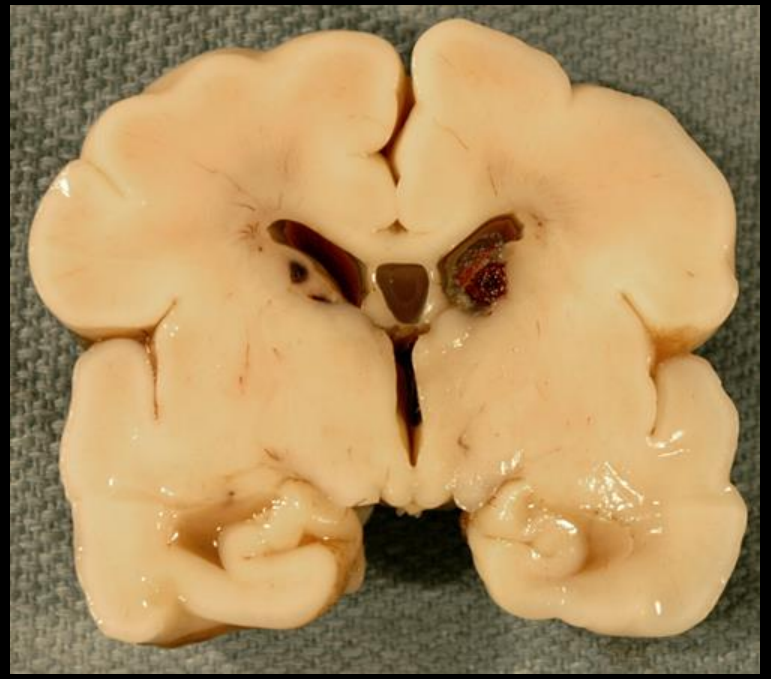




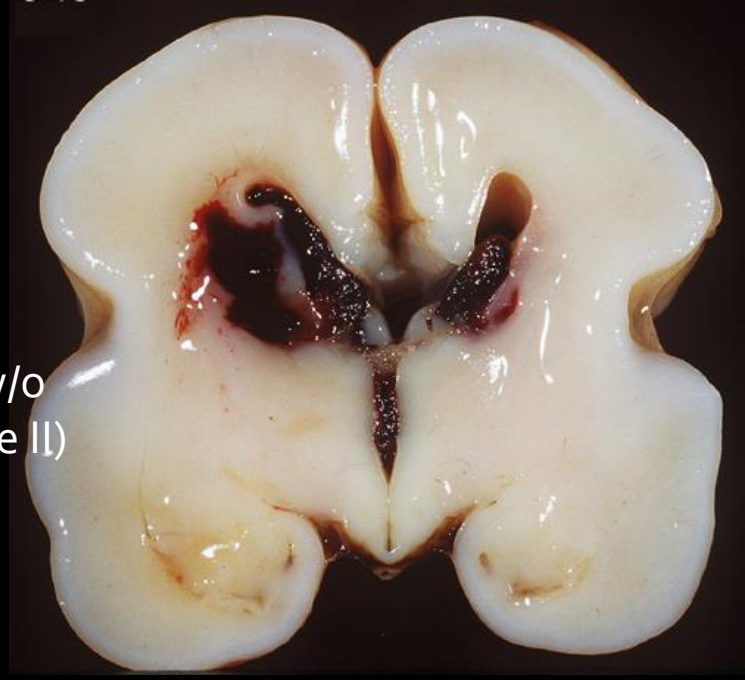


Egesa WI et al. International Journal of Pediatrics Volume 2021

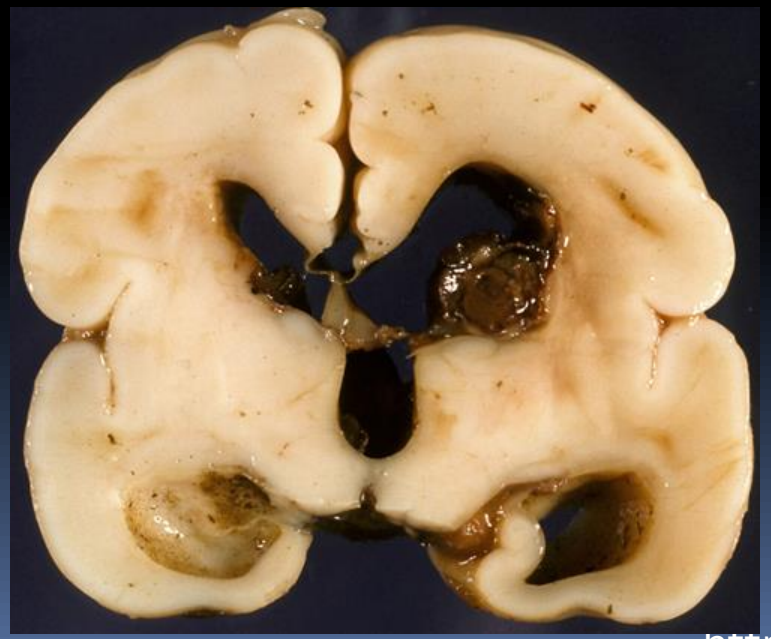
Bilateral small GMH (Grade I)



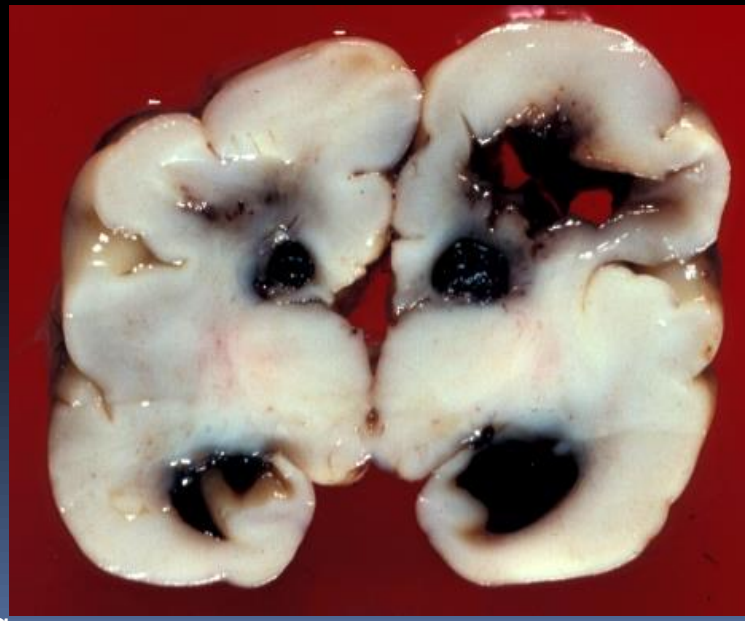
Bilateral GMH-IVH with intraventricular bleeding, w/o ventricular dilatation (Grade II)



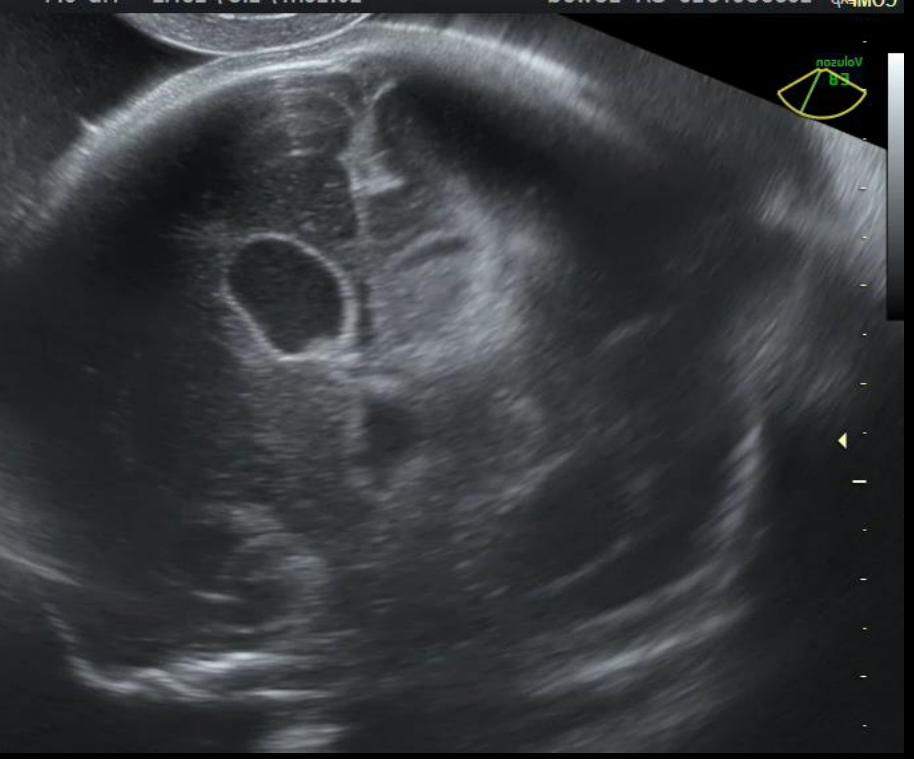
Grade III GMH-IVH lateral ventricles dilated

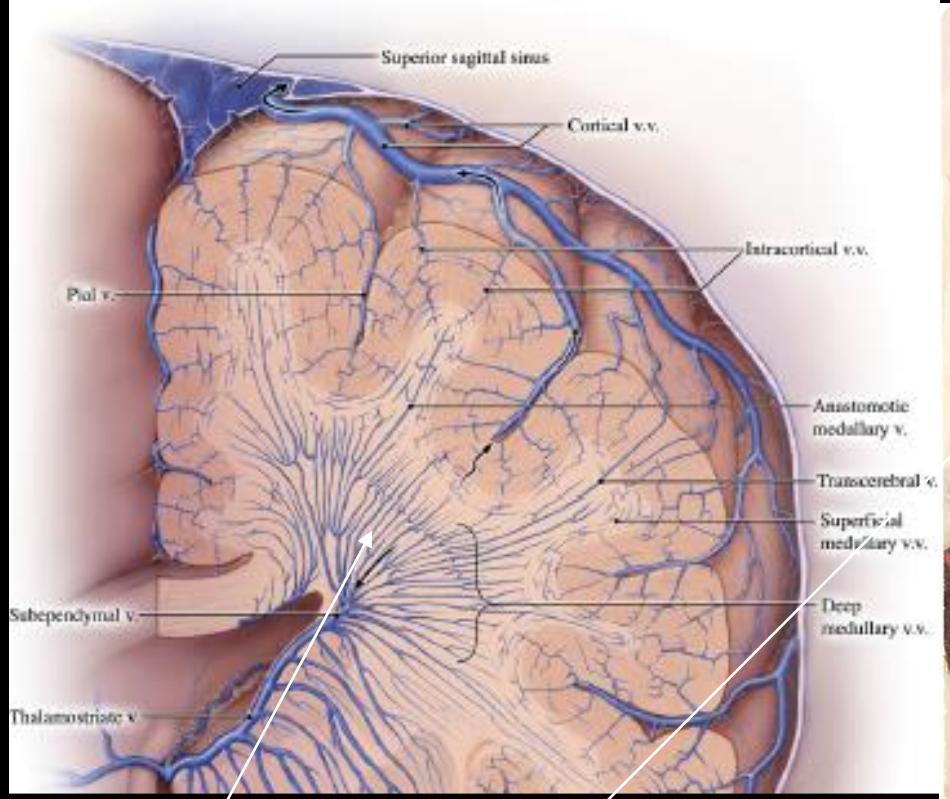


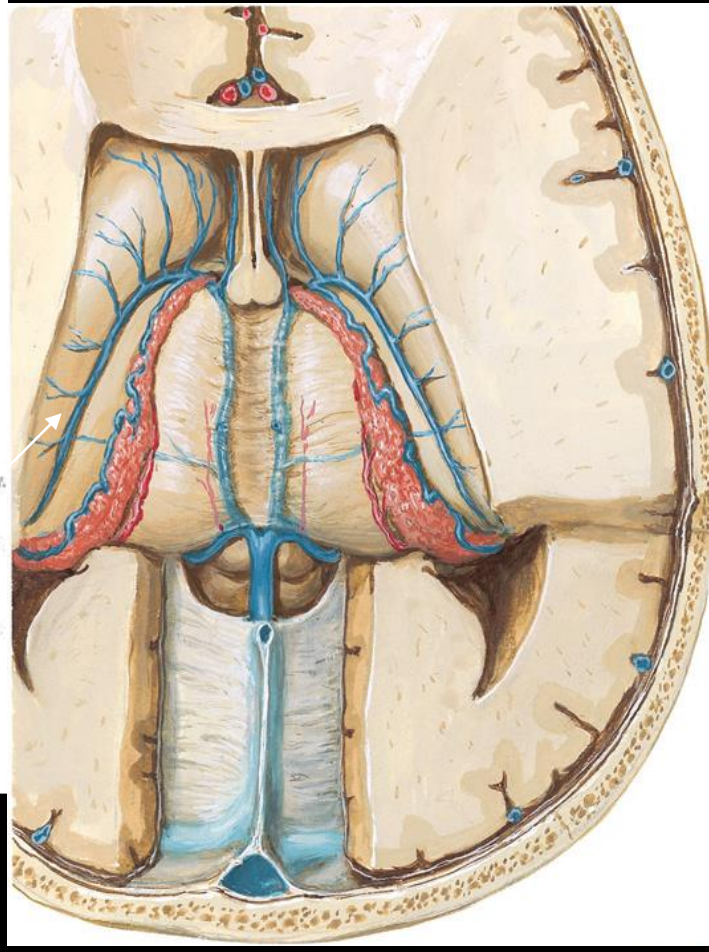
Grade IV GMH-IVH bilateral GM hemorrhage intraventricular rupture extension into PV white matter



https://neuropathology-web.org







Pressure from intraventricular bleeding

Impaired drainage of medullary and terminal veins

Periventricular venous congestion

Periventricular white matter ischemia

Periventricular venous hemorrhagic infarction

FETAL INTRACRANIAL HEMORRHAGE

THE ROLE OF FETAL IMAGING

The Role of Imaging

Diagnosis of the lesion:

- Focal ischemic damage- cavitations, hyperechoic lesions
- MRI useful for diagnosis of ischemic hemorrhagic lesions
- The use of gradient-echo sequences makes it possible to identify old hemorrhagic lesions, which may be missed by US

The role of imaging

Localization of the lesion and evaluation of its extension:

- Cerebral hemispheres/ cerebellum
- Supratentorial damage-precise localization of the lesion and its extension
- Posterior fossa-determine if the cerebellar hemispheres and/or the vermis are damaged

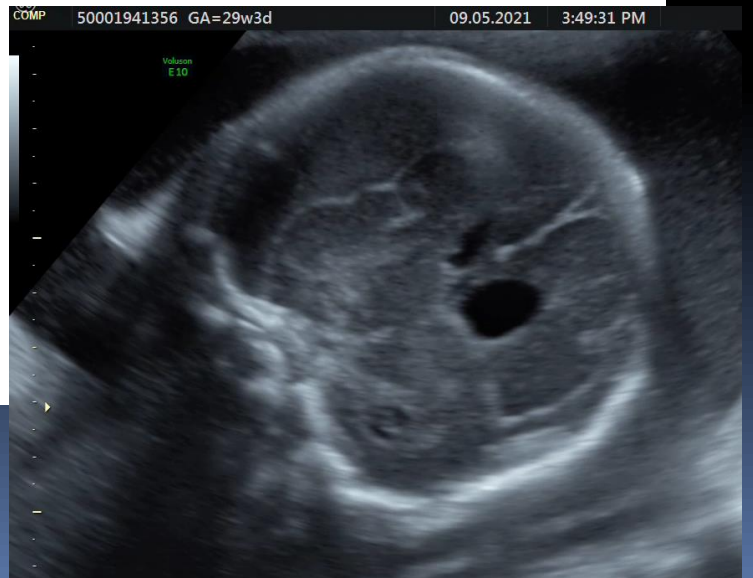
Obstet Gynecol 2020

Intracranial Hemorrhage

in utero

Society for Maternal-Fetal Medicine (SMFM); Ana Monteagudo, MD

- Fetal ICH is rare ~1 in 10,000 pregnancies
- ICH most often is diagnosed late in pregnancy as an incidental finding
- Mean gestational age at diagnosis - 31 weeks
- ICH categories:
  - Intracerebral (Intraventricular, infratentorial)
  - Extracerebral (subdural hematomas)



Causes of Parenchymal Ischemic-Hemorrhagic Insults

- Monochorionic pregnancies
- Maternal trauma
- Maternal history of drug exposure
- Infection (TORCH)
- Platelet alloimmunization
- Thrombophilic disorders
- Underlying inflammatory or metabolic disease
- Genetic- mutations in COL4A1/2

Intracranial Hemorrhage *in utero*

Society for Maternal-Fetal Medicine (SMFM); Ana Monteagudo, MD

Maternal risk factors:

- Trauma
- Seizures
- Hypoxia
- Immune thrombocytopenia (ITP)
- Coagulation disorders
- Infections (CMV, toxoplasmosis)
- Febrile disease
- Medications (warfarin)
- Drugs (cocaine)
- Preeclampsia
- Placental abruption
- Maternal vitamin K deficiency

Fetal risk factors:

- TTTS
- Death of a monochorionic co-twin
- Fetal thrombophilia (factor V Leiden, protein C)
- Thrombosis of the umbilical cord
- Cord entanglement
- Fetal alloimmune thrombocytopenia (FNAIT)

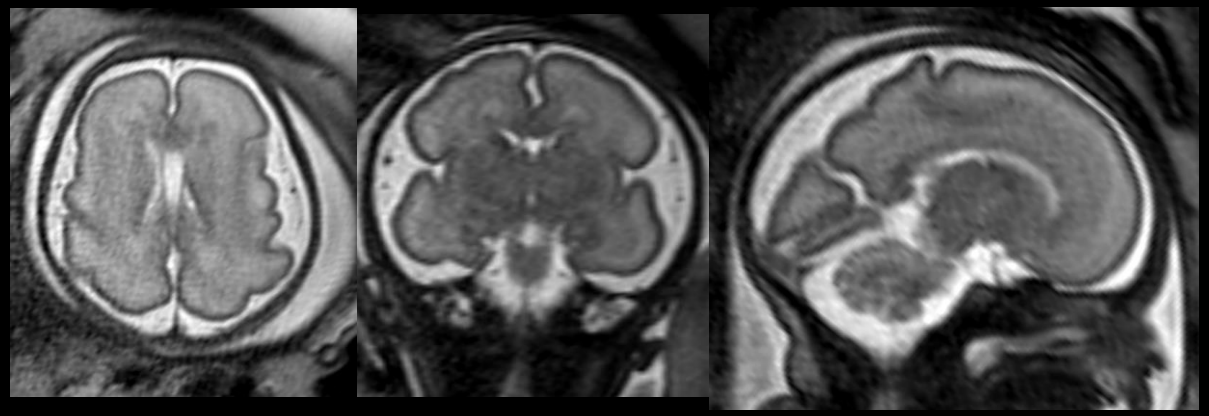
In most cases, the etiology remains unclear

Causes of Parenchymal Ischemic/Hemorrhagic Insults

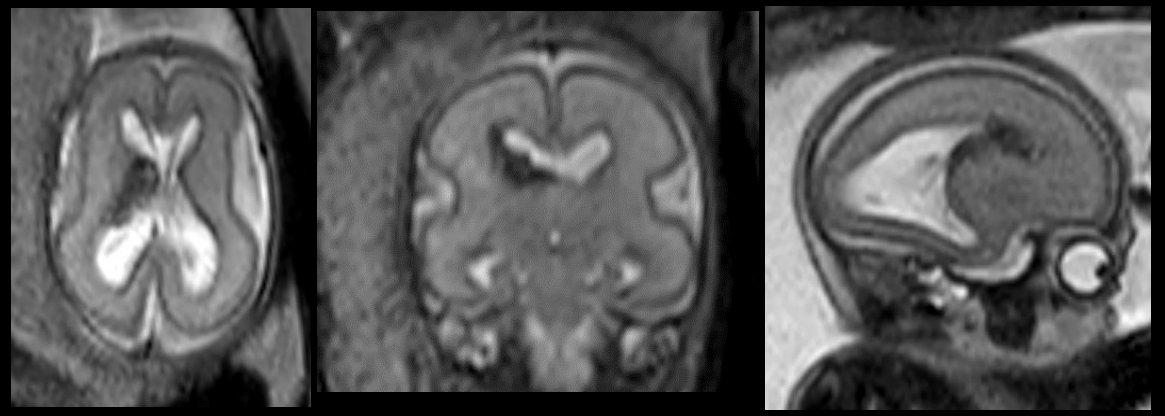
- Monochorionic pregnancies
- Maternal trauma
- Maternal history of drug exposure
- Infection (TORCH)
- Platelet alloimmunization
- Thrombophilic disorders
- Metabolic disease
- Interferonopathy
- Collagenopathy

FETAL INTRAVENTRICULAR HEMORRHAGE

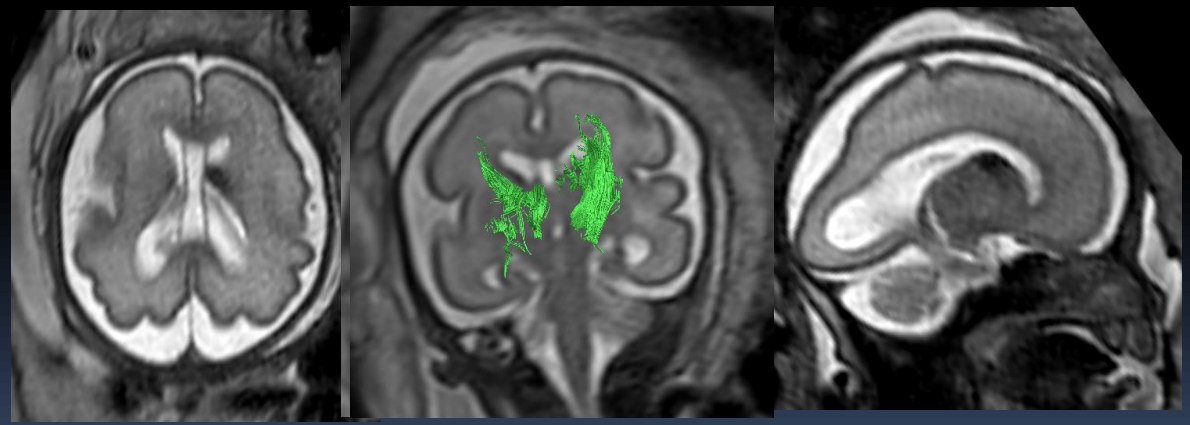
IVH Grading-MRI



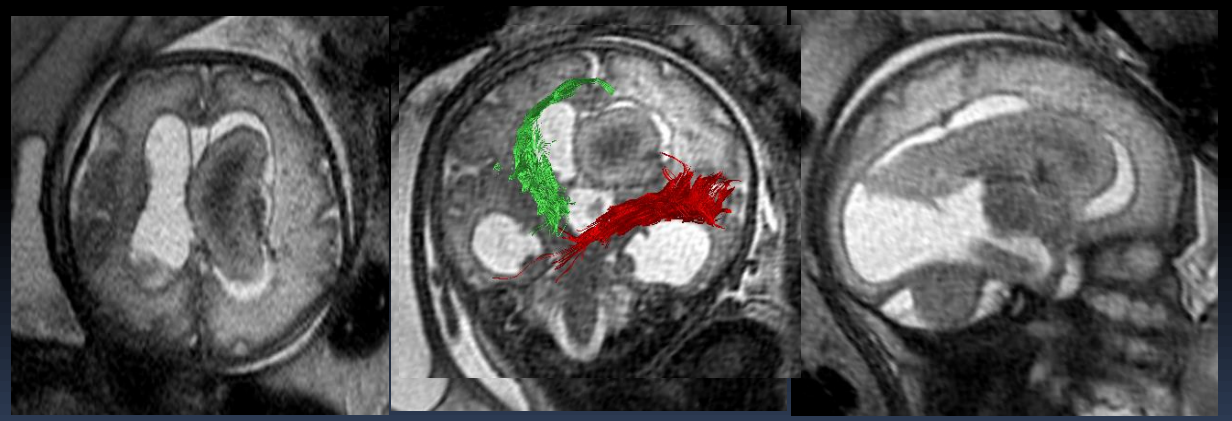
IVH Grade 1



IVH Grade 3

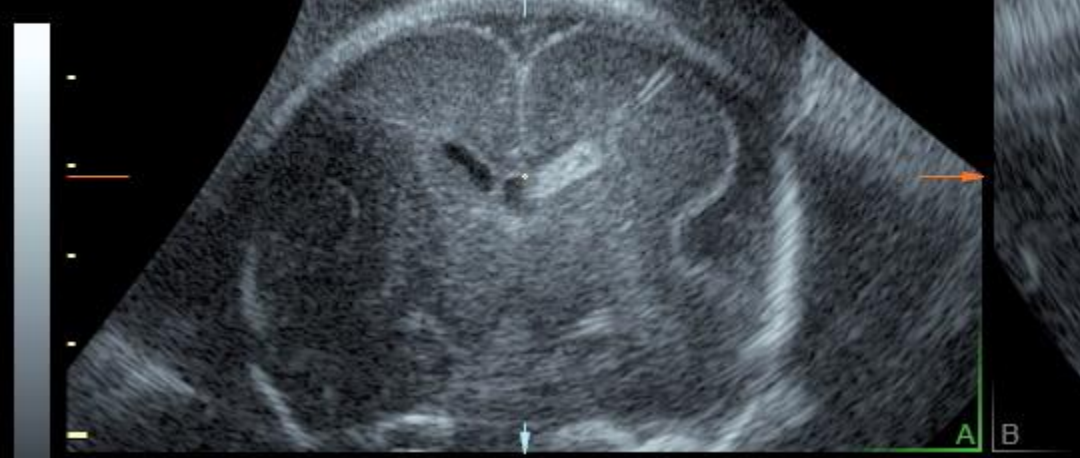


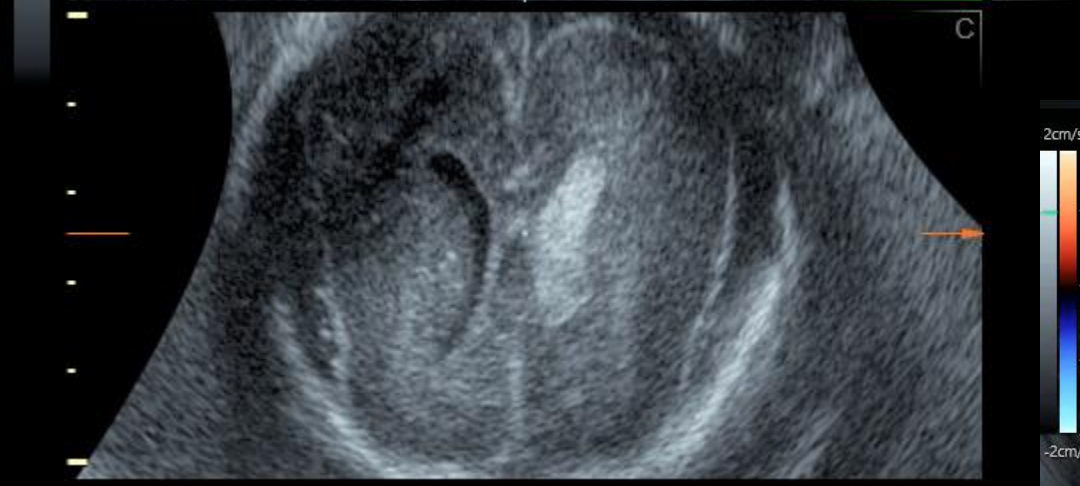
IVH Grade 2



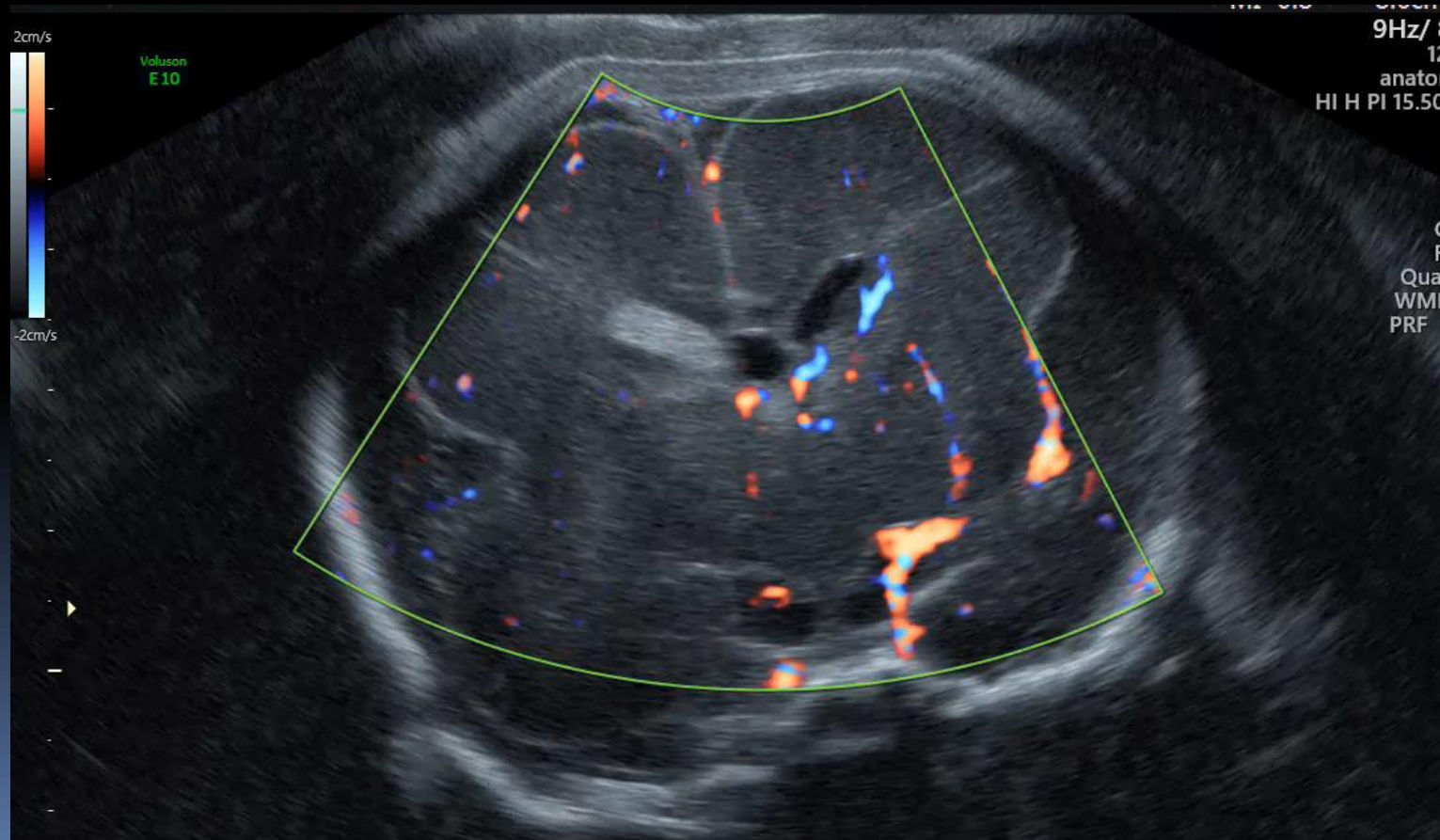
IVH Grade 4

Goeral K, et al. A novel magnetic resonance imaging-based scoring system to predict outcome in neonates born preterm with intraventricular haemorrhage. Developmental Medicine & Child Neurology 2021.

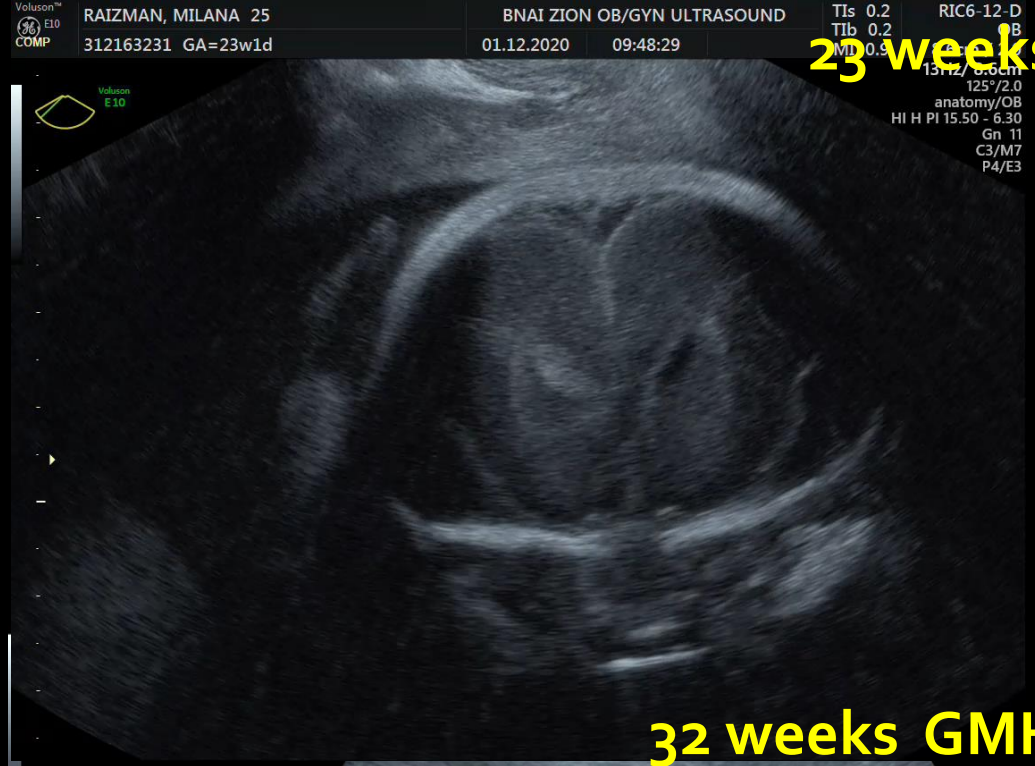


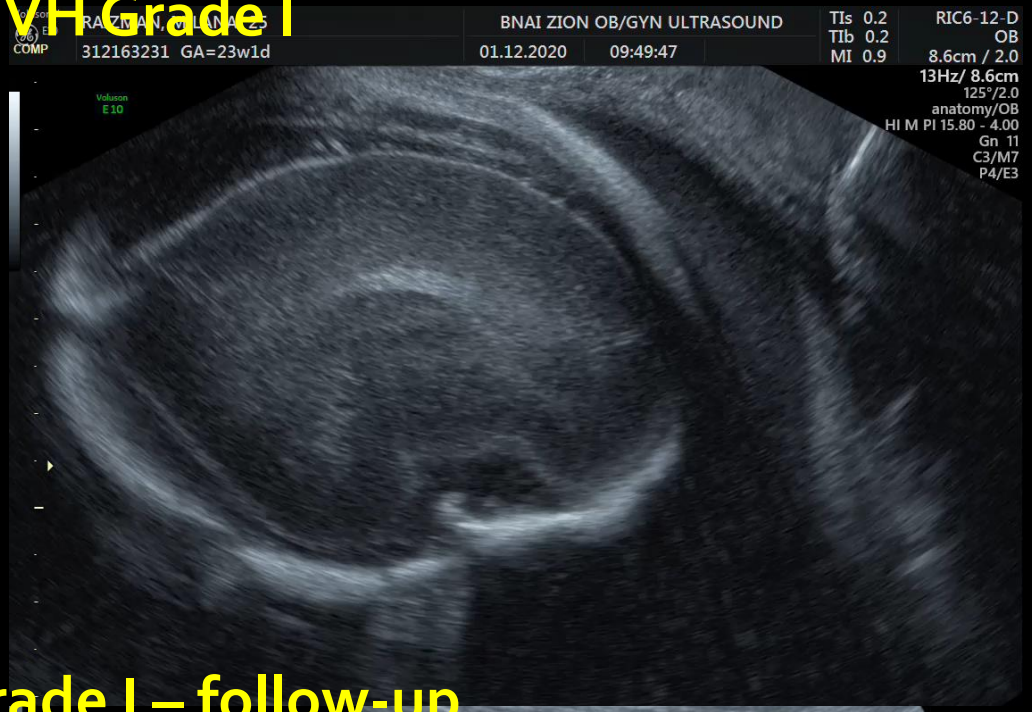


23 weeks GMH-IVH Grade I

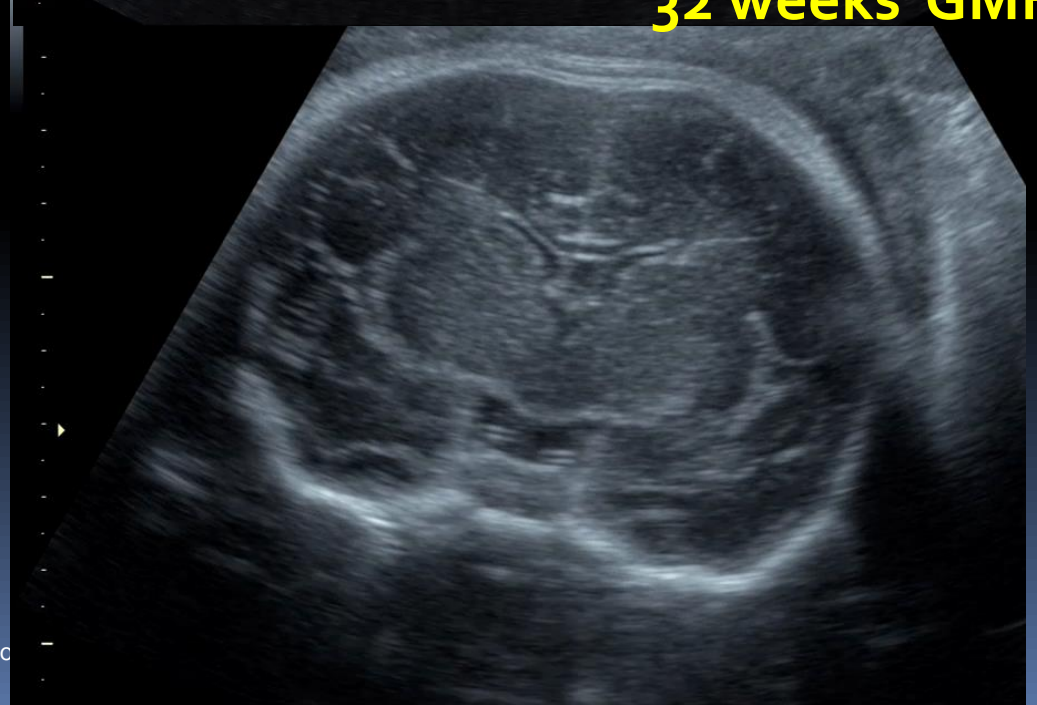


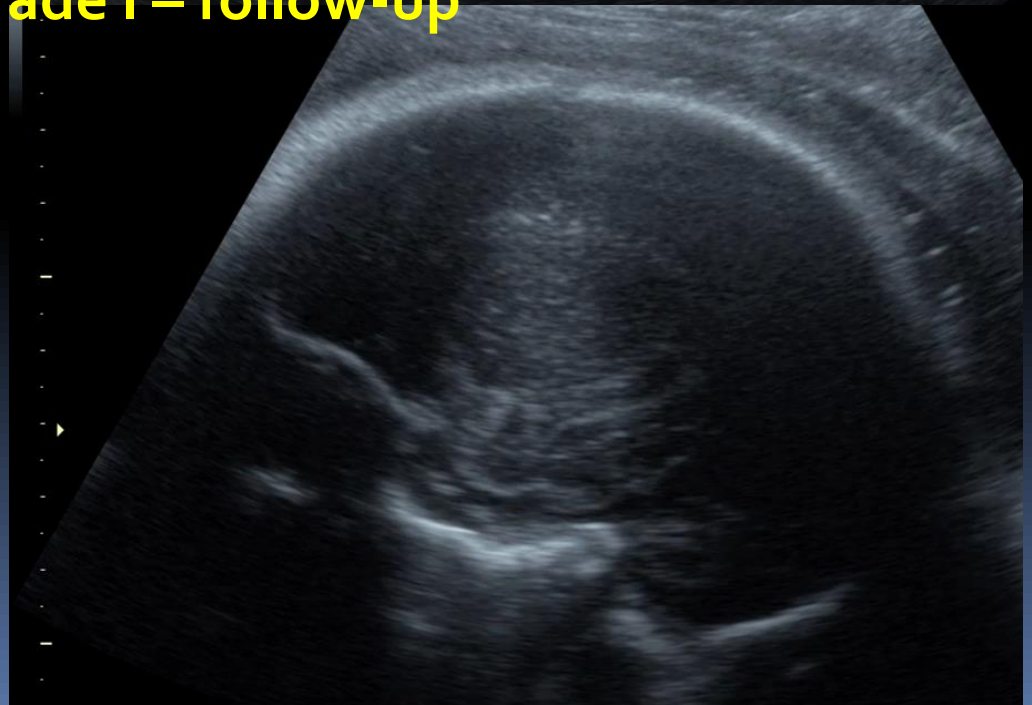
23 weeks GMH-IVH Grade I



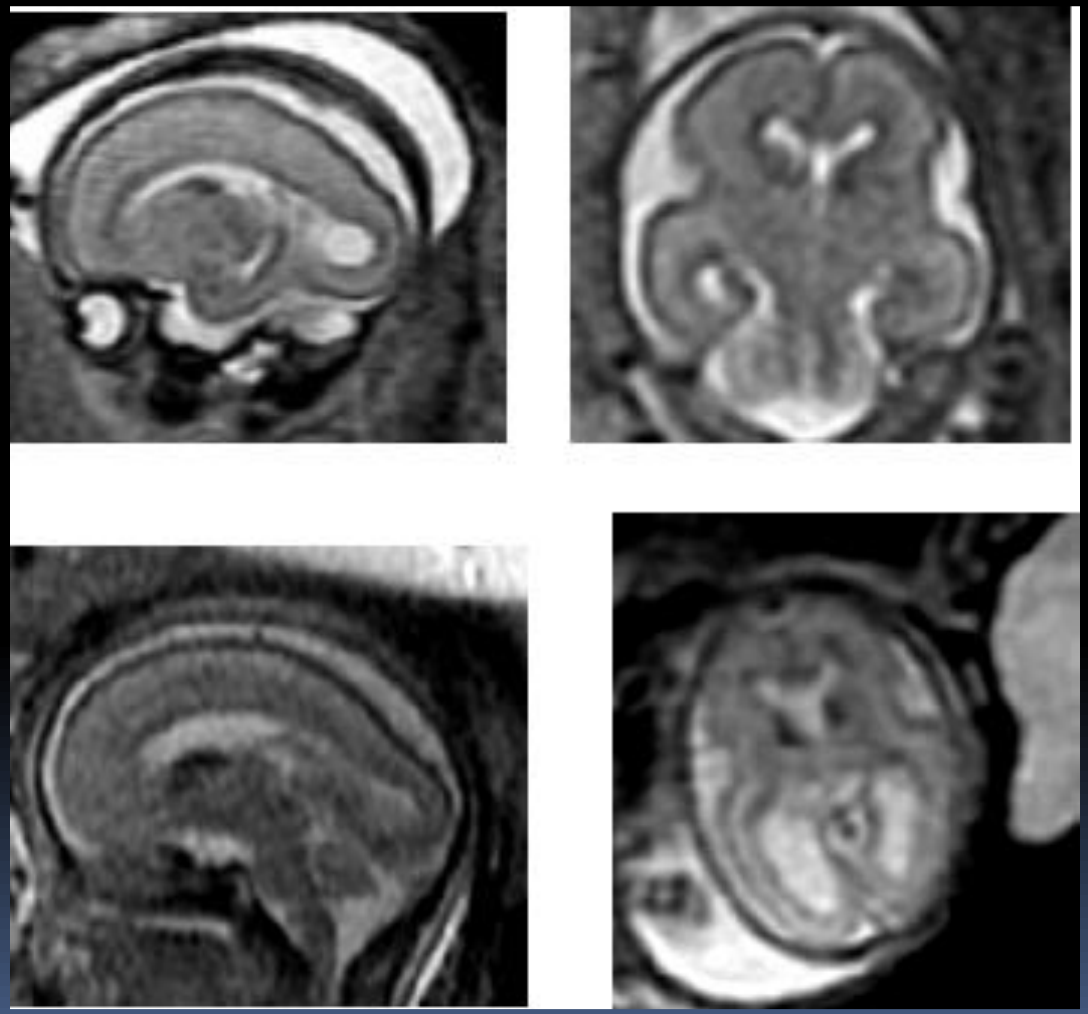


32 weeks GMH-IVH Grade I – follow-up

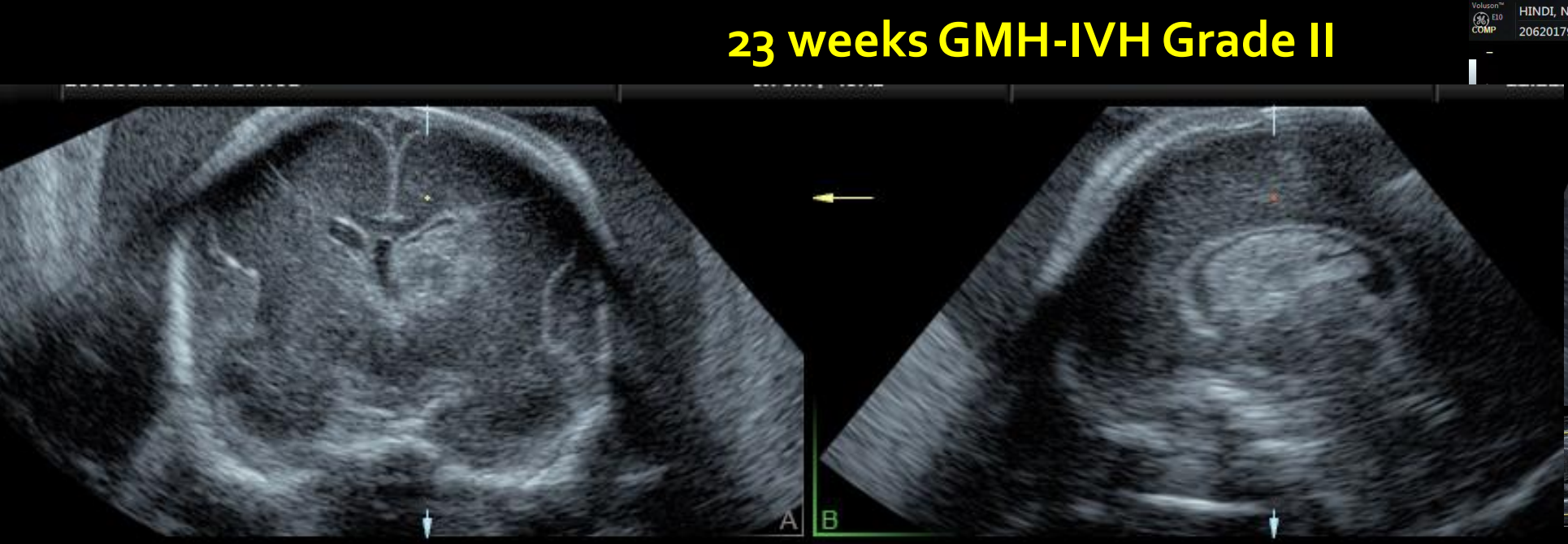


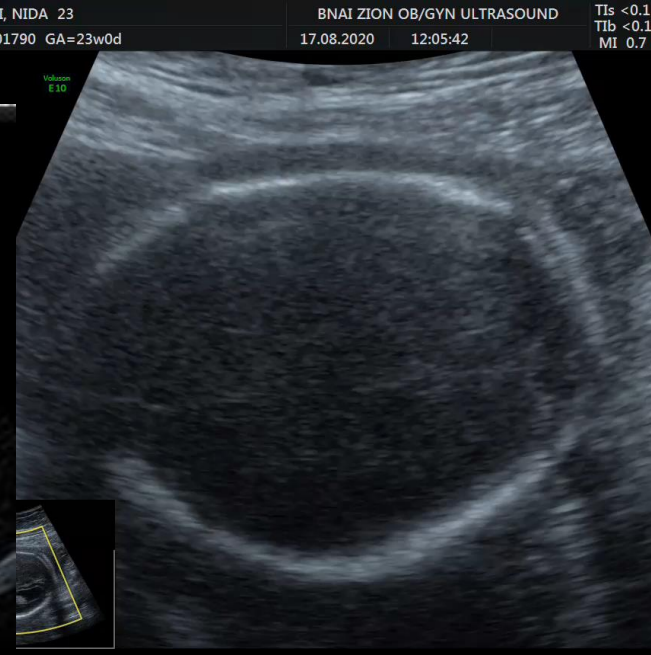


Germinal Matrix Grade 1 Hemorrhage-MRI

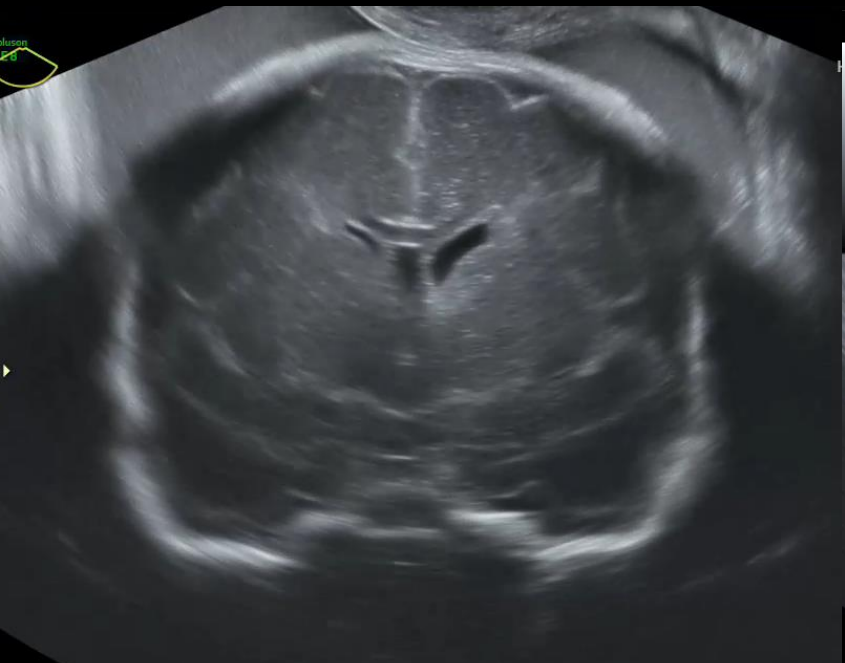


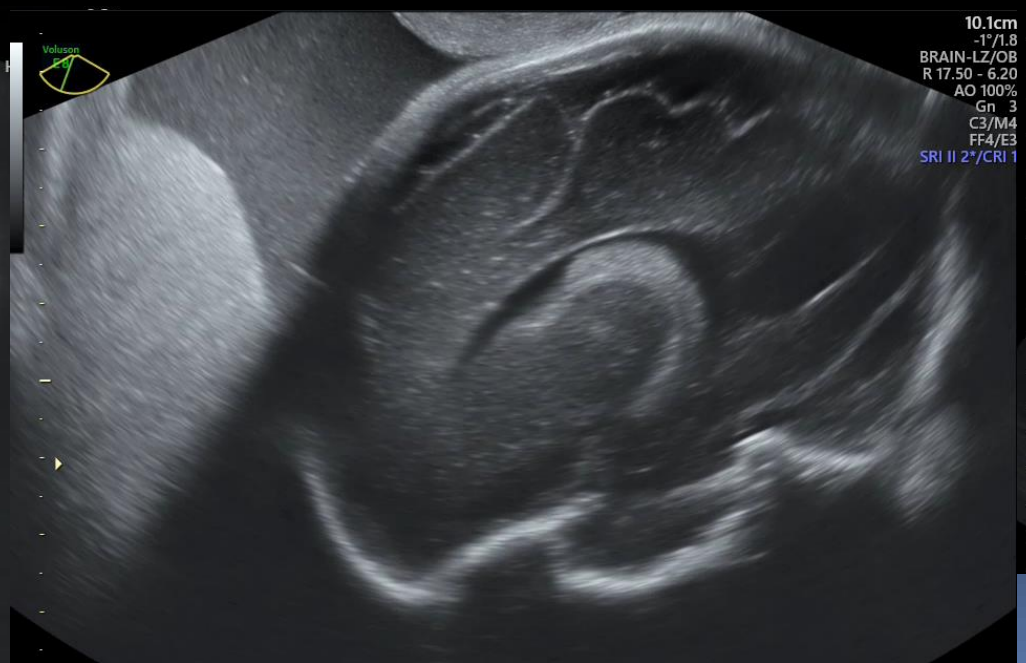
23 weeks GMH-IVH Grade II

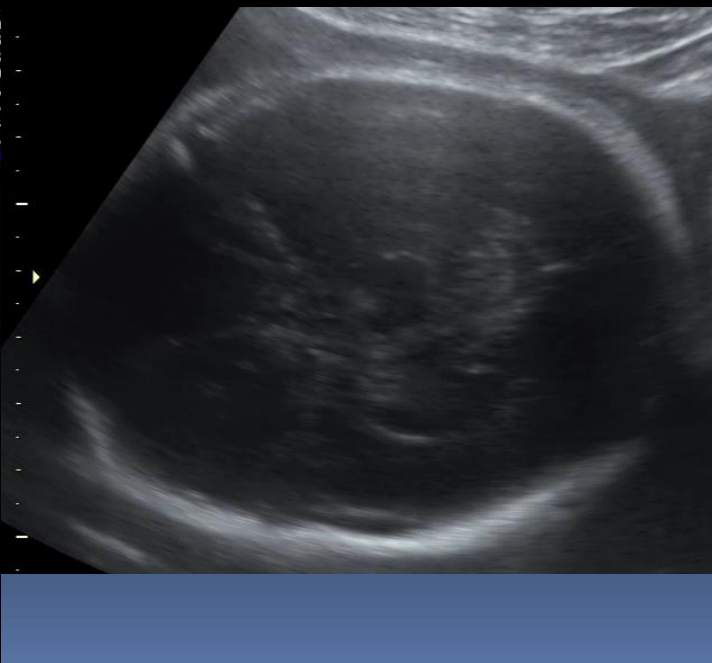


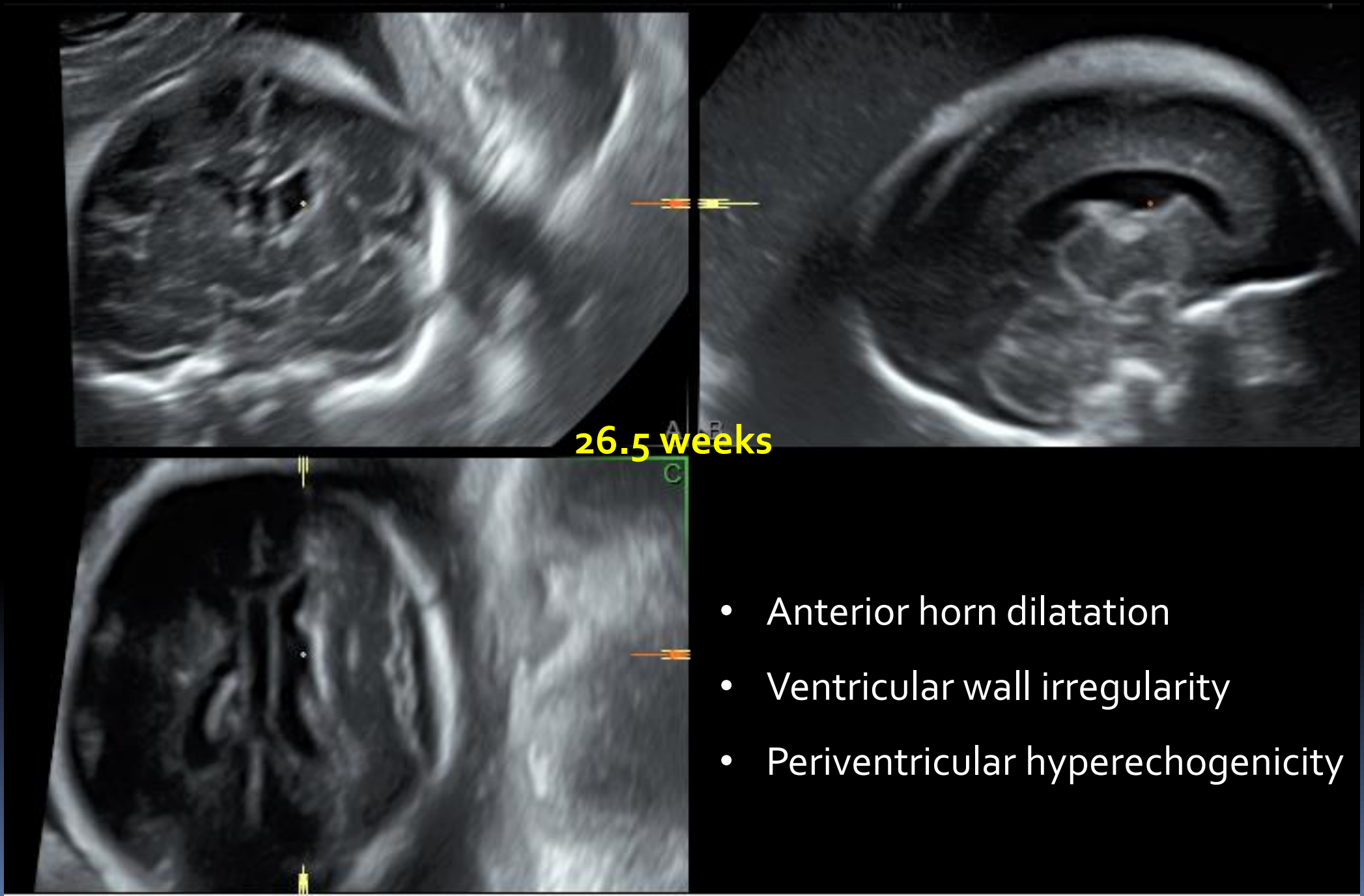


30 weeks follow-up

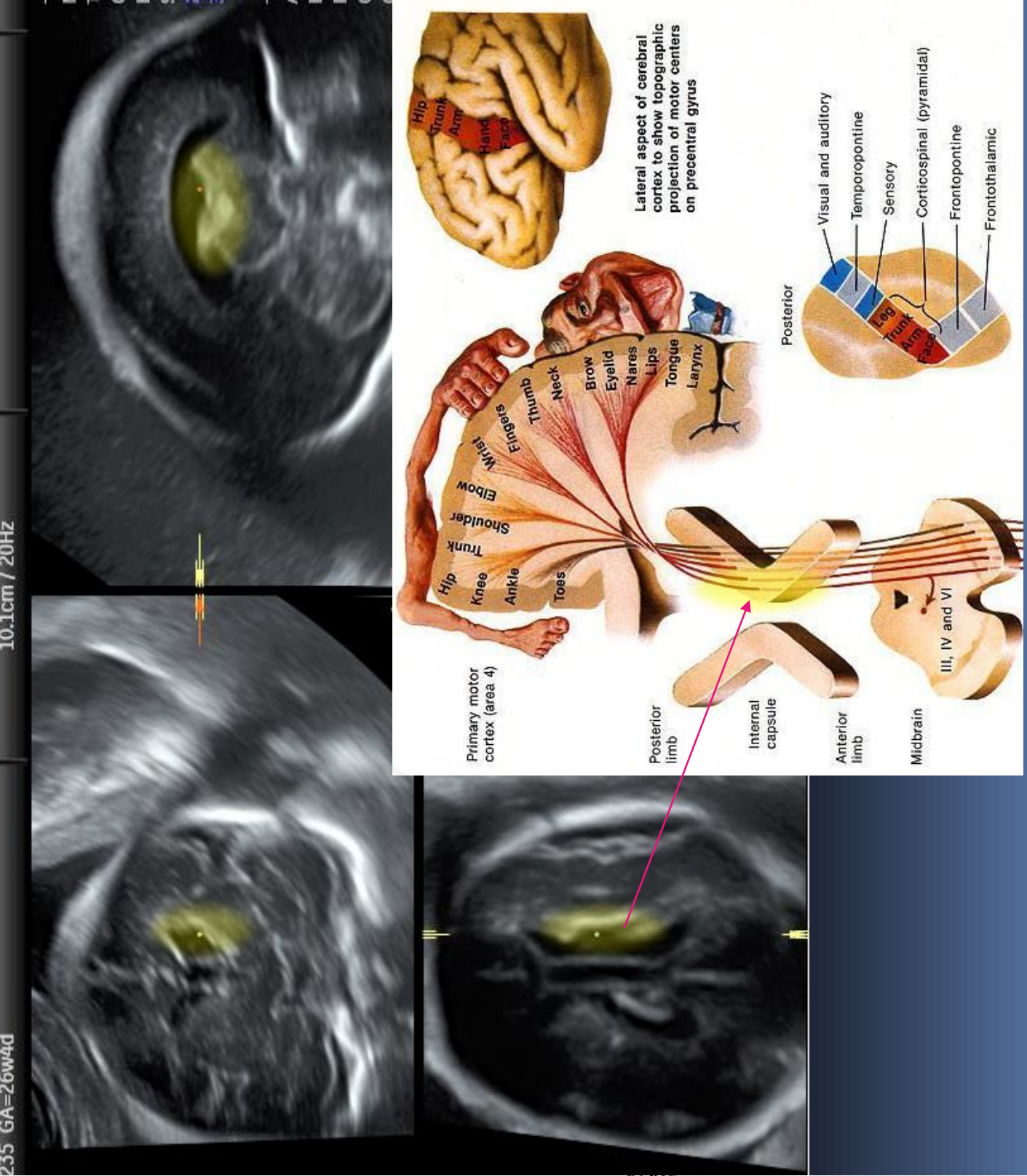


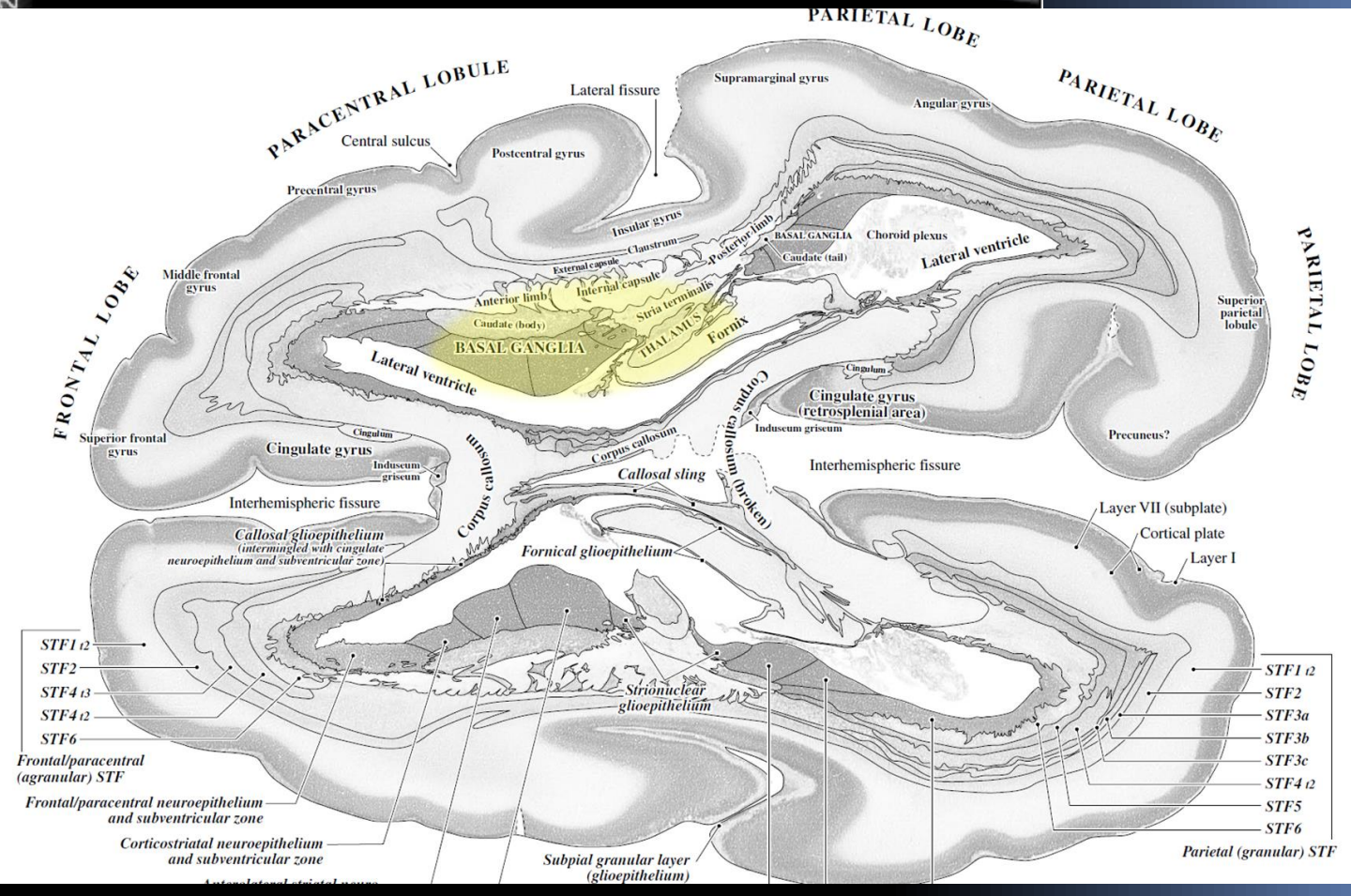


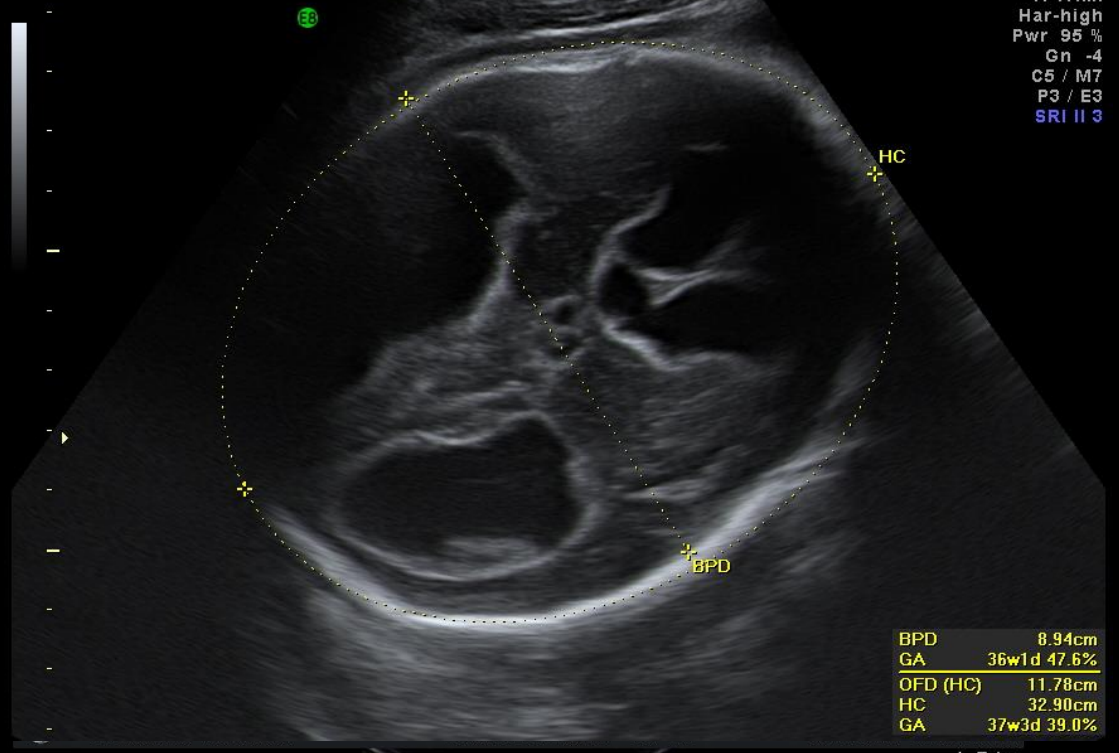


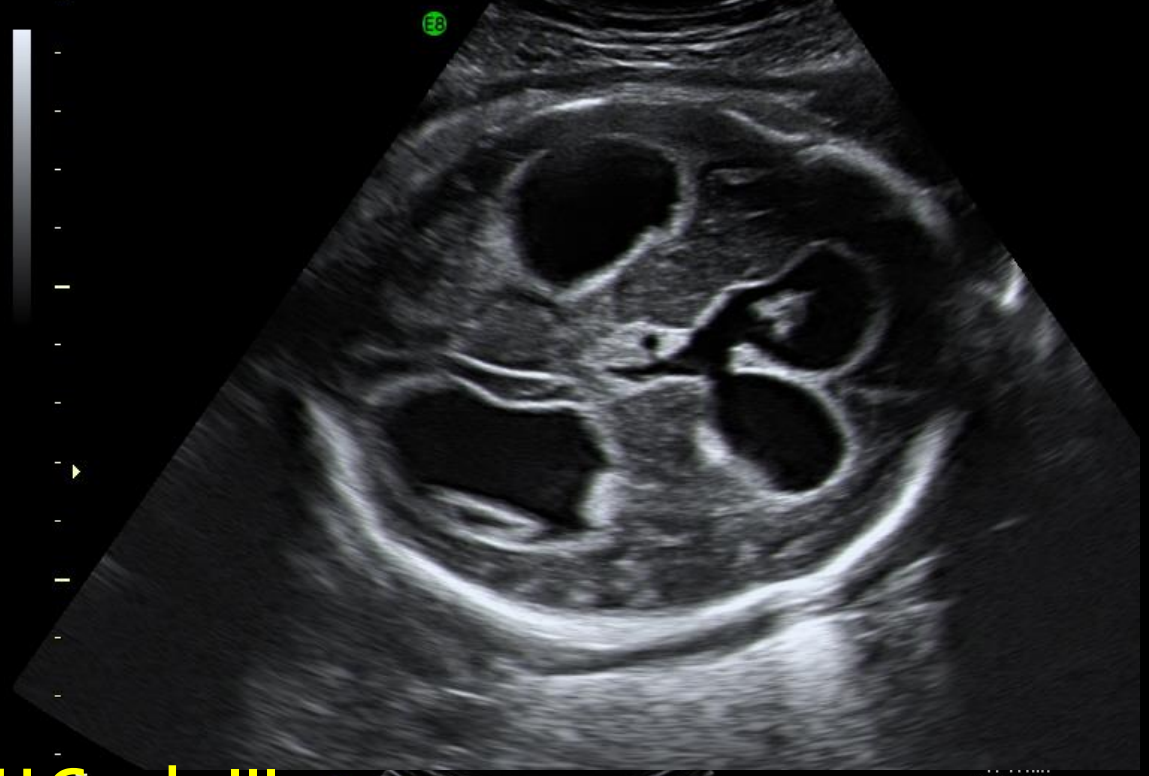


- Anterior horn dilatation
- Ventricular wall irregularity
- Periventricular hyperechogenicity

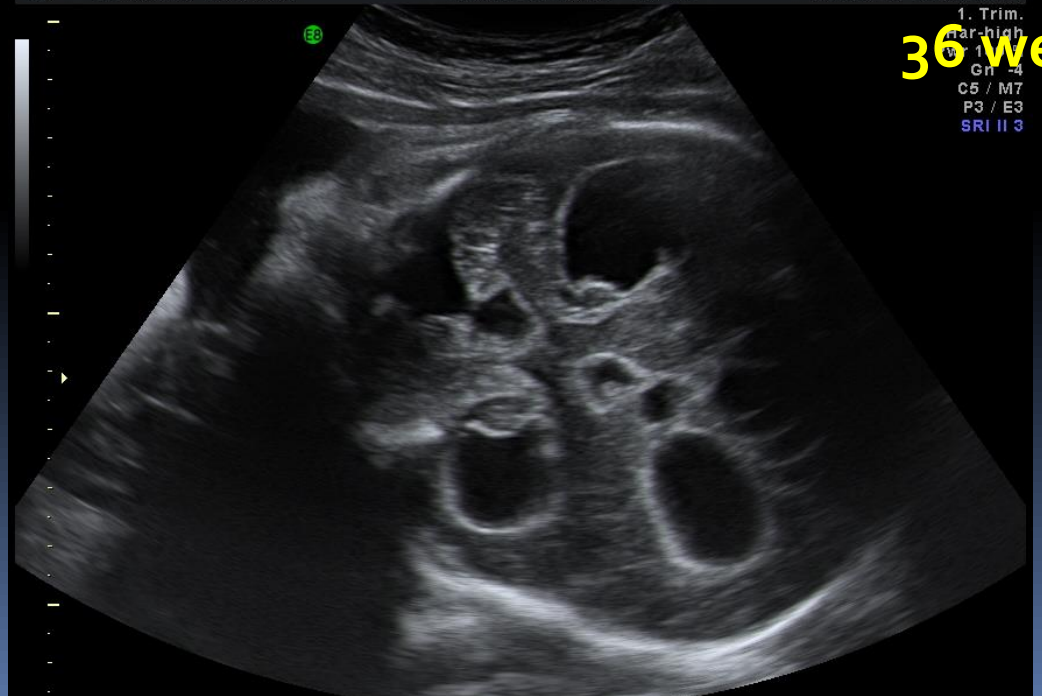

10.1cm / 20Hz
235 GA=26w4d
Lateral aspect of cerebral cortex to show topographic projection of motor centers on precentral gyrus
Hip
Trunk
Arm
Hand
Face
Visual and auditory
Temporopontine
Sensory
Corticospinal (pyramidal)
Frontopontine
Frontothalamic
Posterior
Leg
Trunk
Arm
Face
Toes
Ankle
Knee
Hip
Trunk
Shoulder
Elbow
Wrist
Fingers
Thumb
Neck
Brow
Eyelid
Nares
Lips
Tongue
Larynx
Primary motor cortex (area 4)
Posterior limb
Internal capsule
Anterior limb
Midbrain
III, IV and VI

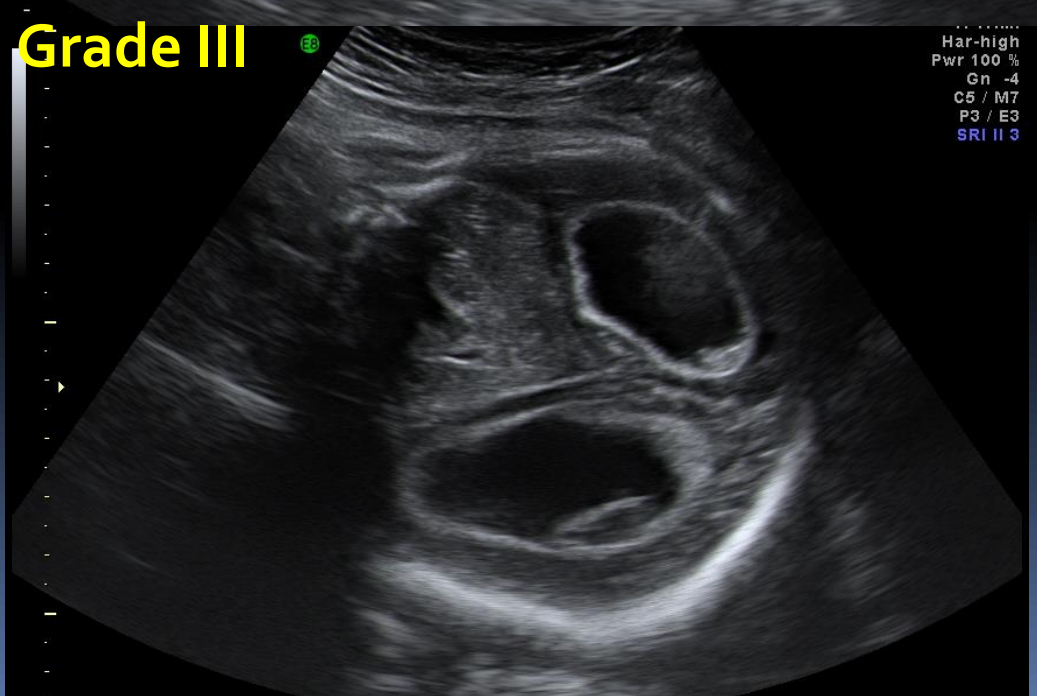

PARIETAL LOBE
PARACENTRAL LOBULE
PARIETAL LOBE
Lateral fissure
Supramarginal gyrus
Angular gyrus
Central sulcus
Postcentral gyrus
Precentral gyrus
Insular gyrus
Claustrum
External capsule
Posterior limb
BASAL GANGLIA
Choroid plexus
Caudate (tail)
Lateral ventricle
Middle frontal gyrus
Internal capsule
Anterior limb
Stria terminalis
Caudate (body)
THALAMUS
Fornix
BASAL GANGLIA
Lateral ventricle
FRONTAL LOBE
PARIETAL LOBE
Superior parietal lobule
Cingulum
Cingulate gyrus (retrosplenial area)
Induseum griseum
Precuneus?
Superior frontal gyrus
Cingulum
Cingulate gyrus
Induseum griseum
Corpus callosum
Corpus callosum
Interhemispheric fissure
Callosal sling
Corpus callosum (broken)
Interhemispheric fissure
Callosal glioepithelium
(intermingled with cingulate neuroepithelium and subventricular zone)
Fornical glioepithelium
Layer VII (subplate)
Cortical plate
Layer I
STF1 t2
STF2
STF4 t3
STF4 t2
STF6
Frontal/paracentral (agranular) STF
Frontal/paracentral neuroepithelium and subventricular zone
Corticostriatal neuroepithelium and subventricular zone
Strionuclear glioepithelium
Subpial granular layer (gliopithelium)
STF1 t2
STF2
STF3a
STF3b
STF3c
STF4 t2
STF5
STF6
Parietal (granular) STF

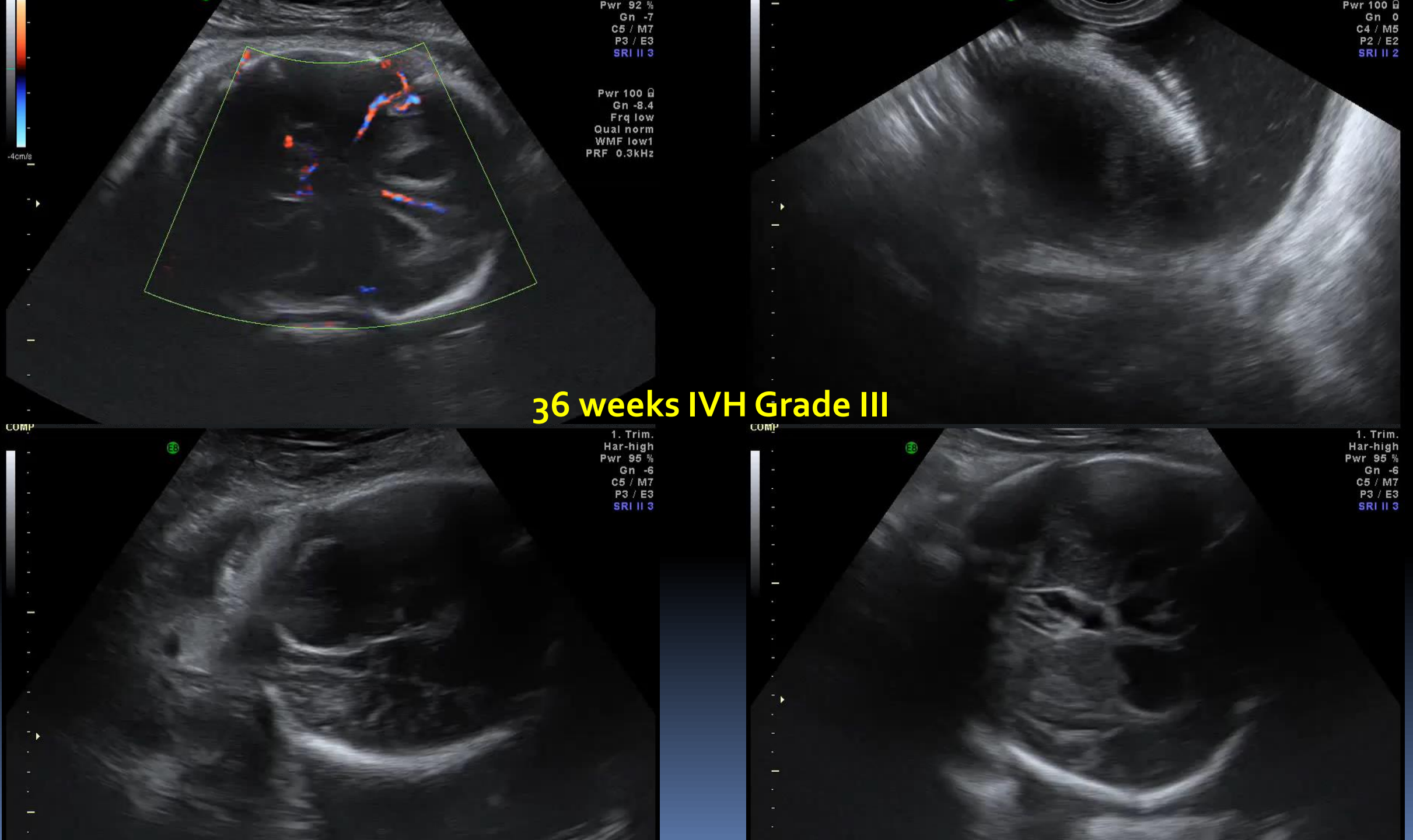


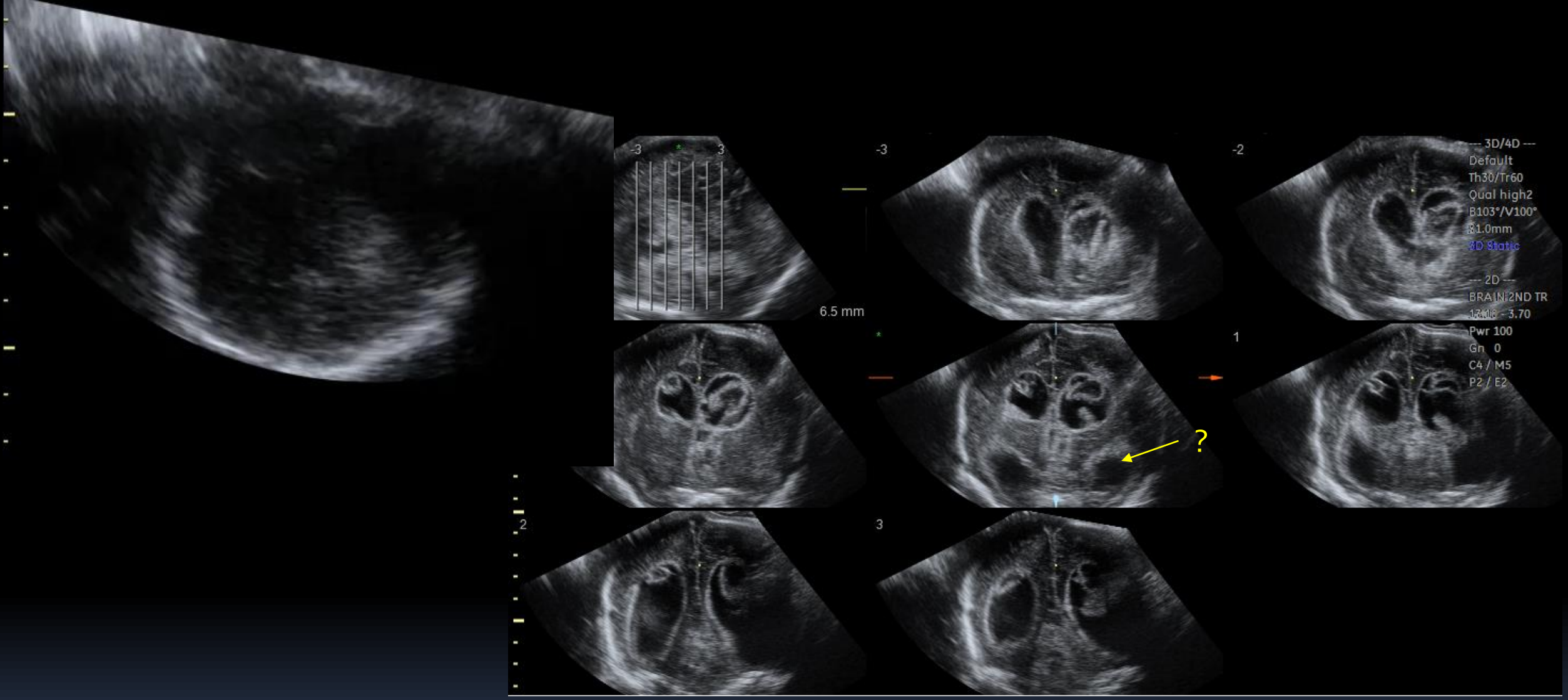


36 weeks IVH Grade III

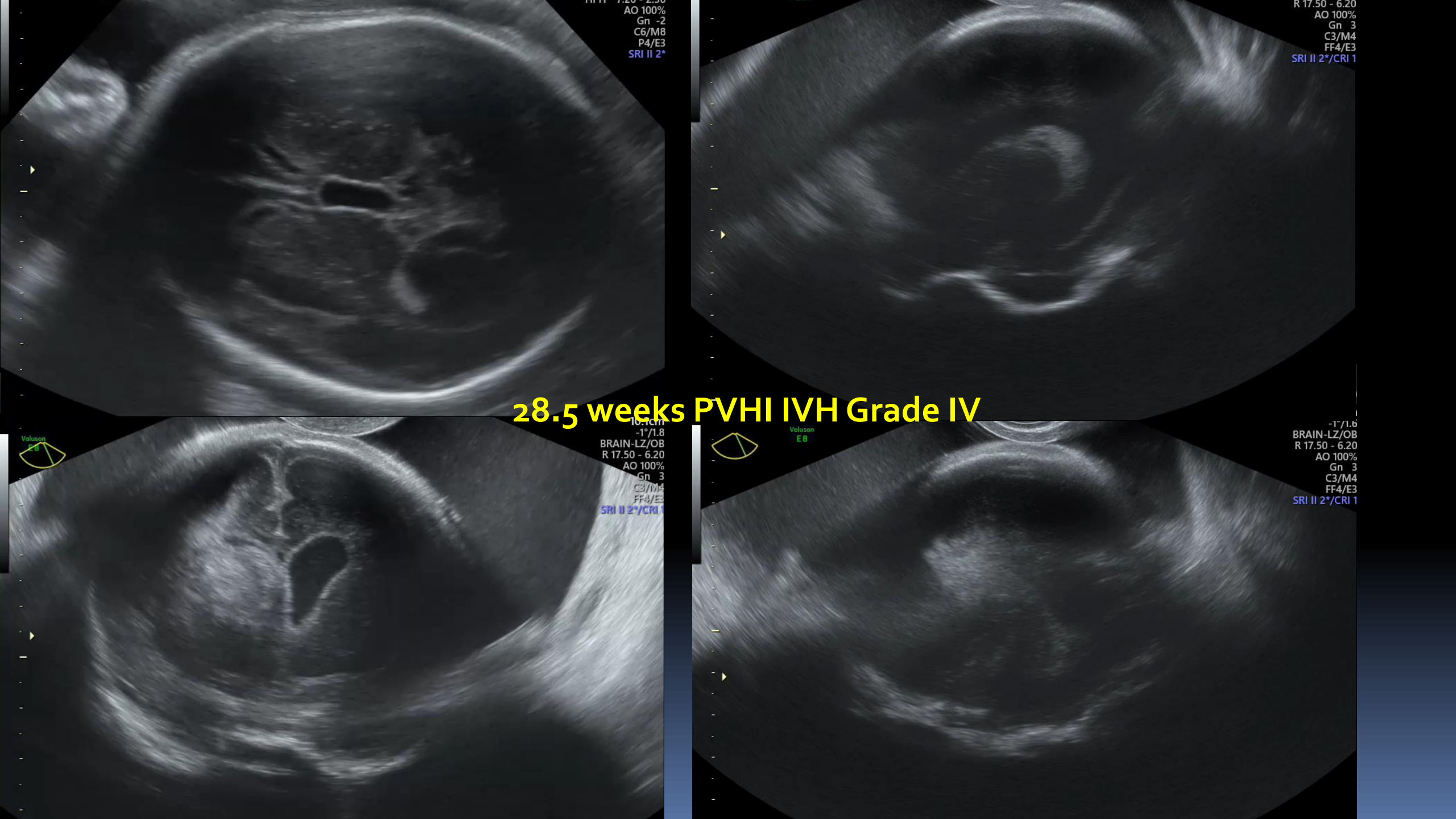




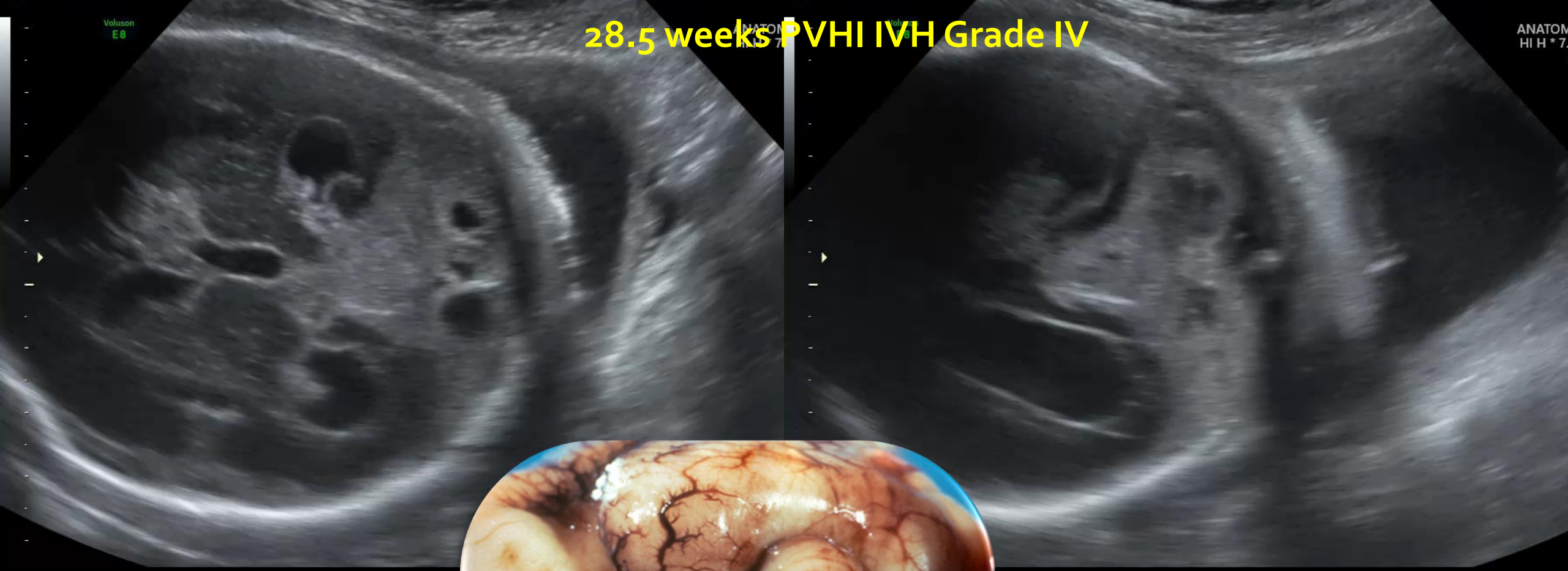

Pwr 92 %
Gn -7
C5 / M7
P3 / E3
SRI II 3
Pwr 100
Gn -8.4
Frq low
Qual norm
WMF low1
PRF 0.3kHz
-4cm/s
Pwr 100
Gn 0
C4 / M5
P2 / E2
SRI II 2
36 weeks IVH Grade III
COMP
1. Trim.
Har-high
Pwr 95 %
Gn -6
C5 / M7
P3 / E3
SRI II 3
COMP
1. Trim.
Har-high
Pwr 95 %
Gn -6
C5 / M7
P3 / E3
SRI II 3

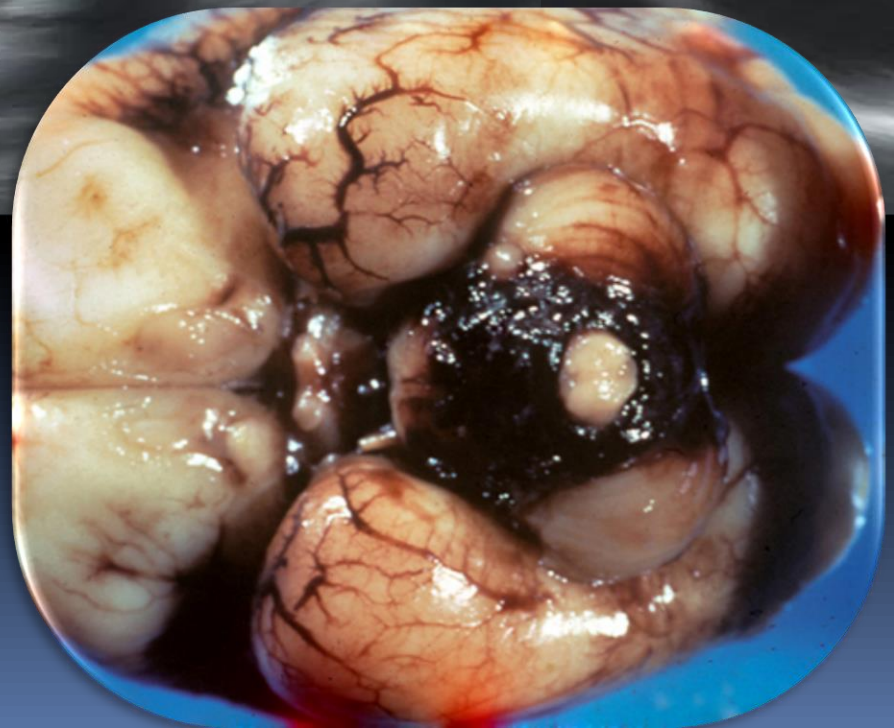


36 weeks Case: GMH-IVH Grade III


AO 100%
Gn -2
C6/M8
P4/E3
SRI II 2*
R 17.50 - 6.20
AO 100%
Gn 3
C3/M4
FF4/E3
SRI II 2*/CRI 1
28.5 weeks PVHI IVH Grade IV
Voluson
E8
-1°/1.8
BRAIN-LZ/OB
R 17.50 - 6.20
AO 100%
Gn 3
C3/M4
FF4/E3
SRI II 2*/CRI 1
Voluson
E8
BRAIN-LZ/OB
R 17.50 - 6.20
AO 100%
Gn 3
C3/M4
FF4/E3
SRI II 2*/CRI 1

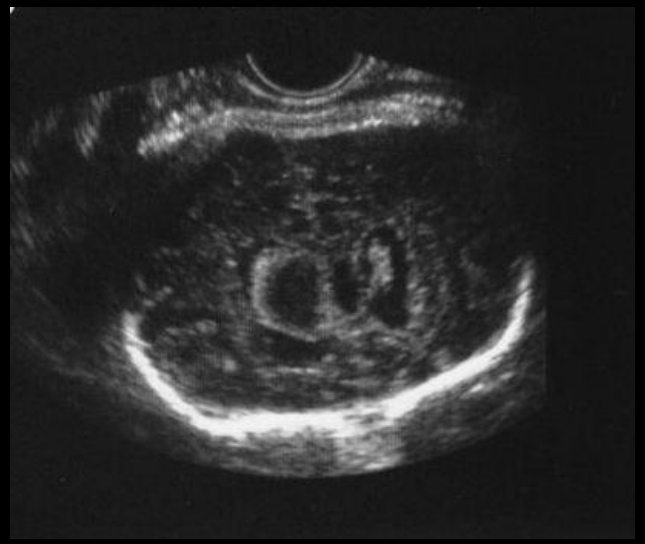
28.5 weeks PVHI IVH Grade IV

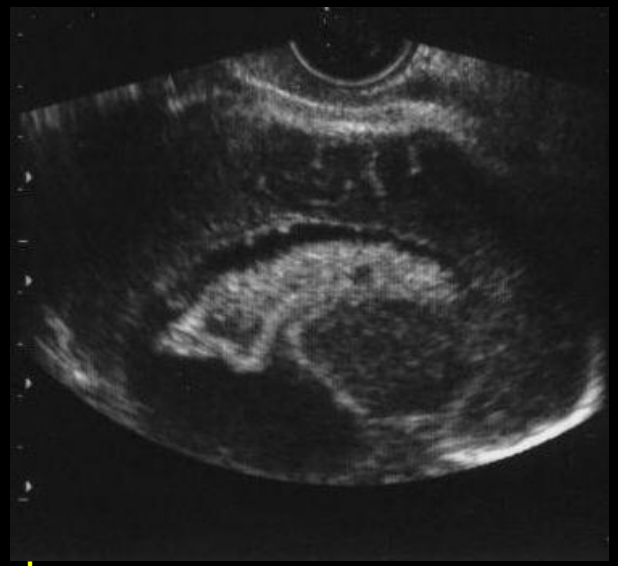




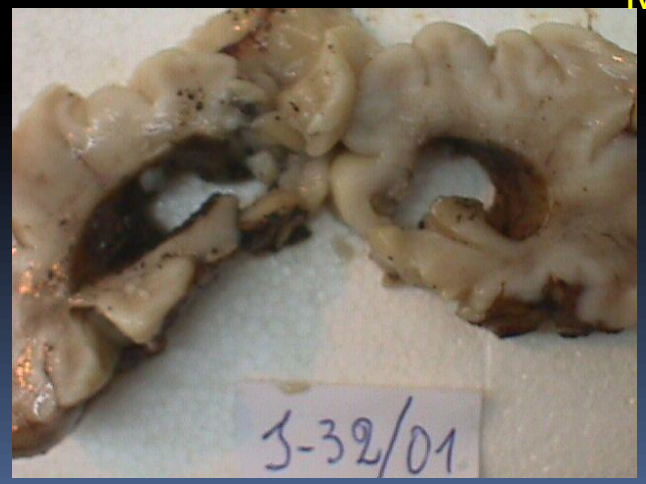
Blood spillage through the Magendi foramen

IVH – Grade III/IV

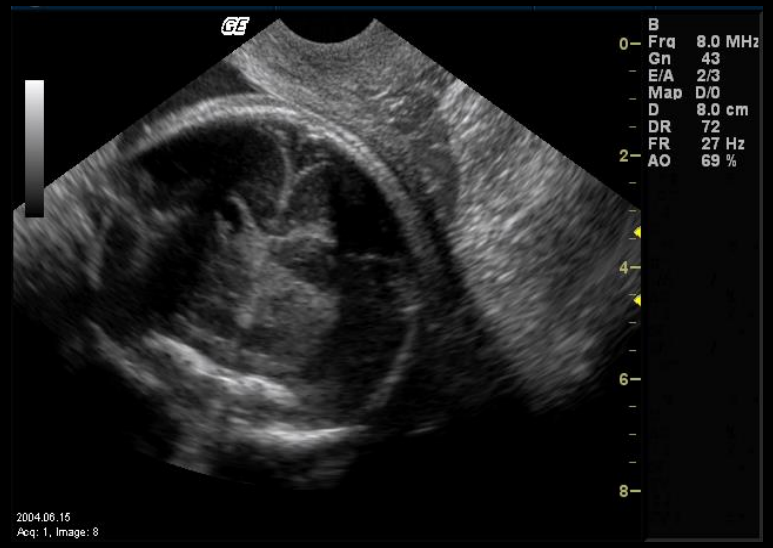


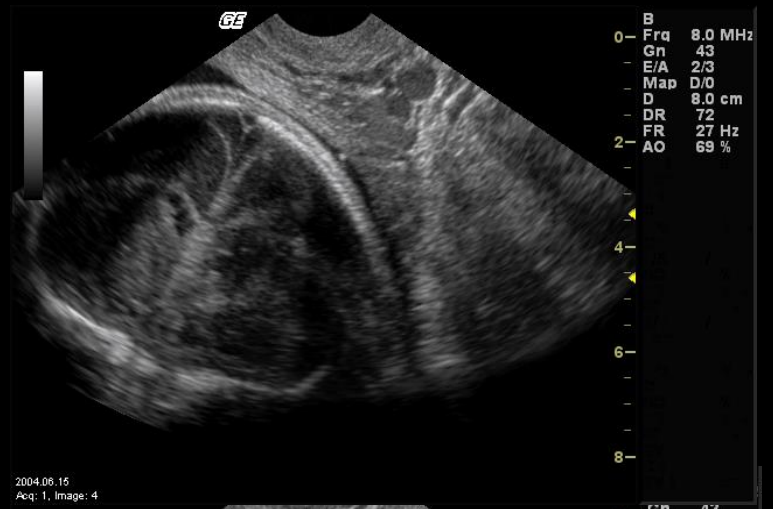


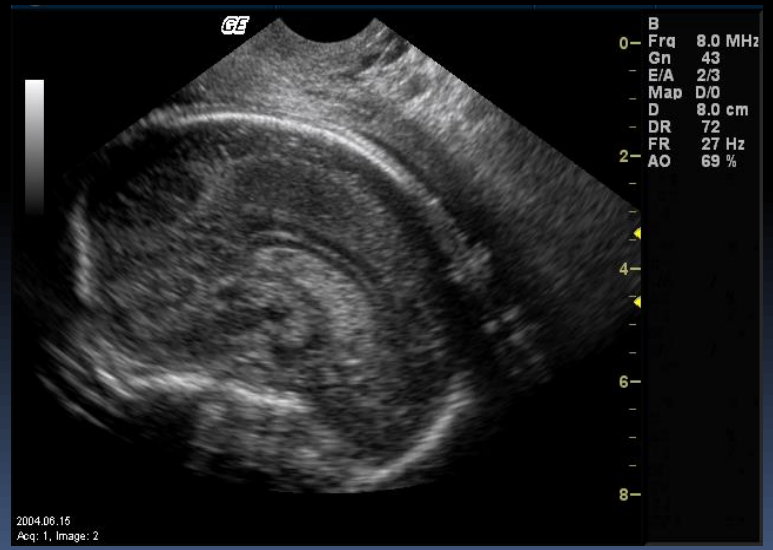
38 weeks
Reduced Fetal Movements

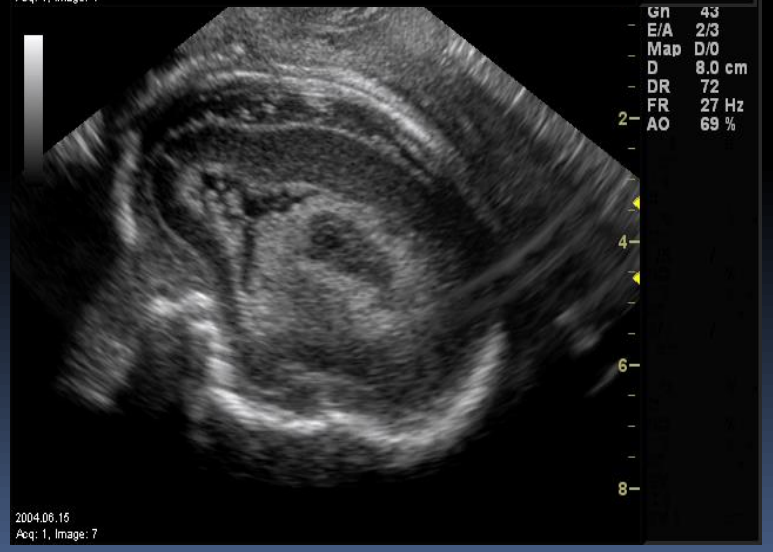


24 weeks IVH grade IV

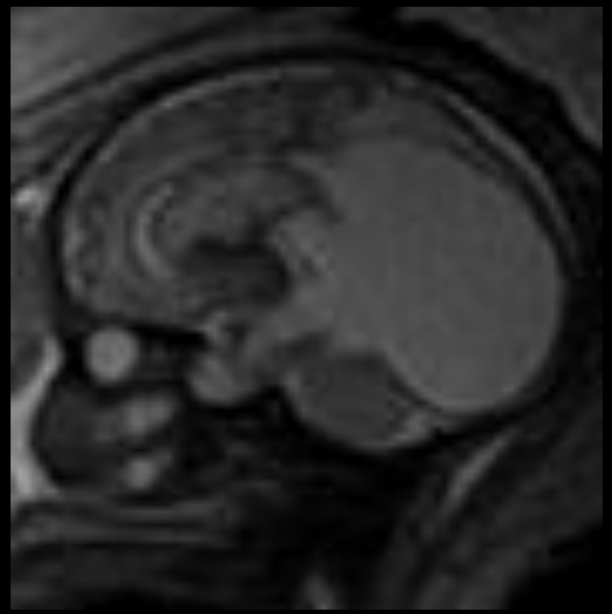


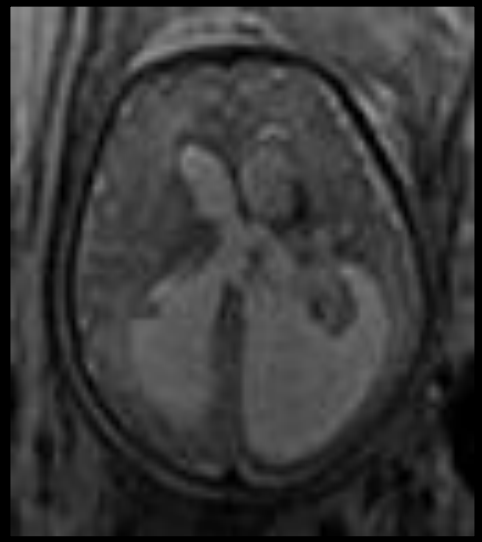


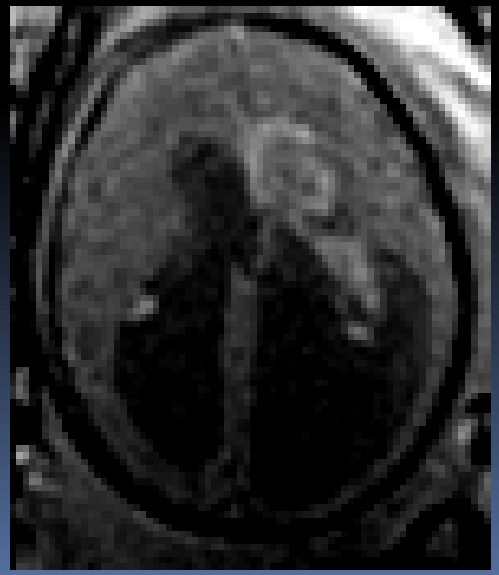


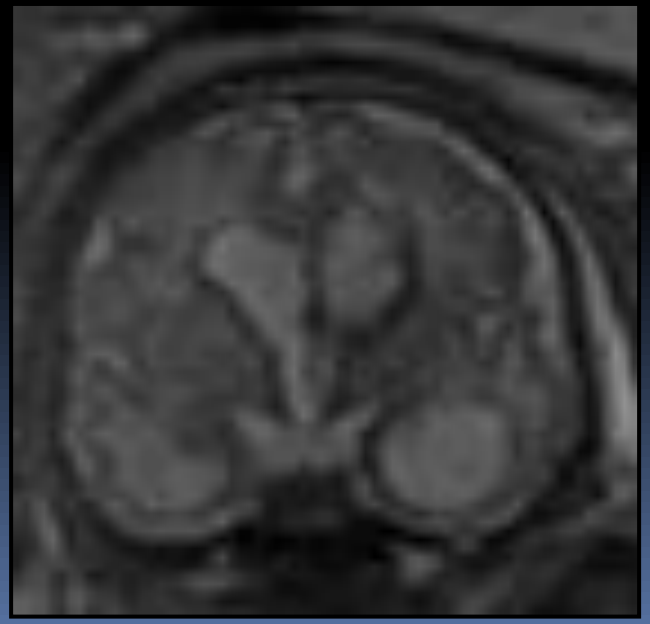


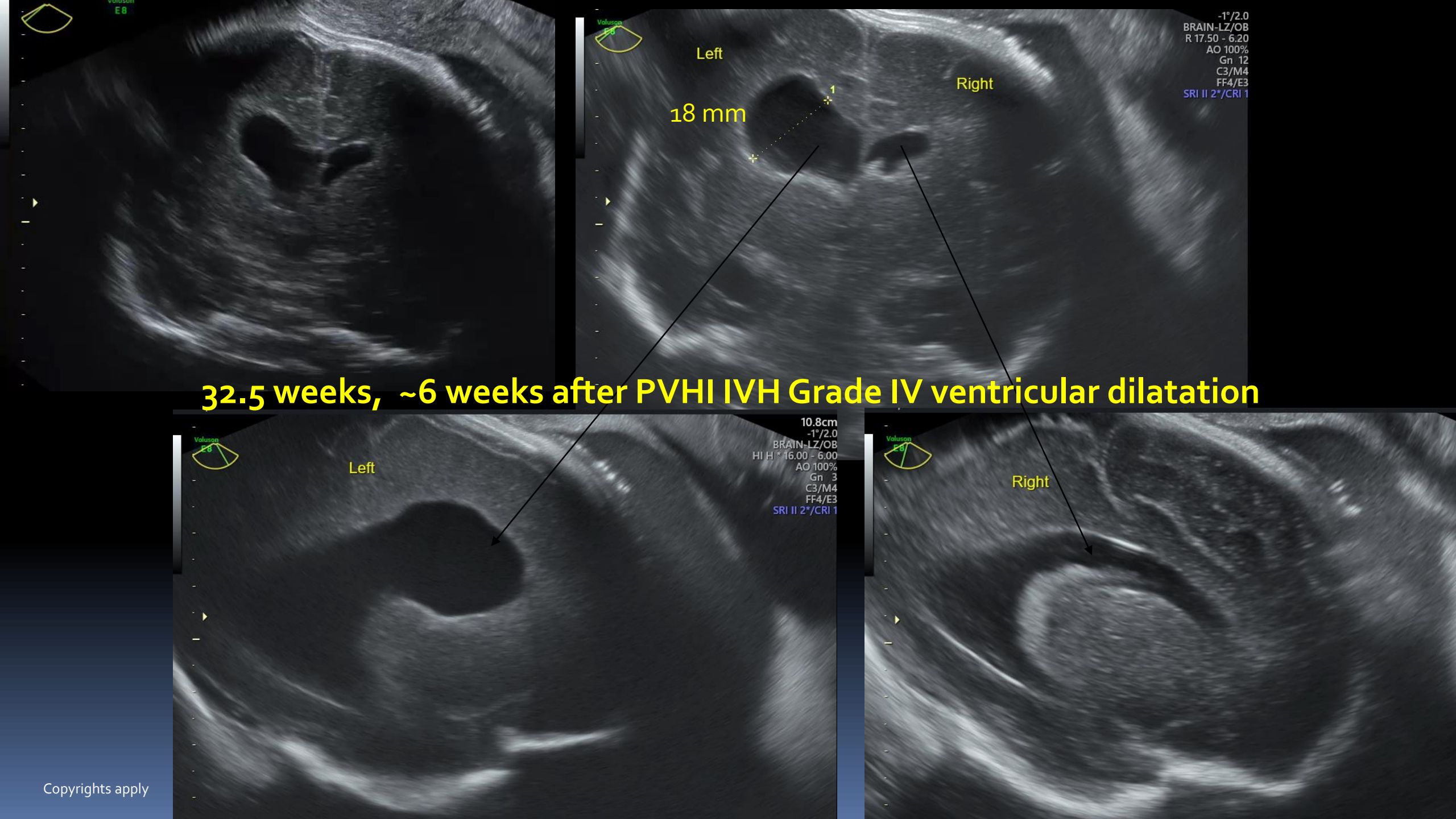
IVH grade IV (34 weeks)-MRI



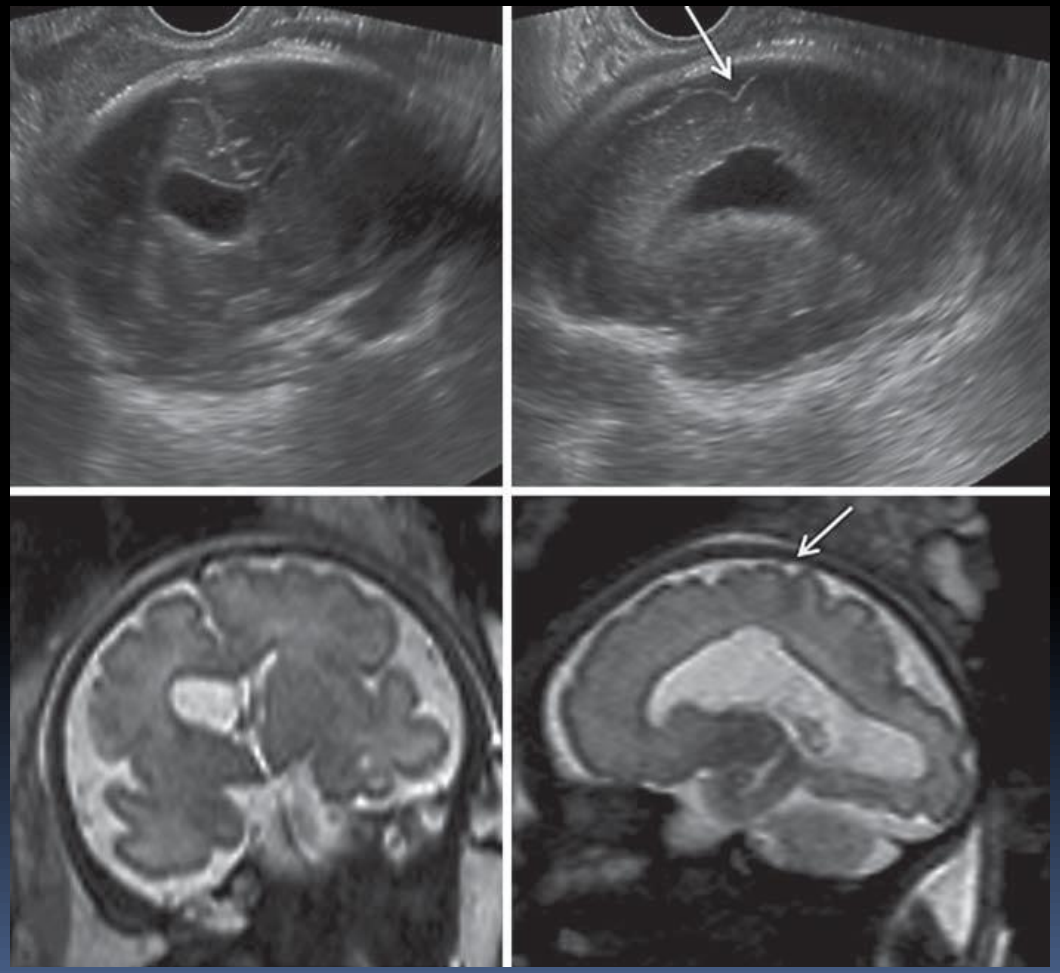




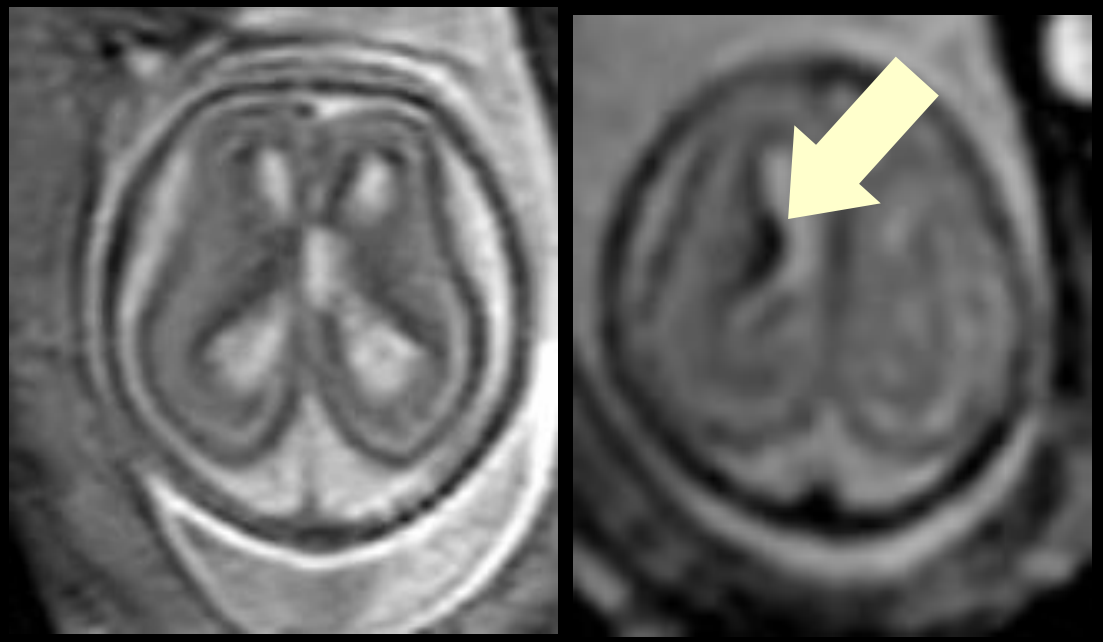


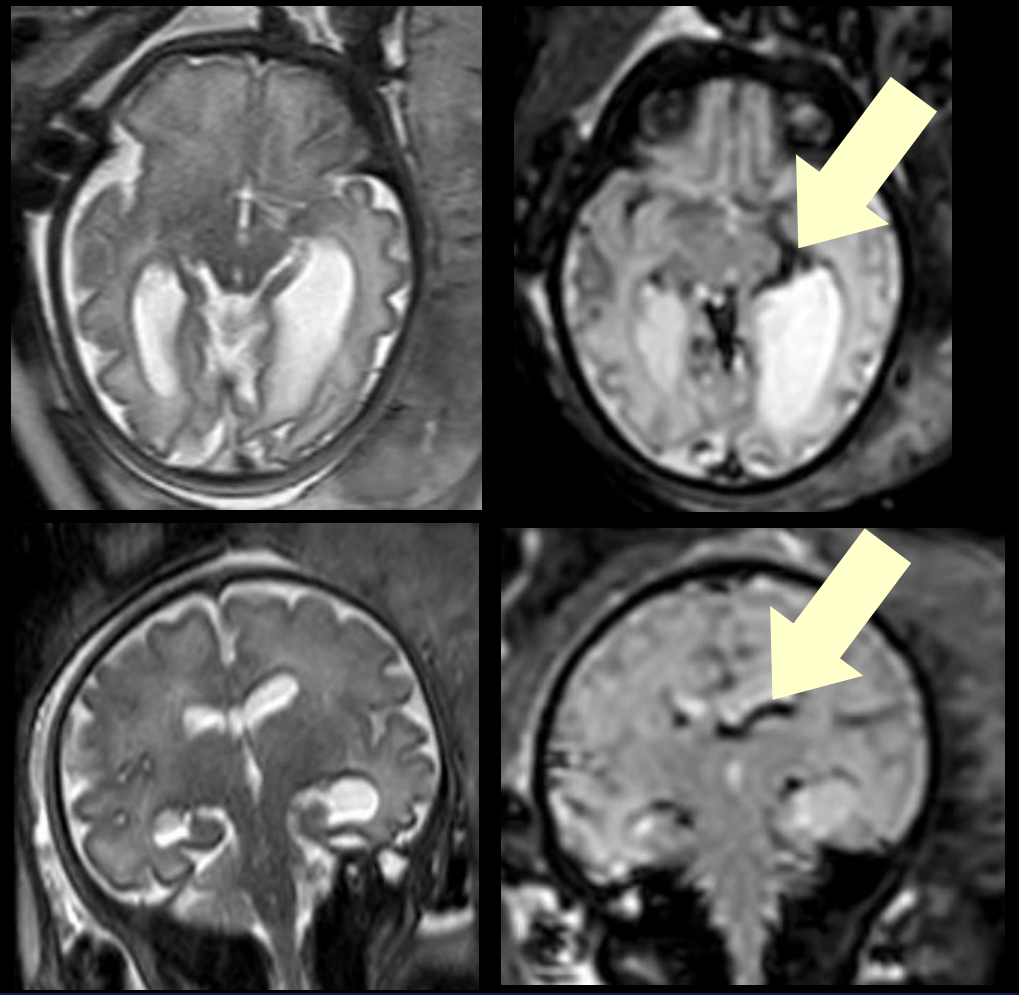


Porencephalic Cyst-MRI



Brain hemorrhages - Prenatal MRI



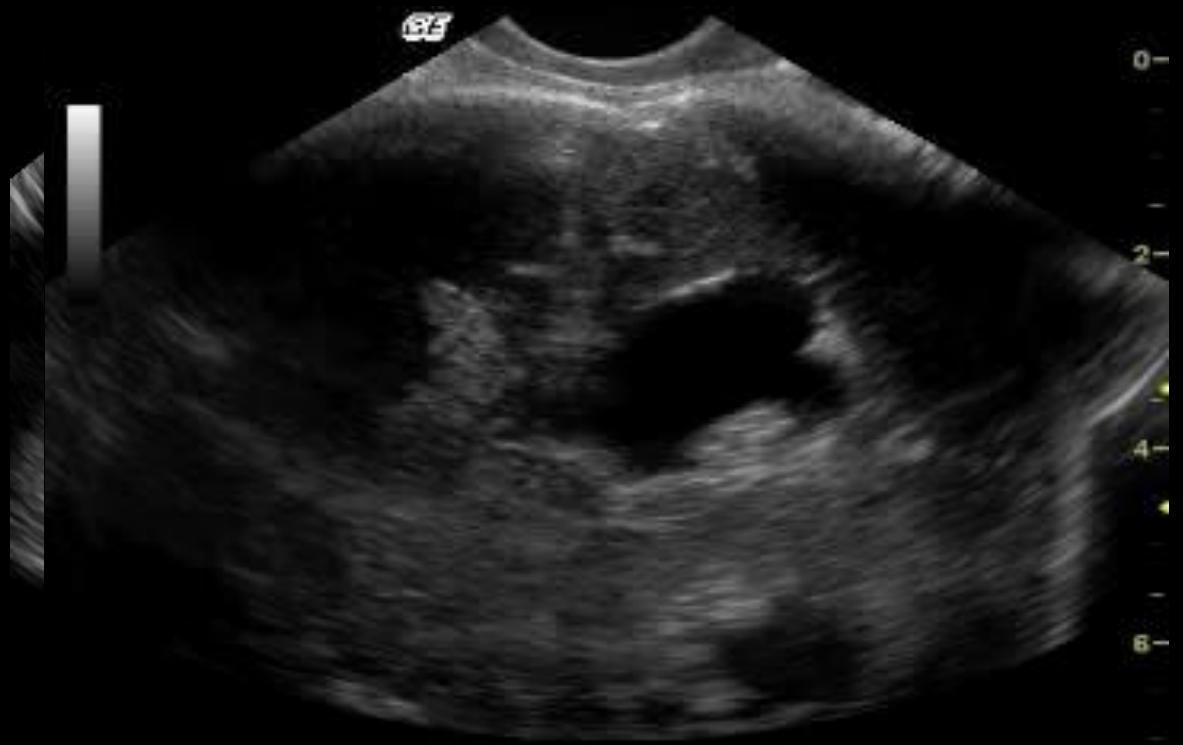


EPI/T2*: 30% more hemorrhages than T2-w!

unpublished

Timing of Occurrence

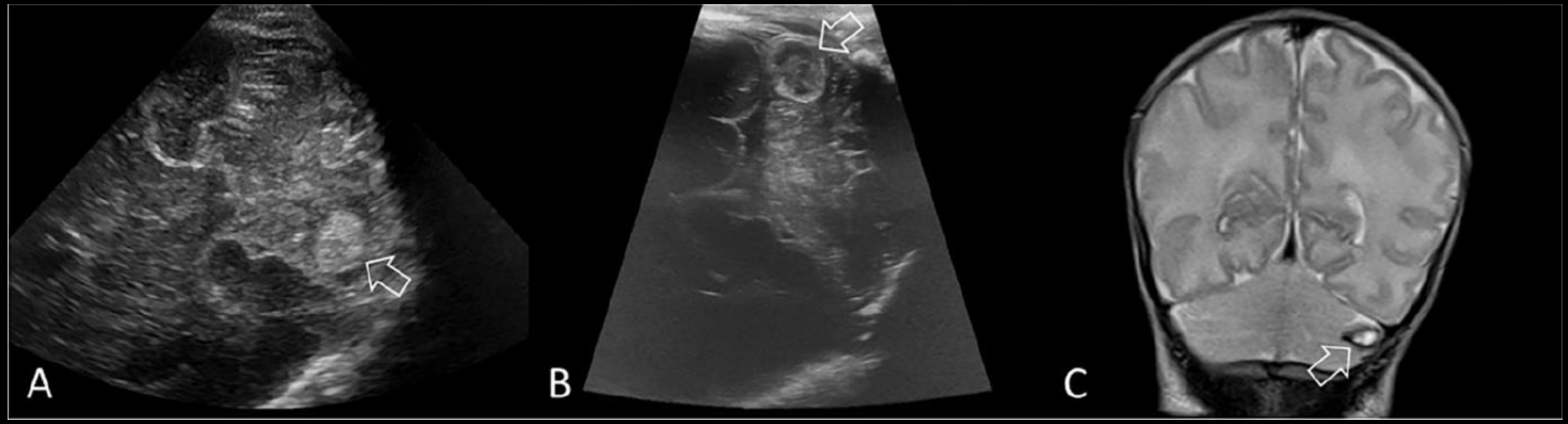
- Hyperacute (0-6 hours) hypoechoic particulate csf w/ motion
- Acute (6 hours – 3 days) hyperechoic – solid
- Early subacute (3-10 days) clot retraction - hypoechogenic central change - fibrin strands
- Late subacute (10-21 days) hyperechogenic ependyma clot w/ gray center & white border
- Chronic (21 days – 6 weeks) cystic changes retracted clots



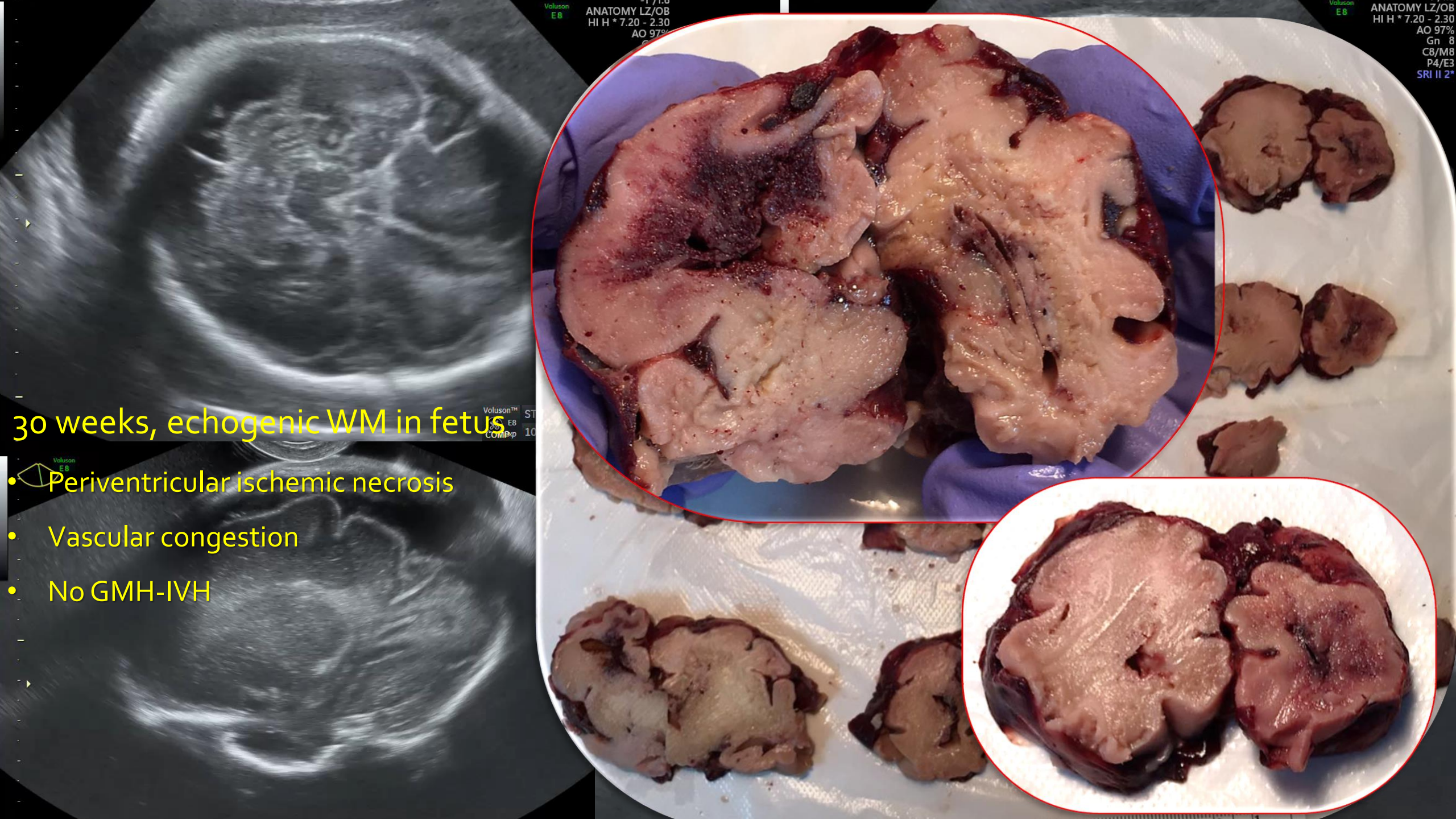
Adapted from Govaert & de Vries, 2010

FETAL CEREBELLAR HEMORRHAGE

Cerebellar Hemorrhage (CBH)

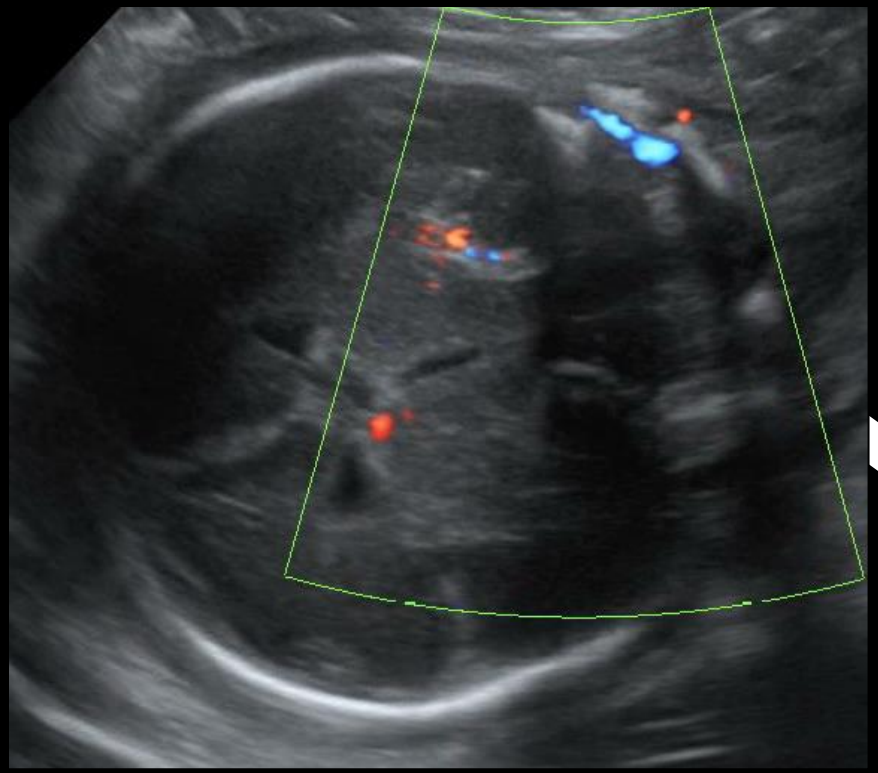


- CBH is frequently associated with a supratentorial hemorrhage (28–71%)
- CBH is confined to one cerebellar hemisphere in 71%, to the vermis in 20%, and affects both the cerebellar hemisphere and vermis in 9%
- CBHs can be divided into three groups: punctate, limited, and large
- Large CBH is associated with high mortality and morbidity (adverse long-term neurodevelopmental and behavioral outcomes)

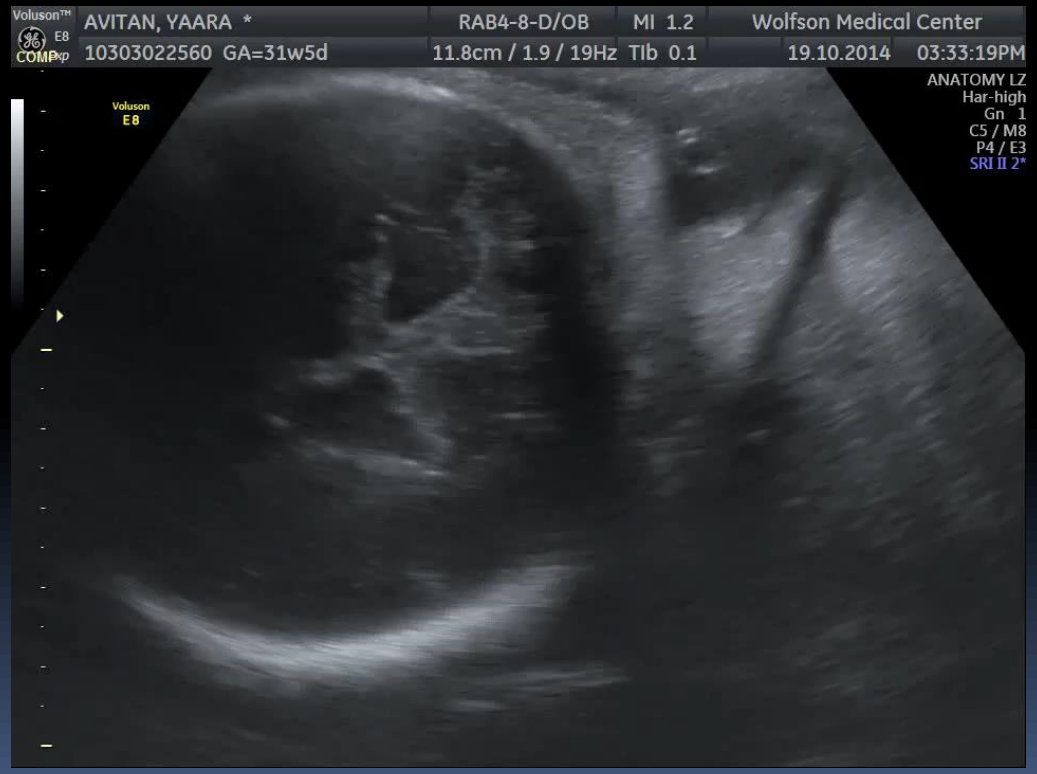


30 weeks, echogenic WM in fetus

- Periventricular ischemic necrosis
- Vascular congestion
- No GMH-IVH



Post-hemorrhagic unilateral cerebellar “hypoplasia” (31w)



Ultrasound Obstet Gynecol 2014; **44**: 447–454
Published online in Wiley Online Library (wileyonlinelibrary.com). **DOI:** 10.1002/uog.13217

Prenatal unilateral cerebellar hypoplasia in a series of 26 cases: significance and implications for prenatal diagnosis

M. MASSOUD*, M. CAGNEAUX*†, C. GAREL‡§, N. VARENE§, M.-L. MOUTARD‡, T. BILLETTE‡, A. BENEZIT¶, C. ROUGEOT¶, J.-M. JOUANNIC**, J. MASSARDIER*, P. GAUCHERAND*, V. DESPORTES‡¶ and L. GUIBAUD*†‡

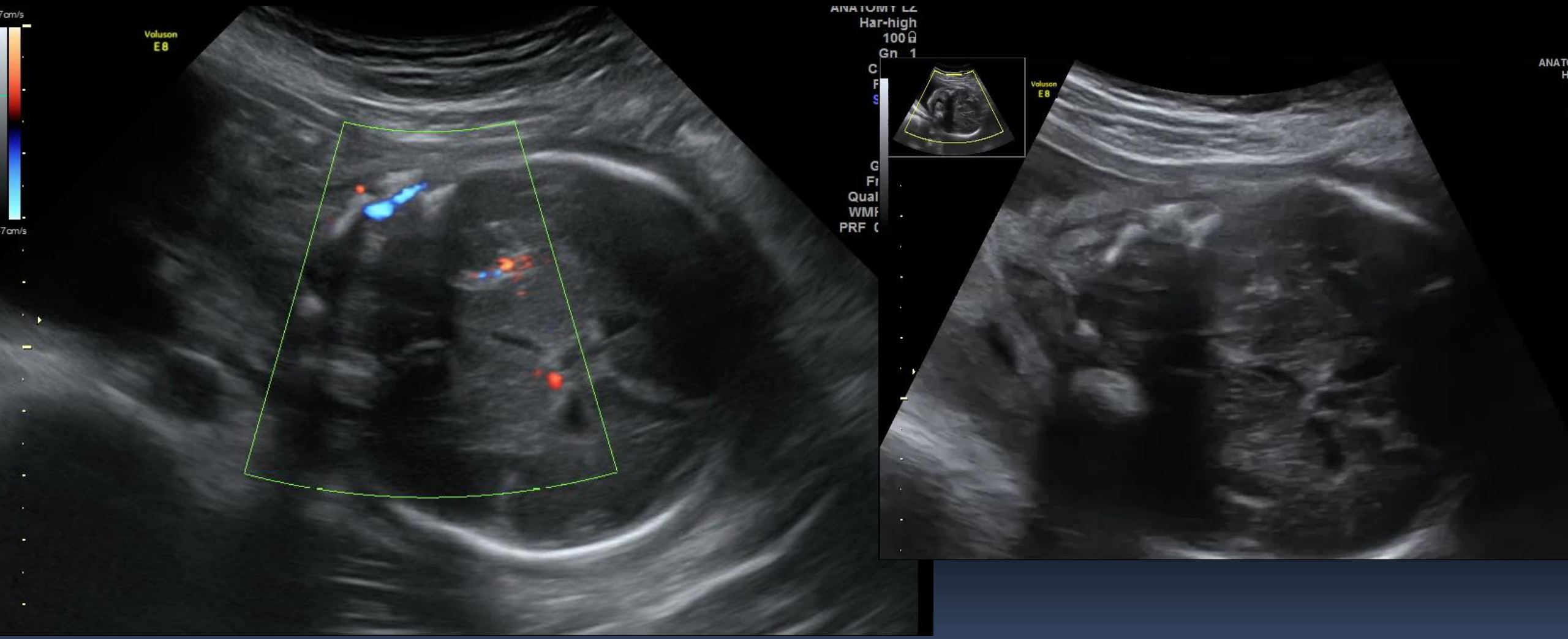
**Centre Pluridisciplinaire de Diagnostic Prénatal, Hôpital Femme Mère Enfant, Université Claude Bernard Lyon I, Lyon, France; †Département d'Imagerie Pédiatrique et Fœtale, Hôpital Femme Mère Enfant, Université Claude Bernard Lyon I, Lyon, France; ‡Centre de Référence des Malformations du Cervelet, Hôpital Femme Mère Enfant, Lyon et Hôpital Armand Trousseau, Paris, France; §Service de Radiologie, Hôpital d'Enfants Armand-Trousseau, Paris, France; ¶Département de Neuropédiatrie, Hôpital Femme Mère Enfant, Université Claude Bernard Lyon I, Lyon, France; **Service d'Obstétrique, Hôpital Armand Trousseau, Paris, France*

- The surface loss of cerebellar hemisphere >50% in 19/24
- The vermis was normal in 19/24

- Predisposing factors for vascular insult were identified in 8/24:
  - Maternal alcohol addiction
  - Diabetes mellitus
  - Congenital CMV
  - Pathological placenta with thrombotic vasculopathy and infarctions

- UCH is a focal cerebellar lesion of clastic origin
- UCH may be a clue for prenatal diagnosis of PHACES syndrome
- The amount of cerebellar surface loss does not correlate with poor prognosis
- UCH with a normal vermis is often associated with a normal outcome

26 weeks, HELLP syndrome



OUTCOME

Ultrasound Obstet Gynecol 2003; 22: 121–130
Published online in Wiley InterScience (www.interscience.wiley.com). DOI: 10.1002/uog.191

Outcome of antenatally diagnosed intracranial hemorrhage: case series and review of the literature

T. GHI*, G. SIMONAZZI*, A. PEROLO*, L. SAVELLI*, F. SANDRI†, B. BERNARDI‡, D. SANTINI§, L. BOVICELLI* and G. PILU*

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- 109 fetal ICHs
- 89 - intracerebral (79 IVH and 10 infratentorial)
- 20 - subdural hematoma

At 12 months follow-up (48 cases)

52% were judged neurologically normal:

- 47% among the IVHs
- 66% among the hematomas
- 66% among the infratentorial ICHs

	Total	*Normal*	*Mild handicap*	*Severe handicap*	*Dead*
Cases	48	25	6	13	4
Intracerebral	39	19	5	12	3
IVH I	4	4	0	0	0
IVH II	7	4	0	3	0
IVH III	18	8	5	5	0
IVH IV	7	1	0	3	3
Infratentorial	3	2	0	1	0
Subdural hematoma	9	6	1	1	1
Both	0	0	0	0	0

Intracranial Hemorrhage

in utero

Obstet Gynecol 2020

Society for Maternal-Fetal Medicine (SMFM); Ana Monteagudo, MD

GMH-IHV	Survival (%)	Neurologic sequelae (%)
Grade II	100	10
Grade III	95	63 (severe: 31.6)
Grade IV	83	85 (severe: 60)

Resolution of ICH is associated with good outcome

It is not known whether the outcomes of fetal GMH-IVH are like neonatal….

Journal of Perinatology (2018) 38:1431–1443
https://doi.org/10.1038/s41372-018-0209-5

REVIEW ARTICLE

Preterm neuroimaging and neurodevelopmental outcome: a focus on intraventricular hemorrhage, post-hemorrhagic hydrocephalus, and associated brain injury

Rebecca A. Dorner[1,2] · Vera Joanna Burton[2,3,4] · Marilee C. Allen[1,2] · Shenandoah Robinson[2,5] · Bruno P. Soares[2,6]

- PHVD an important complication of IVH in preterm infants
- PHVD occurs in 30% to 50% of infants with GMH-IVH grades III or PVHI
- Progressive post-hemorrhagic hydrocephalus is a risk factor for adverse neurodevelopmental outcomes

Prognosis

- The prognosis depends both on the etiology and the lesion
- The postnatal scoring systems for prediction of outcome of IVH , depend mainly on the degree of VM, uni or bilateral and parenchymal involvement
- There are not enough studies on outcome of fetal IVH
- The postnatal classification is not sufficient
- The most important factor is probably the extent and localization of the WM injury
- Tractography is emerging as an important tool for prognostication

Prognosis-Ischemic-hemorrhagic parenchymal insults

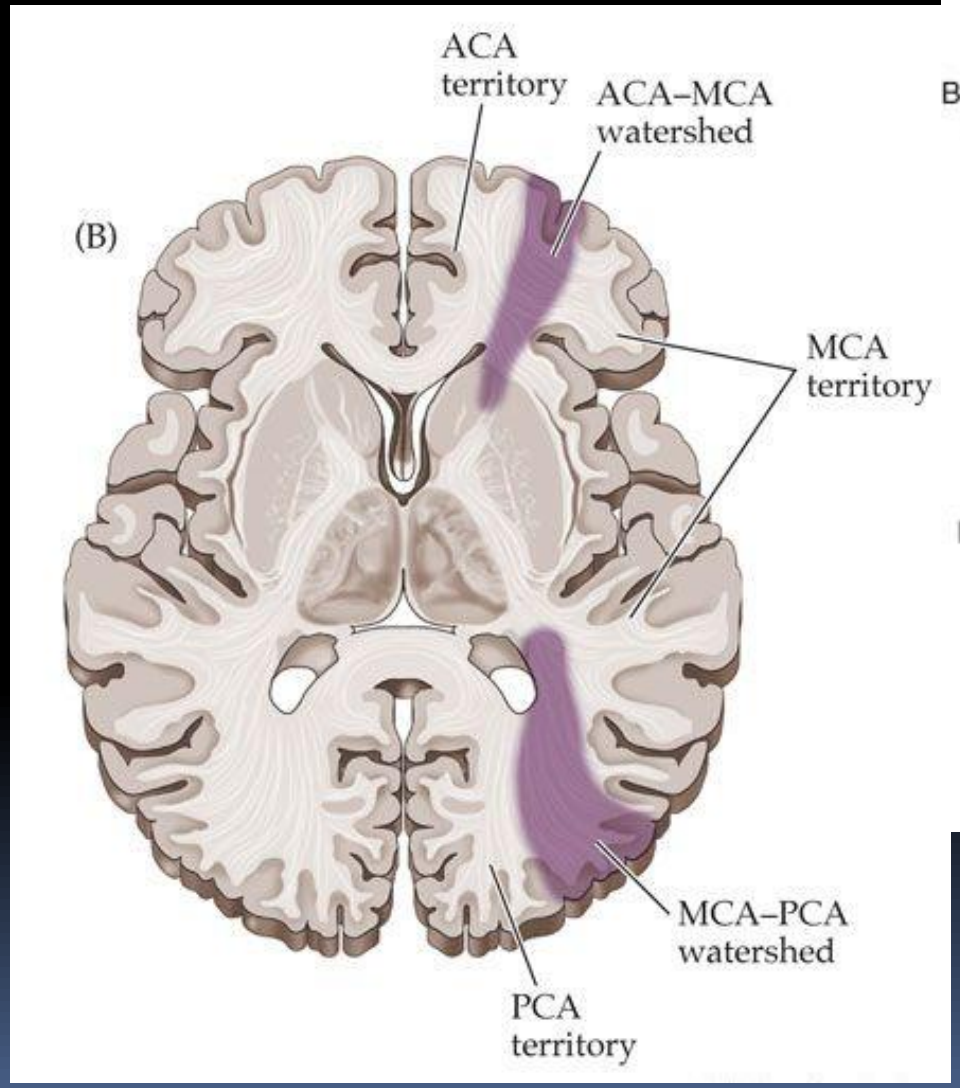
Lesion localization impacts on the prognosis:

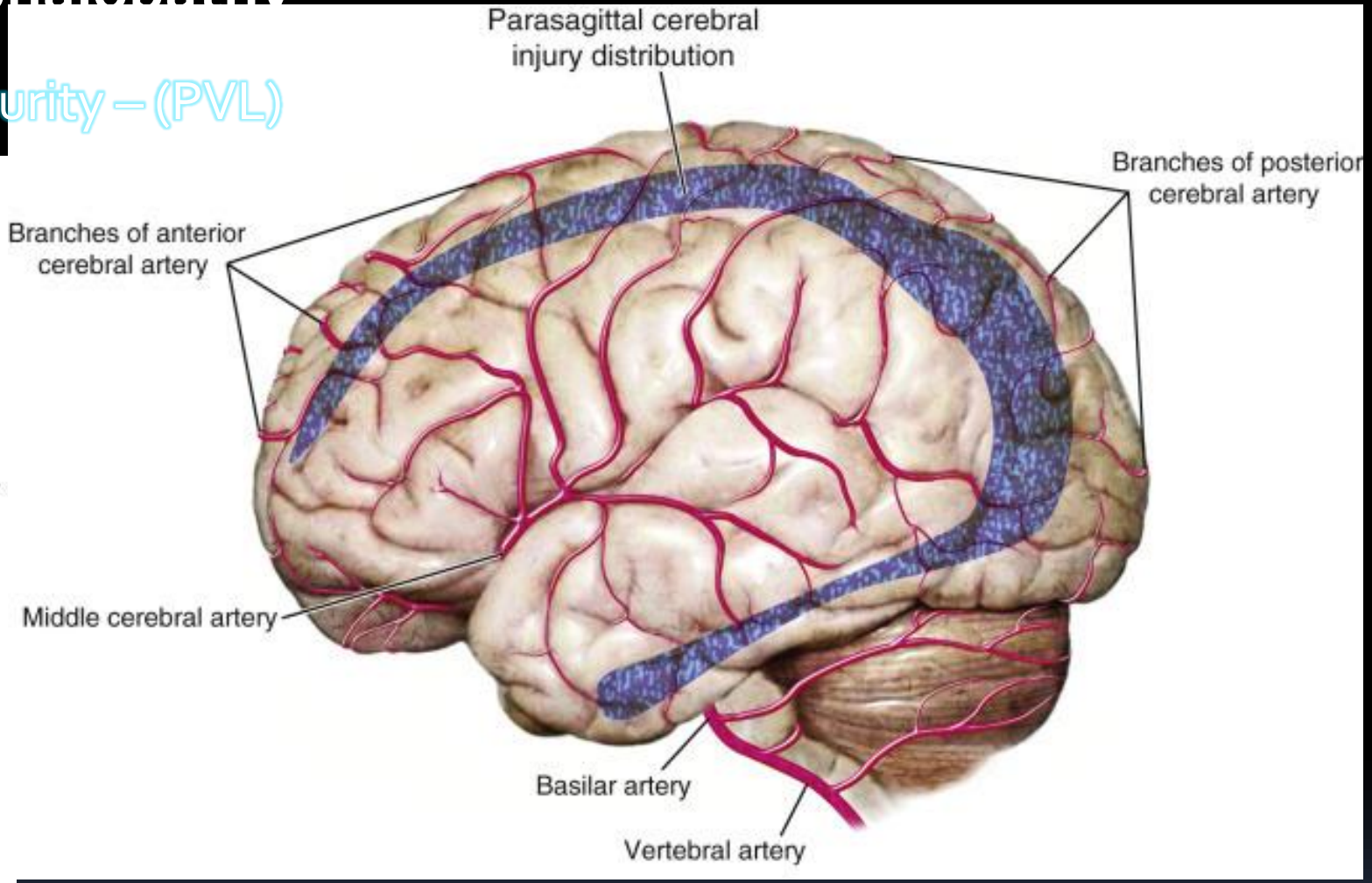
- A unilateral anterior frontal lesion, can carry a good prognosis
- A unilateral cerebellar hemispheric insult may also have a good outcome if the vermis is not involved
- Lesions involving the cortex are at risk for epilepsy
- If the central sulcus is involved, a contralateral congenital hemiparesis is the rule
- Lesions of the occipital lobe may generate visual disorders
- Bilateral, multifocal or extensive lesions worsen the prognosis and may be responsible for intrauterine fetal death

FETAL PERIVENTRICULAR ECHOGENICITY

Hypoxic-ischemic encephalopathy

White matter injury of prematurity – (PVL)





- **PVL in preterm infants is related to:**
- Arterial border zone (watershed) ischemia/infarction
- Vulnerability of oligodendrocyte precursors

frontiers in Pediatrics

METHODS
published: 08 March 2021
doi: 10.3389/fped.2021.618236

White matter injury of prematurity (PVL)

Grade 1: Transient areas of increased PVE* persisting for≥7days

Grade 2: Transient PVE that evolves into small, localized cysts in the fronto-parietal periventricular white matter

Grade 3: PVE evolving into extensive periventricular cystic lesions in the fronto-parieto-occipital white matter

Grade 4: PVE evolving into extensive cystic lesions in the deep white matter or in the subcortical white matter

(L. De Vries et al., Behav Brain Res, 1992)

*, PVE, abnormal periventricular echogenicity. The reference is the echogenicity of the choroid plexus.

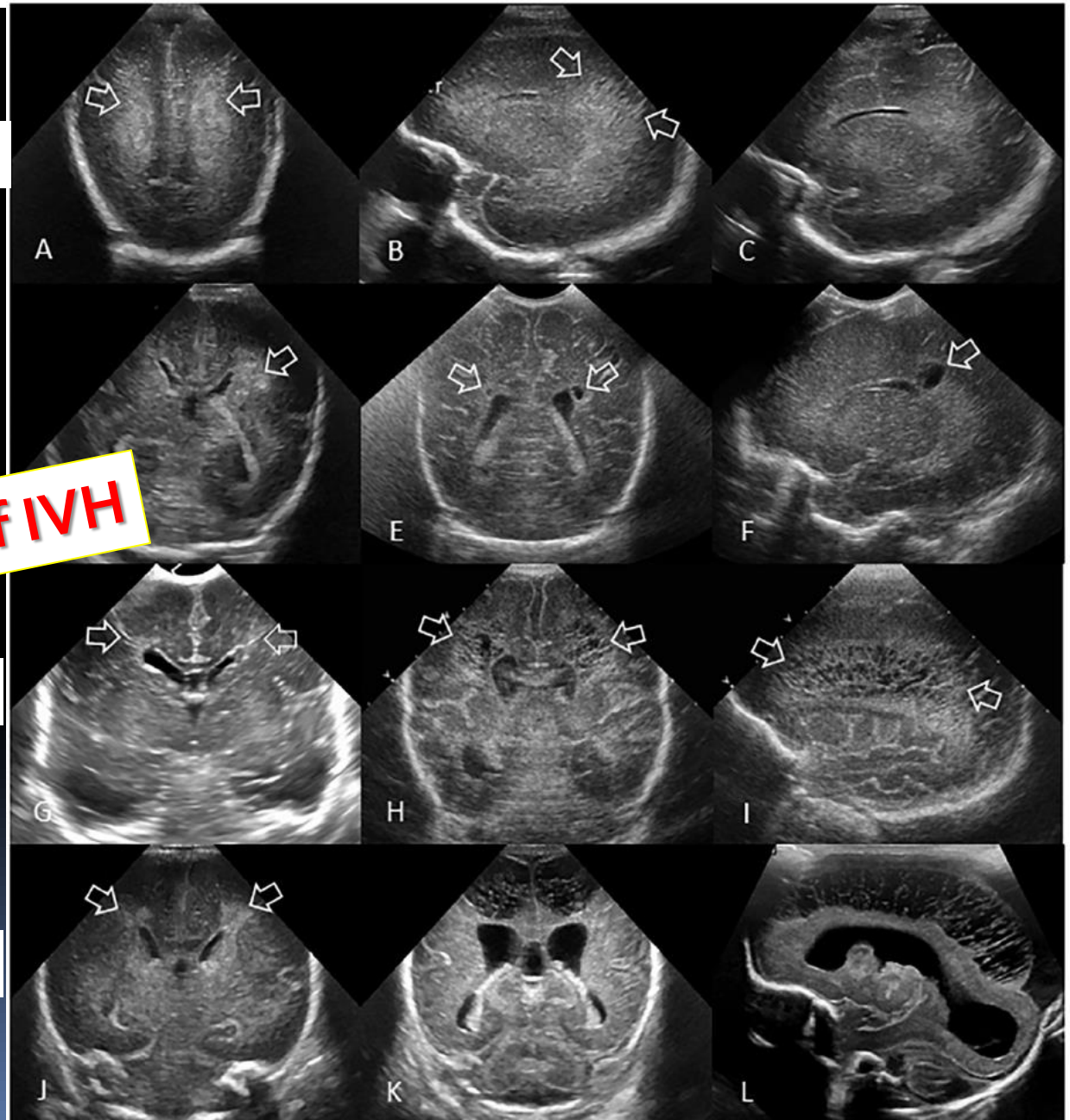
Grade 1

Grade 2

Grade 3

Grade 4

No signs of IVH



Arch Dis Child Fetal Neonatal Ed 2008 Review

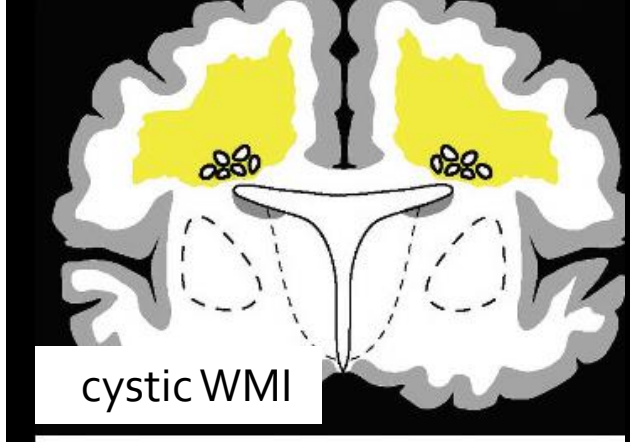
Pathogenesis of cerebral white matter injury of prematurity

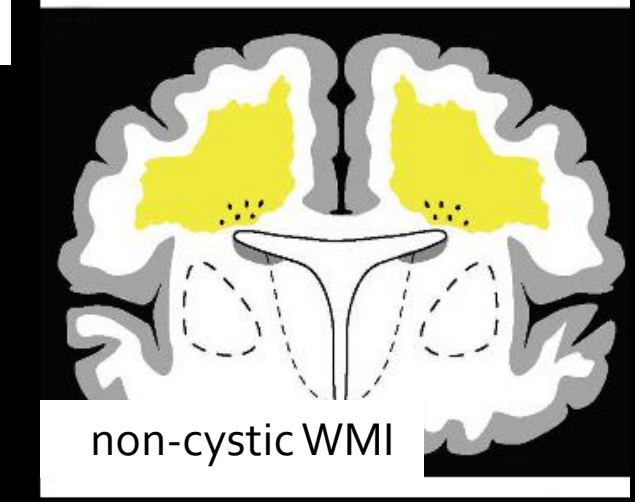
O Khwaja, J J Volpe

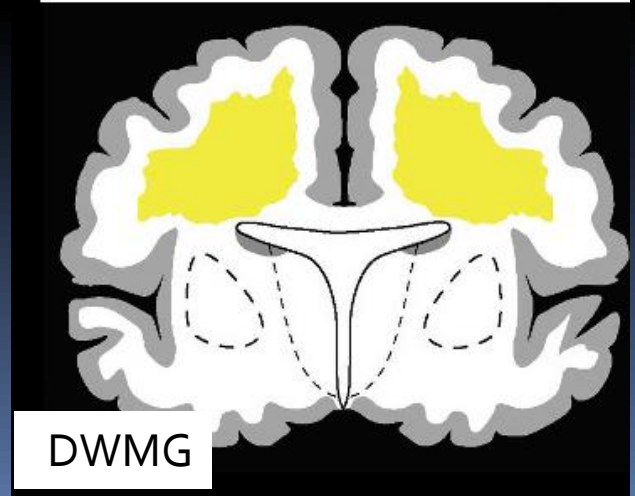
Mechanisms of WMI:

- **<u>focal necrosis deep in the white matter:</u>**
  - cystic WMI: the focal necrotic lesions are macroscopic and evolve to cysts
  - non-cystic WMI: the focal lesions are microscopic and evolve to glial scars

- **<u>diffuse white matter gliosis (DWMG) w/o necrosis:</u>**
  - characterized by a loss of pre-oligodendrocytes and astrogliosis
  - the mildest form of WMI

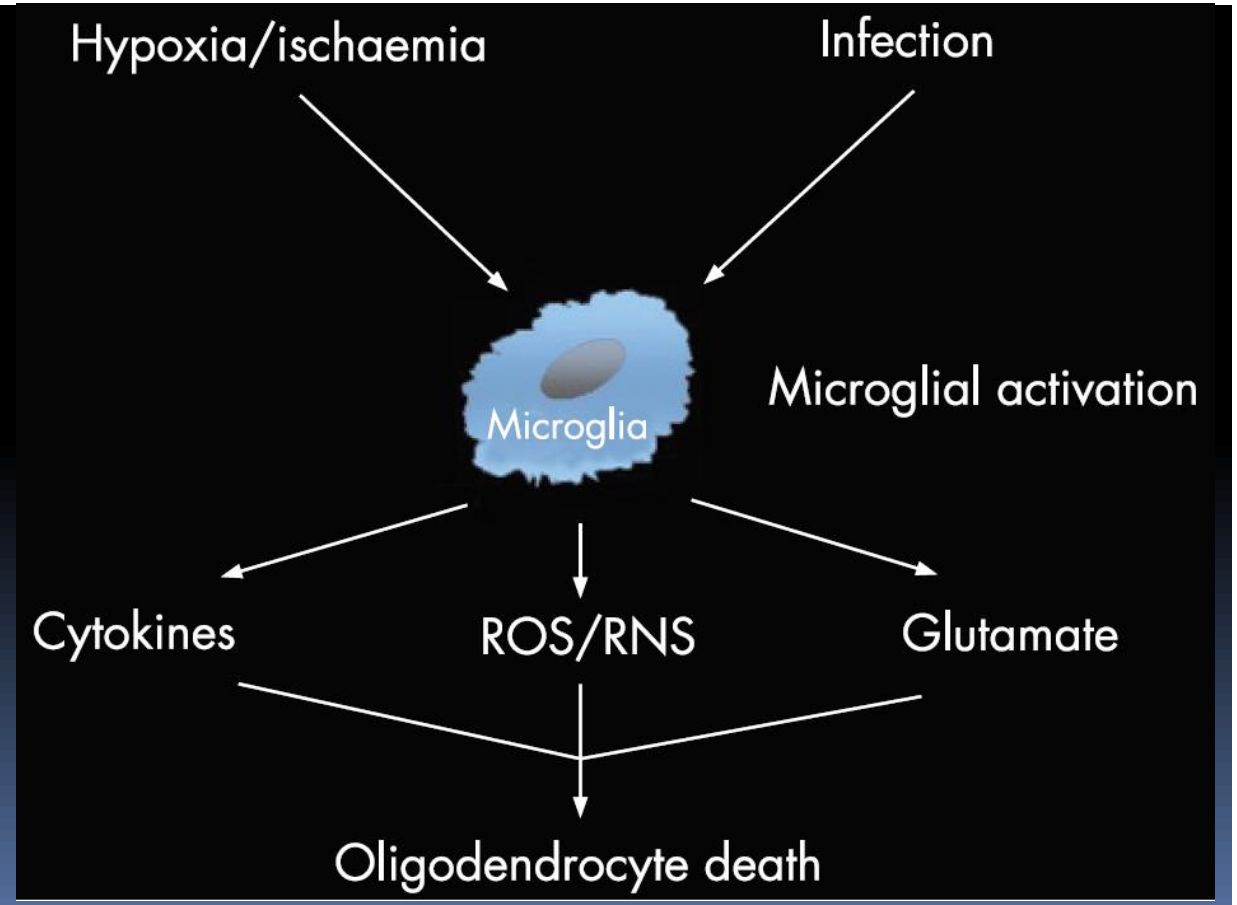


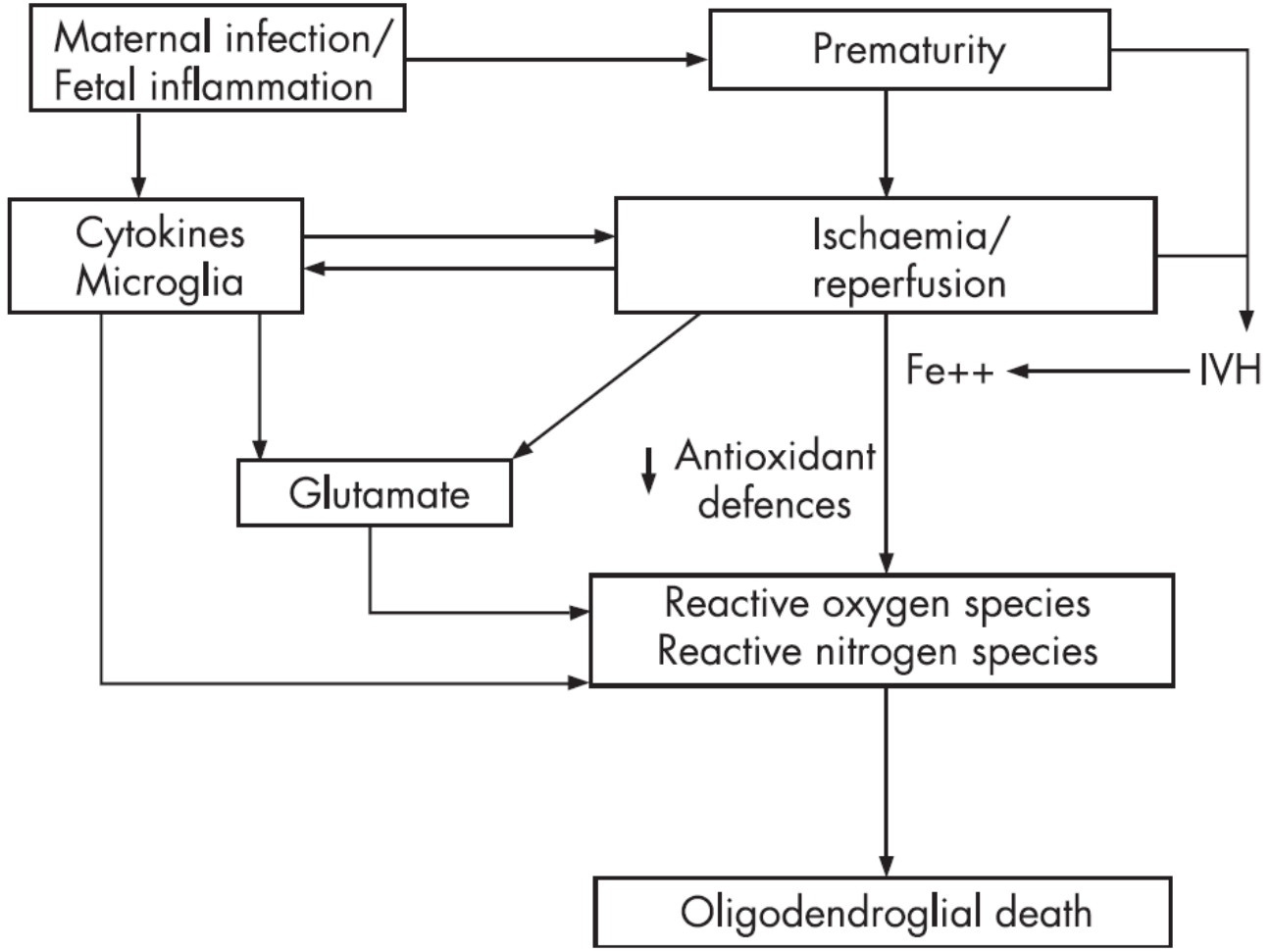




Pathogenesis of cerebral white matter injury of prematurity

O Khwaja, J J Volpe





frontiers in Pediatrics

METHODS
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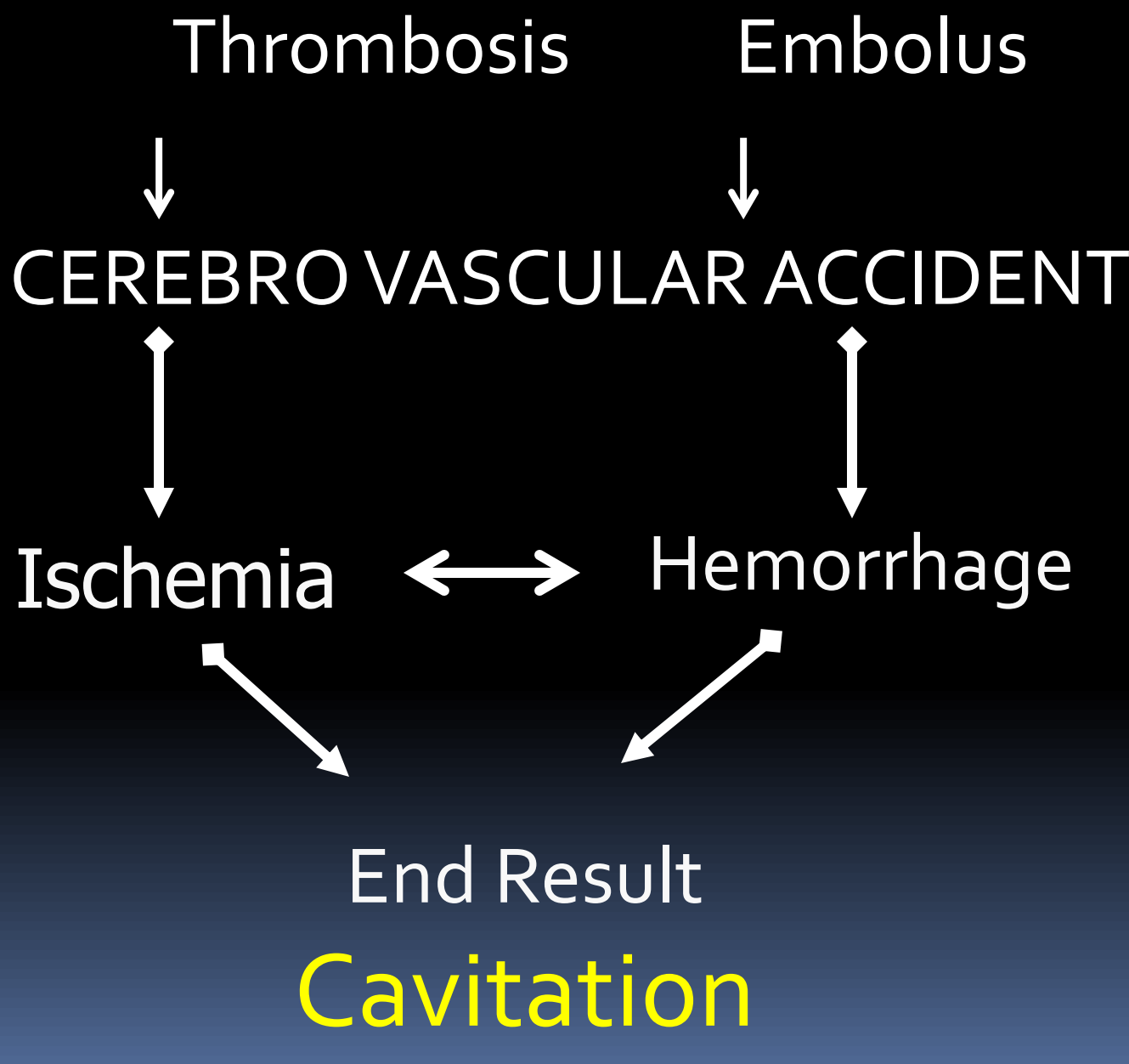
Differentiation of PVHI and Acute White Matter Injury (PVL)

- Non-cystic PVL (non-hemorrhagic WMI) is typically bilateral with ill-defined contour
- PVHI is usually "fan-shaped", unilateral and associated with an ipsilateral IVH

- The key distinction feature is evidence of GMH-IVH:
  - PVHI: blood at the side of the WM lesion, the lesion is asymmetric or unilateral
  - PVL: bilateral WM echogenicities w/o blood in the germinal matrix or ventricles

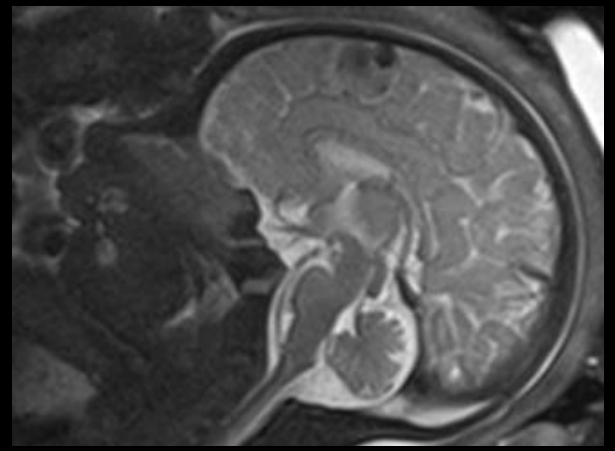
ISCHEMIC FETAL STROKE

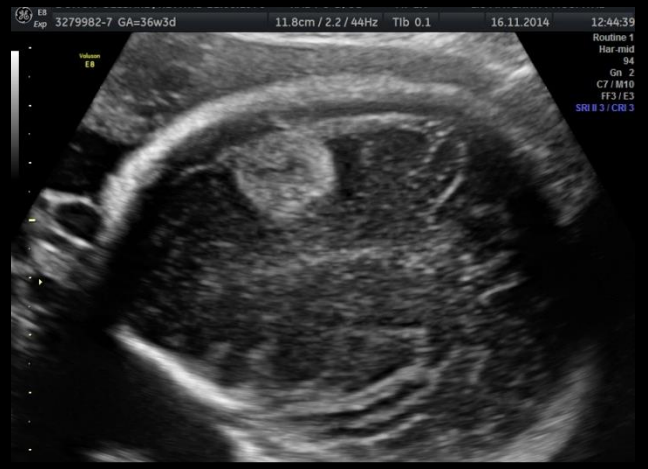

Thrombosis
Embolus
CEREBRO VASCULAR ACCIDENT
Ischemia
Hemorrhage
End Result
Cavitation

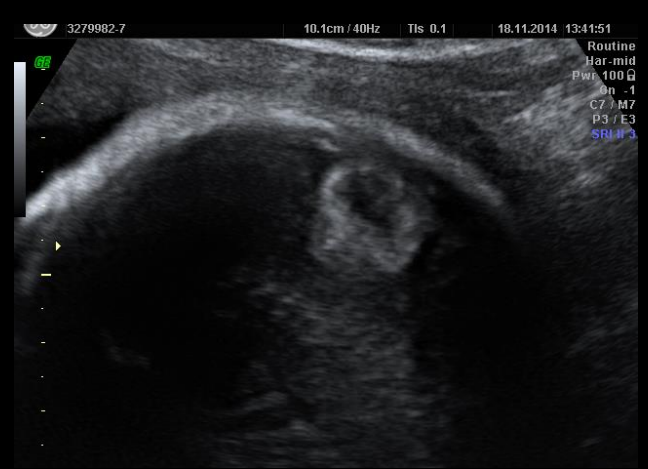
Ischemic Perinatal Stroke

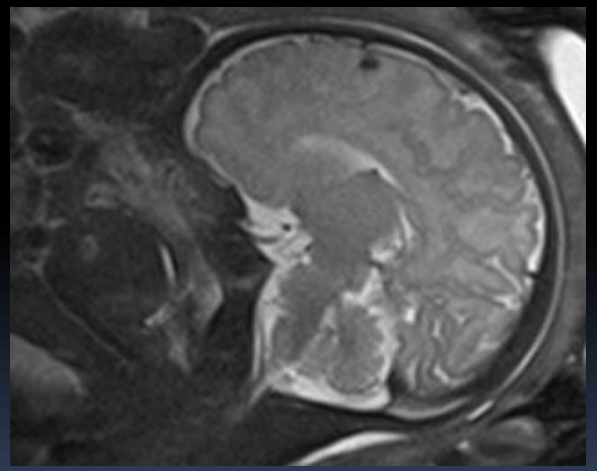
- Fetal ischemic stroke: diagnosed before birth by fetal imaging or in stillbirths on the basis of neuropathological examination (arterial)
- Neonatal ischemic stroke: diagnosed after birth and/or before the 28th postnatal day (including in preterm infants)
- - Presumed perinatal ischemic stroke: diagnosed in infants over 28 days of age in whom it is presumed (but not certain) that the ischemic event occurred sometime between the 20th week of fetal life through the 28th postnatal day

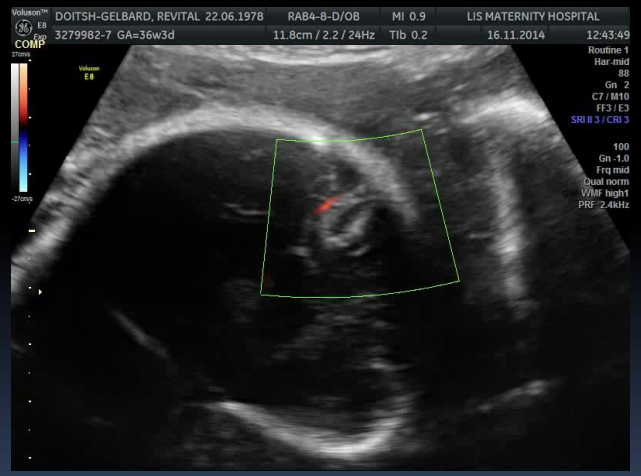
Cortical arterial stroke

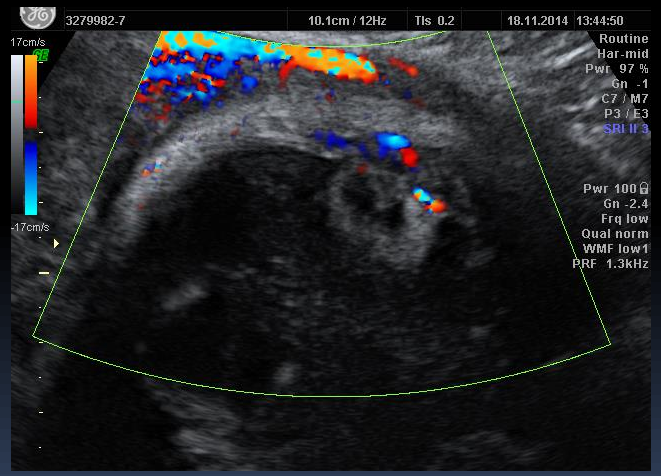












36w0d 36w3d 36w5d

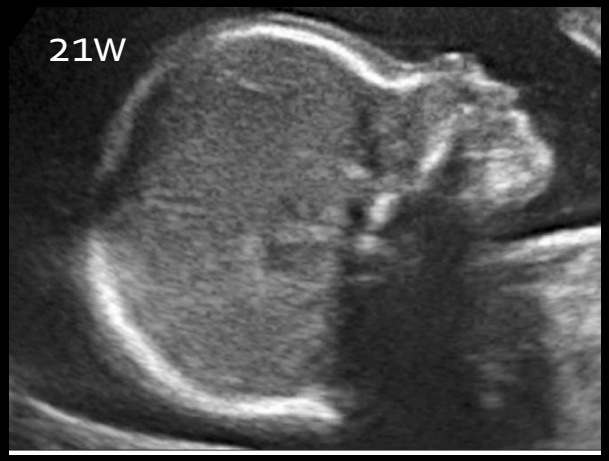
MRI Courtesy Prof. Ben Sira

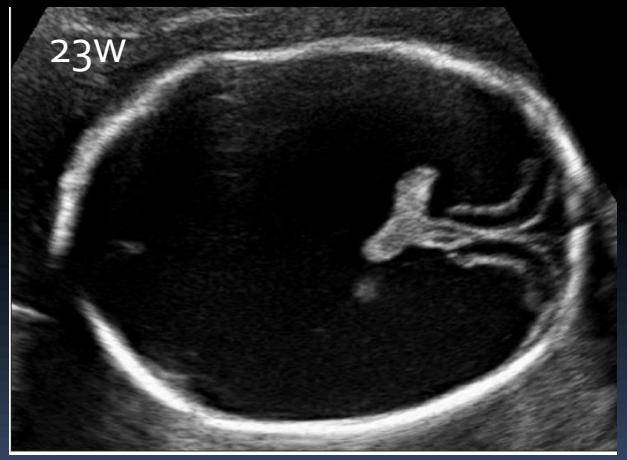
Arterial Ischemic Fetal Stroke Sequelae

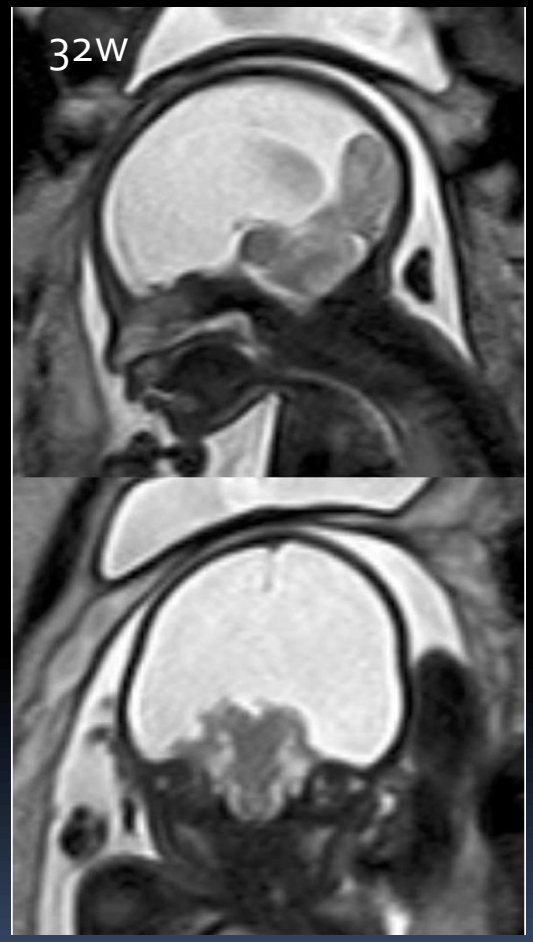
- Porencephaly, cortical-arterial type
- Hydranencephaly
- Schizencephaly

Govaert P, Prenatal Stroke. 2009

Hydranencephaly

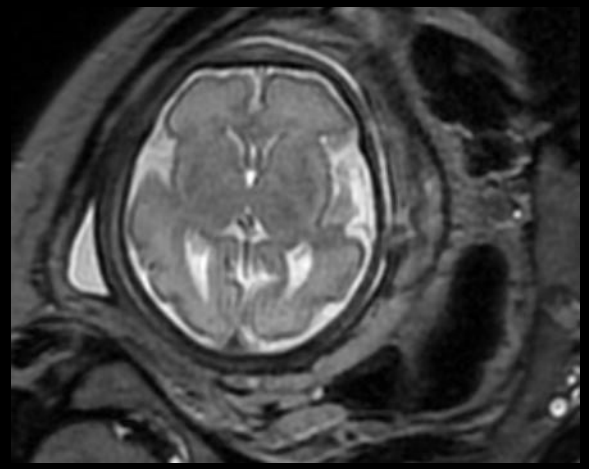


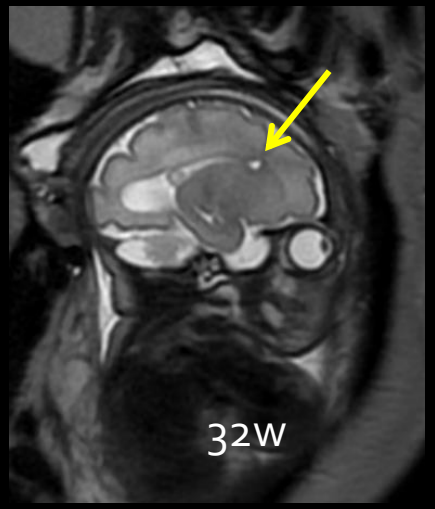


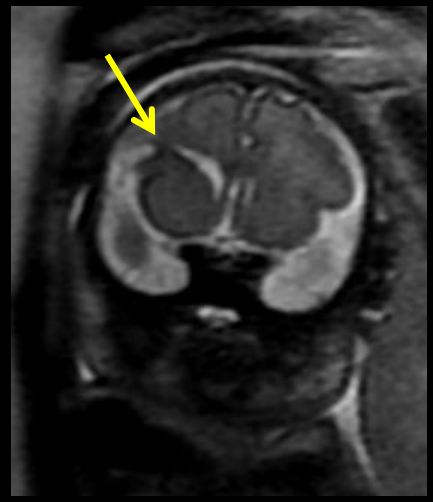


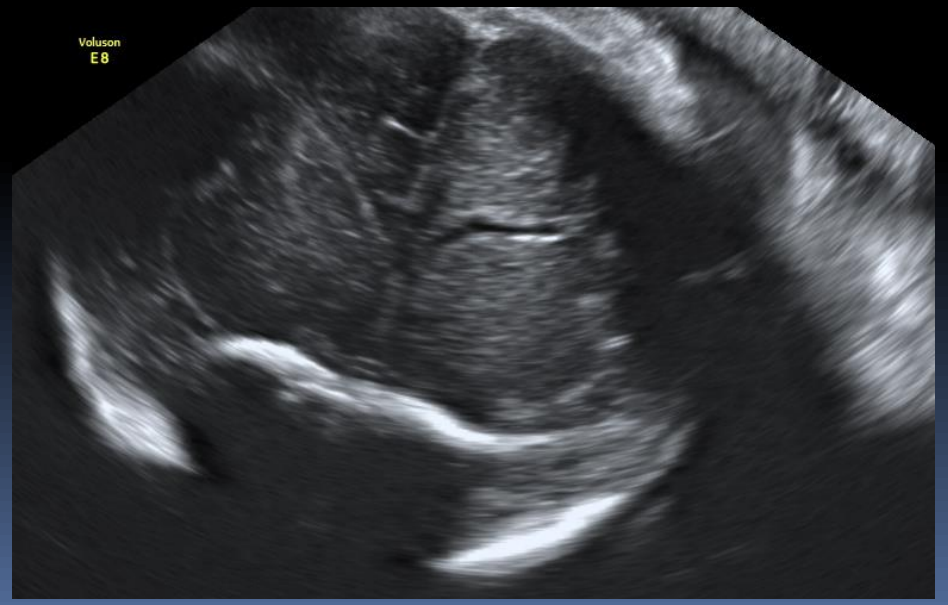
Sepulveda 2012

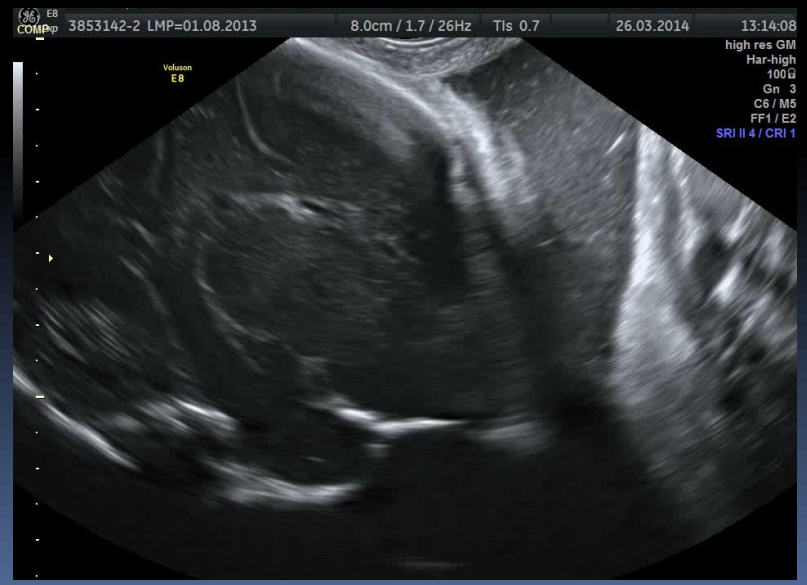
Schizencephaly





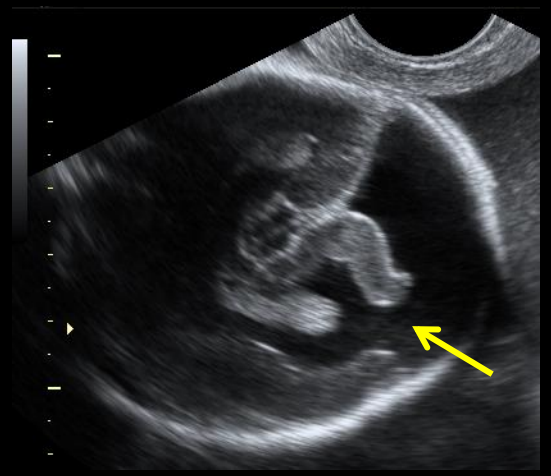




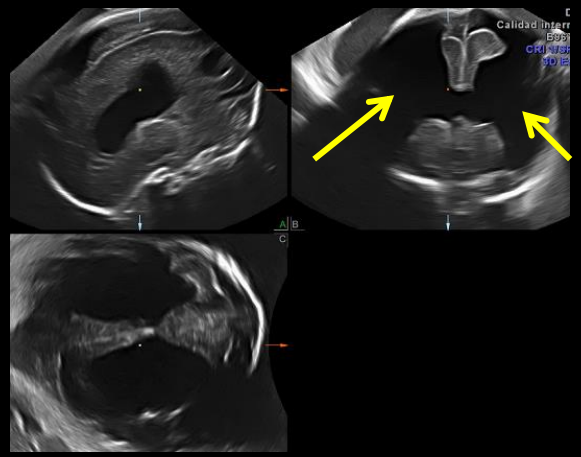


34w

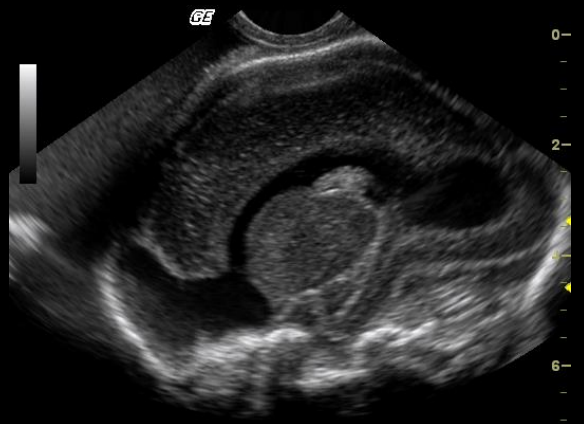
Schizencephaly



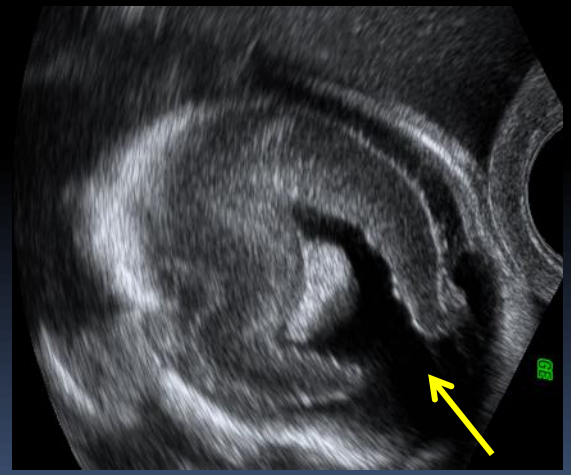
23w



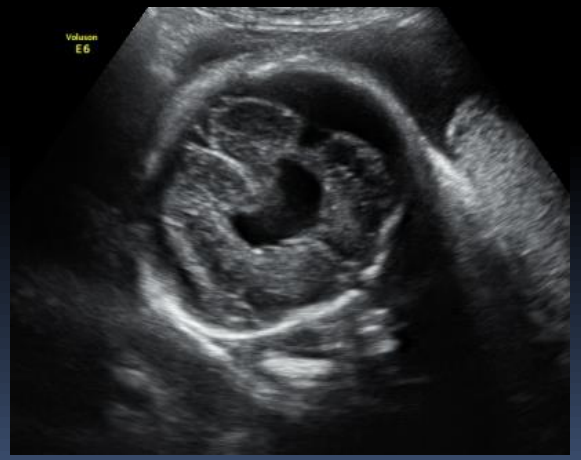
28w



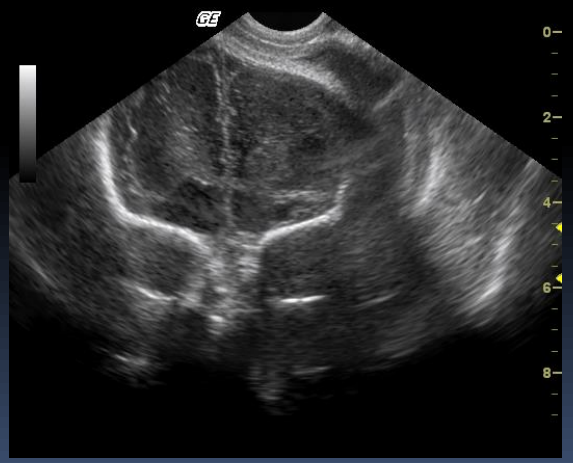
26w



Unilateral Occipital



Bilateral Parietal



Bilateral Frontal

FETAL IVH – PVHI

NEW CLASSIFICATION AND PARAMETERS-US AND MRI

GMH – IVH	Germinal matrix hemorrhage (Caudothalamic/subependymal), Choroid plexus or intraventricular hemorrhage
PVHI	Yes/no
Location of PVHI	**Anterior Frontal** (anterior to the foramen of Monro) **Posterior Frontal** (posterior to the foramen of Monro) With/without involvement of the pre/postcentral gyrus (sensory- motor region) **Parietal** With/without involvement of the pre/postcentral gyrus (sensory-motor region) **Temporal** **Occipital** With/without involvement of the primary visual area)

Basal Ganglia involvement Posterior fossa involvement	Yes/no, Unilateral/bilateral Yes/no
Laterality Ventriculomegaly	Uni/bilateral Yes/no, Unilateral/bilateral, Measurements (mm): Rt: ___ ; Lt: ___
Sub-arachnoid involvement Midline shift	Yes/no (specify) Yes/no
Other findings Evolution of the findings Underlying disorders	Specify table/ progressive/ regressing

Classification

ACUTE/SUBACUTE IVH

Type 1	GMH, Normal lateral ventricle width
Type 2	GMH- IVH[10], Normal lateral ventricle width
Type 3	GMH- IVH[10], with mild ventriculomegaly (<15 mm)
Type 4	GMH- IVH[10], with severe ventriculomegaly (≥ 15 mm)
Type 5	IVH with subarachnoid involvement
Type 6	IVH with posterior fossa involvement

Classification

PARENCHYMAL HEMORRHAGE

Type 1	Focal parenchymal involvement, one territory, unilateral
1 a	Anterior Frontal
1 b	Posterior Frontal With/without involvement of the sensory-motor region
1 c	Parietal With/without involvement of the sensory-motor region
1 d	Temporal
1 e	Occipital With/without involvement of the primary visual area
1 f	Basal Ganglia (other than the caudate nucleus) With/without internal capsula involvement
1 g	Cerebellum

Type 2	Extensive parenchymal involvement
2 a	Unilateral parenchymal involvement of more than 1 territory (specify territories, specify if there is a midline shift)
2 b	Bilateral parenchymal involvement (specify territories, specify if there is a midline shift)

Classification

LATE IVH SEQUELAE / PRESUMED PVHI

Type 1	Ependymal/Subependymal/caudothalamic hemosiderin/ blood products with normal ventricular size
Type 2	Intraparenchymal hemosiderin/ blood products, without parenchymal loss
Type 3	Severe ventriculomegaly (>15 mm) /susp. Hydrocephalus, in the presence of blood products
Type 4	Ventricular border irregularity/ change in ventricular morphology (note location and laterality)
Type 5	Periventricular porencephalic cysts (note location, extension, laterality)