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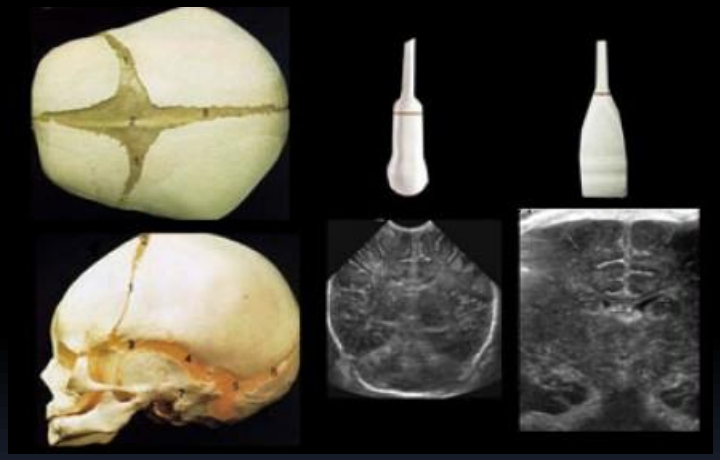
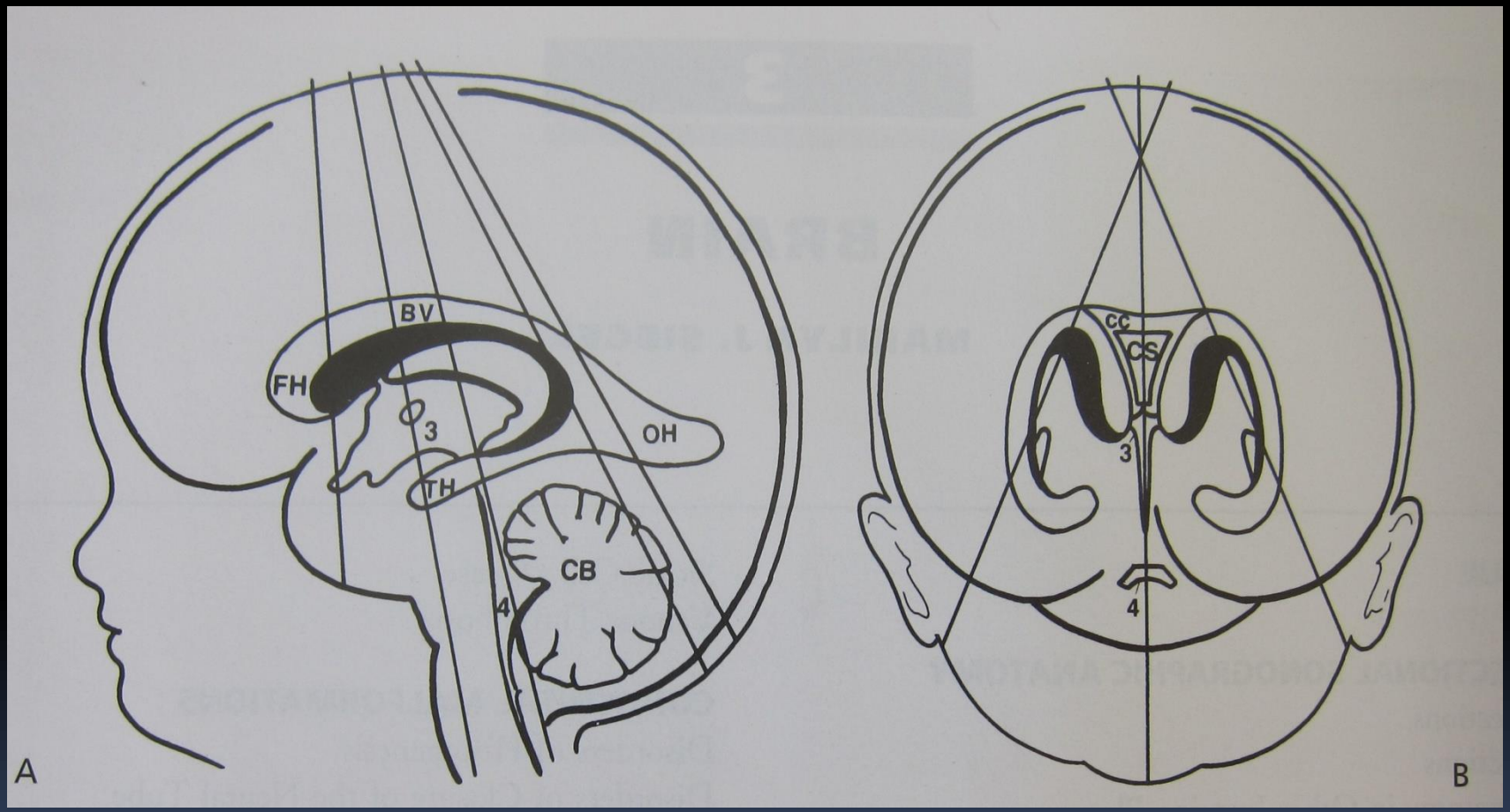
Μιχάλης Παπαδόγιαννης
Παιδοακτινολόγος
Διδάκτωρ Πανεπιστημίου Αθηνών



- Ultrasound is a fast and bedside examination which makes it ideal for premature infants.
  Try to get all the information you can.
  Do not limit yourself to only one transducer or only one acustic window (figure).
  Generally the large fontanel is used as acoustic window.
  The small fontanel however is a good window to the occipital lobes.
  This can be usefull in patients with borderline hyperechogenicity in these areas.

CROSS-SECTIONAL SONOGRAPHIC ANATOMY

- Coronal Sections
- Sagittal Sections
- Normal Anatomy in Other Imaging Planes
- Normal Variants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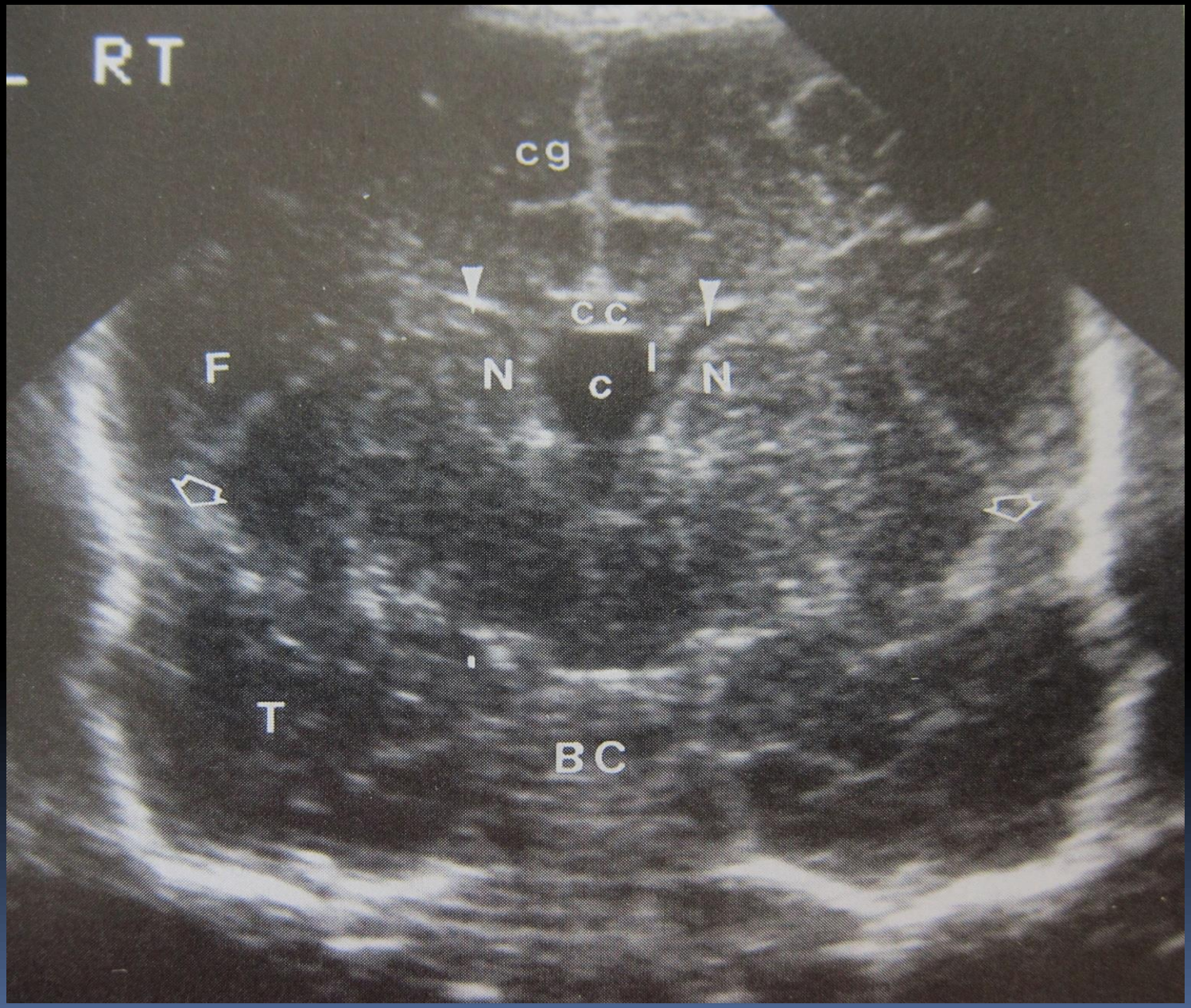
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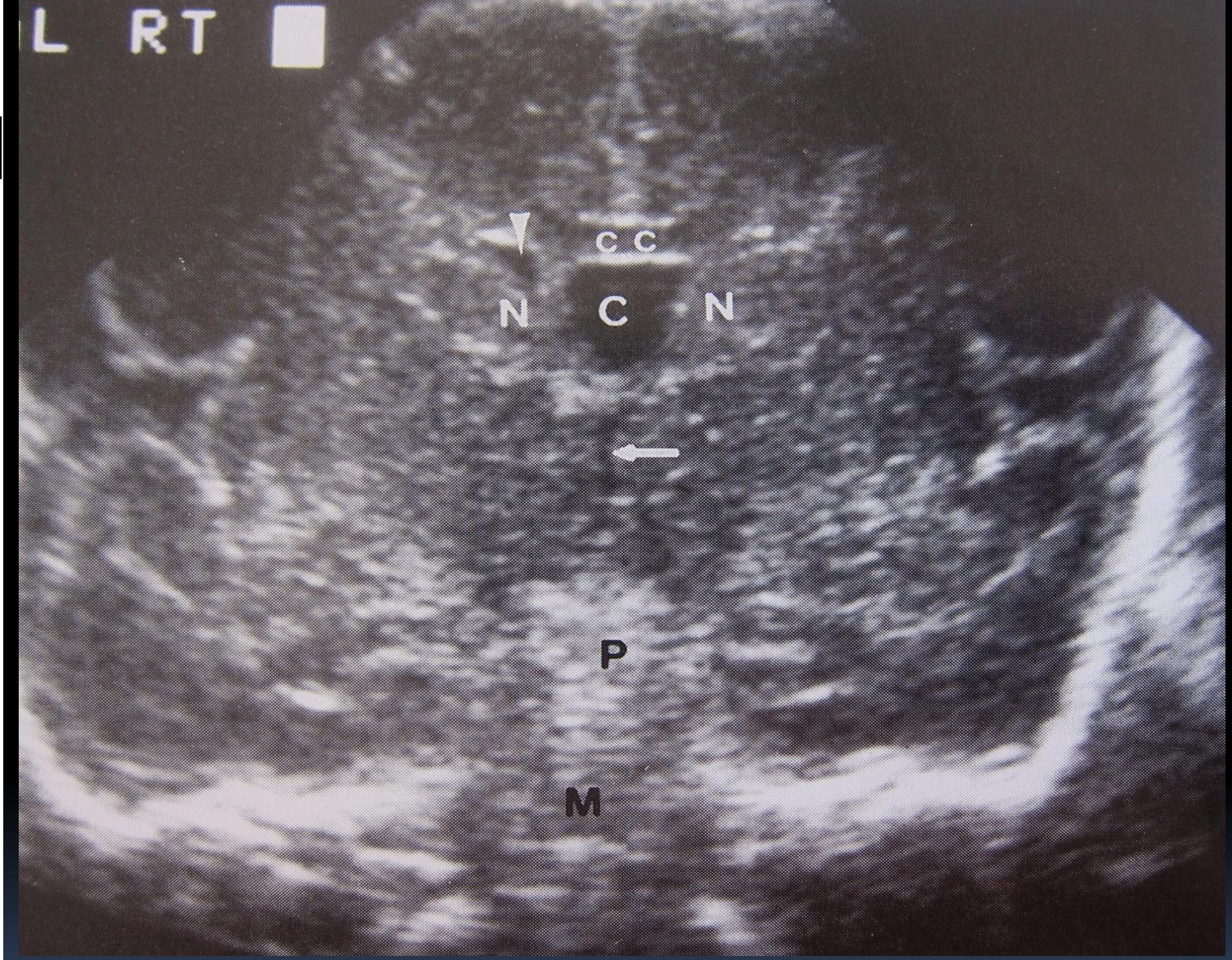
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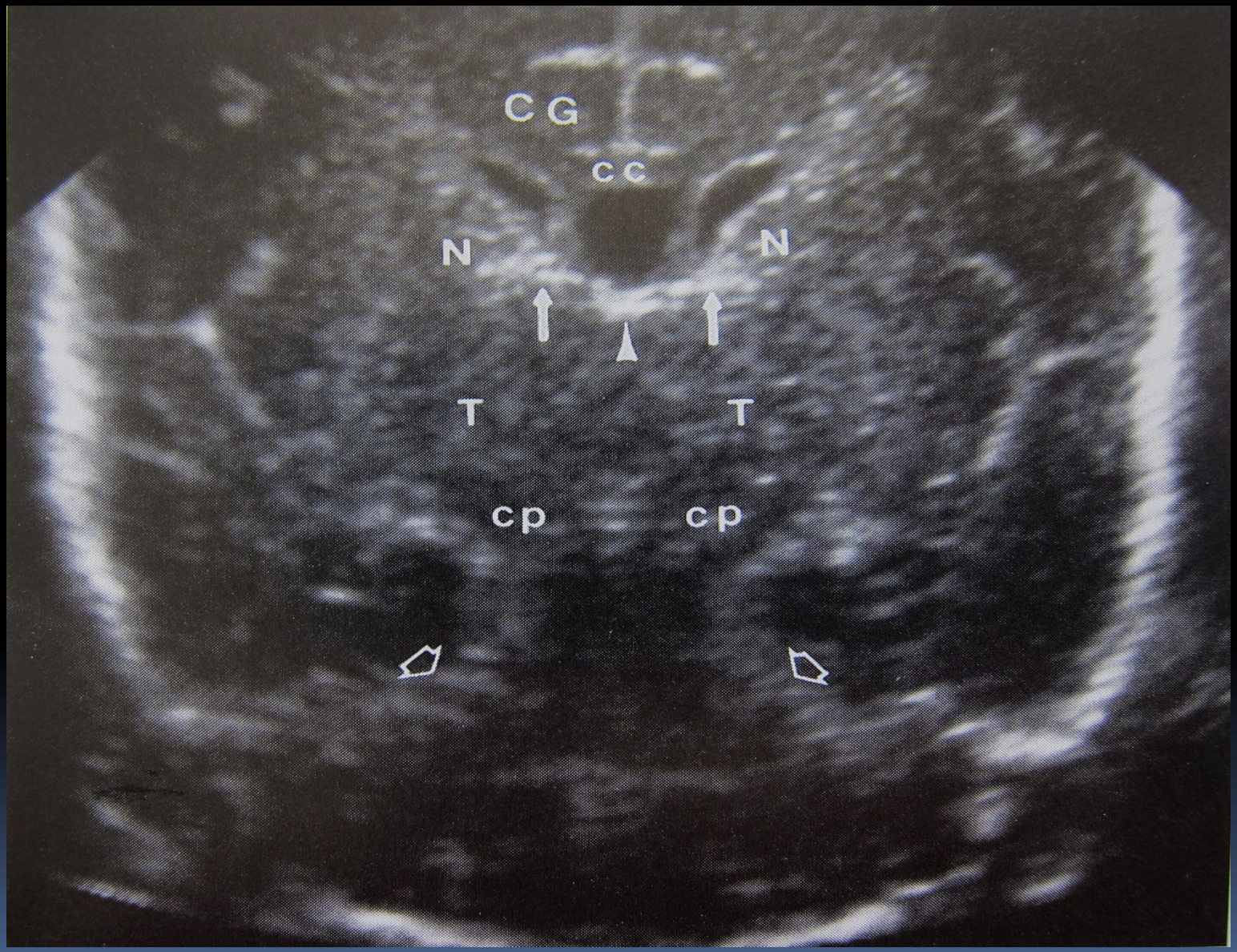
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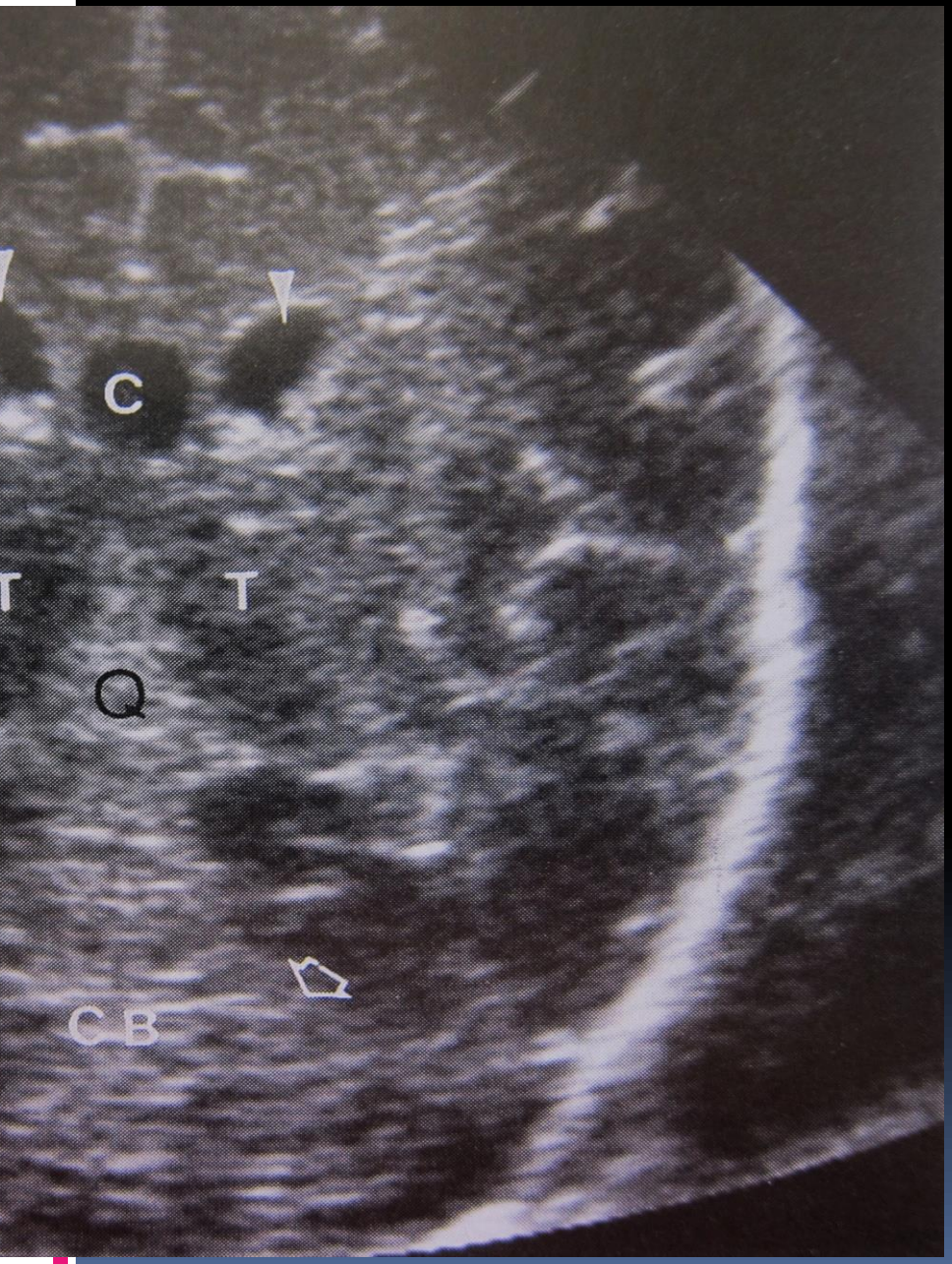
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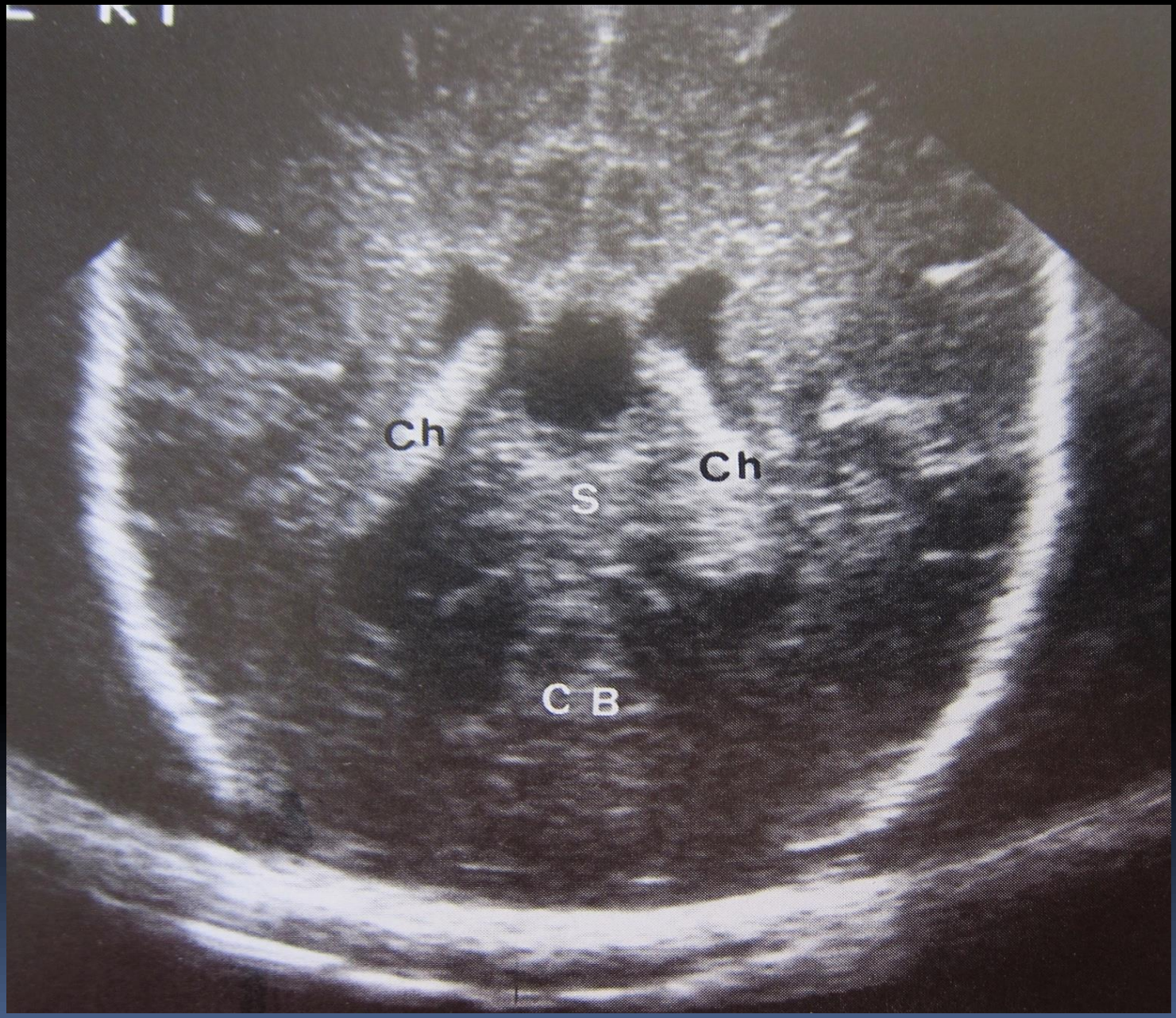
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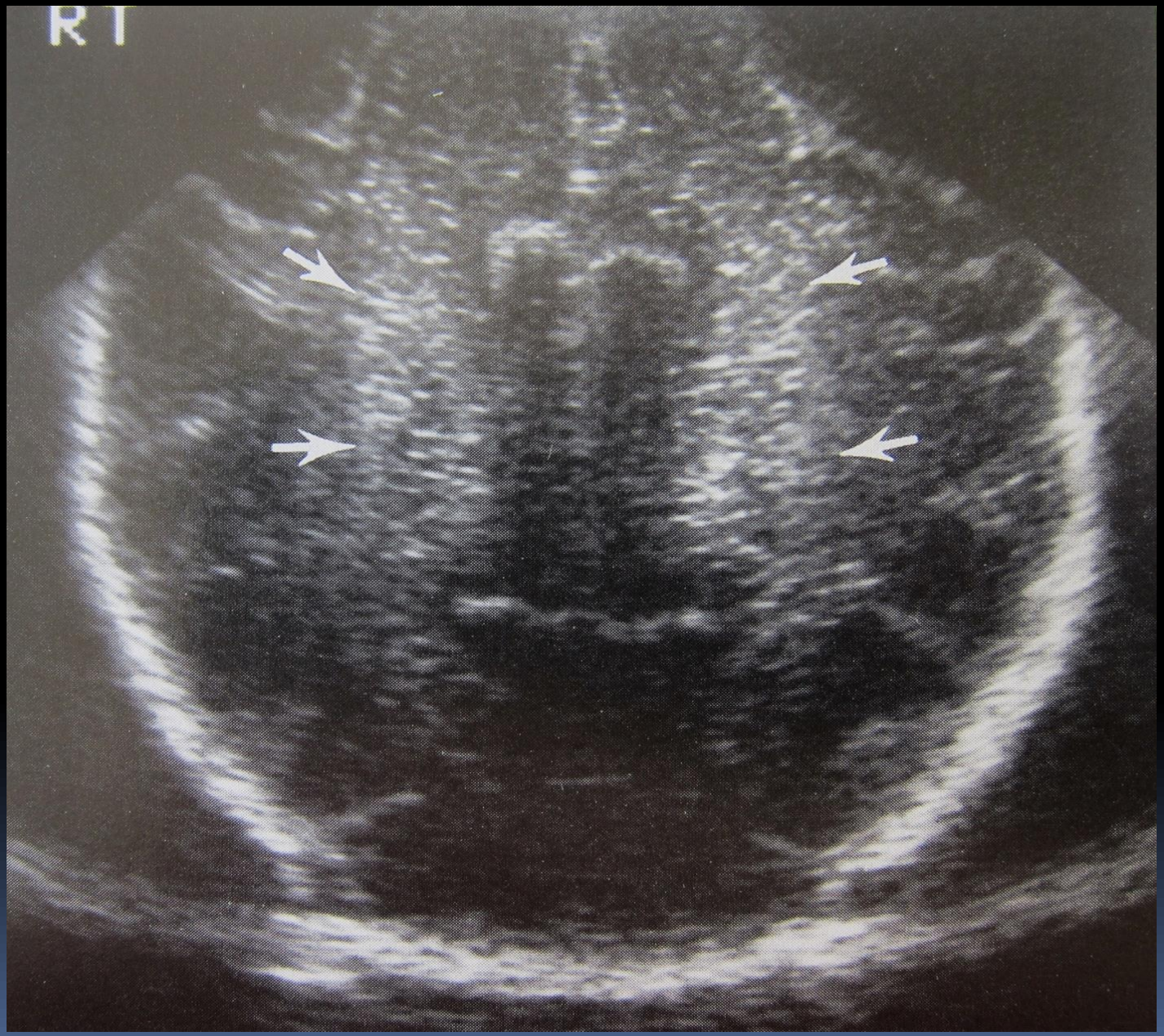
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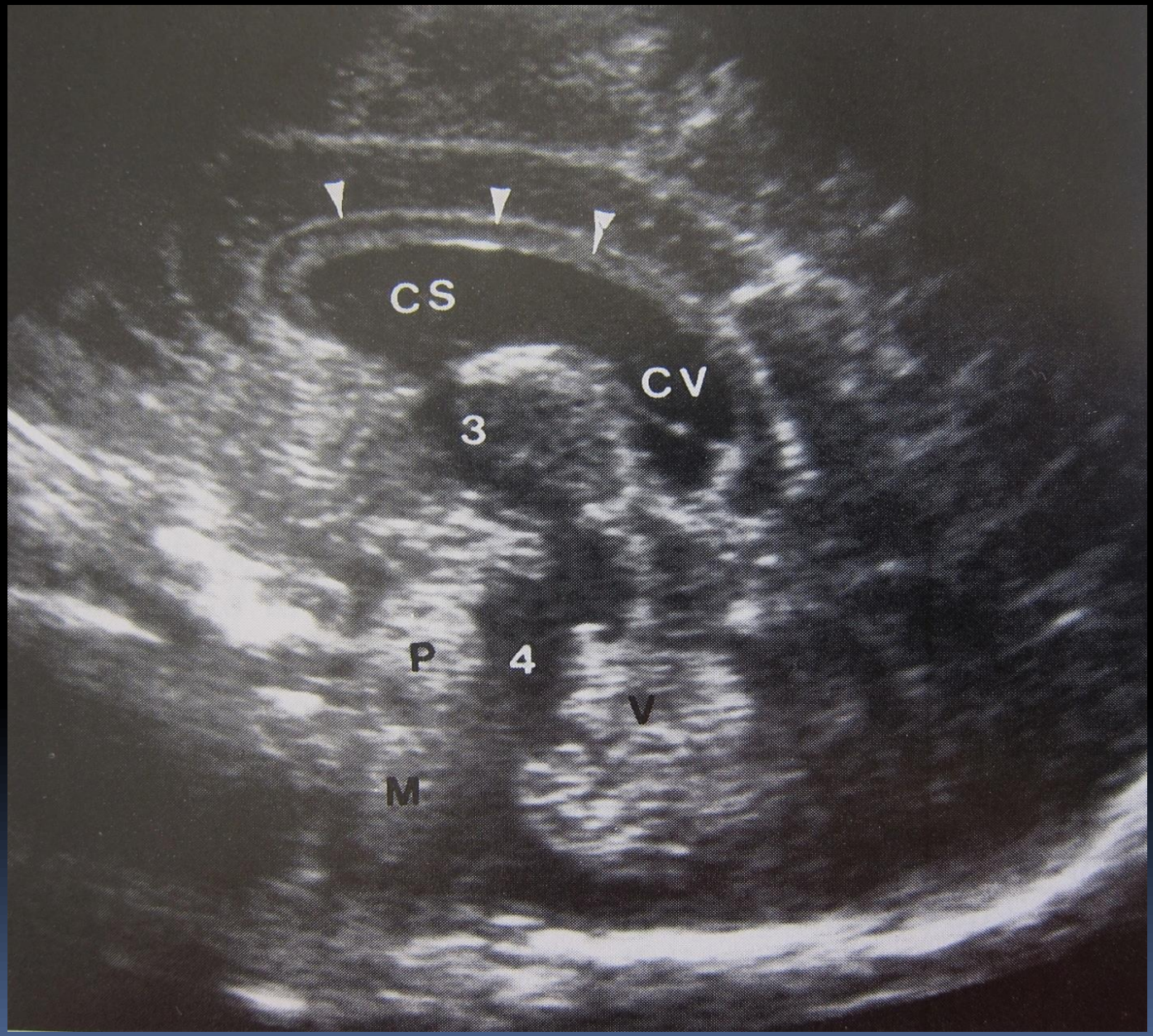
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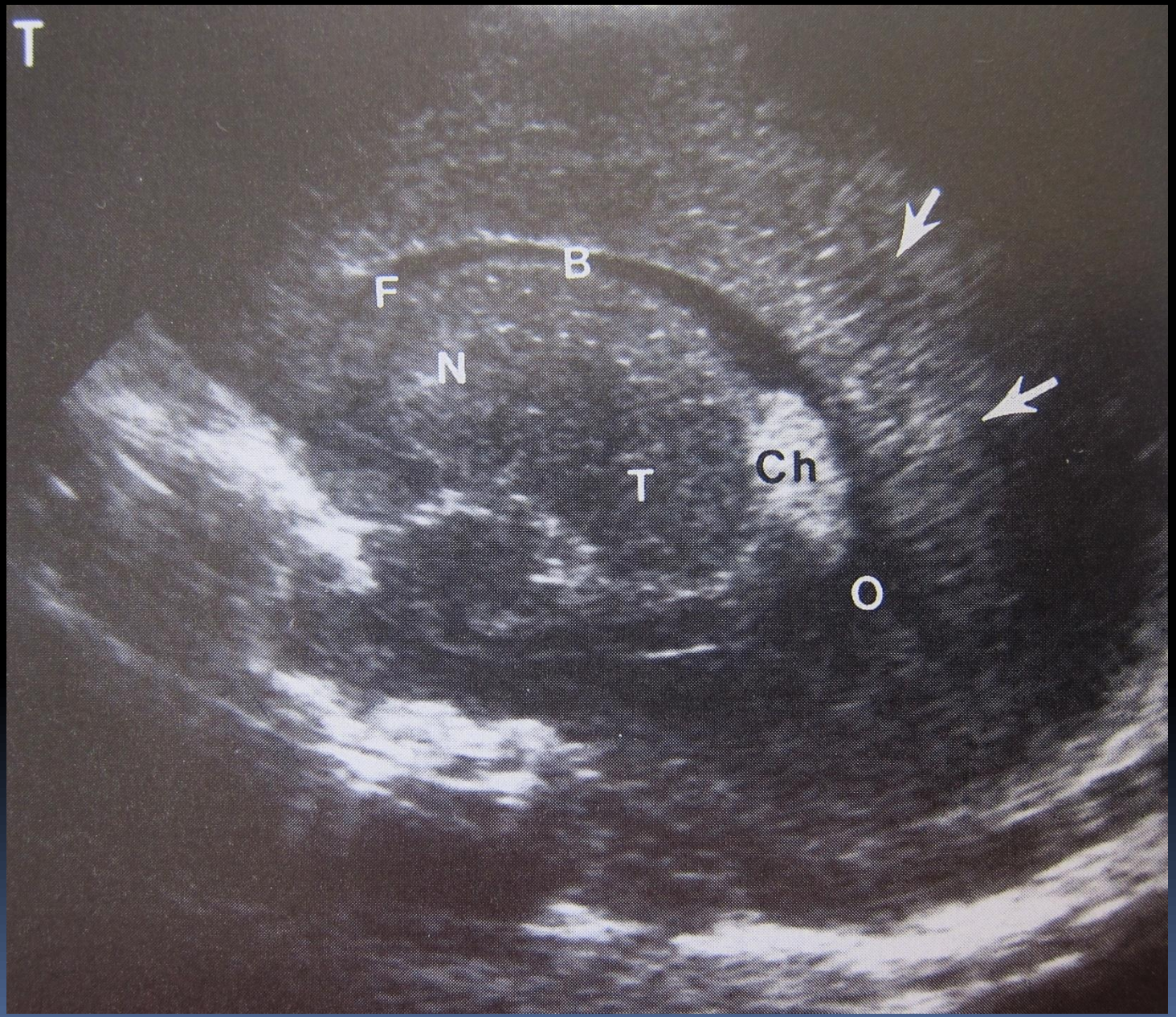
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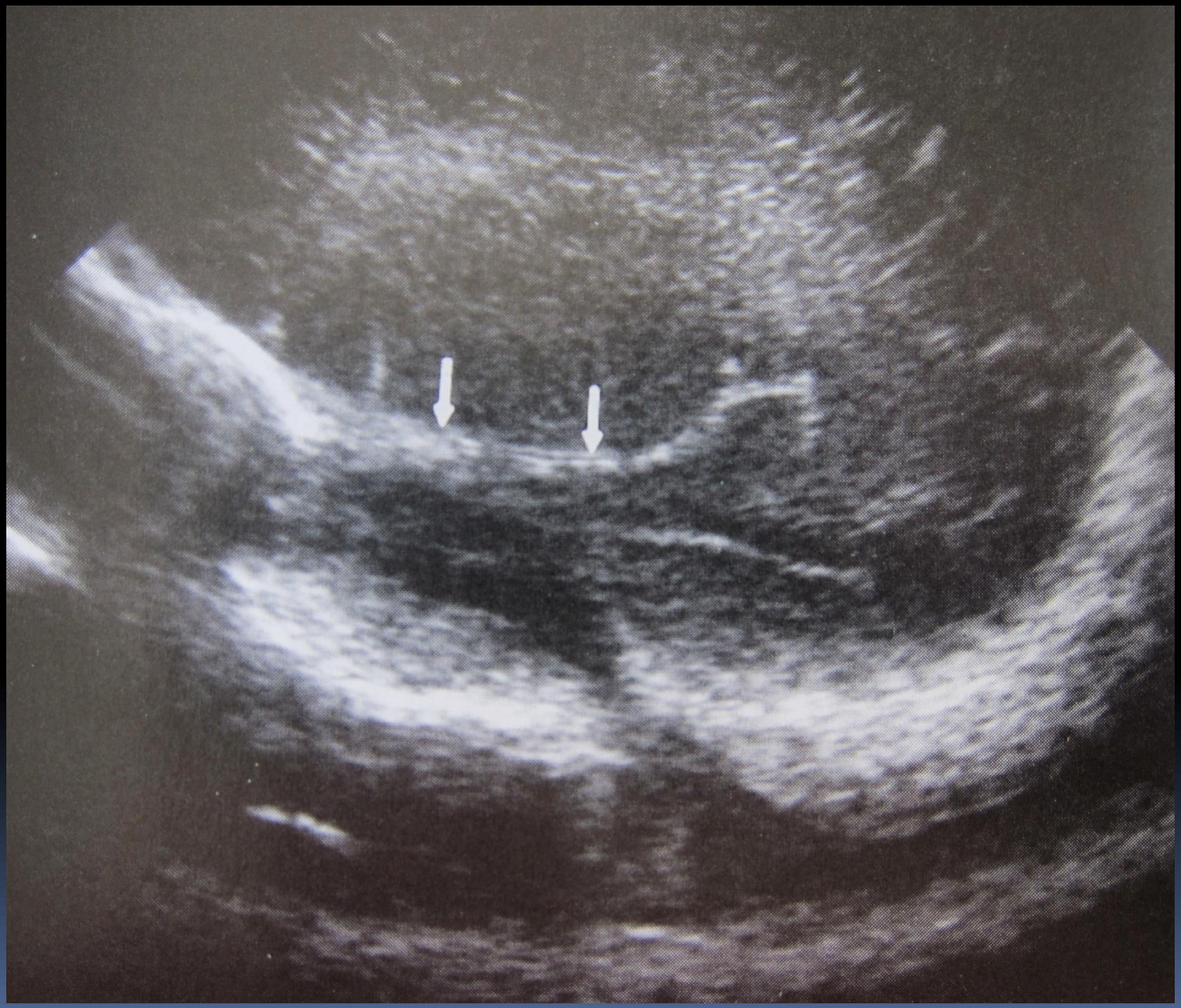

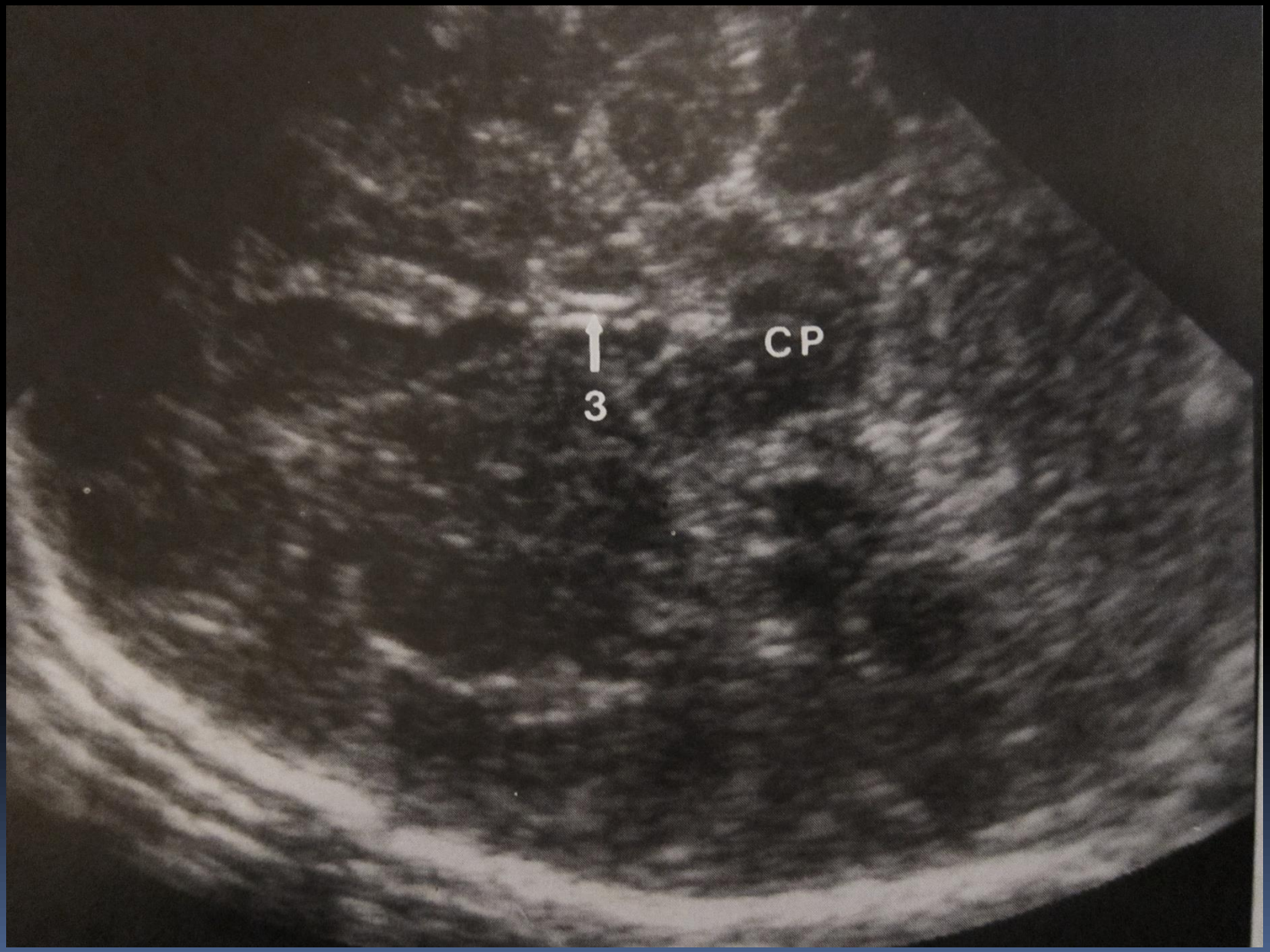
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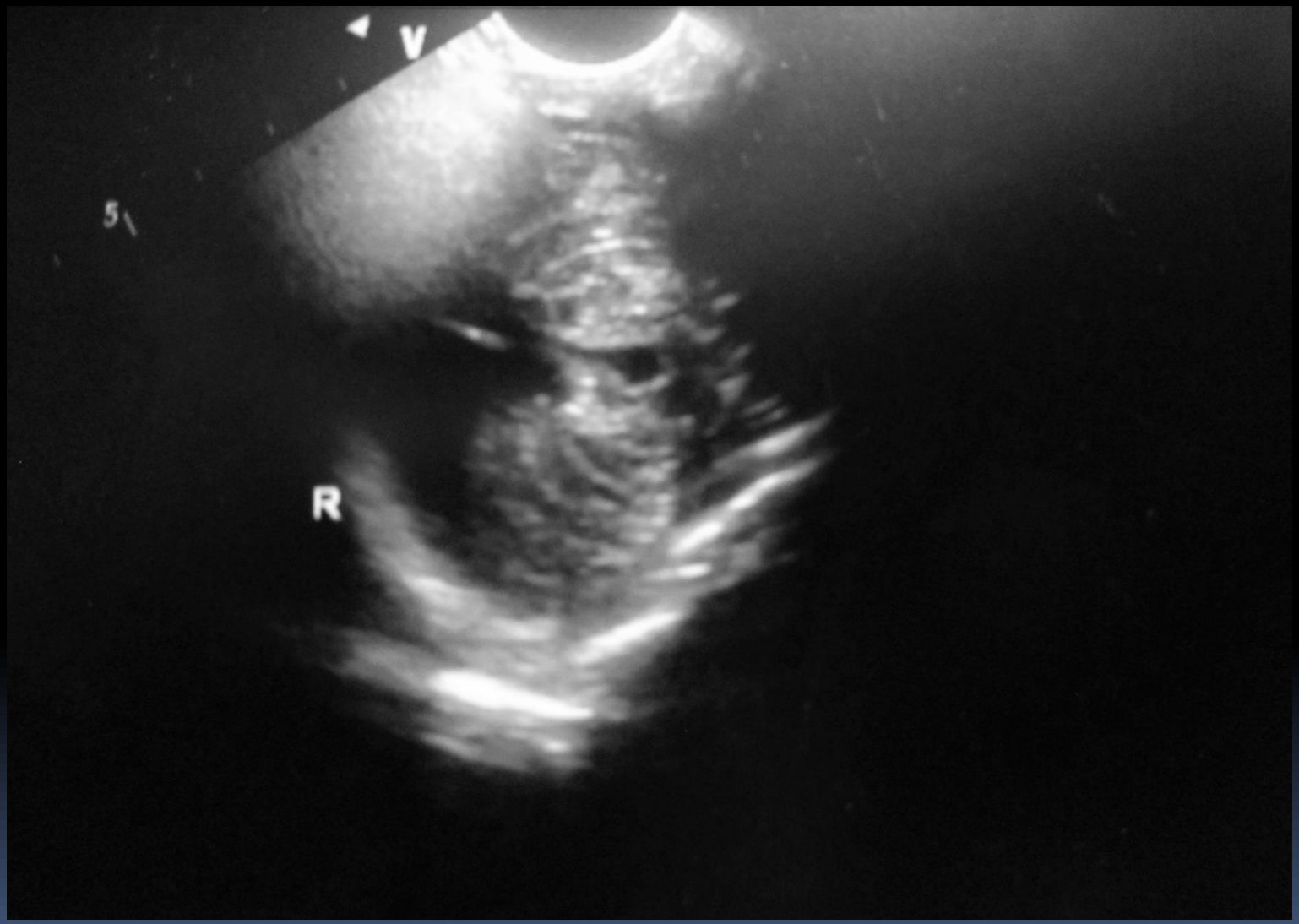
Disadvantages of US are:

- Limited overview in posterior fossa and convexity of the brain
- Absence of US-signs in ischemia in full-terms in first 24 hours
- Difficulty in detecting migration disorders, cortical dysplasia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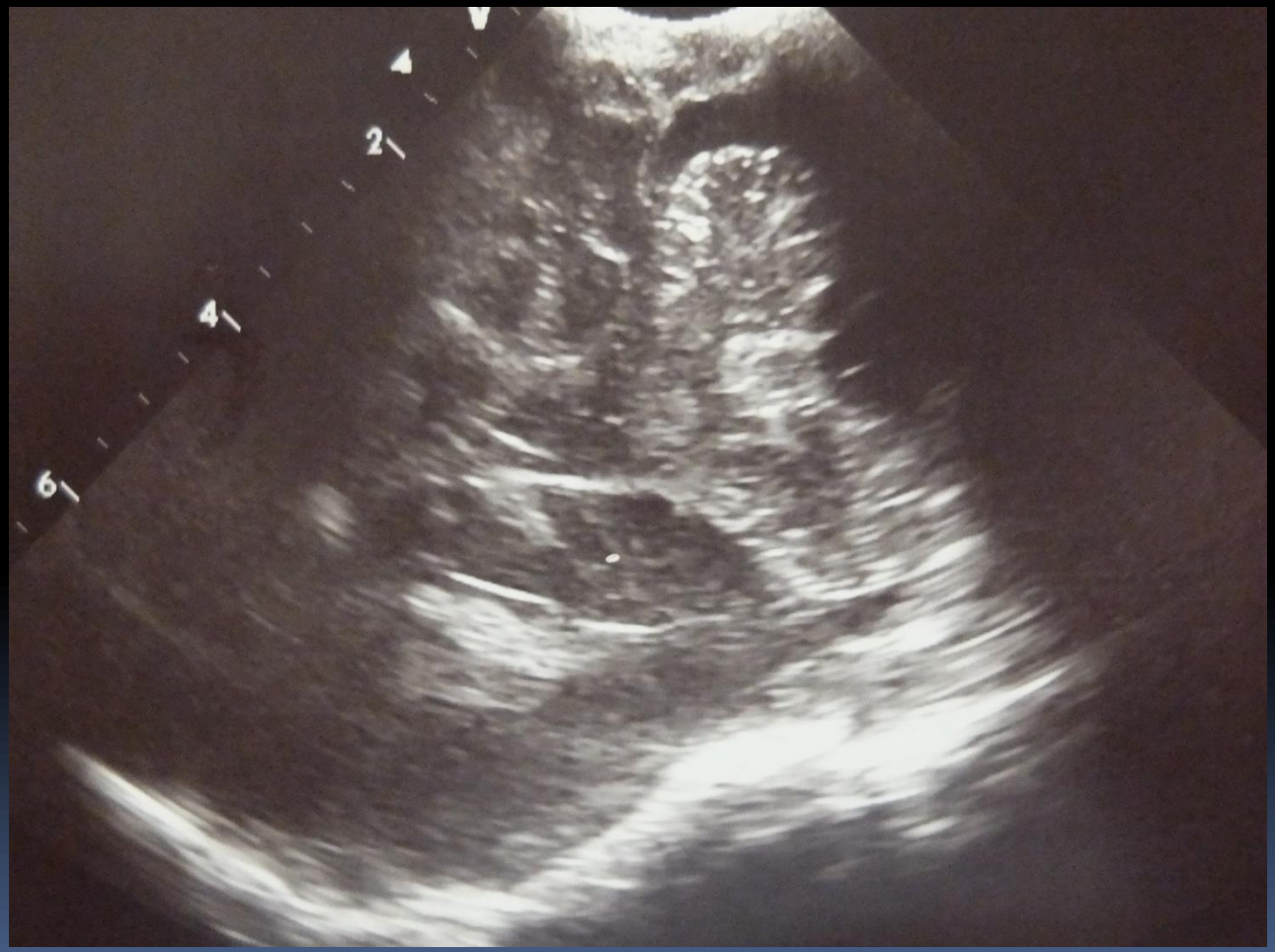
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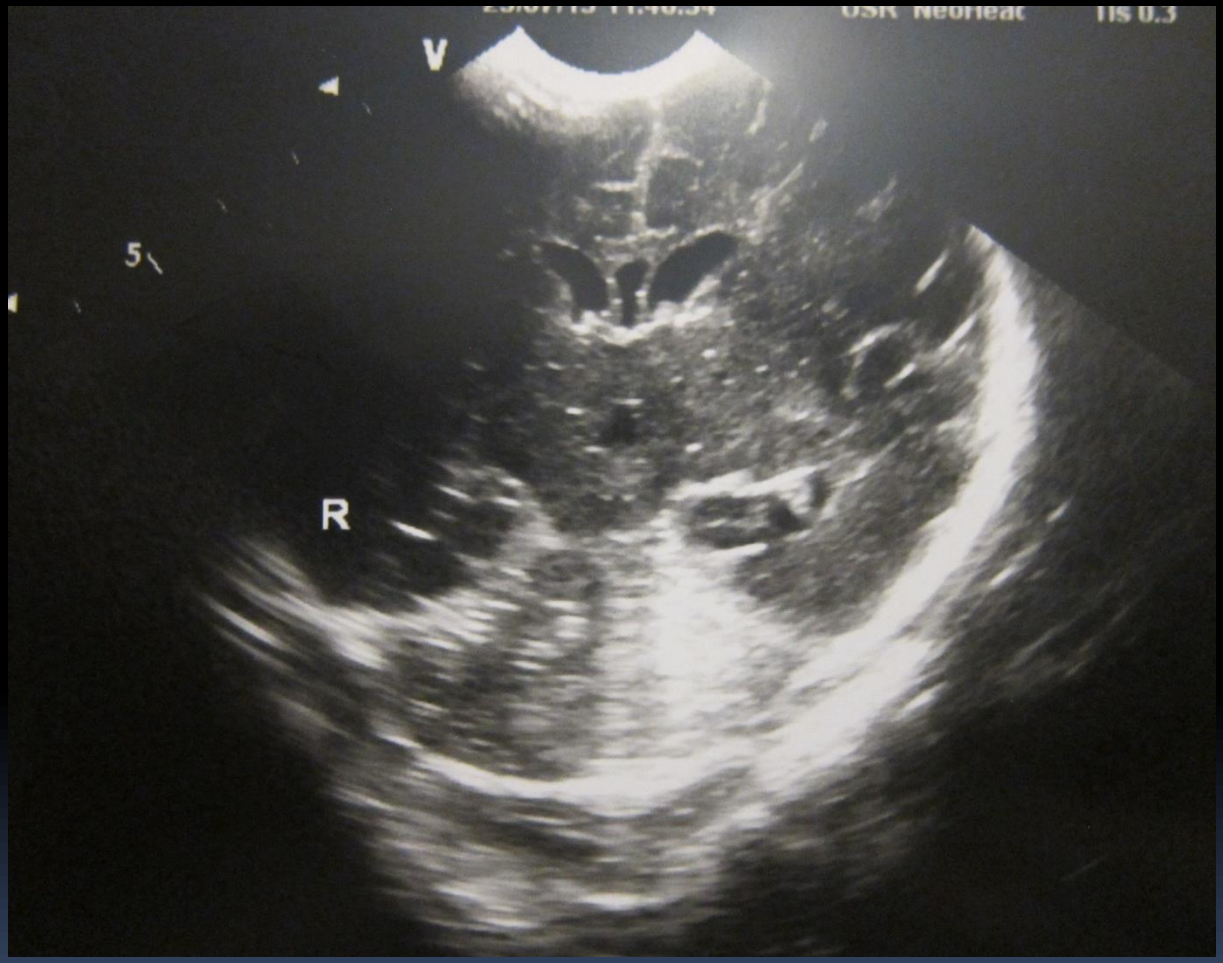
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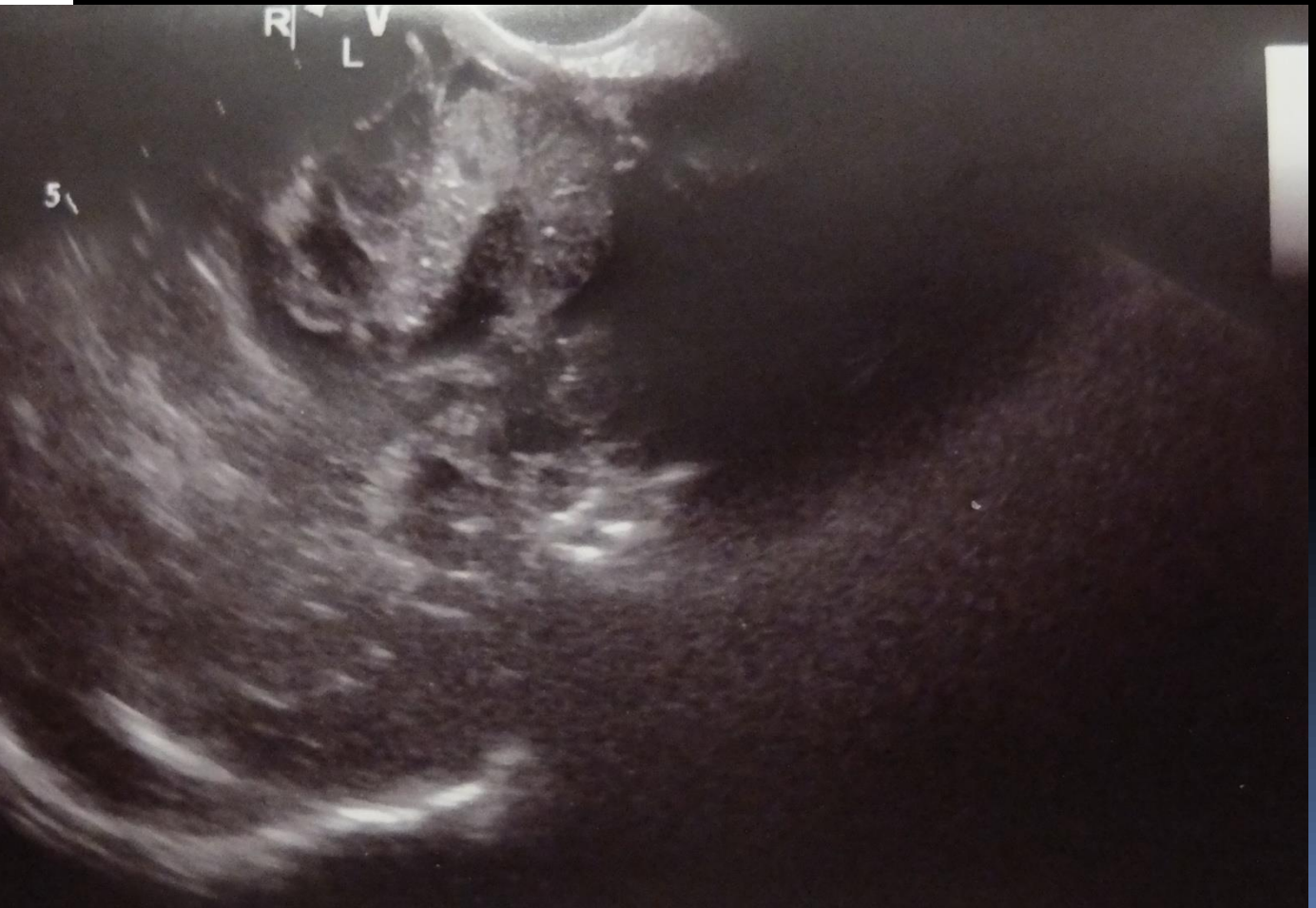
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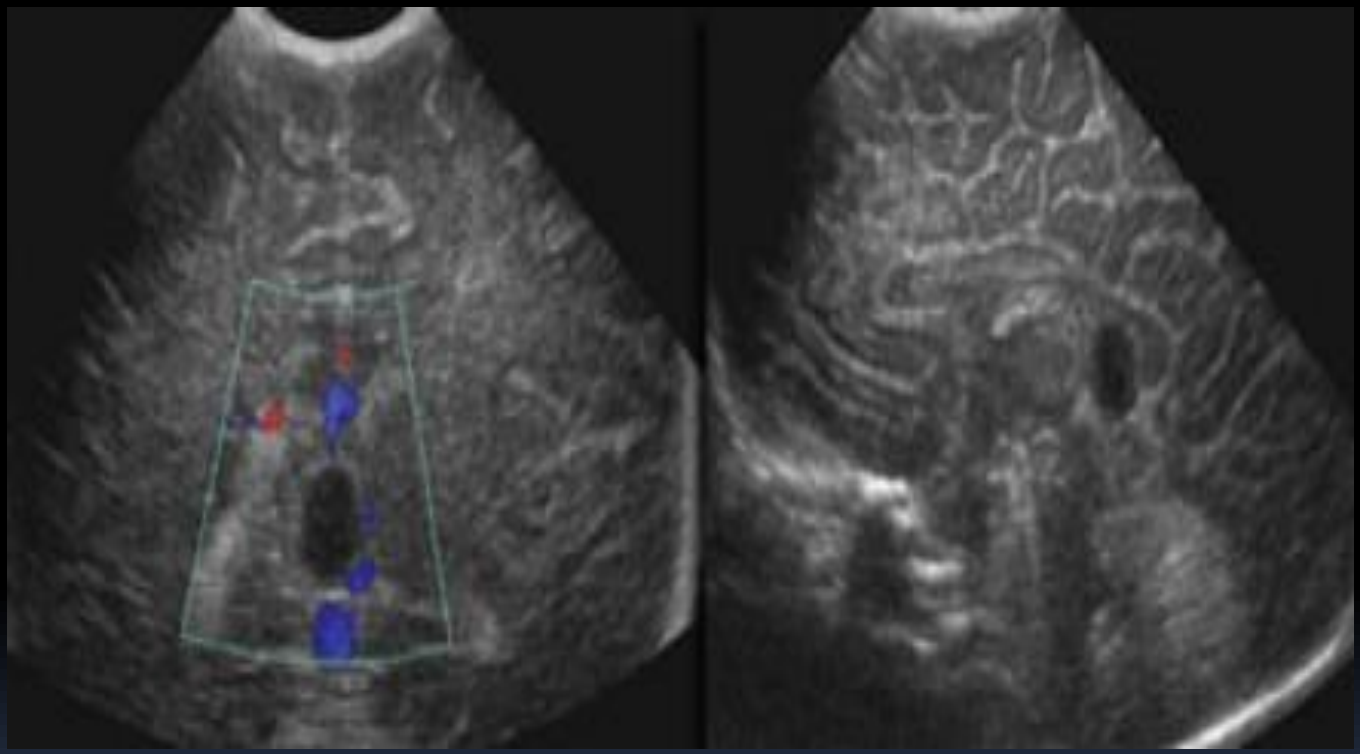
Normal Variants

Cavum septi pellucidi, vergae and velum interpositum
Chorioid plexus cyst
Benign macrocrania
Mineralizing vasculopathy
Pseudocyst

Cavum septi pellucidi, cavum vergae and cavum of the velum interpositum

- Well known variants are the cavum of the septum pellucidum and the cavum vergae.
  The more premature the baby, the more frequently these cavities are present.
  They can persist until adulthood.
- A less frequently seen variant is the cavum of the velum interpositum.
  This presents as a cyst-like structure in the region of the tectum.
  It's shape is compared to a helmet.
  It can easily be confused with a subarachnoid cyst or a cyst of the pineal gland.



Chorioid plexus cyst

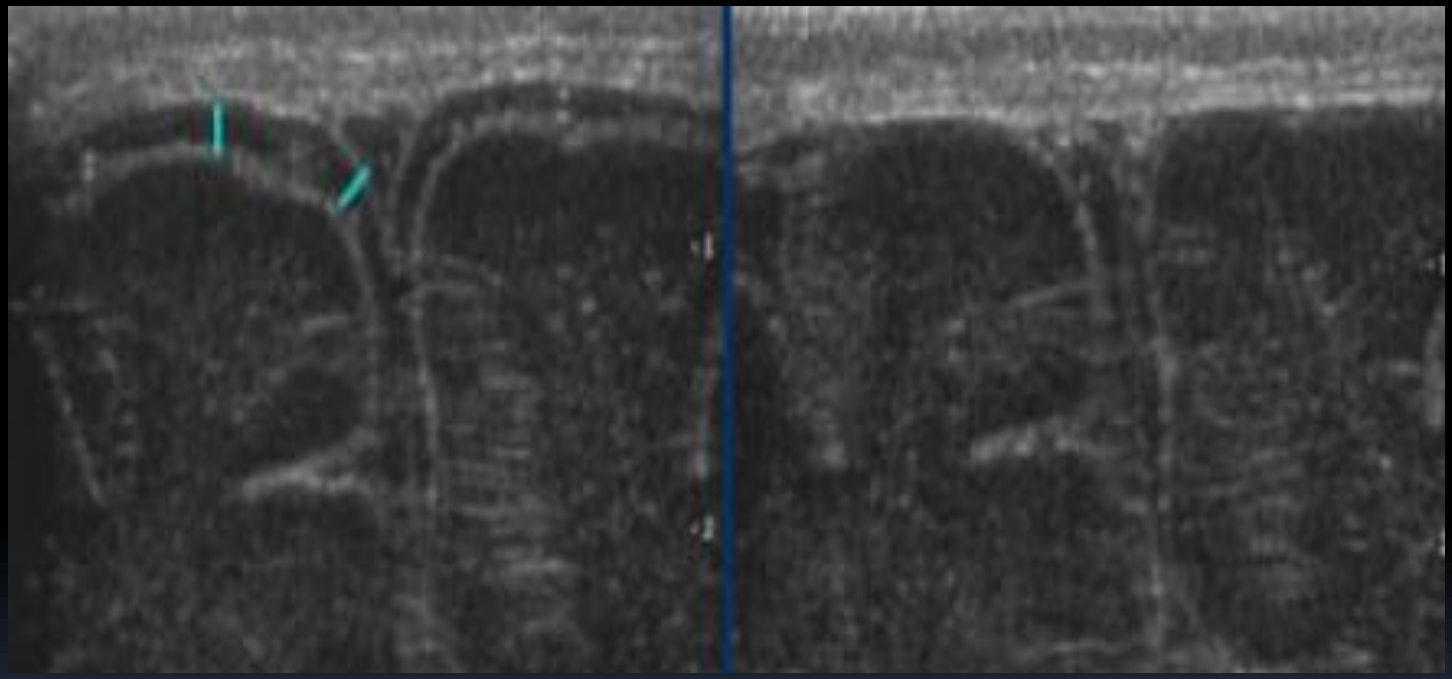
- In postnatal US these cysts of the chorio?d plexus are often incidental findings without clinical consequences.
  Chorio?d plexus cysts (CPC) are however of importance for obstetricians.
  At prenatal US these cysts can be predictive of trisomy 18.
  About half of babies with Trisomy 18 show a CPC on ultrasound, but nearly all of these babies will also have other abnormalities on the ultrasound, especially in the heart, hand, and feet.

- An exeption must be made for cysts that arise close to the foramen of Monro.
  Although these cysts often disappear spontaneously, follow up US is necessary to ensure disappearance.
  Some may produce symptoms of raised intracranial pressure due to obstruction to the cerebrospinal fluid (CSF) flow.

Benign macrocrania

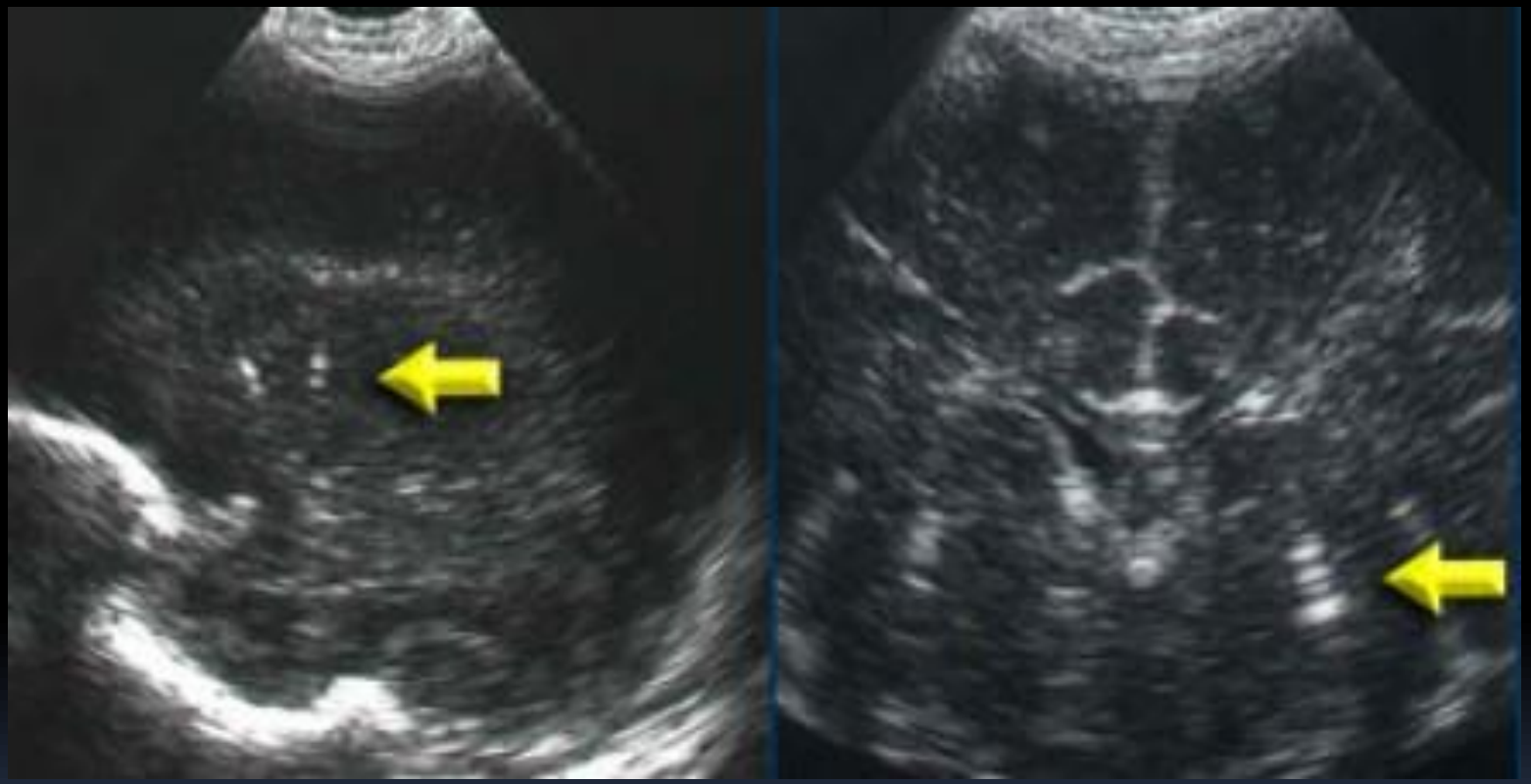
Benign macrocrania is also known as extraventricular obstructive hydrocephalus.
This is seen in children between 6 months and 2 years.
The head circumference is above the 97th percentile.
After the age of 2 years the head size normalizes.
Often the mother or father of the child had large heads at that age.
The cause is not known. Most state that it is a normal condition, although some state that these children have a slight developmental delay.

- When children with a large head are presented for US, examine the superficial subarachnoid space and the ventricles. Normal subarachnoid space measures The ventricles are often slightly enlarged. Thes prominent subarachnoid space and ventricular system in these children should not be interpreted as cerebral atrophia, as in atrophia there is a small head circumpherence.

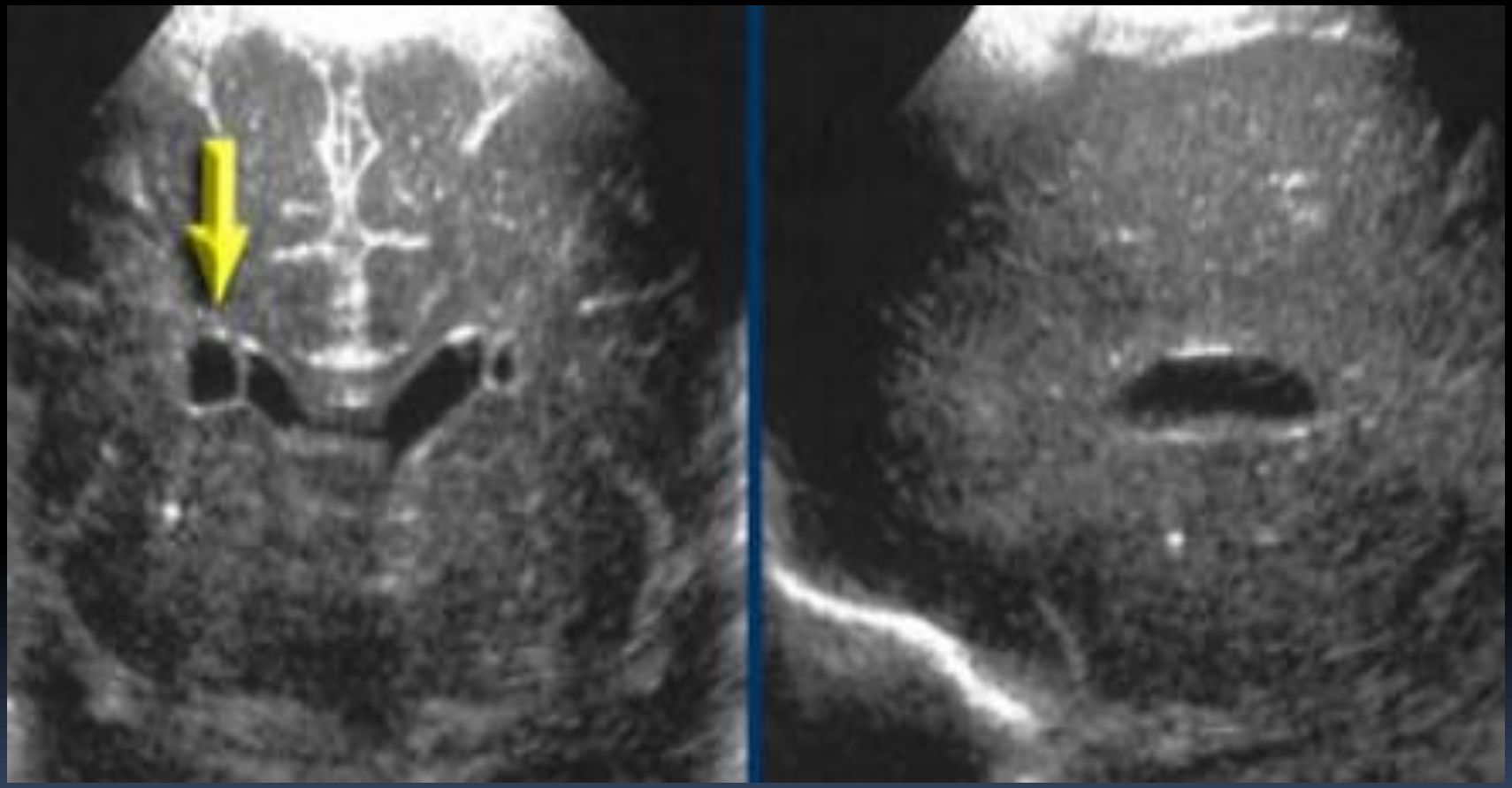


Mineralizing vasculopathy

- Mineralizing vasculopathy can be seen in the thalamostriatal and lenticulostriatal arteries and is caused by calcification of the arterial wall.
  A wide range of perinatal, acquired, and nonspecific clinical conditions may result in this sonographic finding.
  In the Wilhelmina Children's Hospital these children used to be tested for TORCH-infection, but currently the only test that is done is a urine-test for CMV.



- **Germinolytic cysts**
  Are located at the caudothalamic groove.
  They are tear shaped.
  There are no signs of intracerebral hemorrhage and these children have no neurological sequelae.
  The etiology is not known.
- **Pseudocyst**
  These are also called coarctation of the lateral ventricle.
  They are often bilaterally and have no neurological sequelae



Cysts

- If cysts are seen around the lateral ventricles, it is important to determine their position in regard to the upper part of the lateral ventricle (figure).
- 1+2 = Germinolytic cysts and Pseudocysts are below or at the level of the upper part of the lateral ventricle.
- 3 = Cystic periventricular leukomalacia is mostly above this level
- 4 = Cysts as a result of a venous infarct is large and can be either above, at or below this level

INTRACRANIAL HEMORRHAGE

- Premature Infants
- Term Infants

Germinal Matrix Hemorrhage

Germinal matrix hemorrhage (GMH) is also known as periventricular hemorrhage or preterm caudothalamic hemorrhage.
These germinal matrix hemorrhages occur in the highly vascular but also stress sensitive germinal matrix, which is located in the caudothalamic groove. This is the subependymal region between the caudate nucleus and thalamus.
The germinal matrix is only transiently present as a region of thin-walled vessels, migrating neuronal components and vessel precursors

It has matured by 34 weeks gestation, such that hemorrhage becomes very unlikely after this age.

Most GMHs occur in the first week of life

Most common in infants

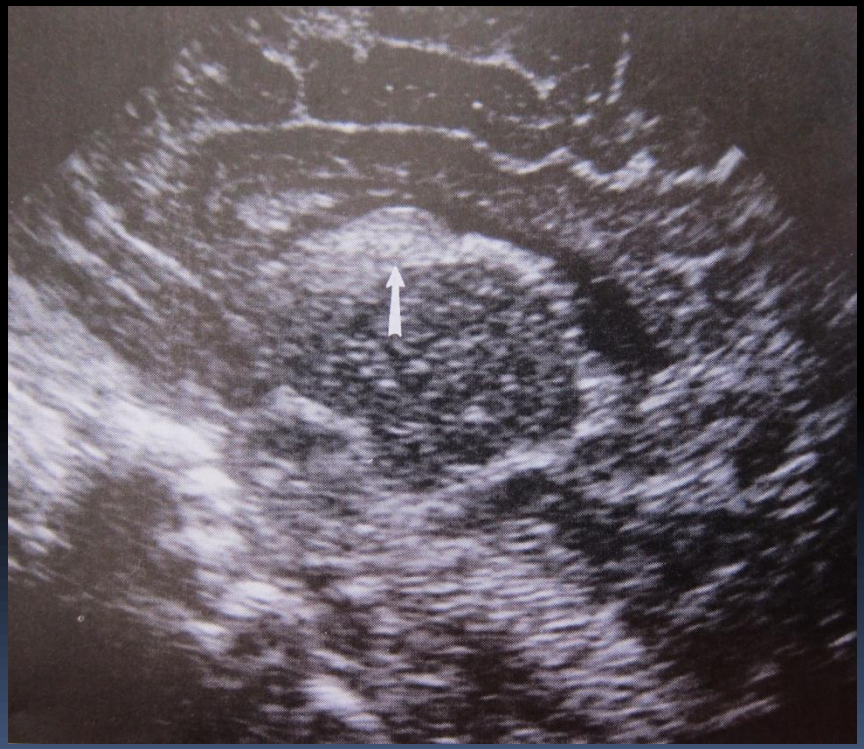
These hemorrhages start in the caudothalamic groove and may extend into the lateral ventricle and periventricular brain parenchyma.

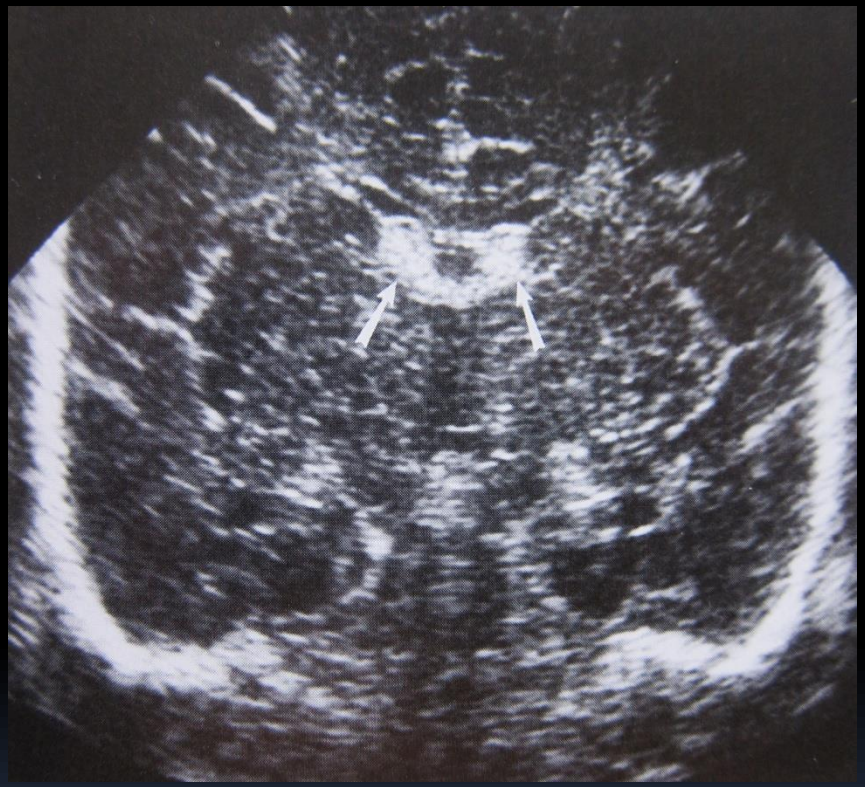
Most infants are asymptomatic or demonstrate subtle signs that are easily overlooked. These hemorhages are subsequently found on surveillance sonography.

Grade 1 intracranial hemorrhage

An intracranial hemorrhage confined to the caudothalamic groove.
It is staged as grade 1 hemorrhage.

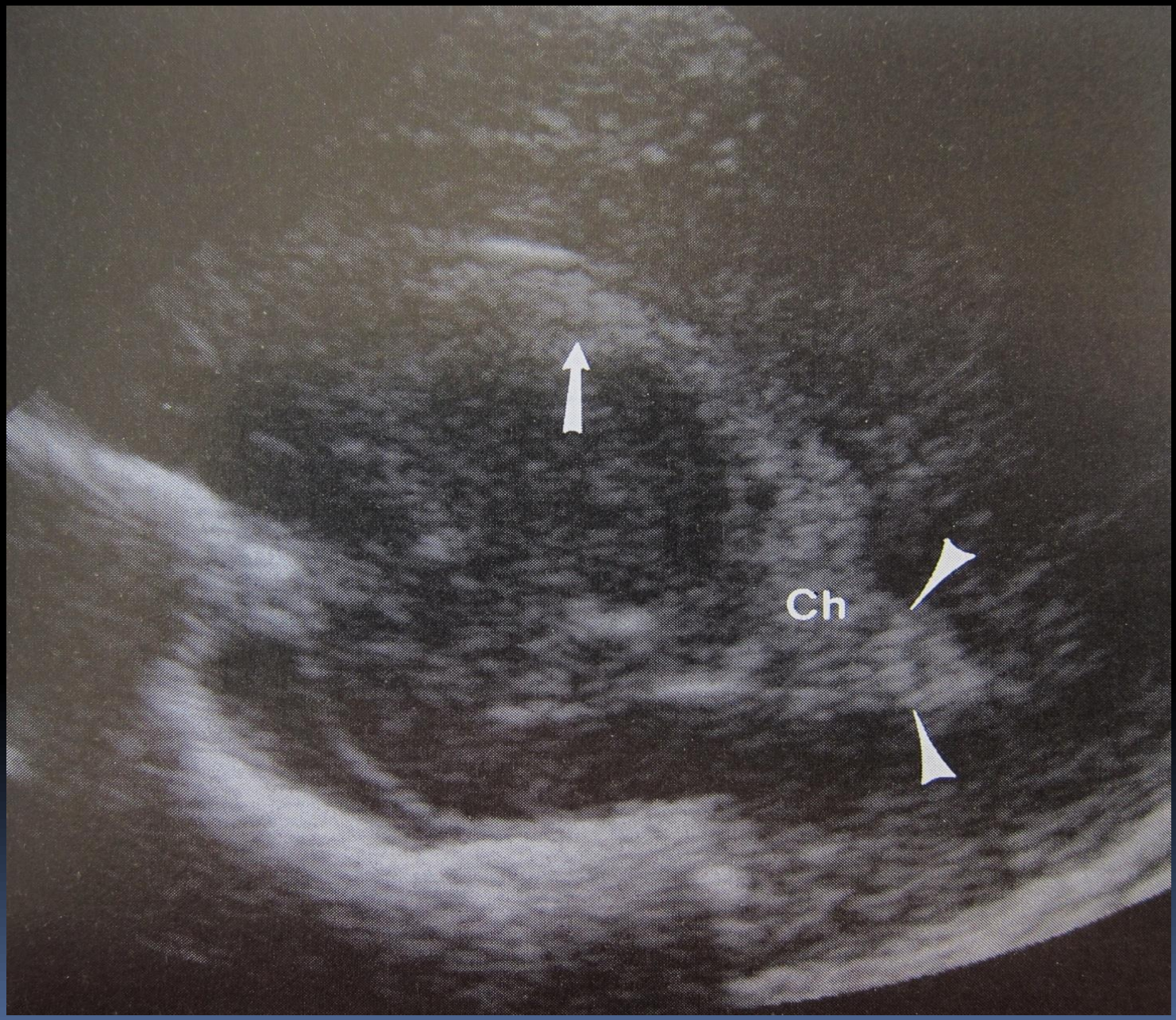
In the acute phase these bleedings are hyperechoic, changing to iso- and hypo-echoic with time.





Grade 2 intracranial hemorrhage

Blood in lateral ventricles, but there is no ventricular dilatation.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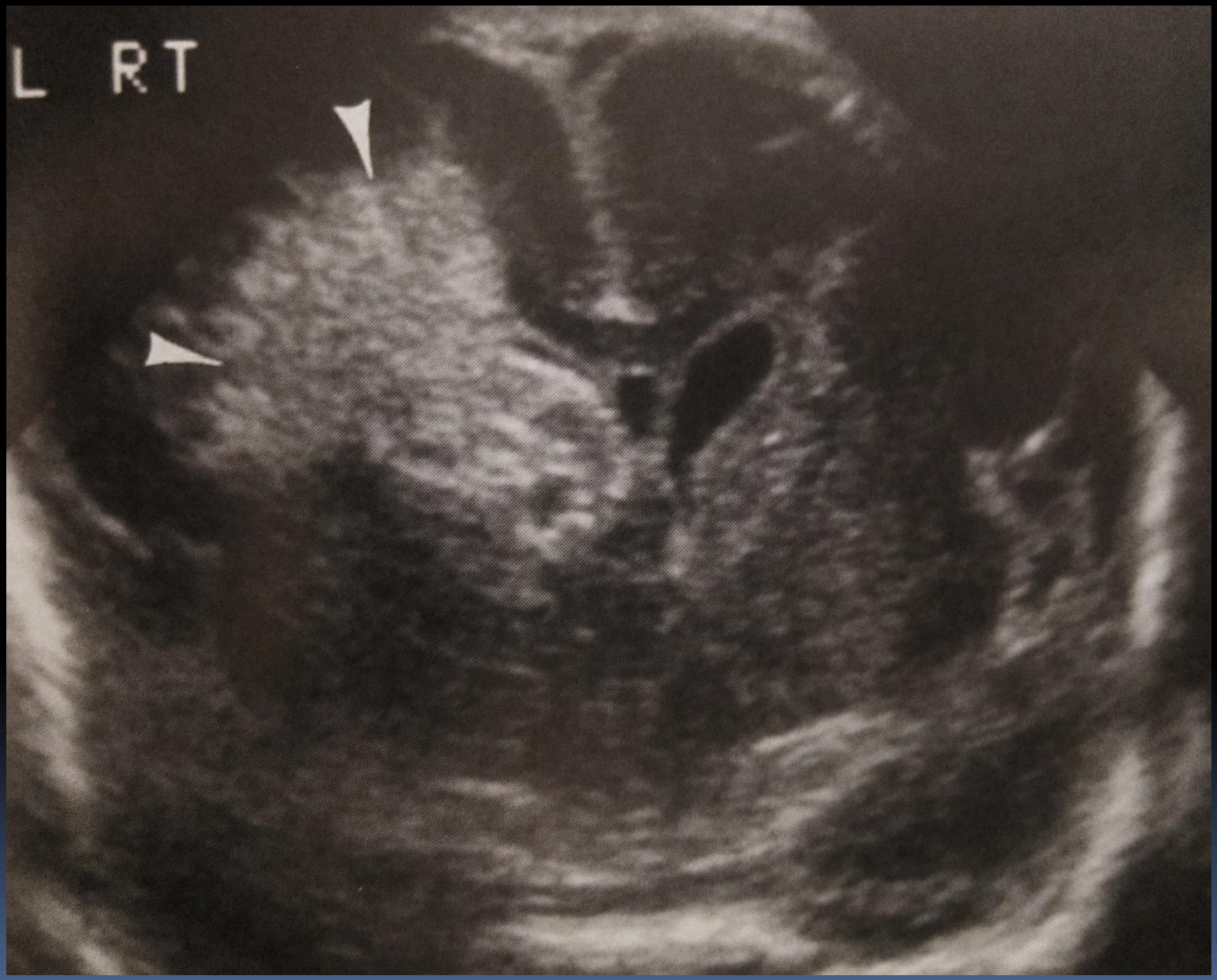
Grade 3 intracranial hemorrhage

In grade 3 intracranial hemorrhage the blood is filling the whole lateral ventricle with subsequent dilatation.


AL RT

Grade 4 intracranial hemorrhage

Originally these grade 4 hemorrhages were thought to result from subependymal bleeding into the adjacent brain.
Today however most regard these grade 4 hemorrhages to be venous hemorrhagic infartions, which are the result of compression of the outflow of the veins by the subependymal hemorrhage. These venous infarctions resolve with cyst formation. These cysts can merge with the lateral ventricle, finally resulting into a **porencephalic cyst**.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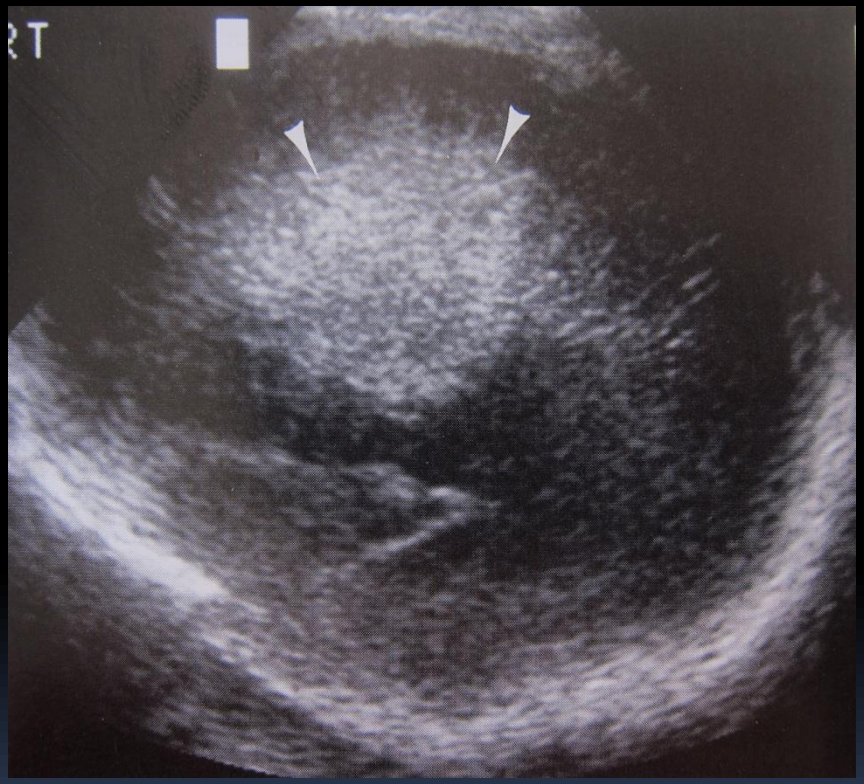

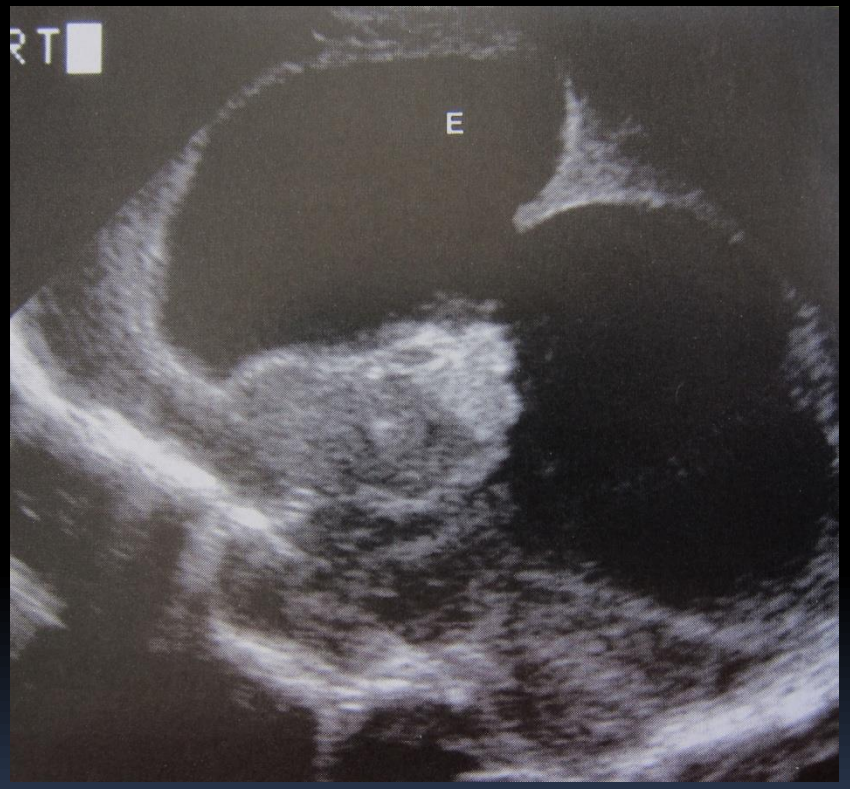
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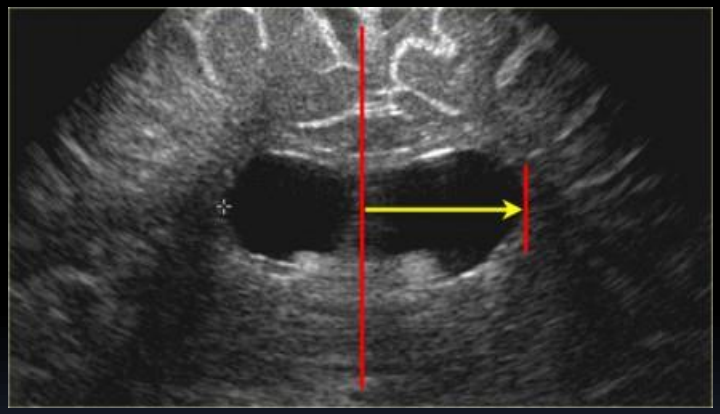
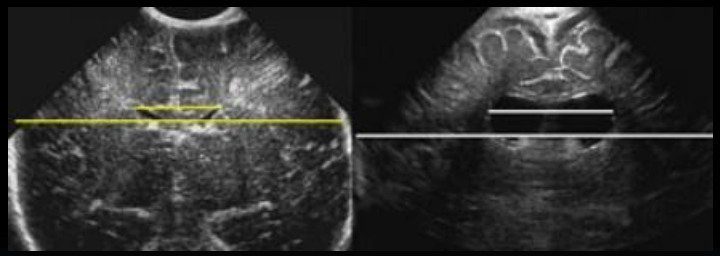
- Grade 1 and 2 bleeds generally have a good prognosis. Grade 3 and 4 bleeds have variable long-term deficits, but outcome in grade 3 hemorrhages is usually good when no parenchymal injury has occurred.
- Hydrocephalus is a common complication and many infants require ventriculoperitoneal shunting.
  The mechanisms by which hydrocephalus develop include:
- **Communicating hydrocephalus**: decreased absorption of cerebral spinal fluid (CSF) secondary to obstruction of arachnoid villi by blood and debris or the development of arachnoiditis
- **Obstructive hydrocephalus**: obstruction to CSF circulation.



Levene index

- Up to 40 weeks of gestational age the Levene-index should be used and after 40 weeks the ventricular index.
  The Levene index is the absolute distance between the falx and the lateral wall of the anterior horn in the coronal plane at the level of the third ventricle.
  This is performed for the left and right side.
  These measurements can be compared to the reference curve and are quite usefull for further follow-up.

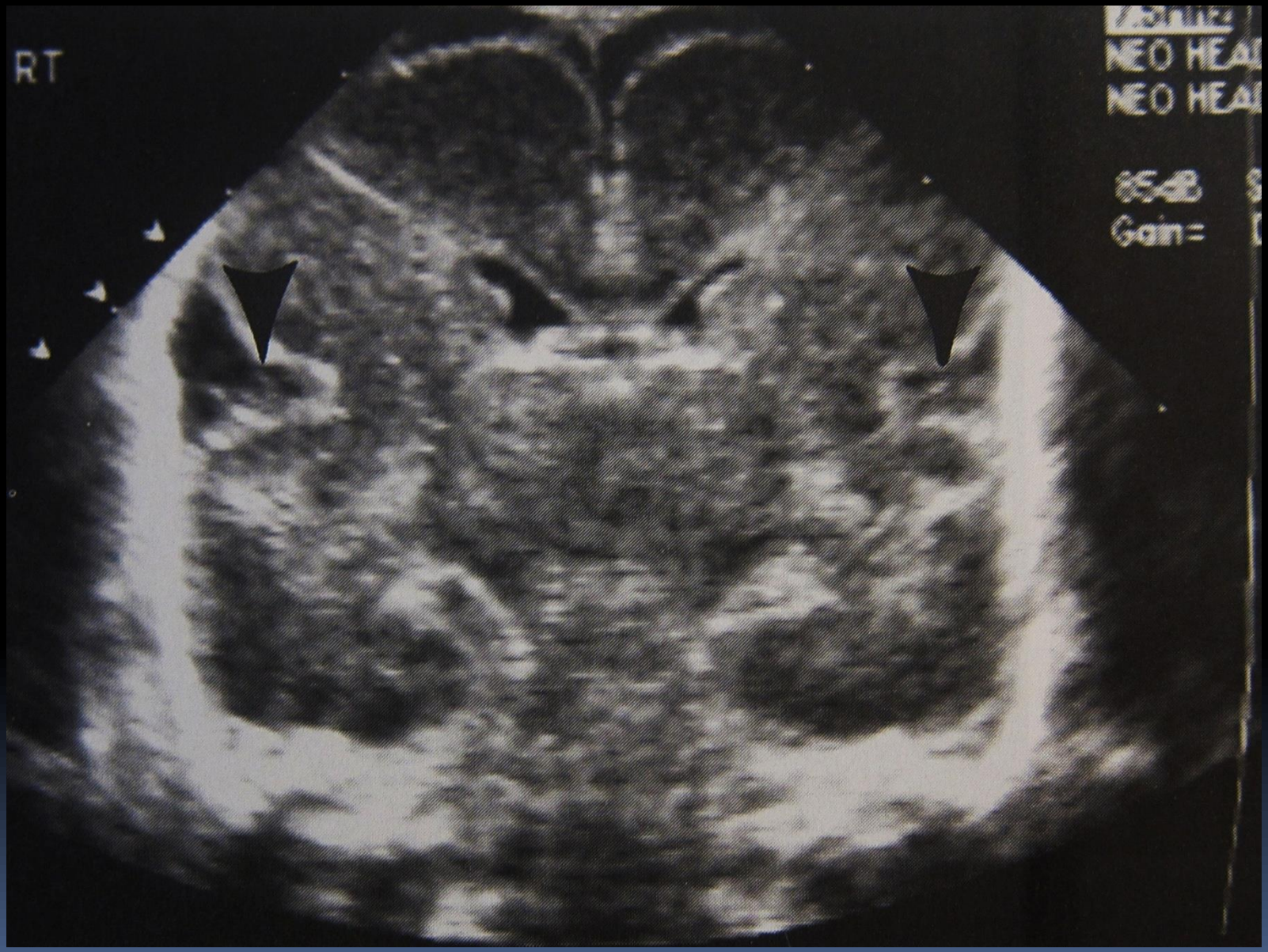


Ventricular index

- After 40 weeks the ventricular index or frontal horn ratio should be used, i.e. the ratio of the distance between the lateral sides of the ventricles and the biparietal diameter.
  When using this ratio you have to realise, that when the ventricular system widens, the frontal horns tend to enlarge in the craniocaudal direction more than in the left to right dimension.

INTRACRANIAL HEMORRHAGE

- Term Infants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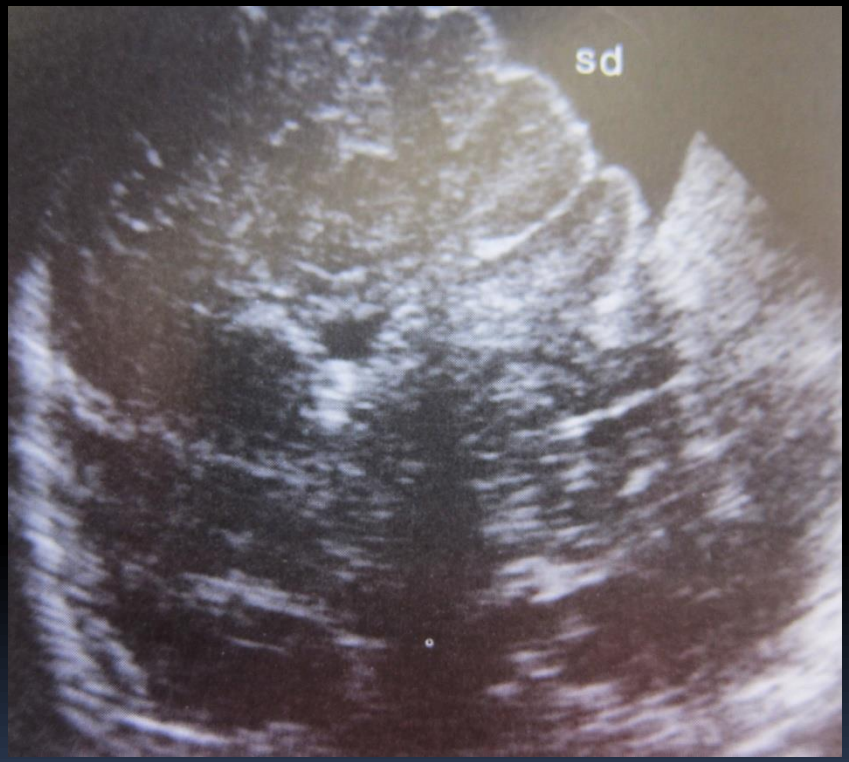
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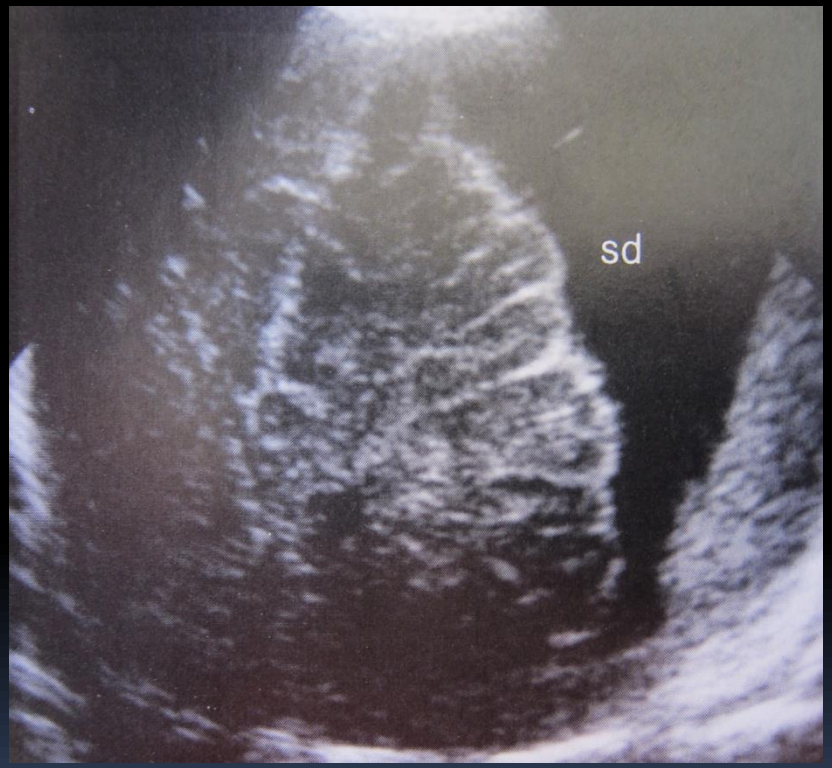
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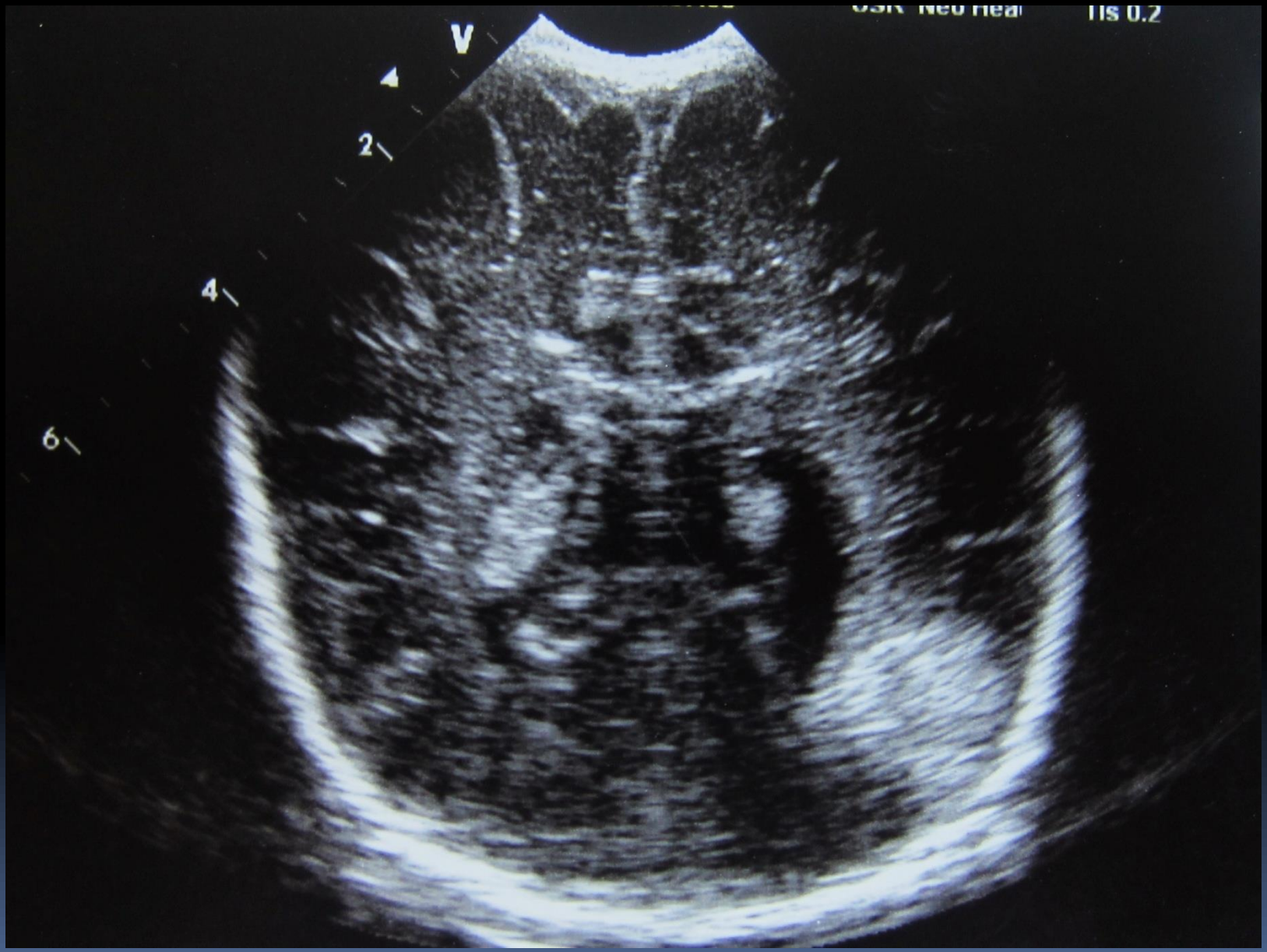
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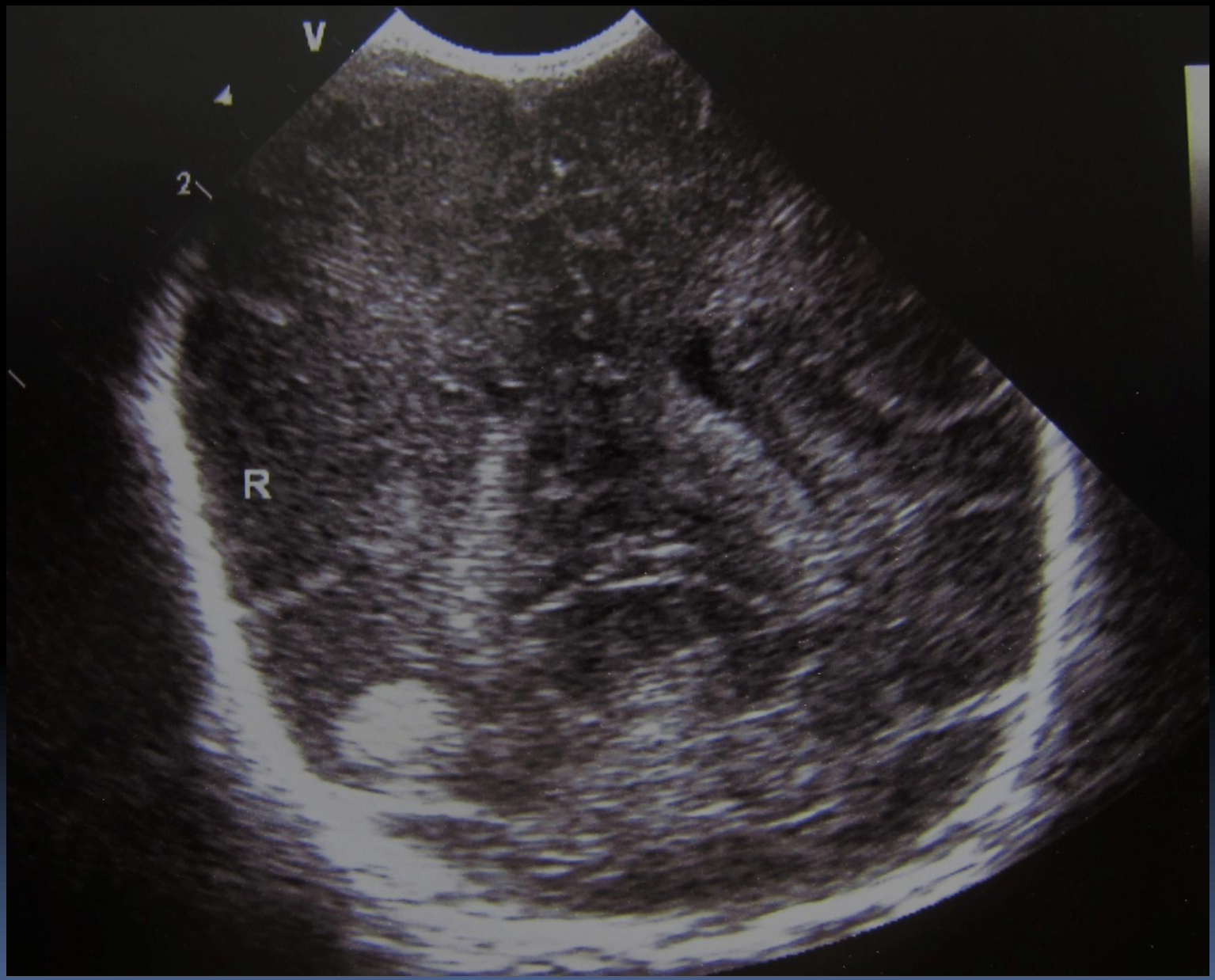
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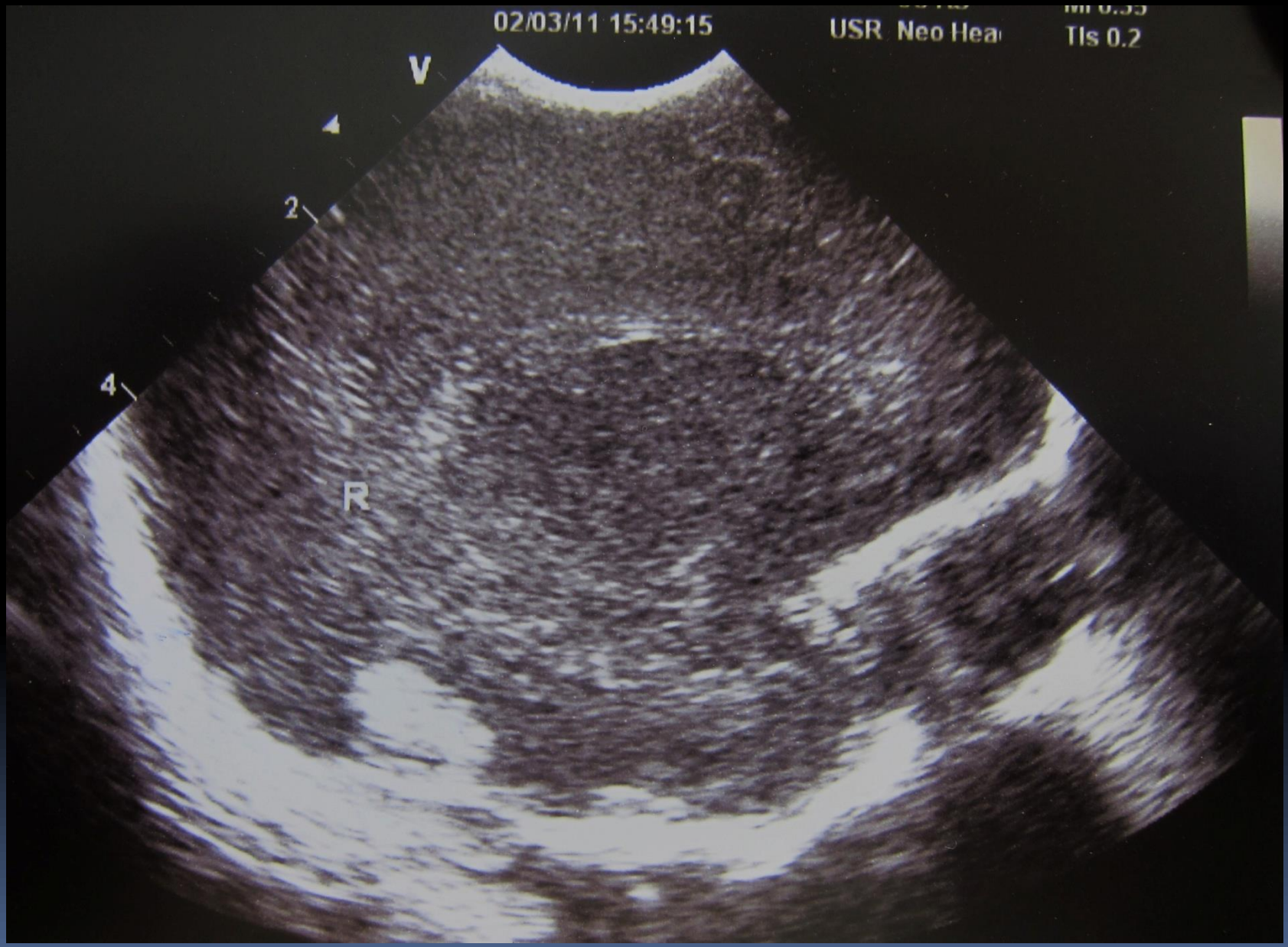
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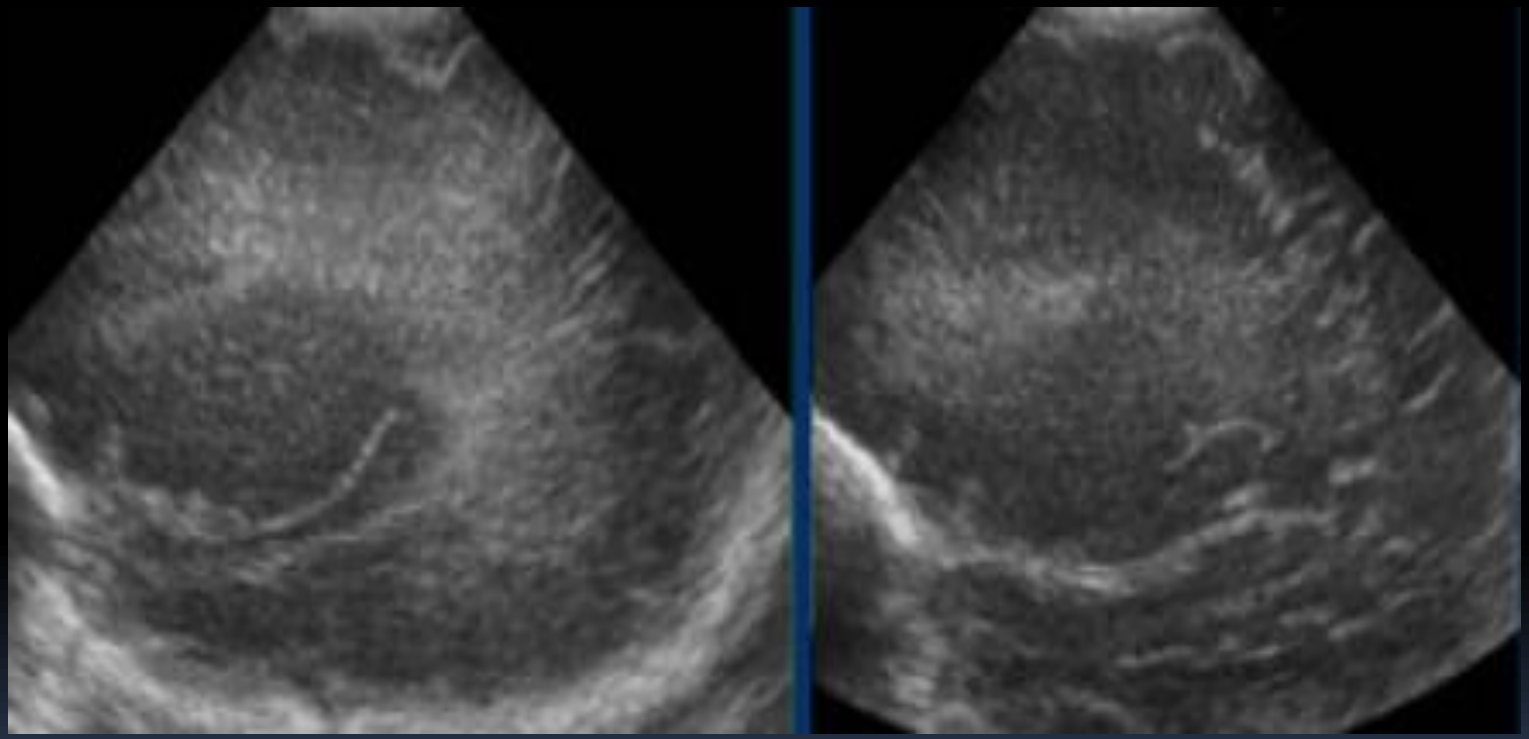
HYPOXIC-ISCHEMIC INJURY

- ***Periventricular Leukomalakia of Prematurity***
- Ischemic Lesions in Term Infants
- Diffuse Ischemic Injury

Flaring

- Transverse and sagittal images demonstrating flaring in a premature infant.
- The term flaring is used to describe the slightly echogenic periventricular zones, that are seen in many premature infants in the first week of life.
  During this first week it is not sure if this is a normal variant or a sign of PVL grade 1.
  Flaring persisting beyond the first week of life is by definition PVL grade 1.

- Follow up is needed to differentiate flaring from PVL grade I.
  The next case shows a premature infant with flaring.
  At follow up no cyst formation was found and after the first week a normal periventricular white matter was seen.



Periventricular Leukomalacia (PVL)

- PVL is also known as Hypoxic-Ischemic Encephalopathy (HIE) of the preterm.
  It is a white matter disease that affects the periventricular zones.
  In prematures this white matter zone is a watershed zone between deep and superficial vessels.
  Until recently ischemia was thought to be the single cause of PVL, but probably other causes (infection, vasculitis) play an additional role.
  PVL presents as areas of increased periventricular echogenicity.
  Normally the echogenicity of the periventricular white matter should be less than the echogenicity of the choroid plexus.

- PVL occurs most commonly in premature infants born at less than 33 weeks gestation (38% PVL) and less than 1500 g birth weight (45% PVL).
  Detection of PVL is important because a significant percentage of surviving premature infants with PVL develop cerebral palsy, intellectual impairment or visual disturbances.
  More than 50% of infants with PVL or grade III hemmorrhage develop cerebral palsy.

Grading PVL

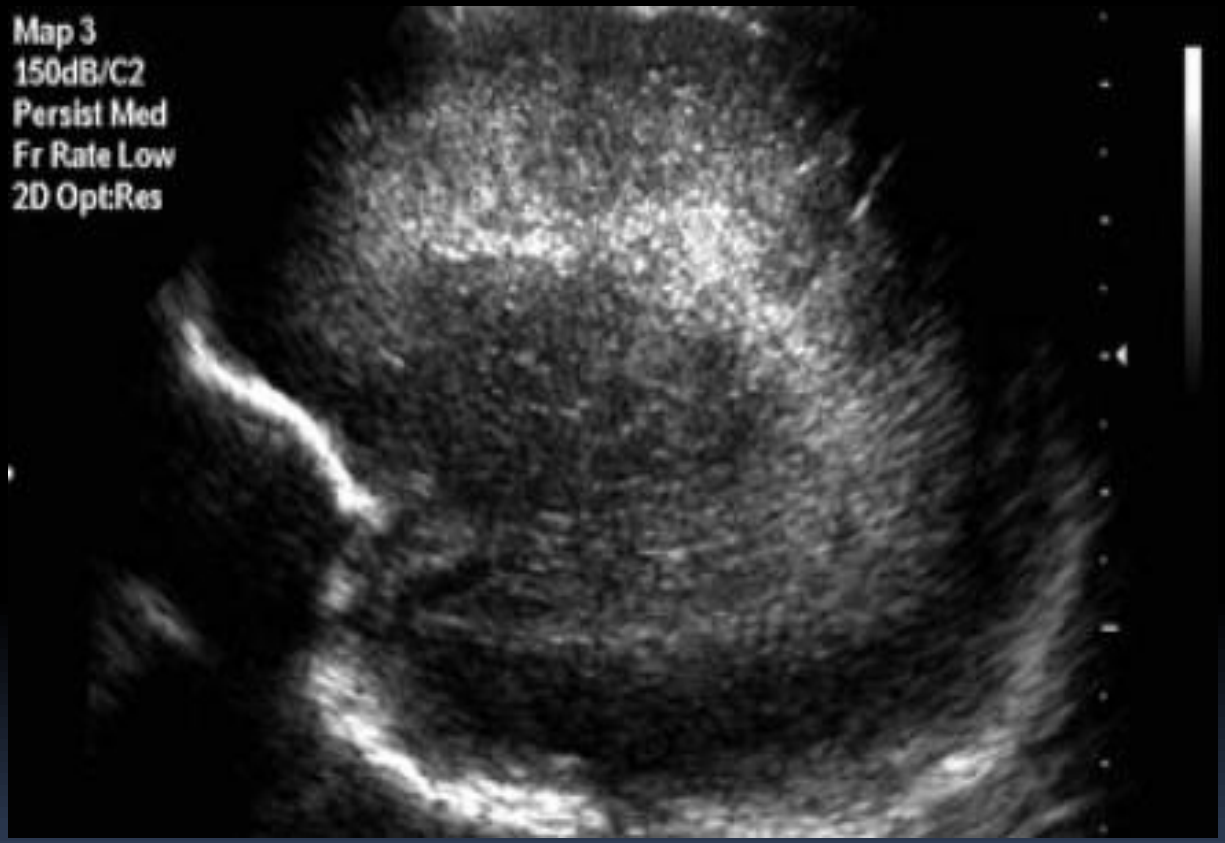
- Regular sonographic examination is mandatory as cysts in PVL can develop as long as 4 weeks after birth (especially in prematures Cranial ultrasonographic findings may be normal in patients who go on to develop clinical and delayed imaging findings of PVL. A good protocol is US-examination at least once a week until discharge and at the age of 40 weeks.

CLASSIFICATION OF PERIVENTICULAR AND SUBCORTICAL LEUKOMALAKIA BASED ON CRANIAL ULTRASOUND FINDINGS (De Vries)

- Grade I Periventricular echodense area, present for 7 days or more
- Grade II Periventricular echodense areas evolving into localized frontoparietal cysts
- Grade III Periventricular echodense areas evolving into multiple cysts in the parieto-occipital white matter
- Grade IV Echodense areas in the deep white matter, with evolution into multiple subcortical cysts

PVL grade 1

PVL is diagnosed as grade 1 if there are areas of increased periventricular echogenicity without any cyst formation persisting for more than 7 days.
Increased periventricular echogenicity is however a nonspecific finding that must be differentiated from the normal periventricular halo or normal hyperechoic 'blush' posterosuperior to the ventricular trigones.
Suspect PVL if the echogenicity is asymmetric, coarse, globular or more hyperechoic than the choroid plexus.
The abnormal periventricular echotexture of PVL usually disappears at 2-3 weeks.
PVL can be differentiated from hemorrhages because PVL lacks mass effect.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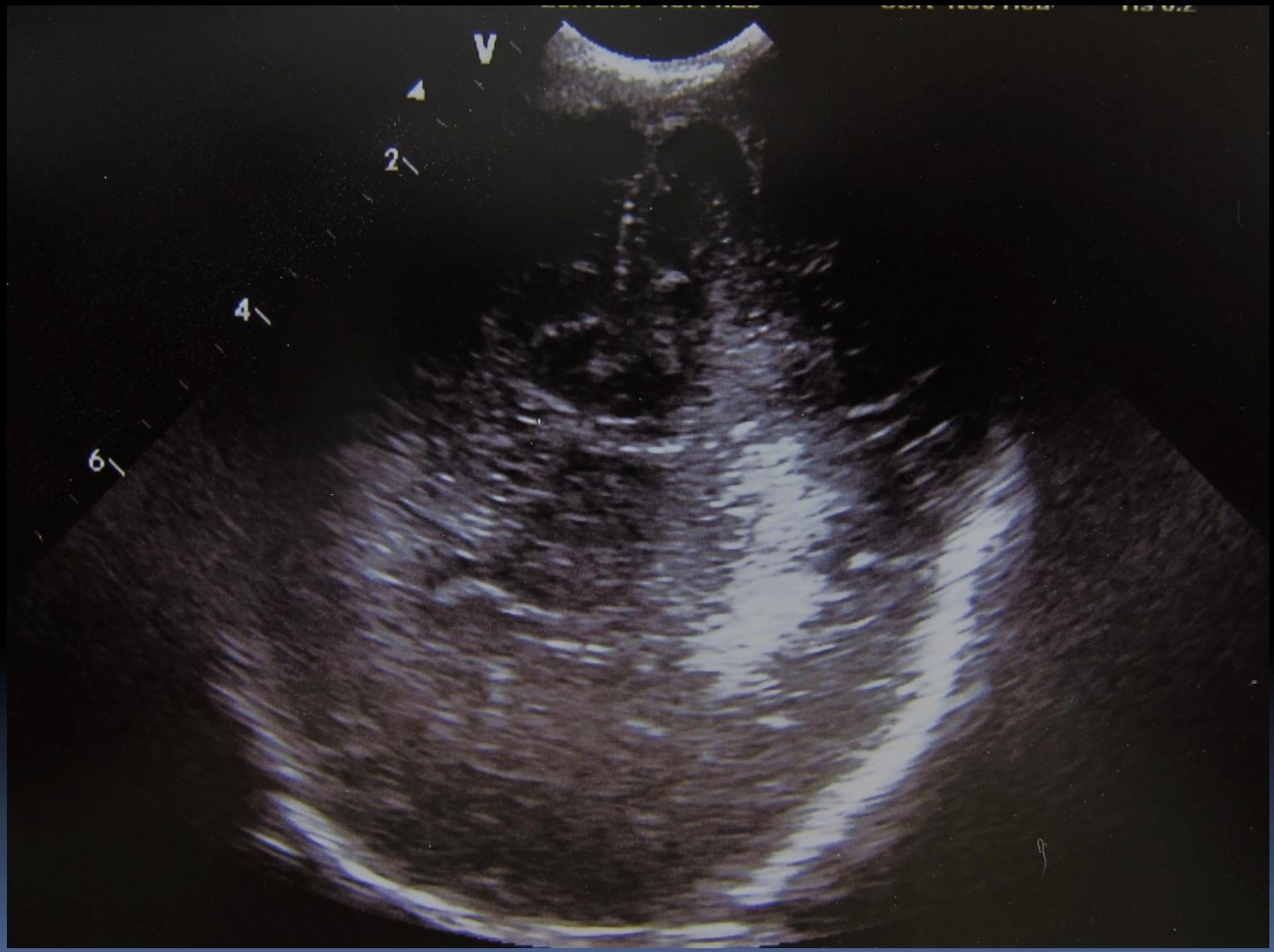
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PVL grade 2

The echogenicity has resolved at the time of cyst formation.
2% of the preterm neonates born before 32 weeks develop cystic PVL.
The severity of PVL is related to the size and distribution of these cysts.
Cystic PVL has been identified on cranial ultrasounds on the first day of life, indicating that the adverse event was at least 2 weeks prenatal rather than perinatal or postnatal.

US is highly reliable in the detection of cystic WM injury (PVL grade II or more), but has significant limitations in the demonstration of noncystic WM injury (PVL grade I).
This deficiency of neonatal cranial US is important, because noncystic WM injury is considerably more common than cystic WM injury.


17/12/08 14:29:52
USR Neo Hea
TIs 0.2
V
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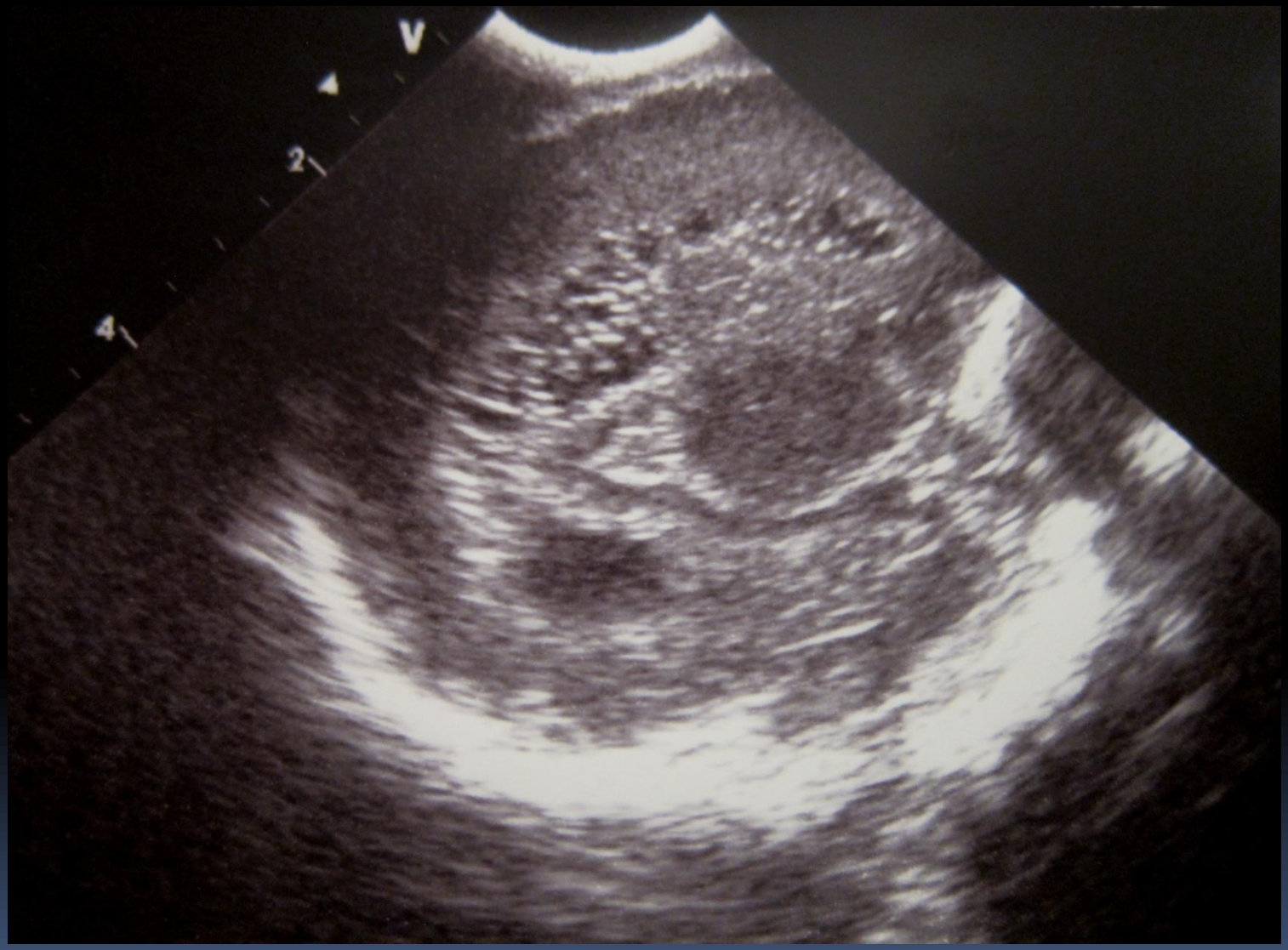
L

PVL grade 3

PVL is diagnosed as grade 3 if there are areas of increased periventricular echogenicity, that develop into extensive periventricular cysts in the occipital and fronto-parietal region.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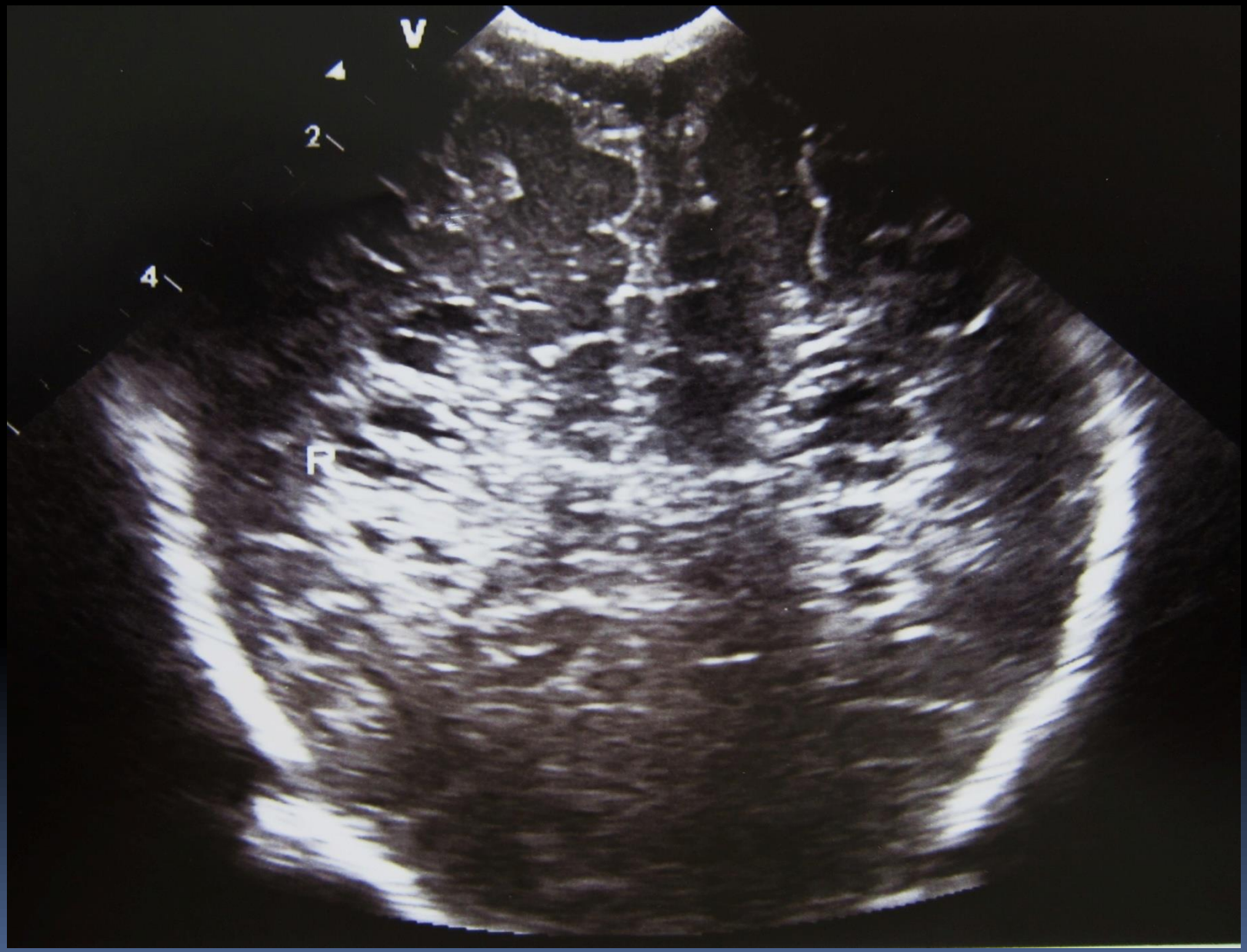
V
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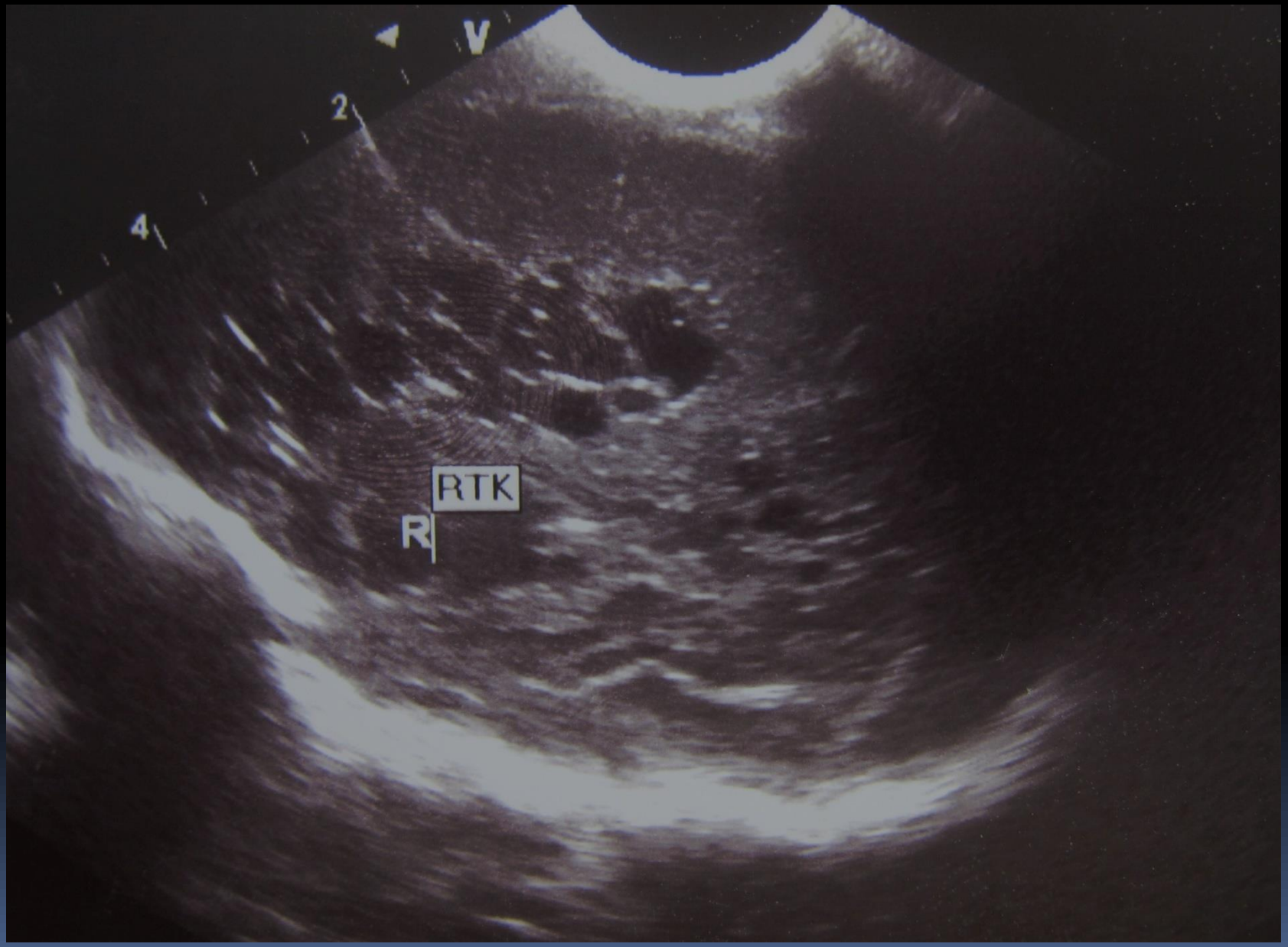
PVL grade 4

PVL is diagnosed as grade 4 if there are areas of increased periventricular echogenicity in the deep white matter developing into extensive subcortical cysts.
PVL grade 4 is seen mostly in fullterm neonates as opposed to PVL grade 1-3, which is a disease of the preterm neonate. Flaring persisting beyond the first week of life is by definition PVL grade 1.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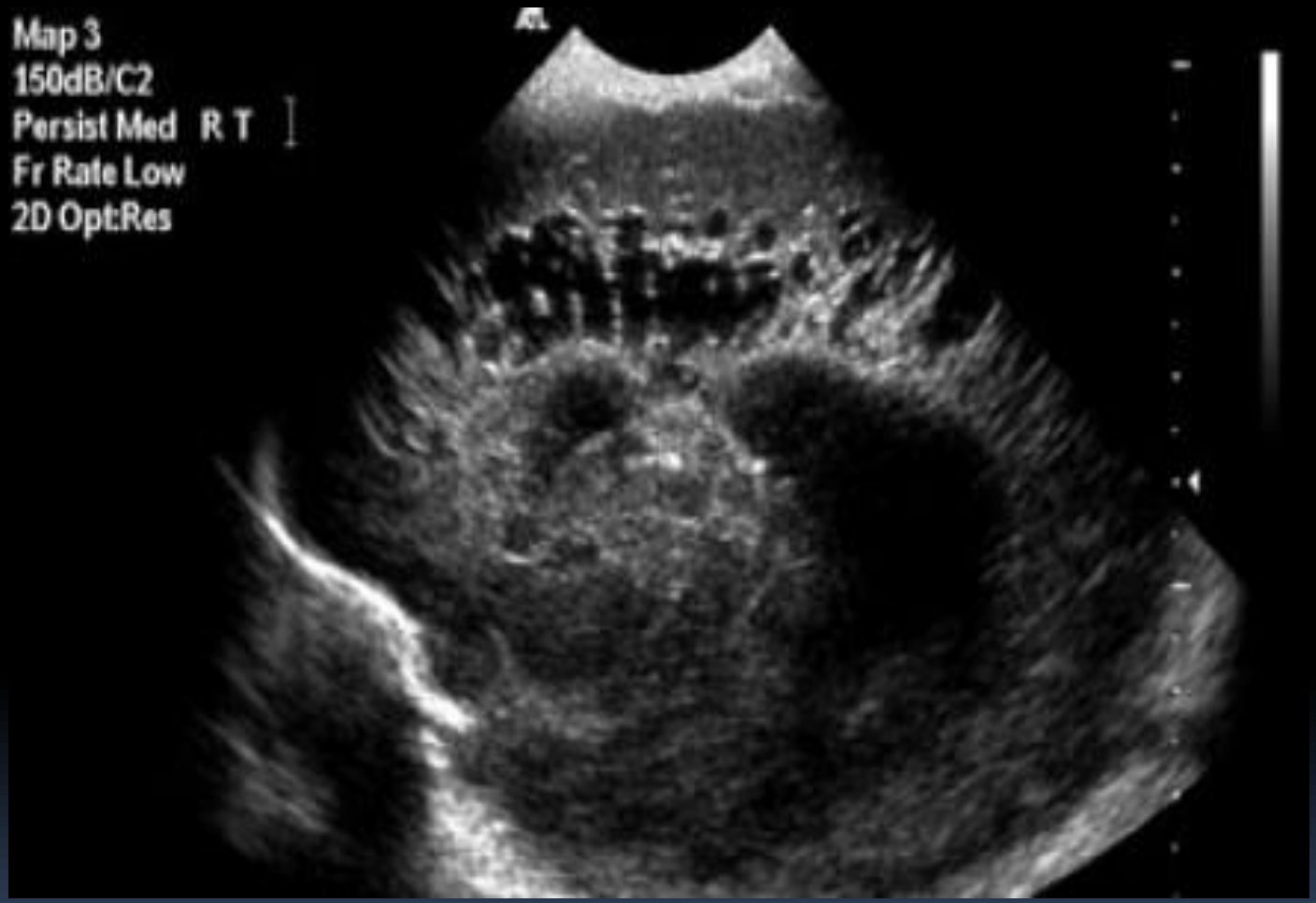
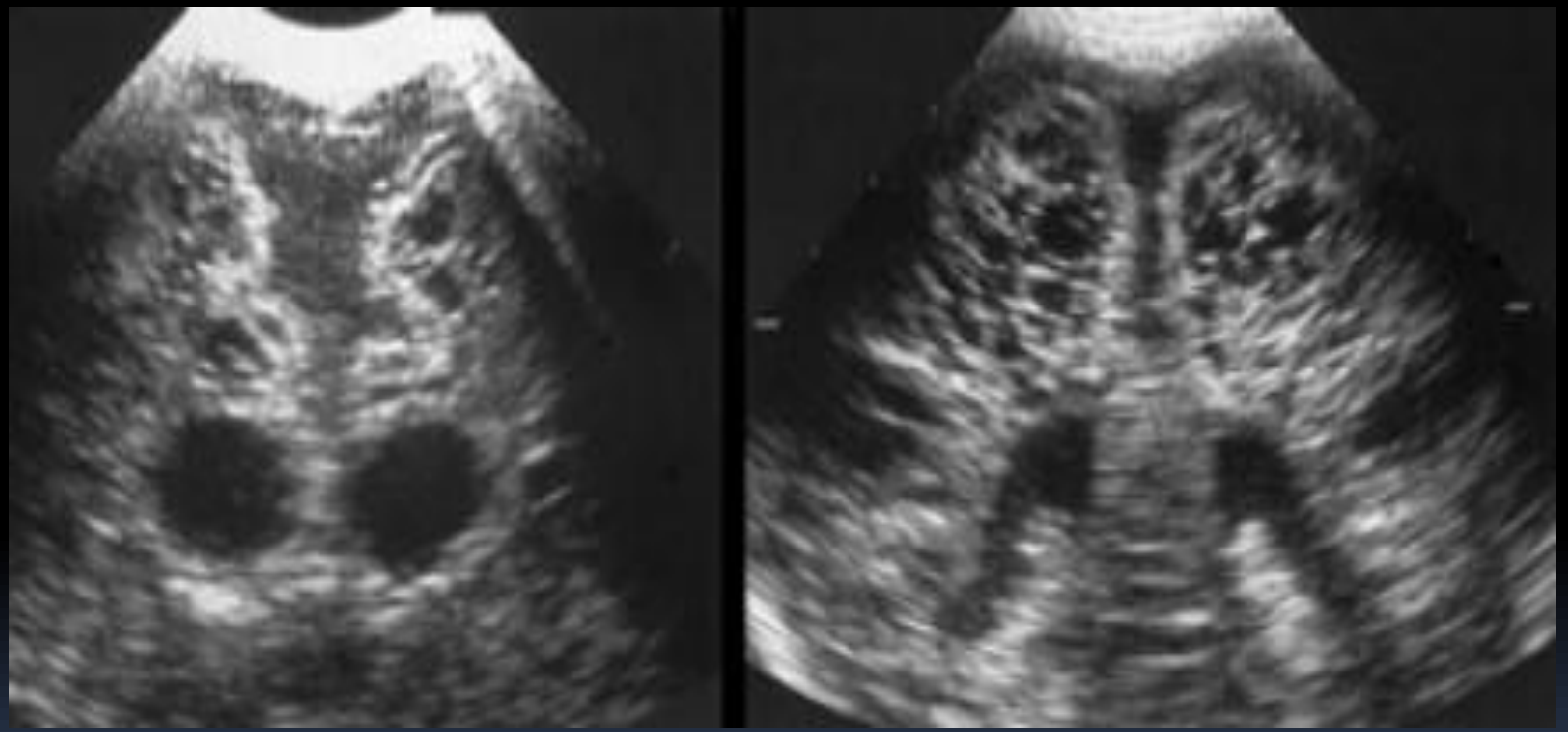
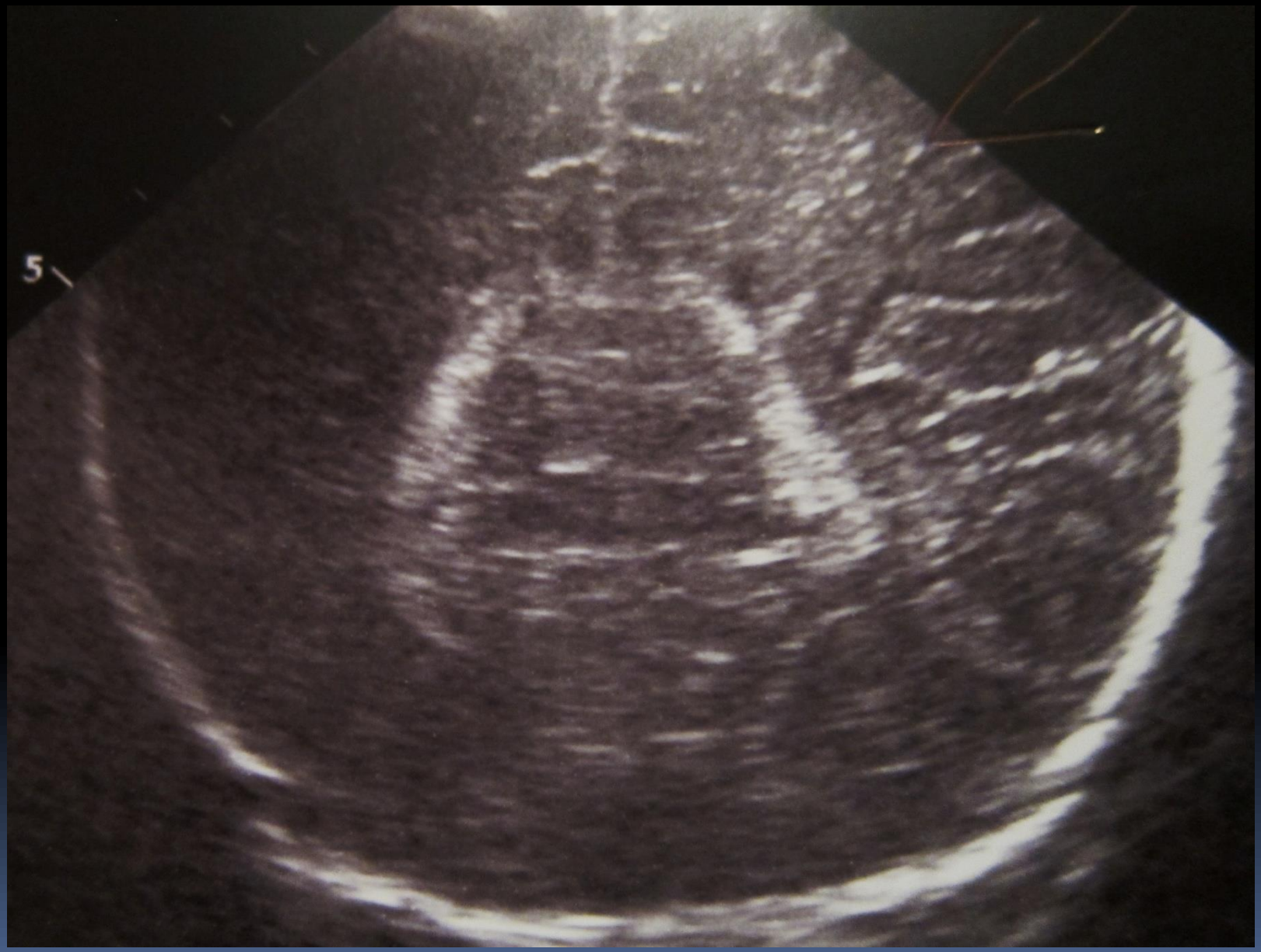

Map 3
150dB/C2
Persist Med R T
Fr Rate Low
2D Opt:Res



HYPOXIC-ISCHEMIC INJURY

- *Ischemic Lesions in Term Infants*


V
5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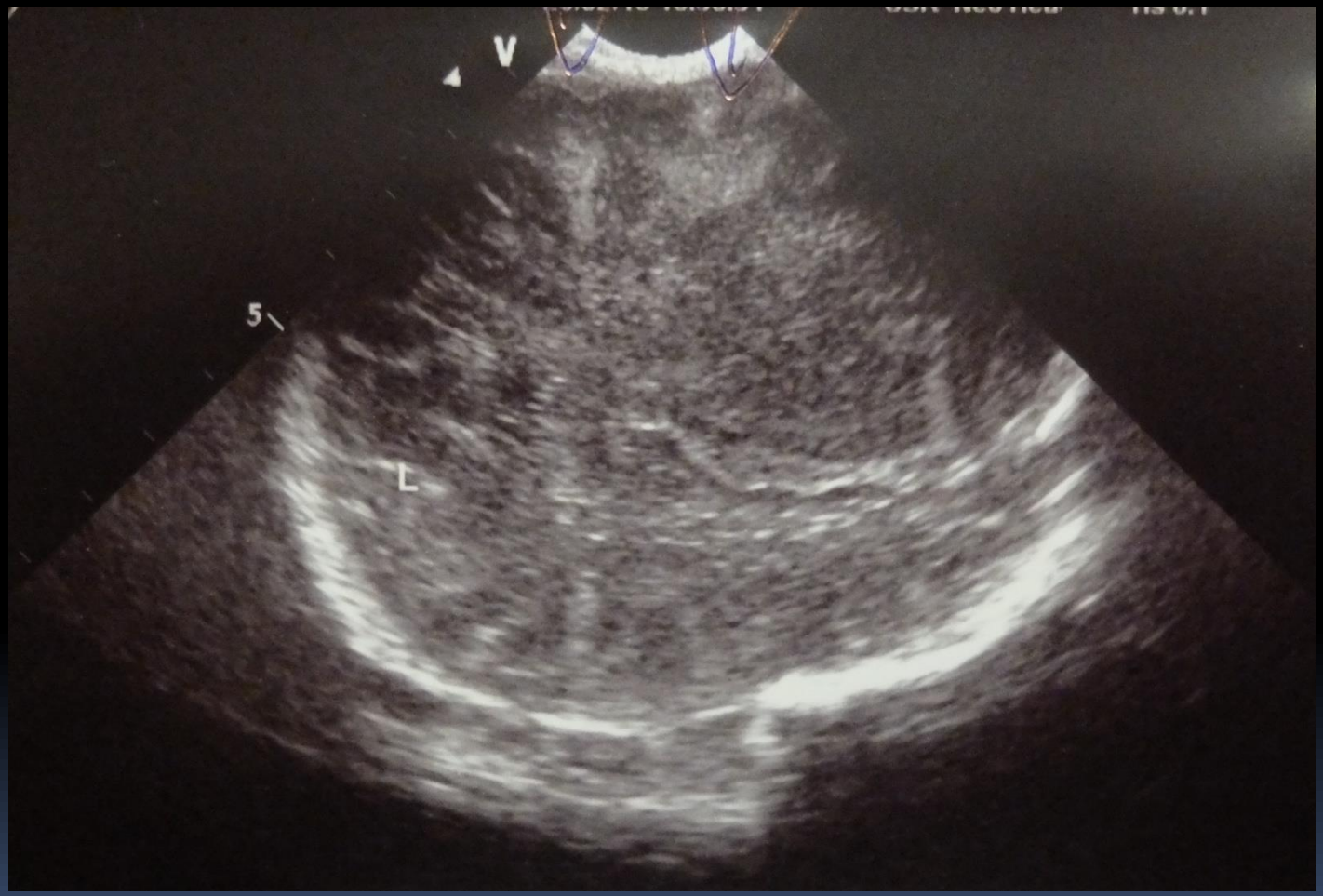
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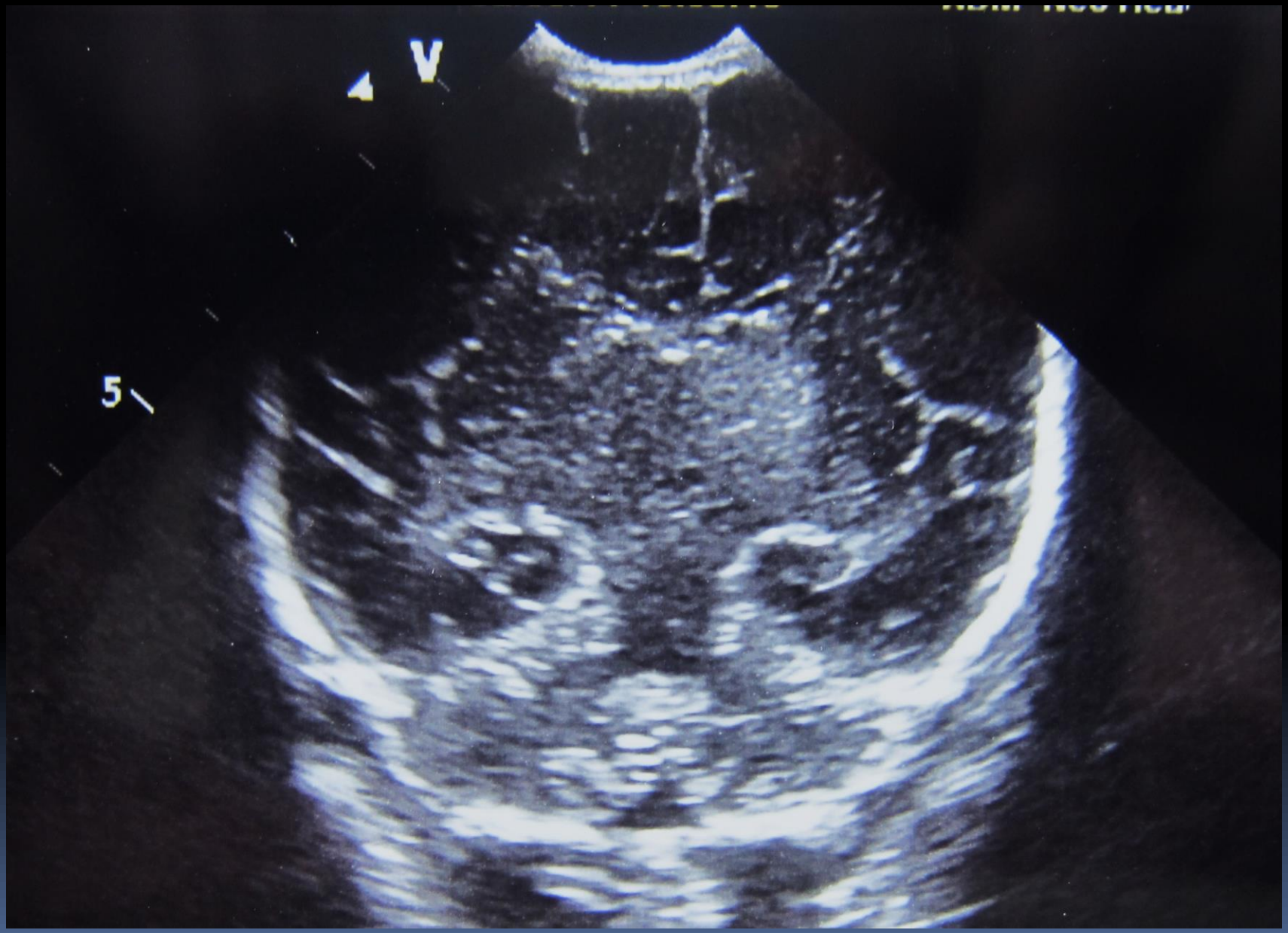
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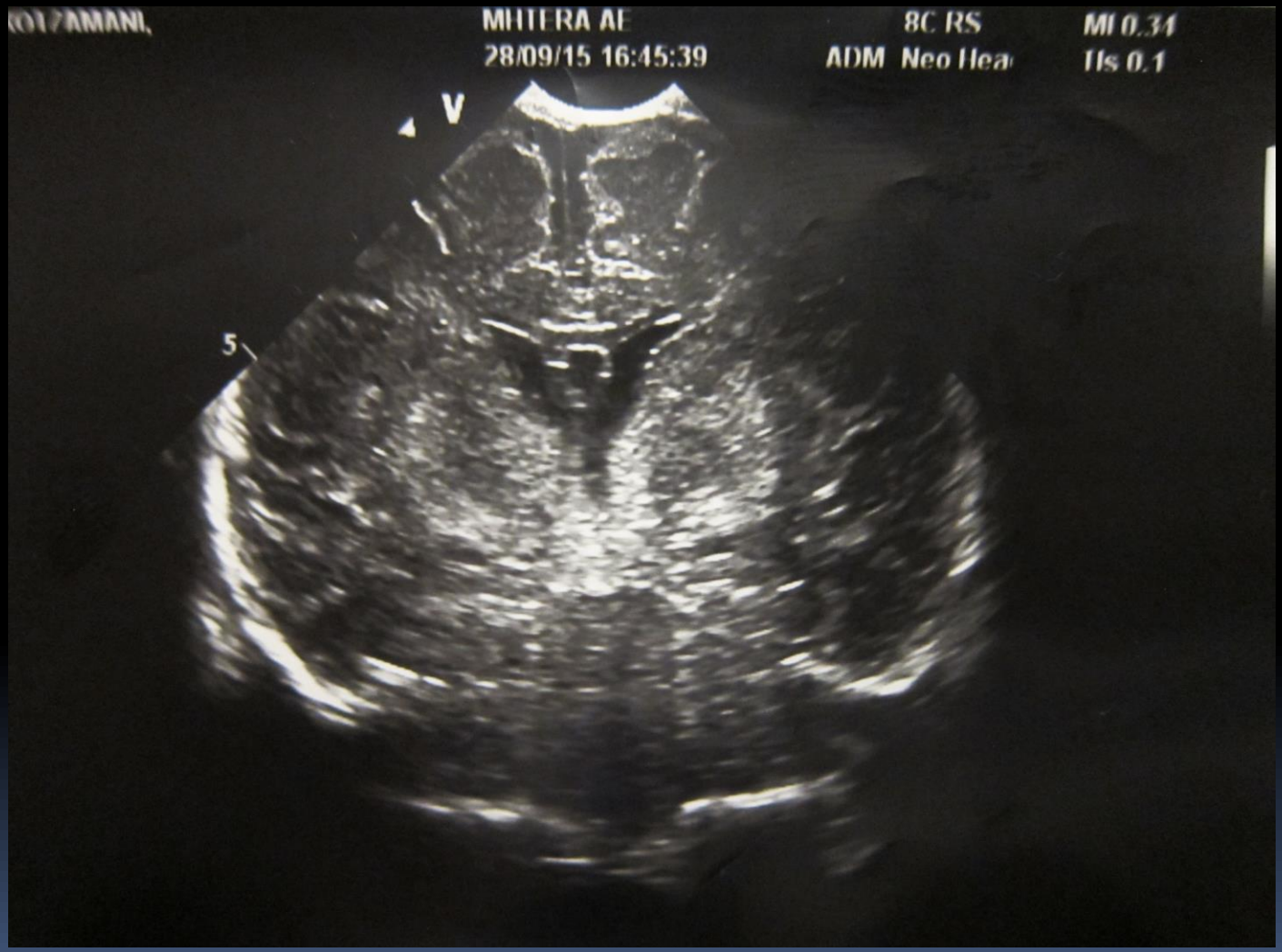
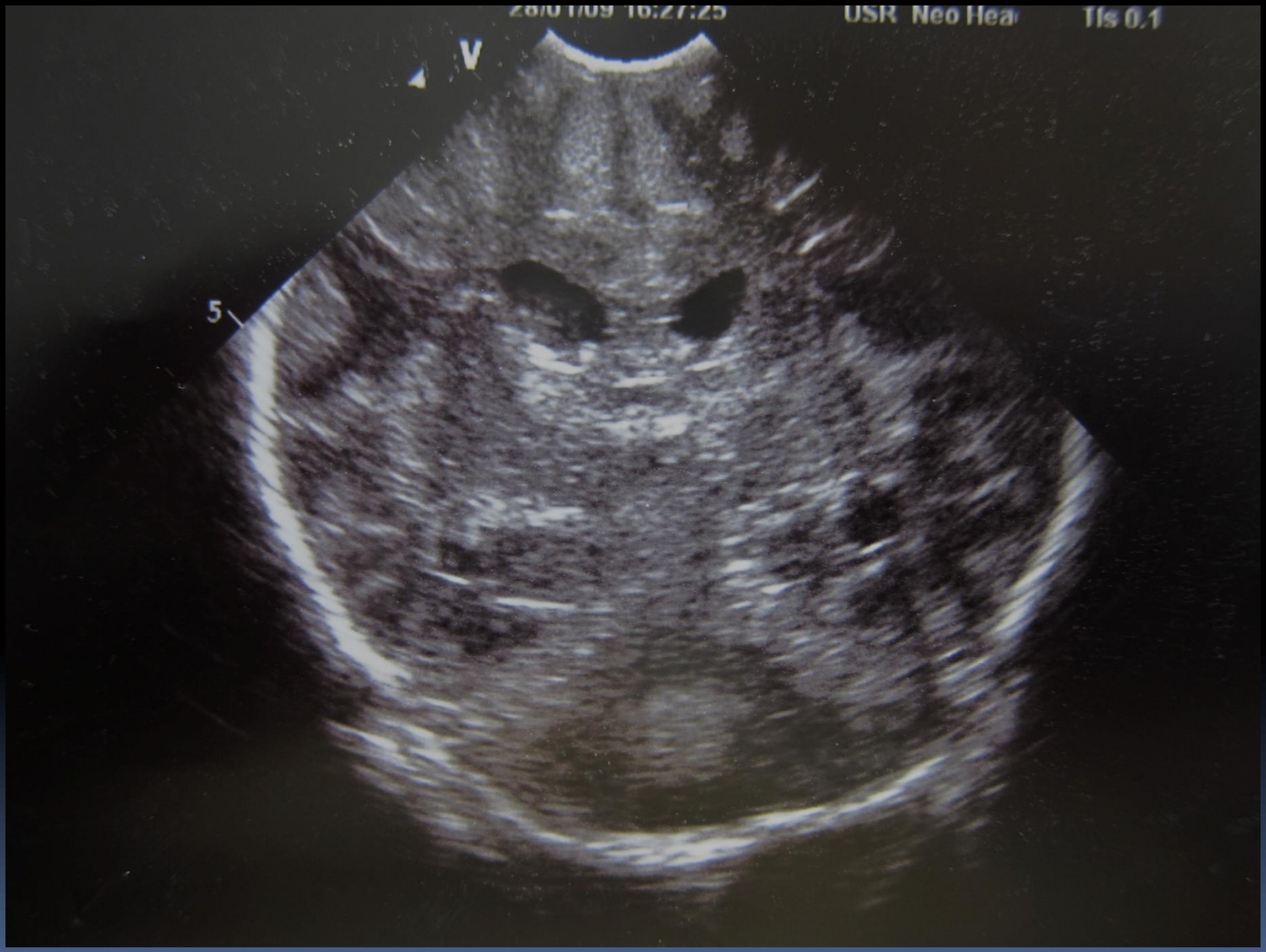

MHTERA AE
28/09/15 16:45:39
8C RS
ADM Neo Hea
MI 0.34
V
5

HYPOXIC-ISCHEMIC INJURY

- *Diffuse Ischemic Injury*


V
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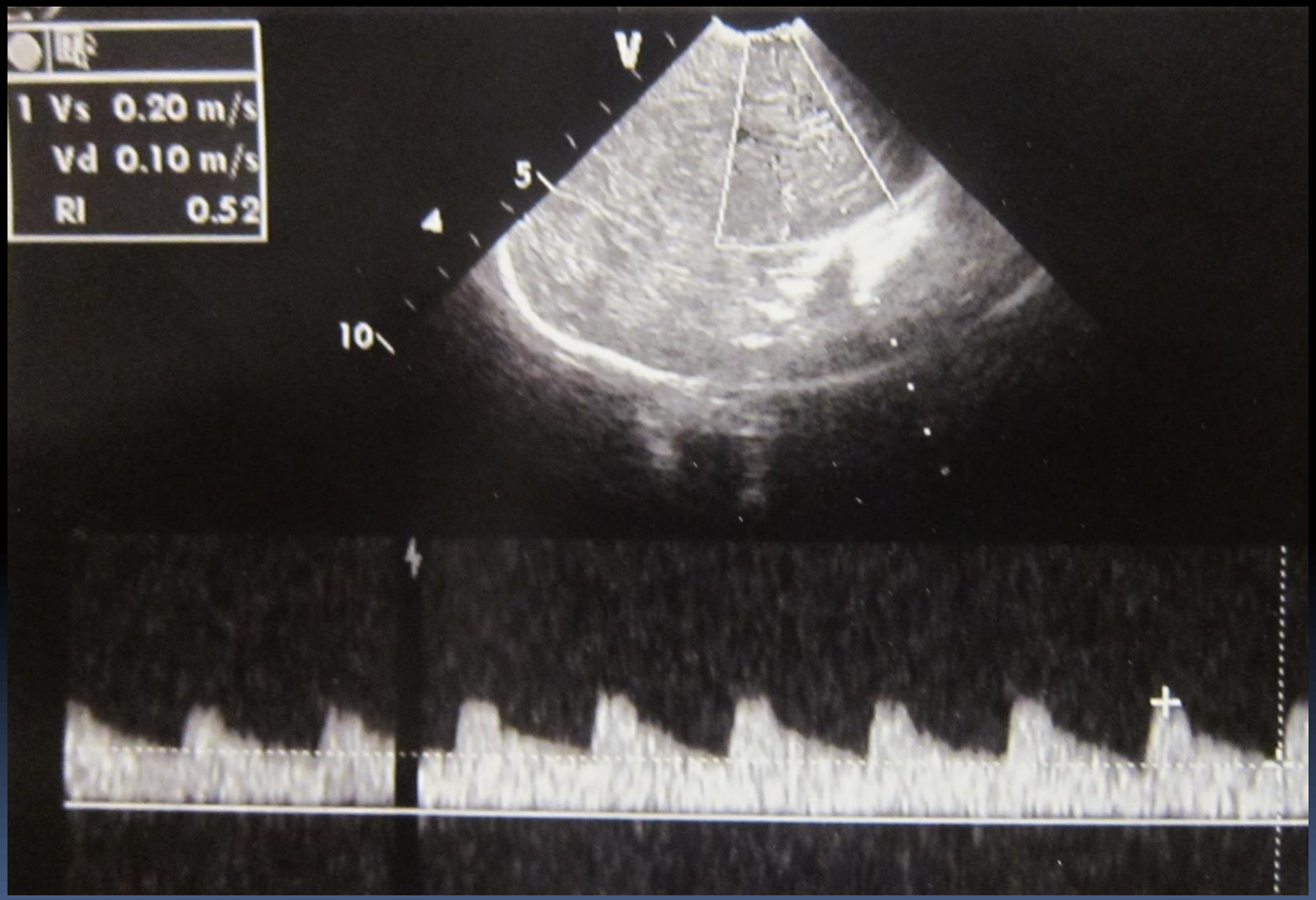

1 Vs 0.20 m/s
Vd 0.10 m/s
RI 0.52
V
5
10

Table 3.1 Mean Arterial Velocities and Mean Resistance Indices in Intracranial Arteries*

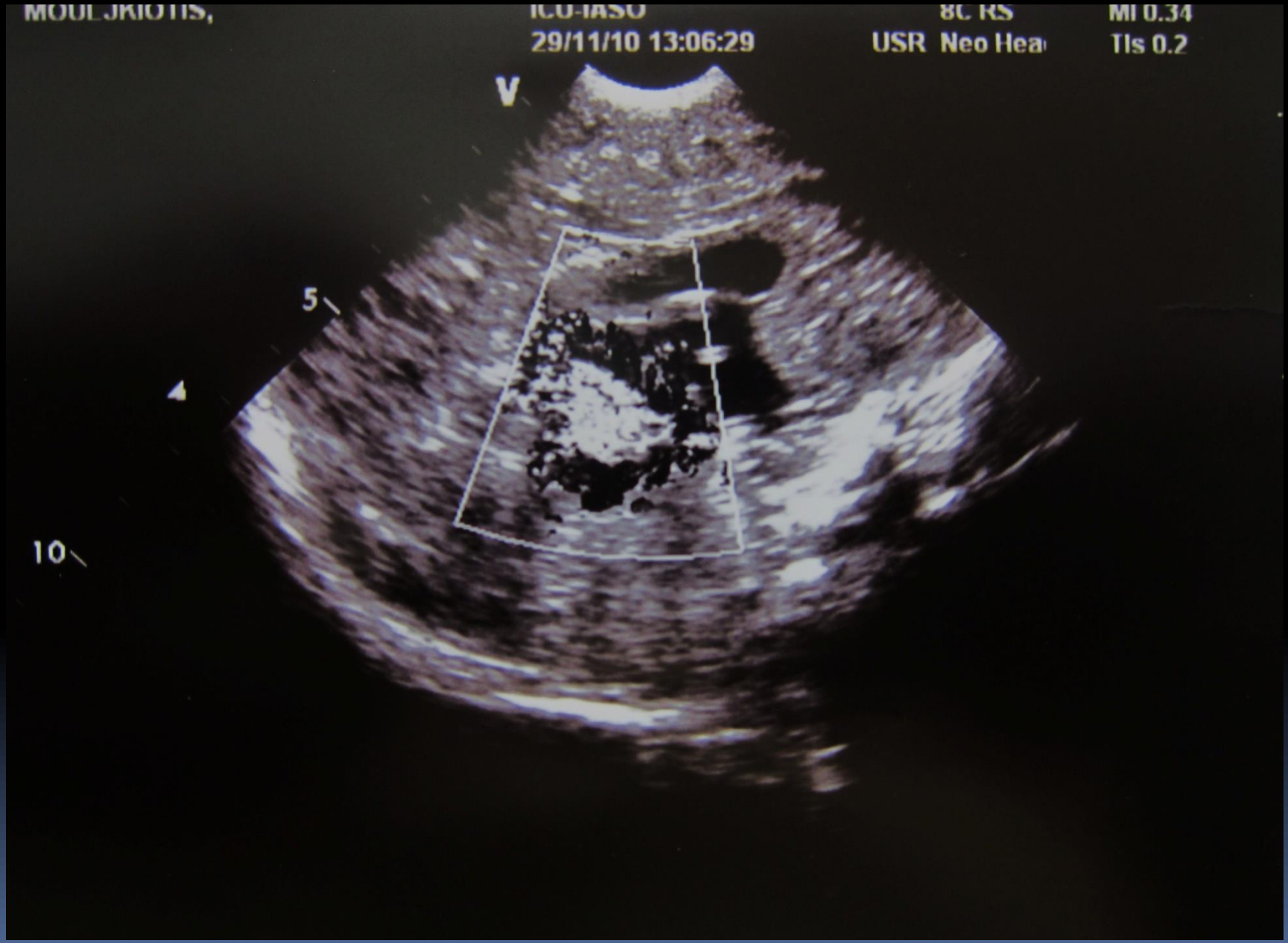
Vessel	Mean Systolic	End Diastolic	Resistive Index
Internal carotid	48 (14)	11 (5)	0.77 (0.08)
Basilar	41 (12)	10 (5)	0.76 (0.07)
Anterior cerebral	38 (13)	9 (5)	0.76 (0.09)
Middle cerebral	45 (14)	11 (5)	0.75 (0.08)
Posterior cerebral	39 (12)	10 (5)	0.74 (0.08)

CONGENITAL MALFORMATIONS OF THE BRAIN

- Disorders of Cytogenesis
- Disorders of Histogenesis
- Disorders of Organogenesis

DISORDERS OF HISTOGENESIS

- Vein of Galen Malformation
- Tuberous Sclerosis
- Sturge-Weber Syndrome
- Neurofibromatosis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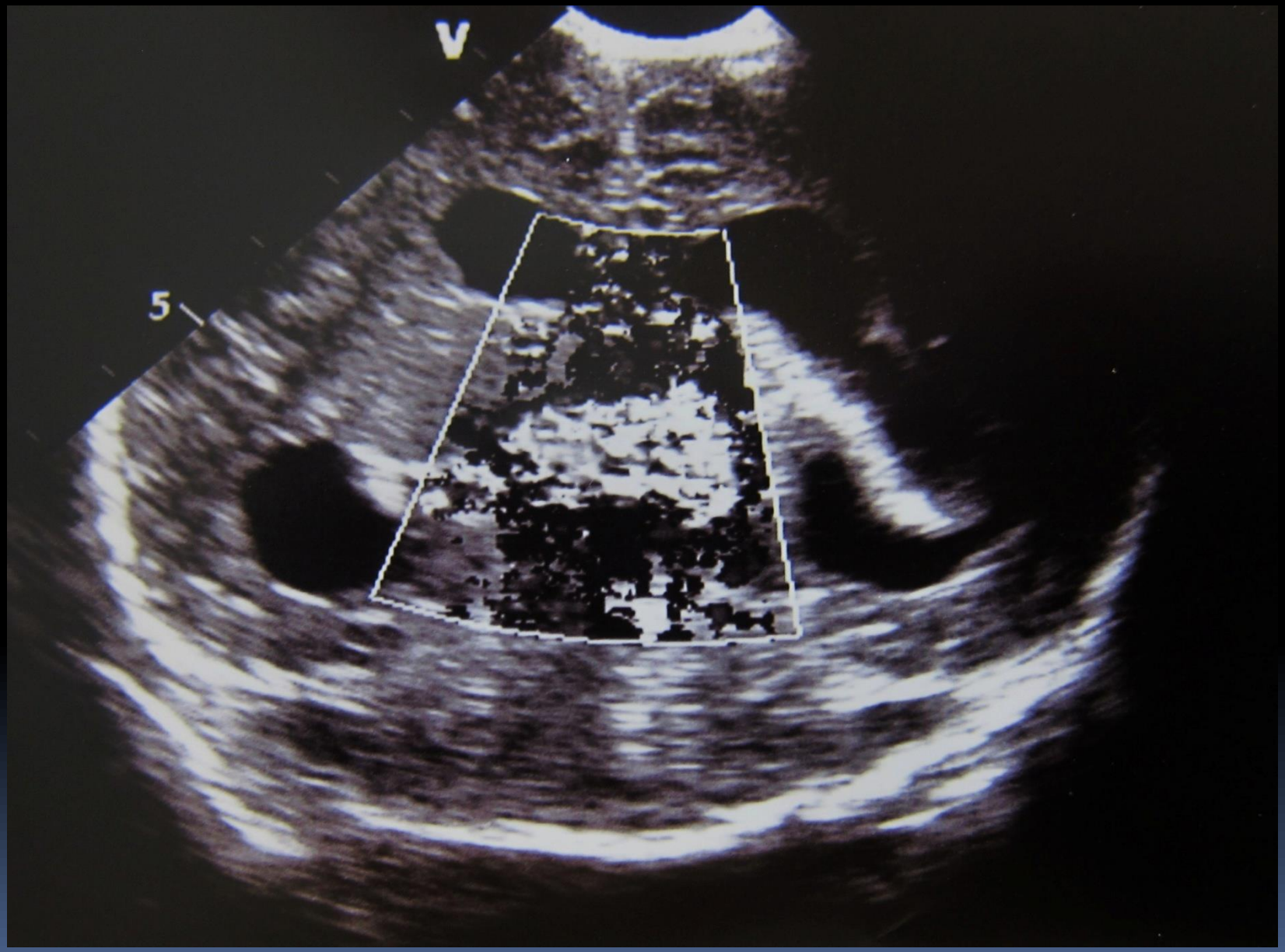
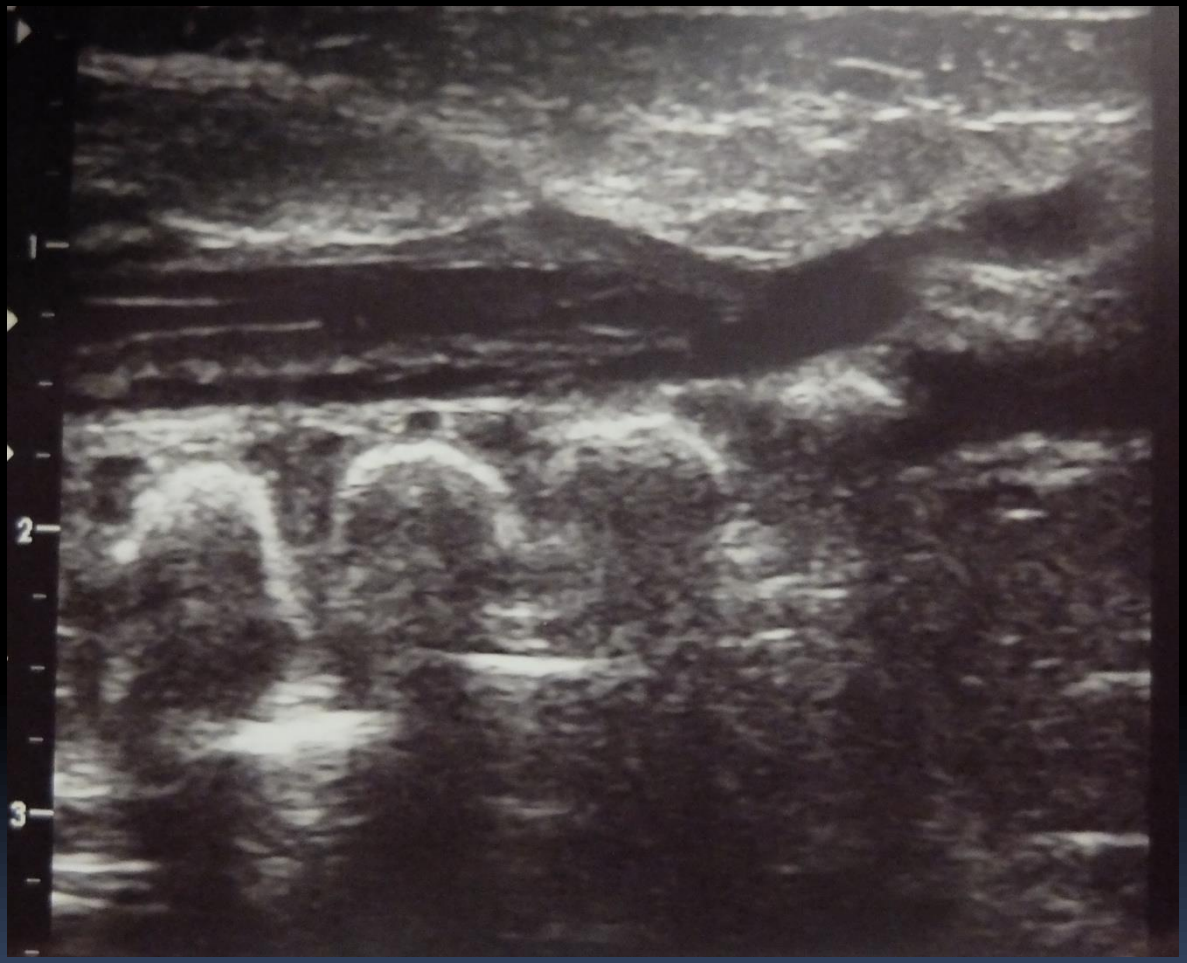
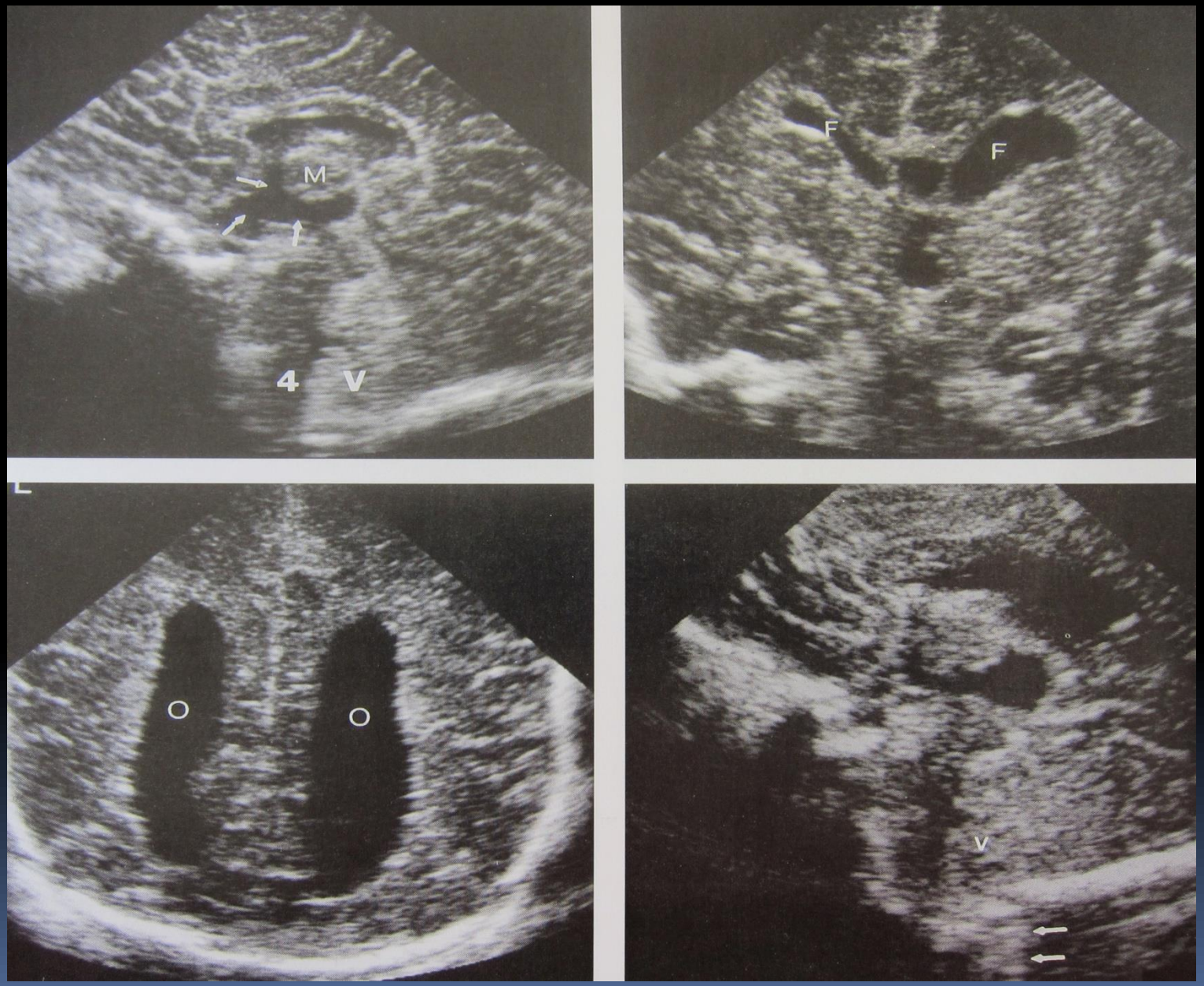
MI 0.34
TIs 0.2
5
10


V
5

DISORDERS OF NEURAL TUBE CLOSURE (3-4w)

- Anencephaly
- Encephalocele
- Meningocele
- Myelomeningocele
- Chiari Malformation Type I, II, and III
- Dandy-Walker Complex
- Anomalies of the Corpus Collosum


1
2
3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4 V
F
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O
O
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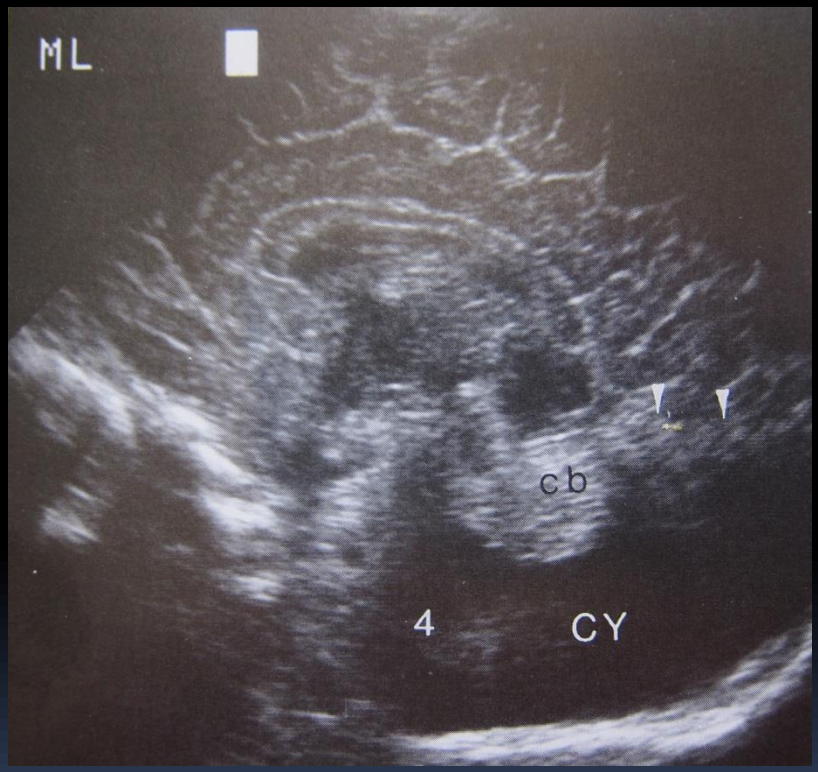
ML
cb
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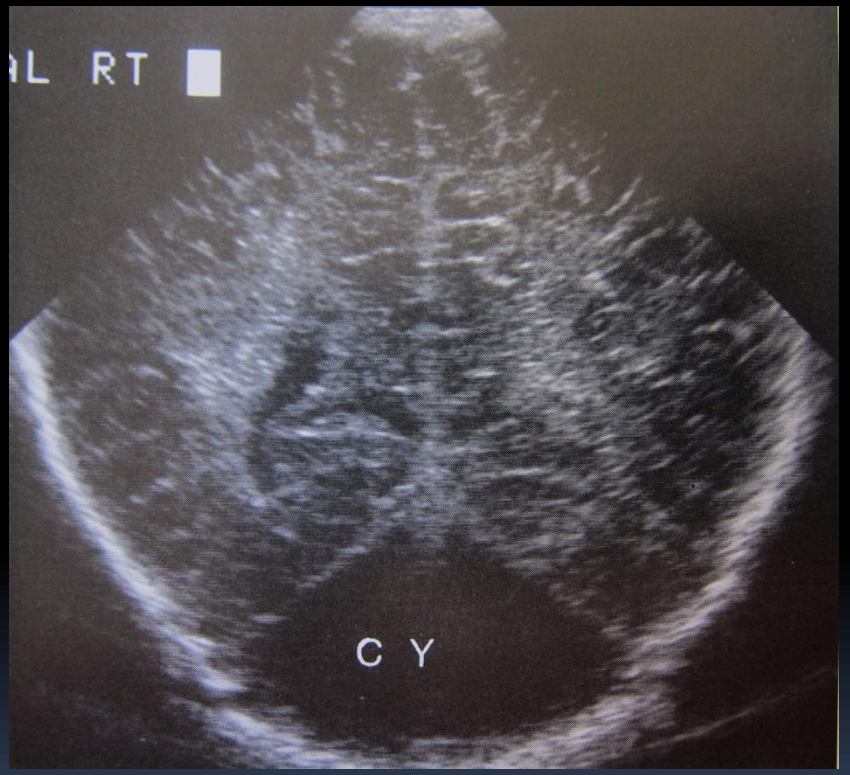
RT
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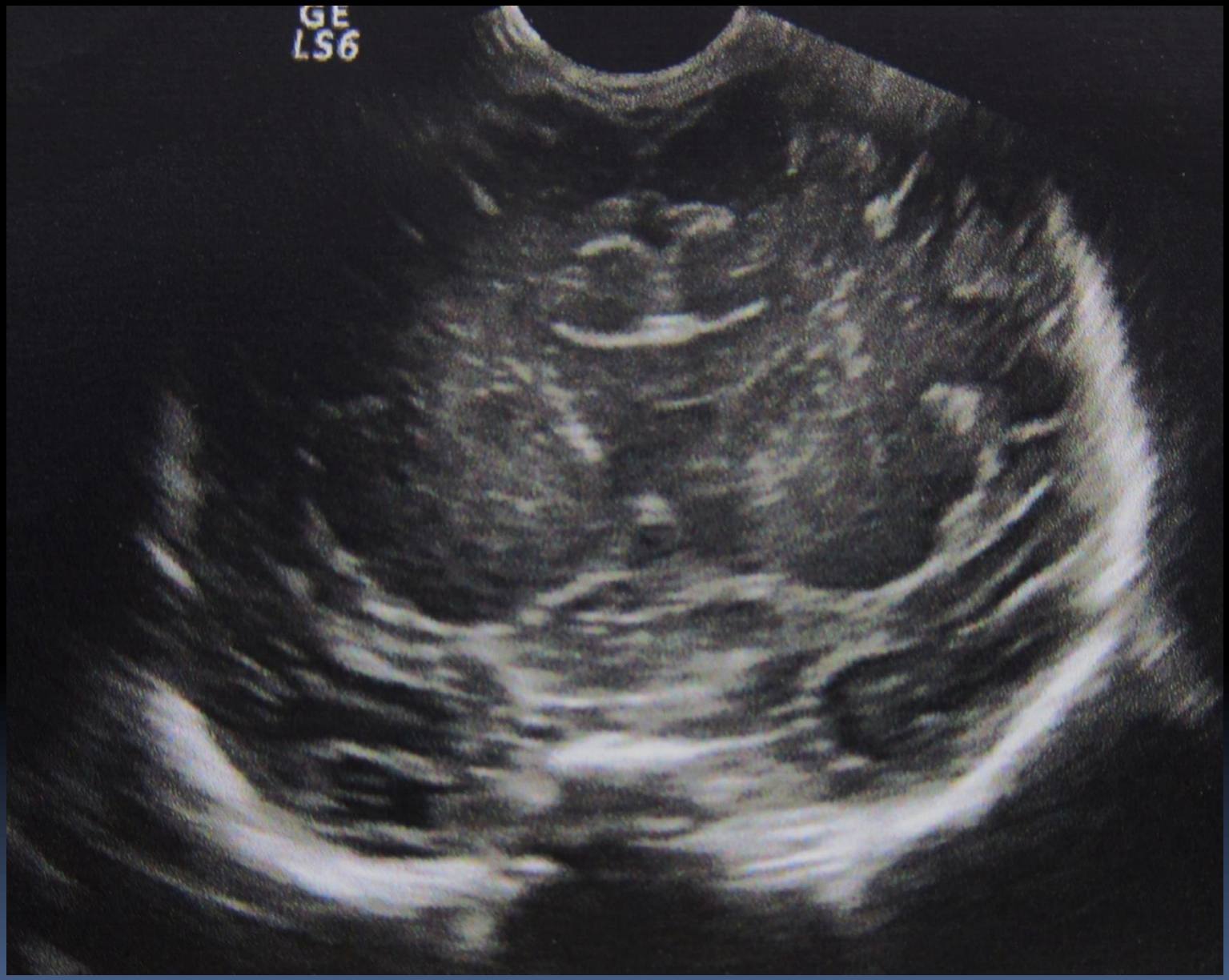
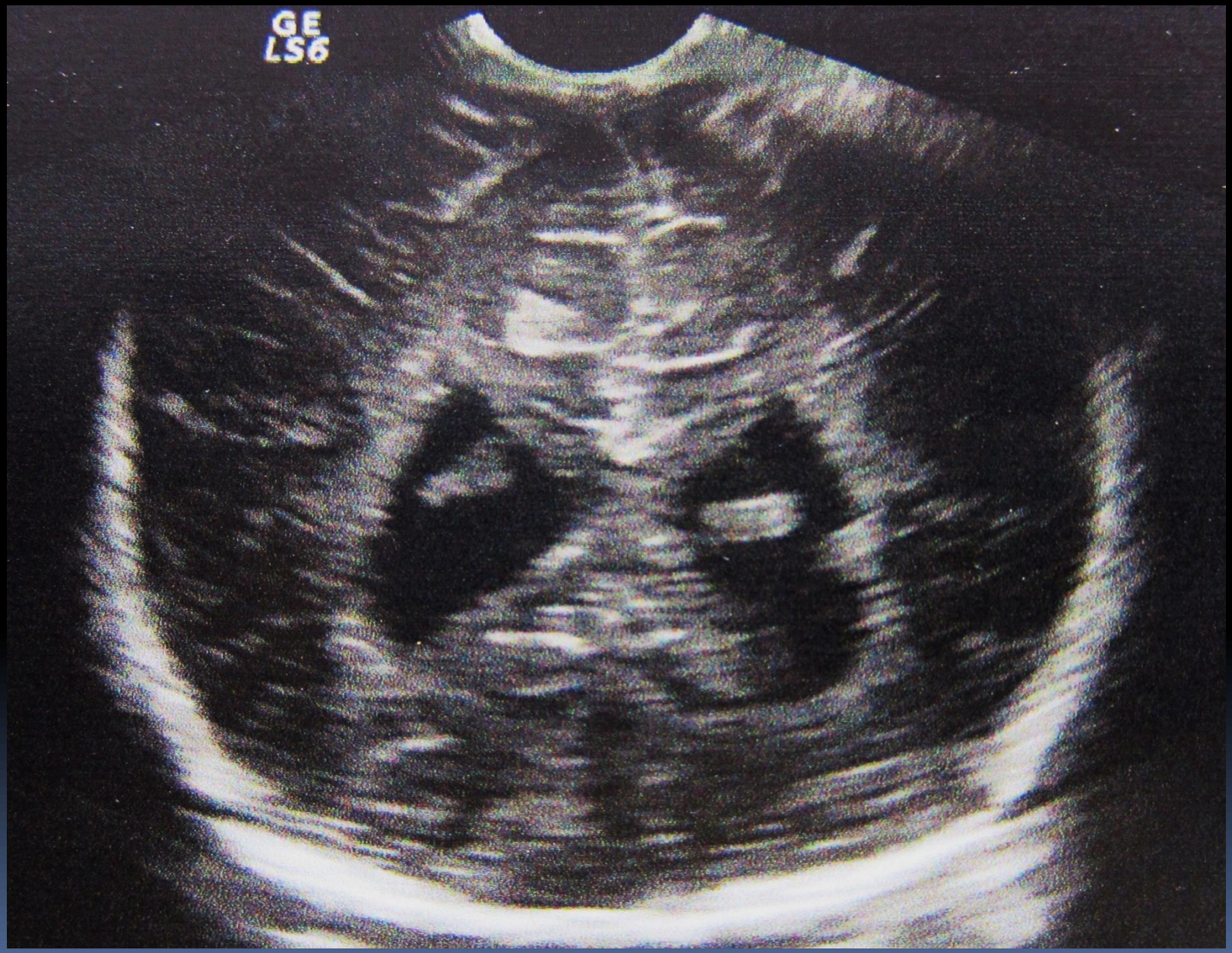

GE
LS6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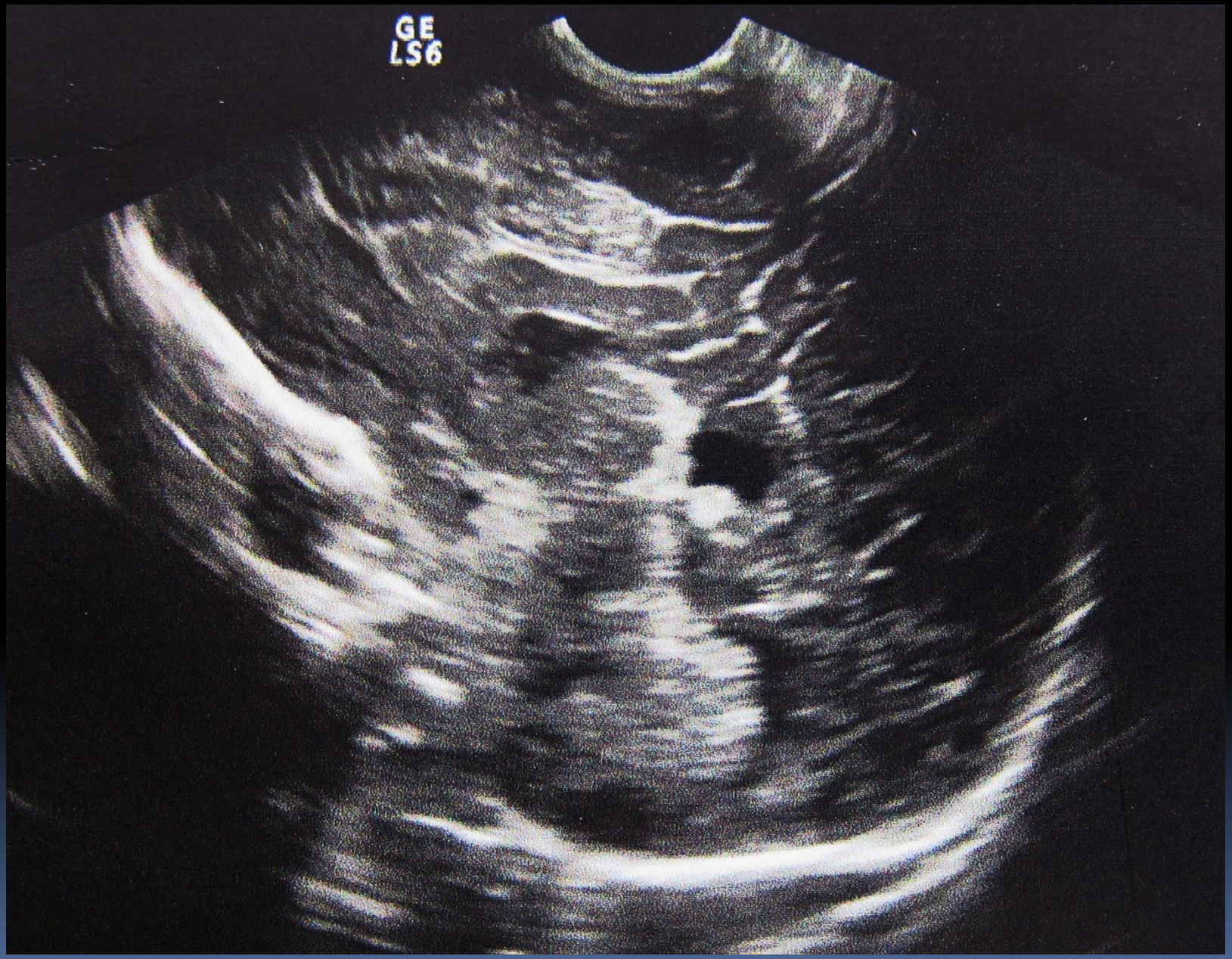
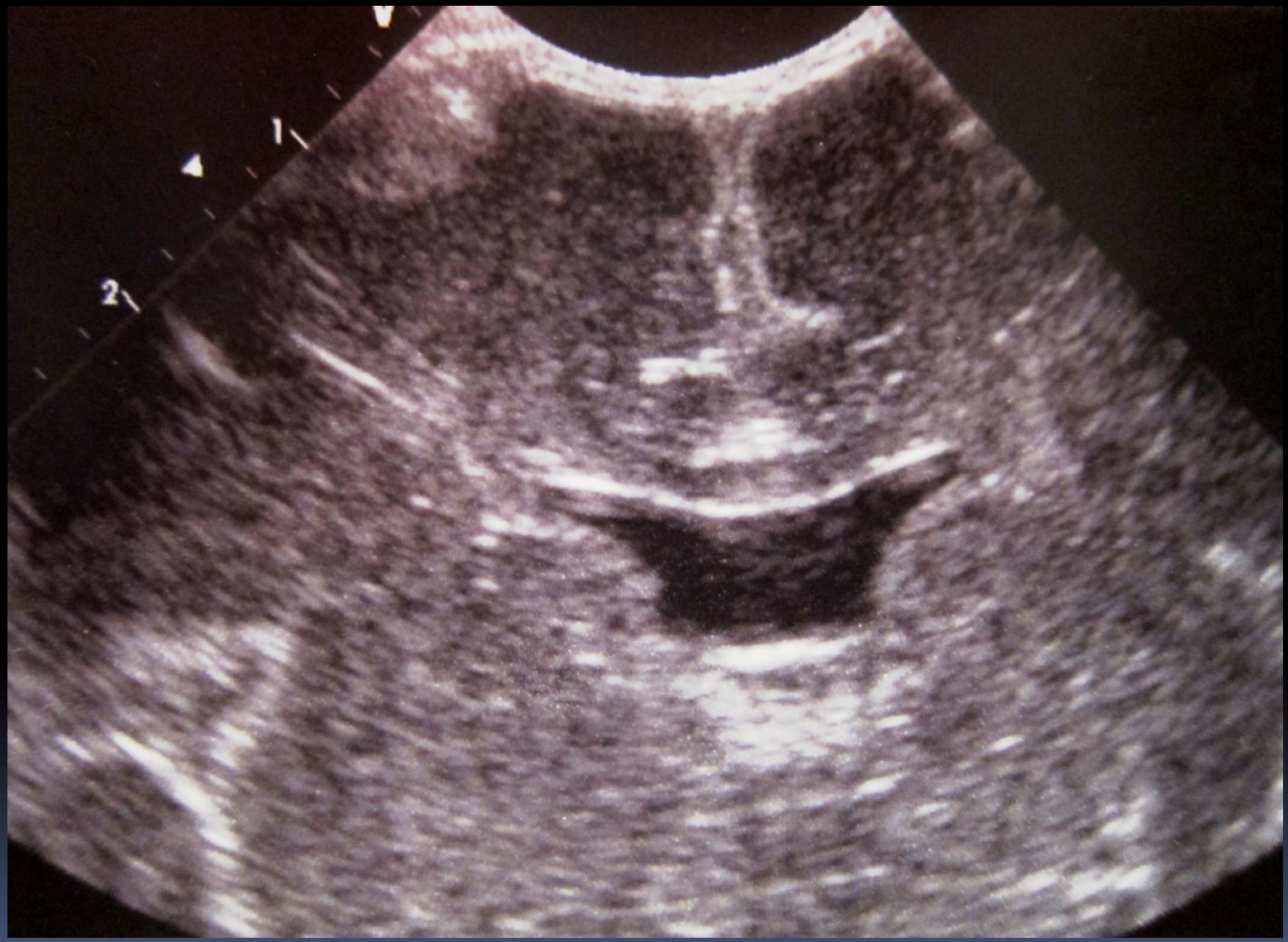

GE
LS6

DISORDERS OF DIVERTICULATION (5-10w)

Holoprosencephaly

- Lobar
- Semilobar
- A Lobar


1
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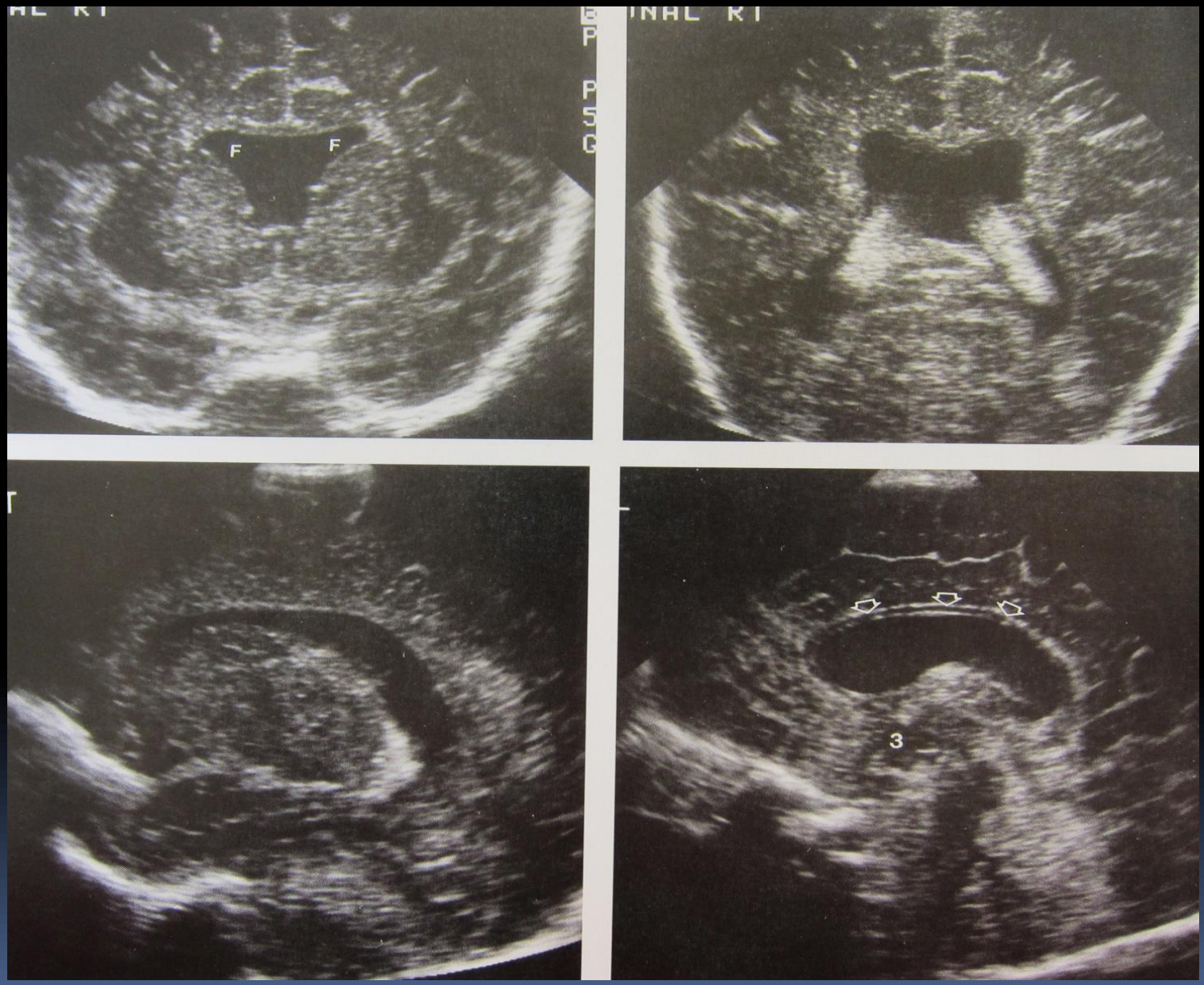
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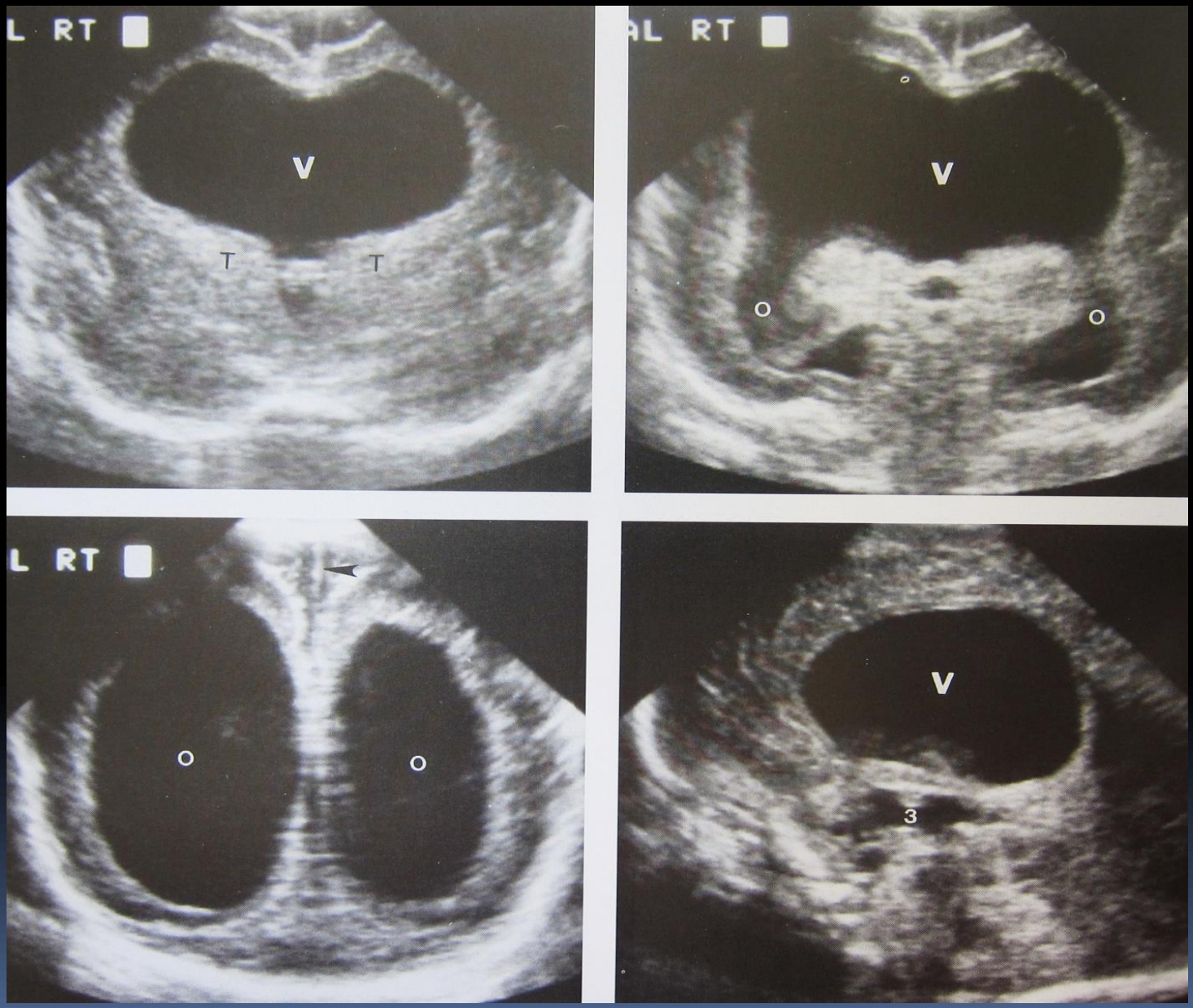
3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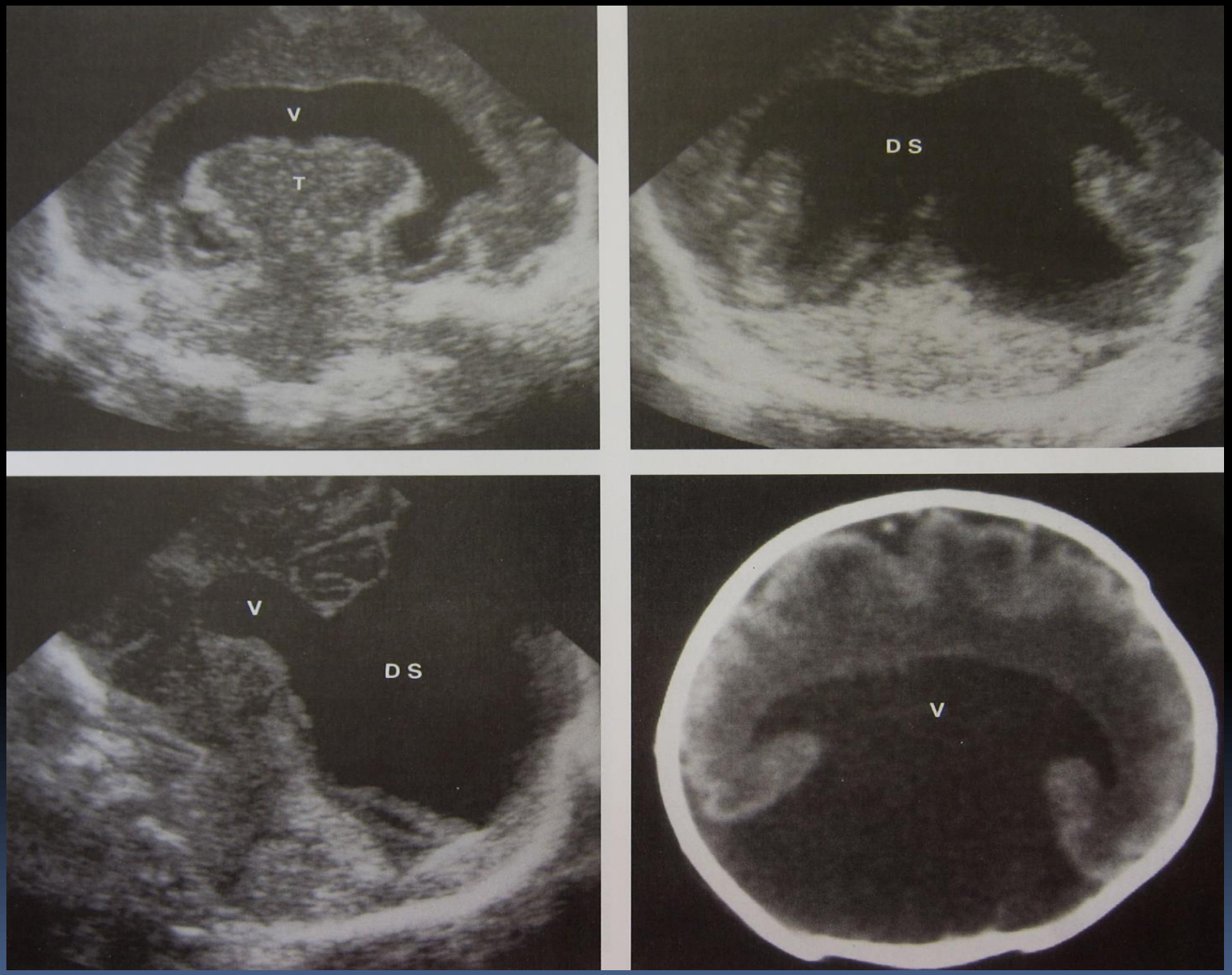
V
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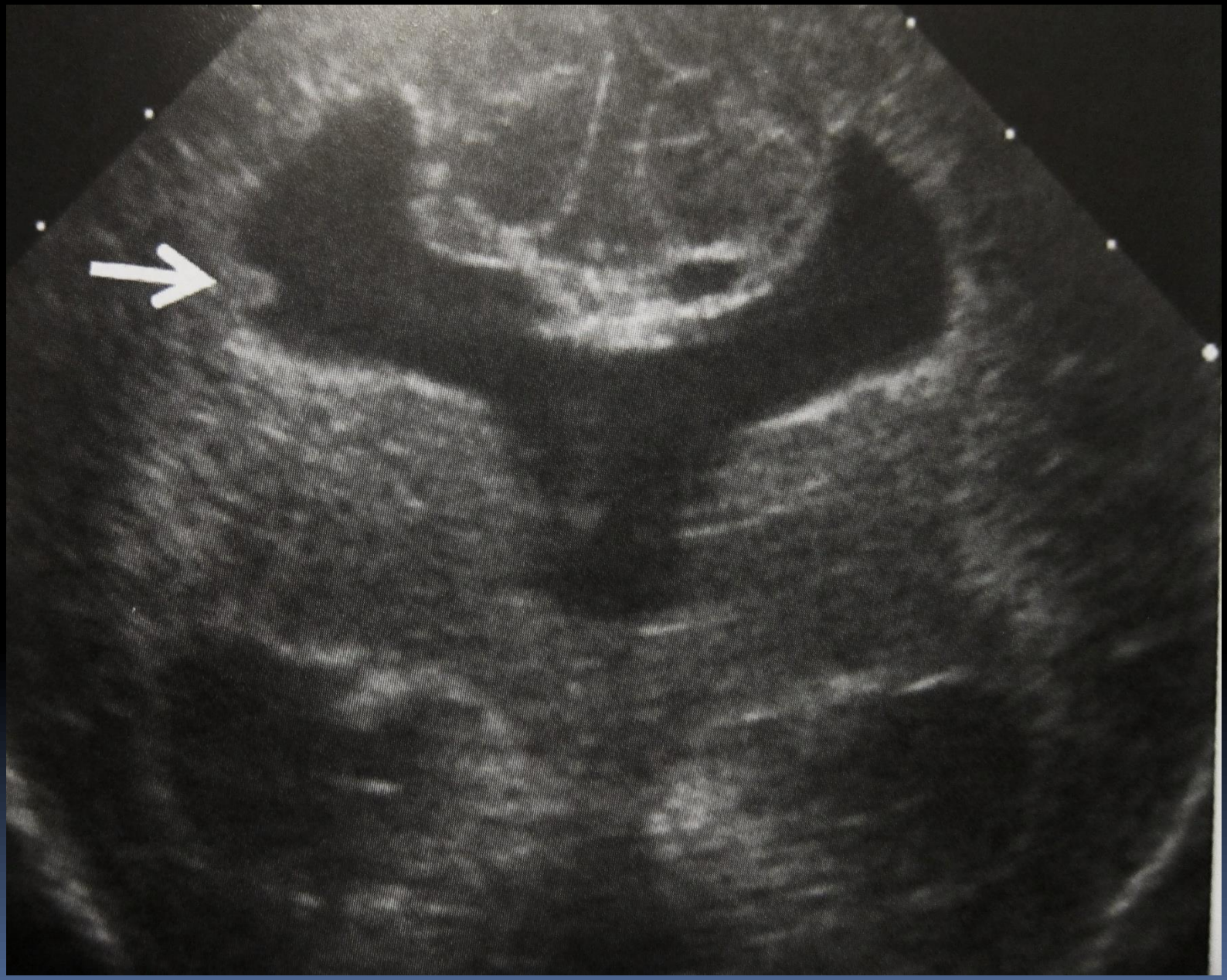
DS
V
DS
V

DISORDERS OF MIGRATION, SULCATION AND PROFILARATION (2-6 months)

- Hemimegalencephaly
- Lissencephaly
- Schizencephaly
- Heterotopic Gray Matter
- Polymicrogyria

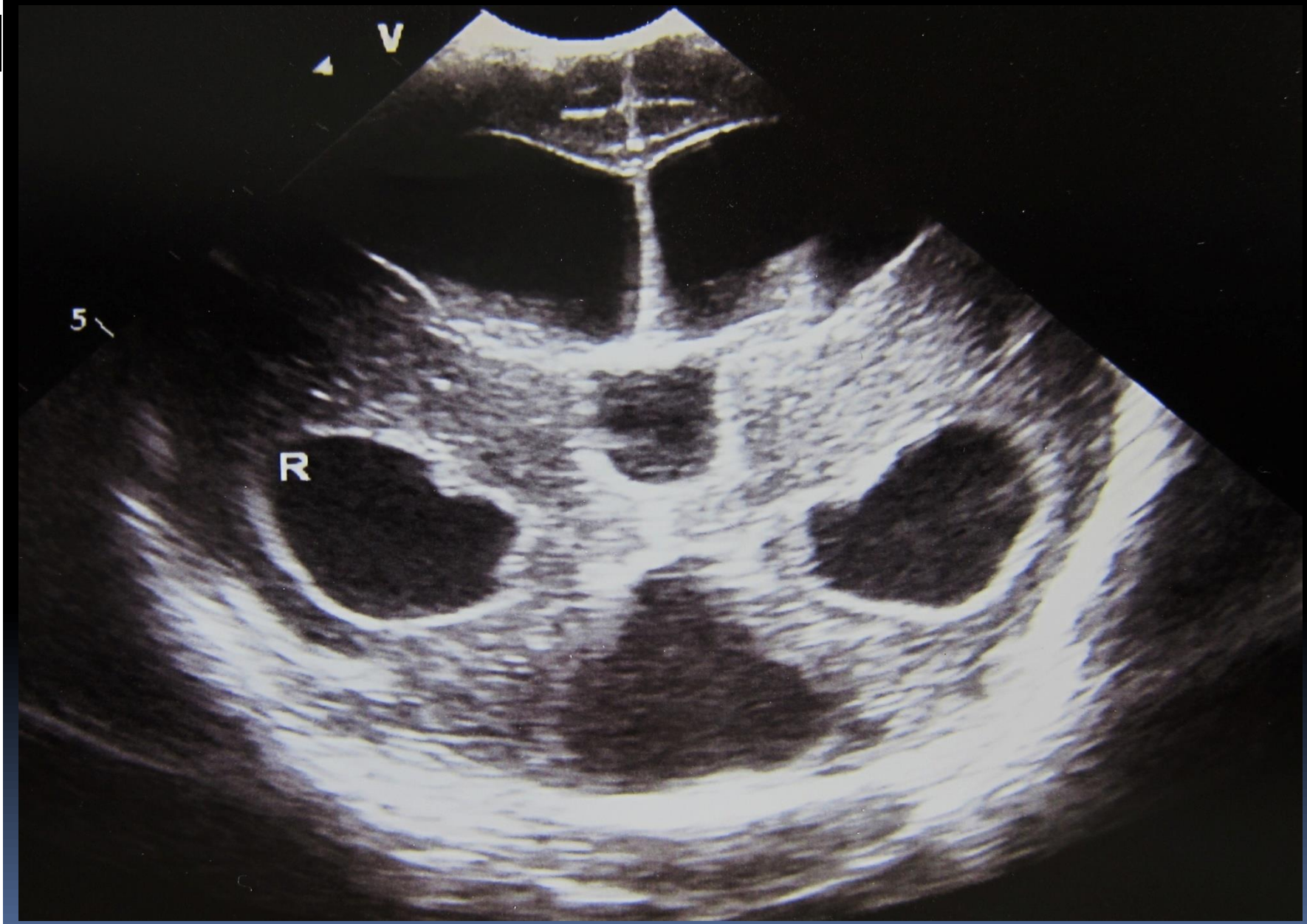


DISORDERS OF MYELINATION

(7months until 2 years postnatal)

HYDROCEPHALUS

- Normal Cerebrospinal Fluid Production
- Disturbances In Cerebrospinal Fluid Production and Circulation
- Sonographic Diagnosis
- Doppler Interrogation
- Bening Infantile Hydrocephalus


V
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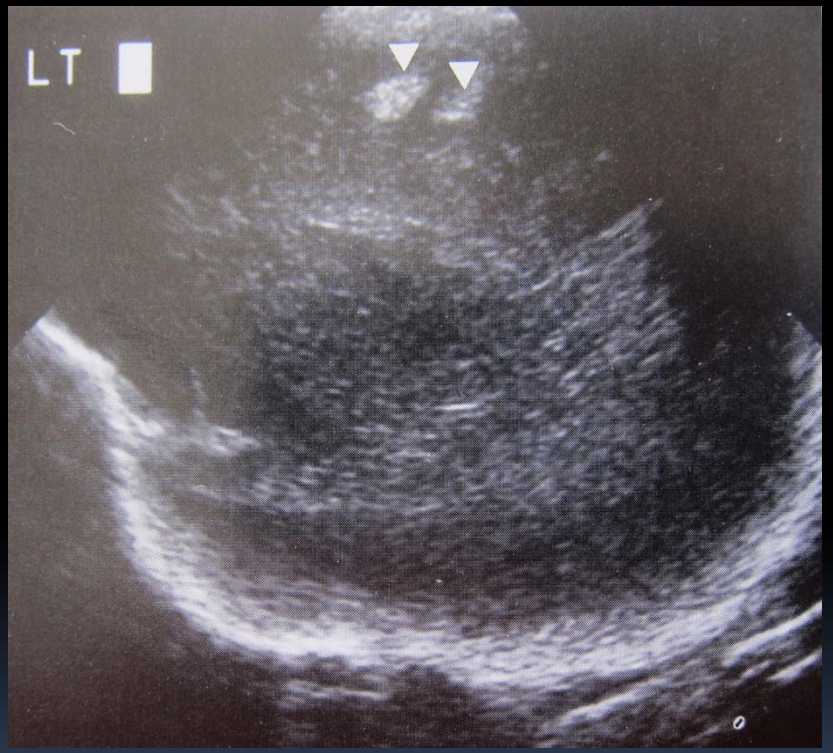
R

INTRACRANIAL INFECTIONS

- Congenital Infections
- Neonatal Meningitis


AL RT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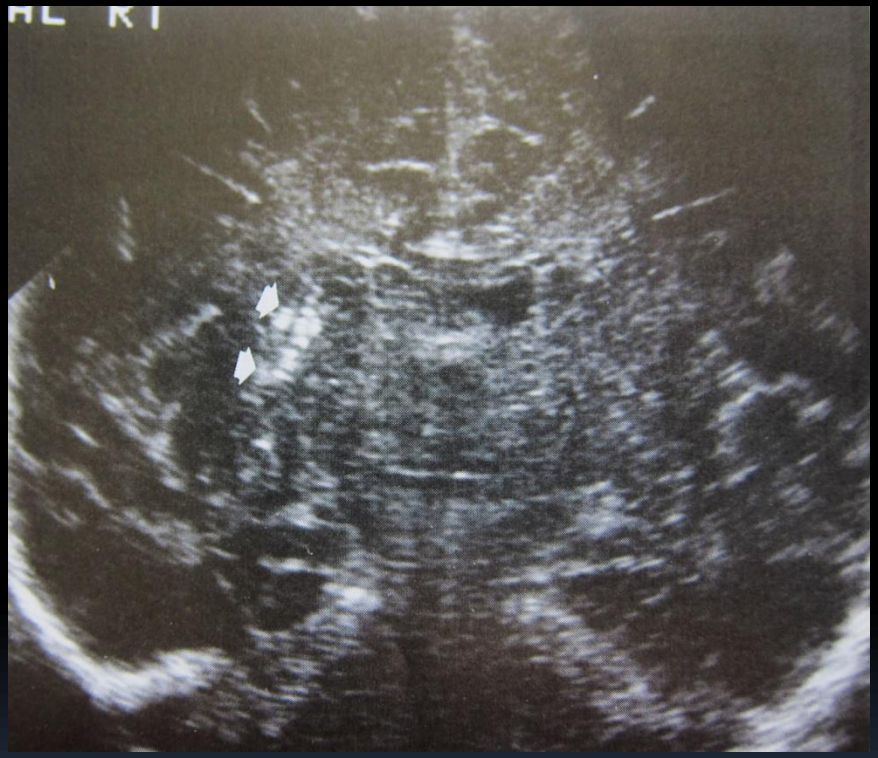

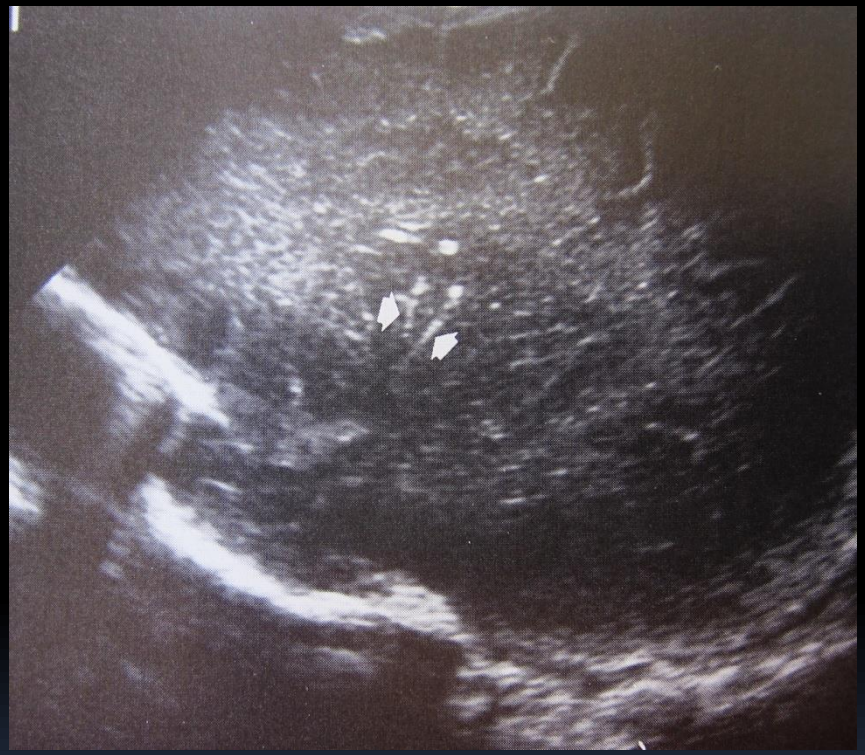

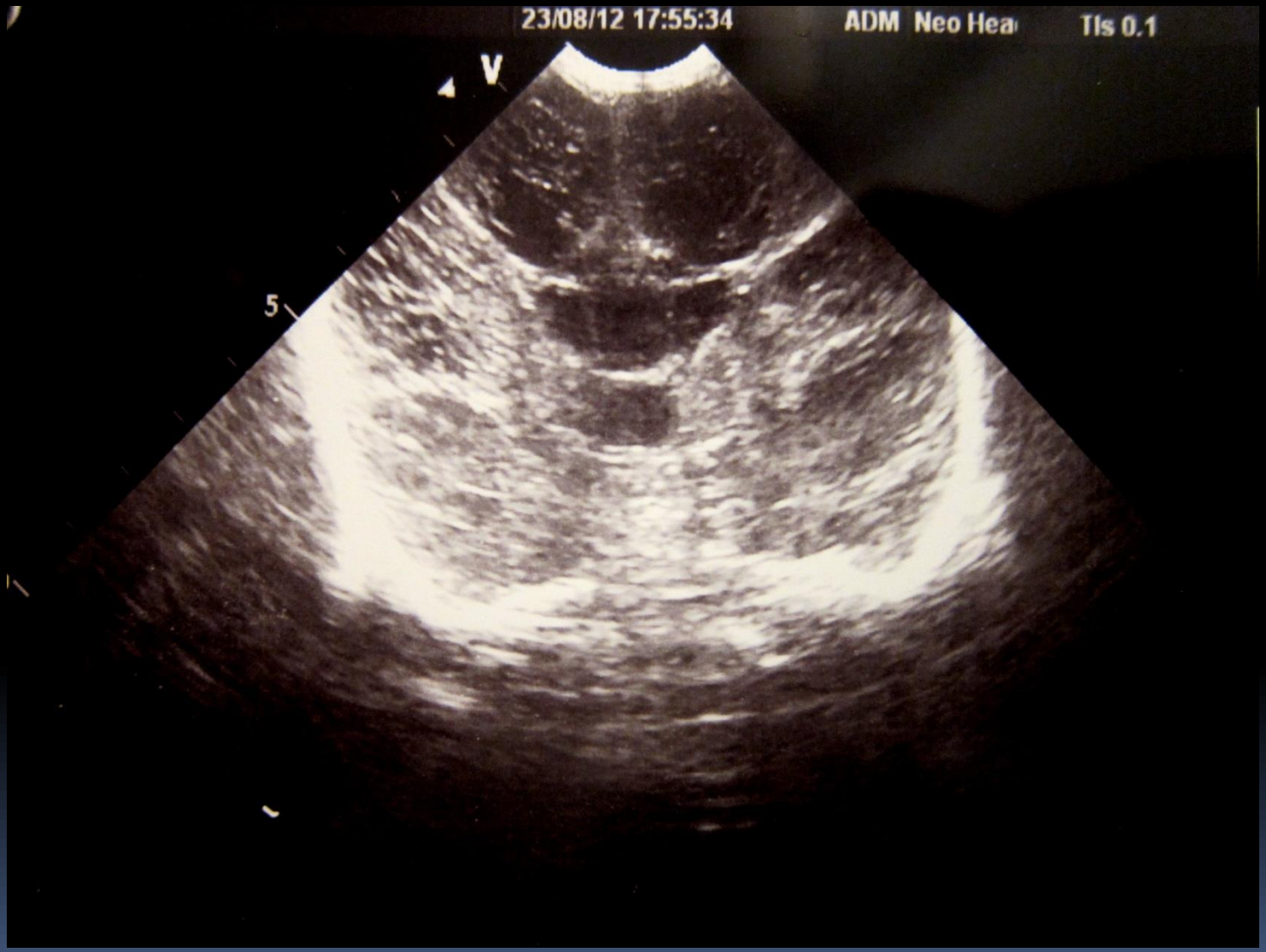
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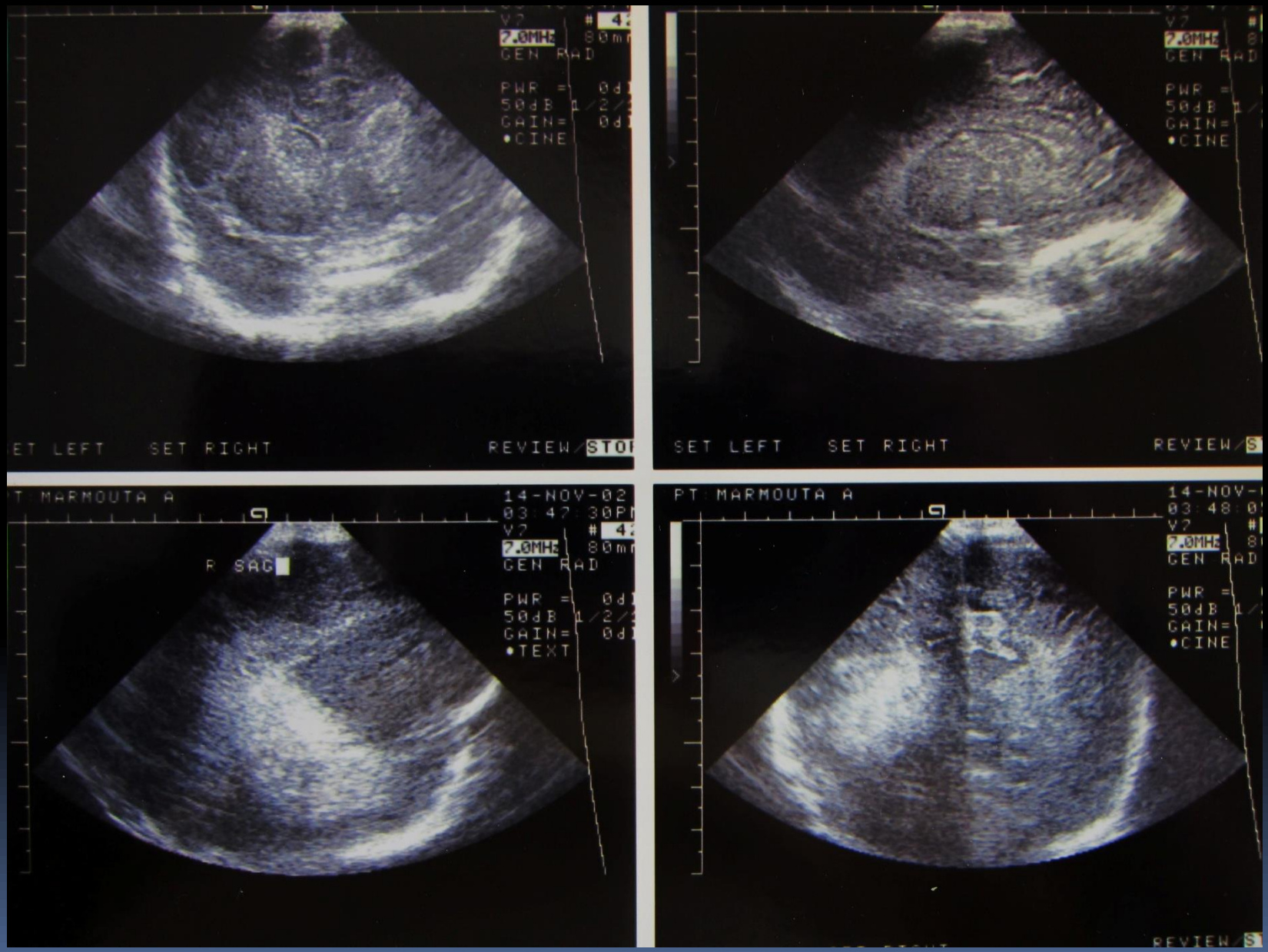
5


22/12/10 17:27:22
USR Neo Hea
Tls 0.1
V
5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50dB
GAIN=
•CINE
SET LEFT
SET RIGHT
REVIEW
PT MARMOUTA A
14-NOV-02
03:47:30PM
R SAG
•TEXT
14-NOV-
03:48:0


ICU-IASO
25/05/15 12:49:18
8C RS
USR SmallPar
MI 0.5
TIs 0.3
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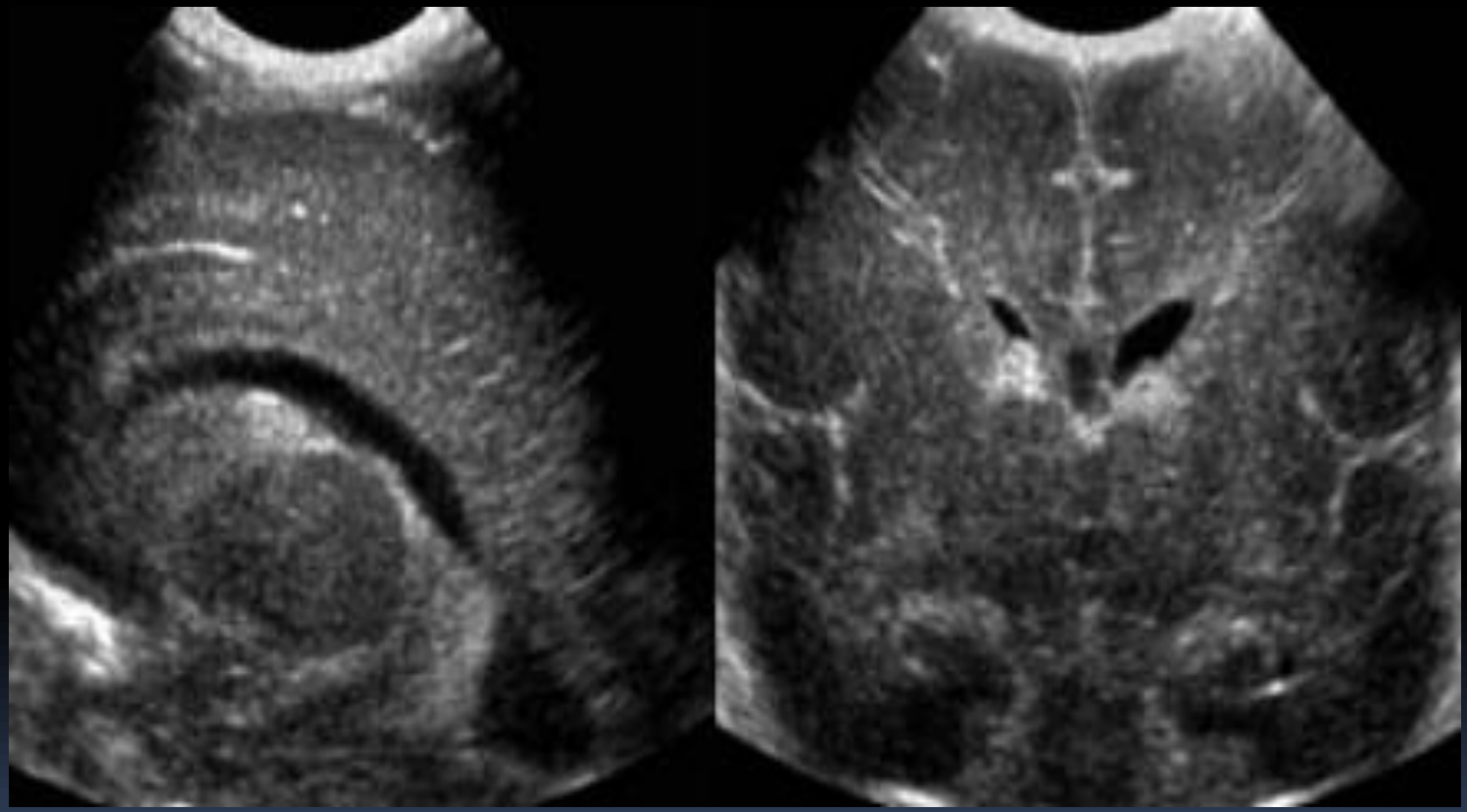

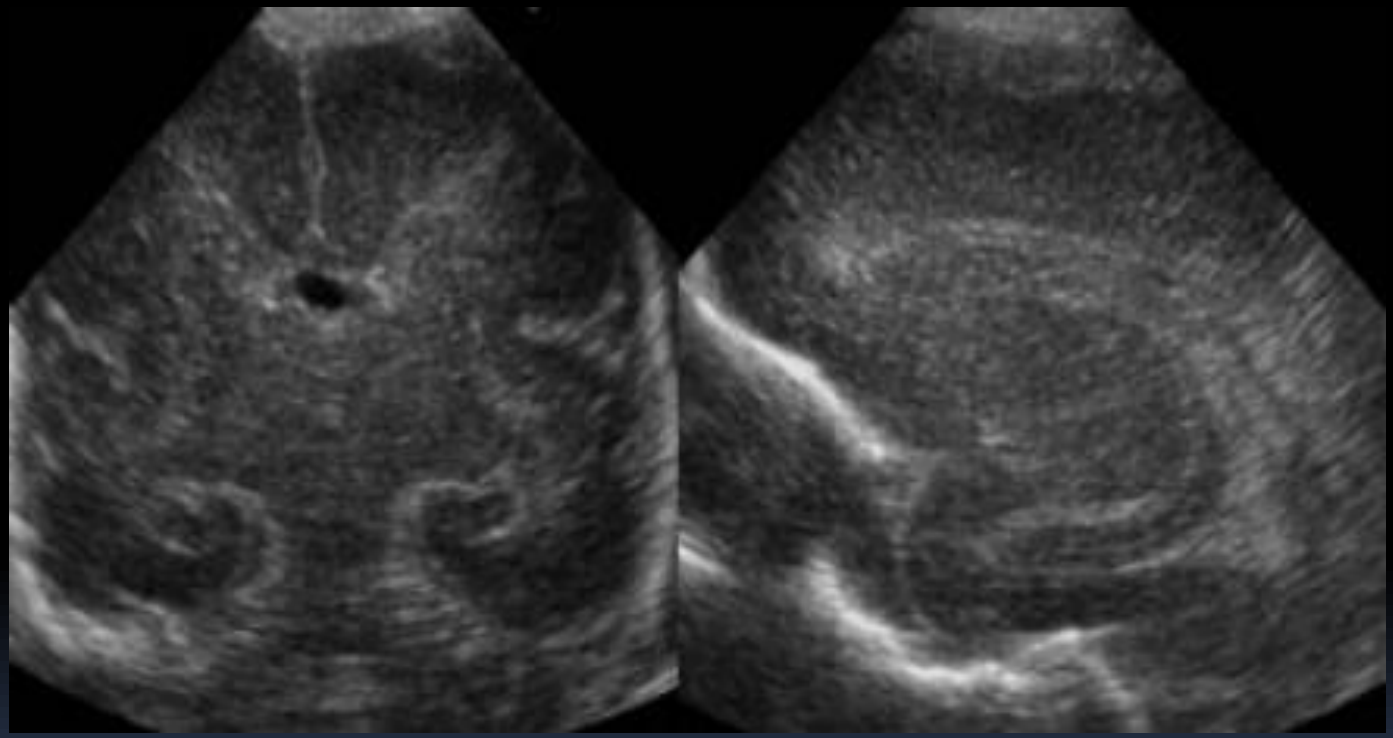

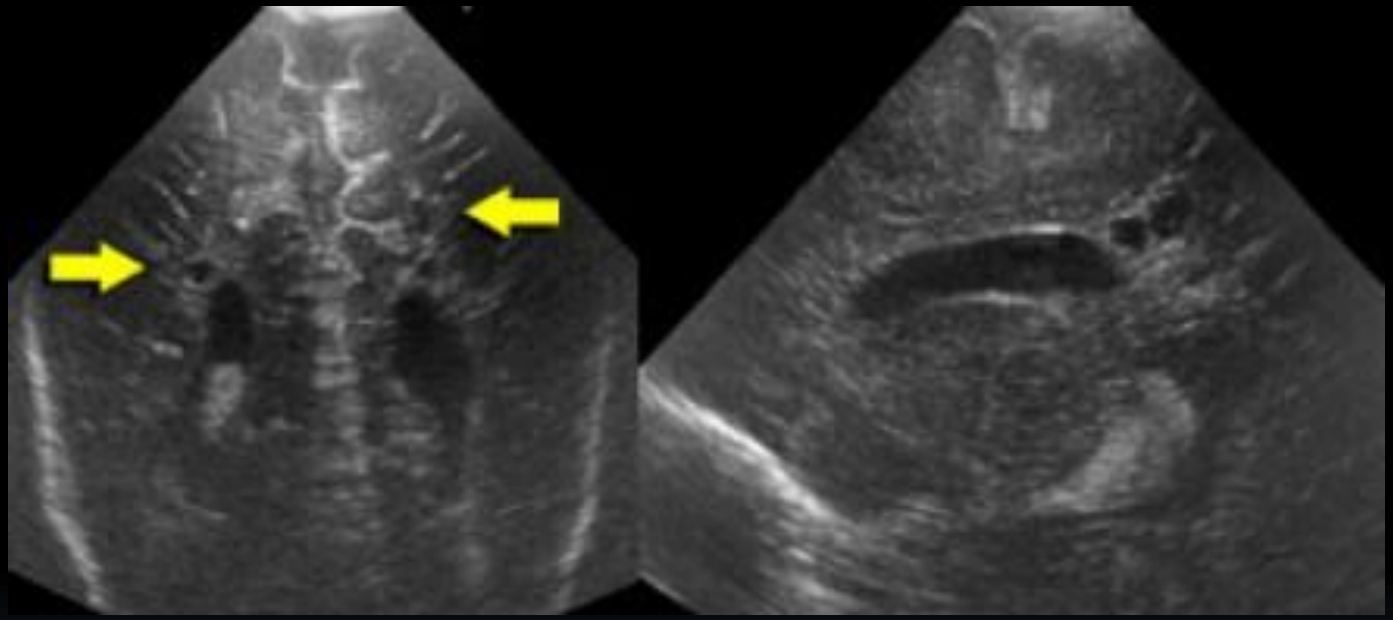

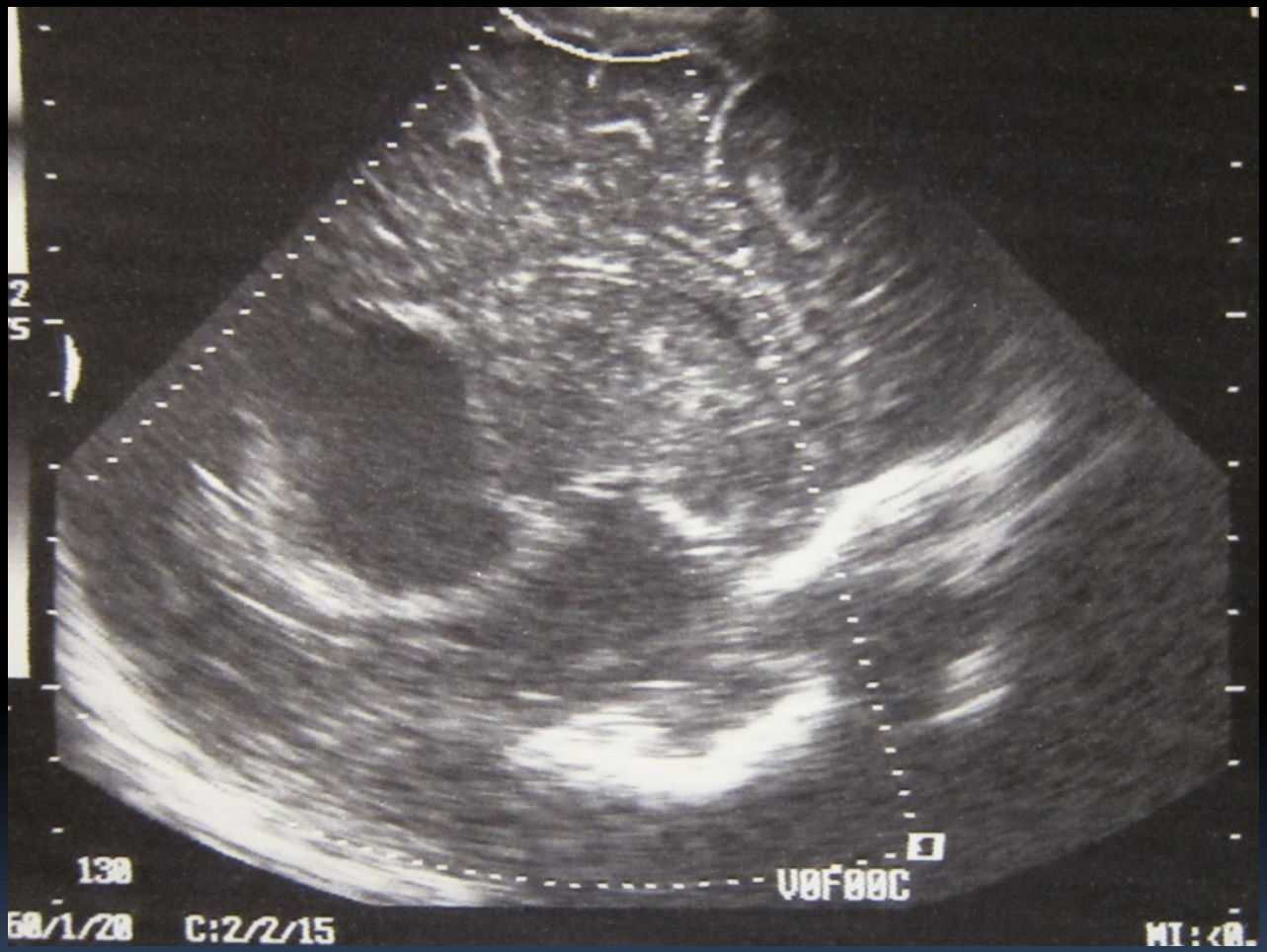
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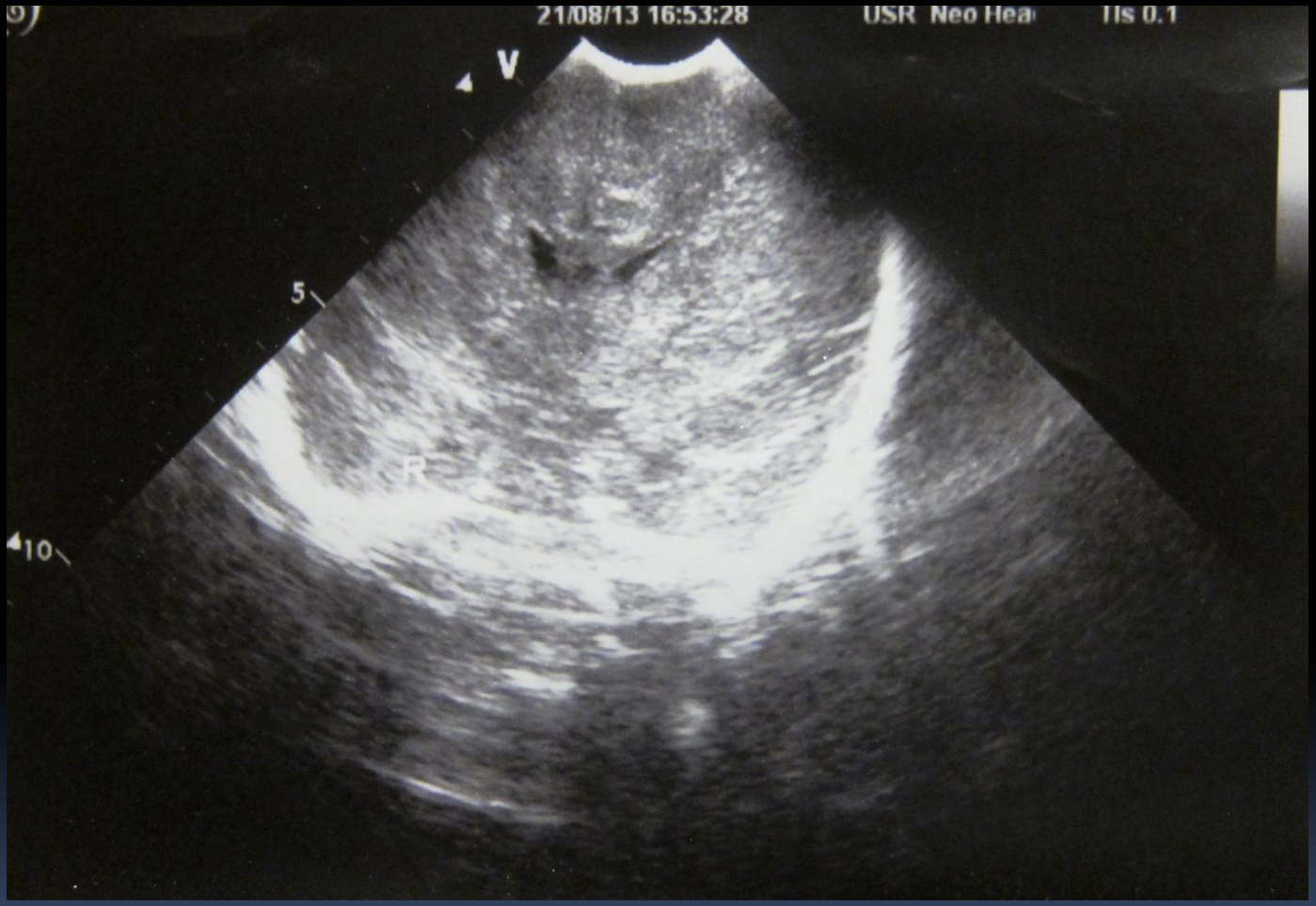
138
V8100C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V
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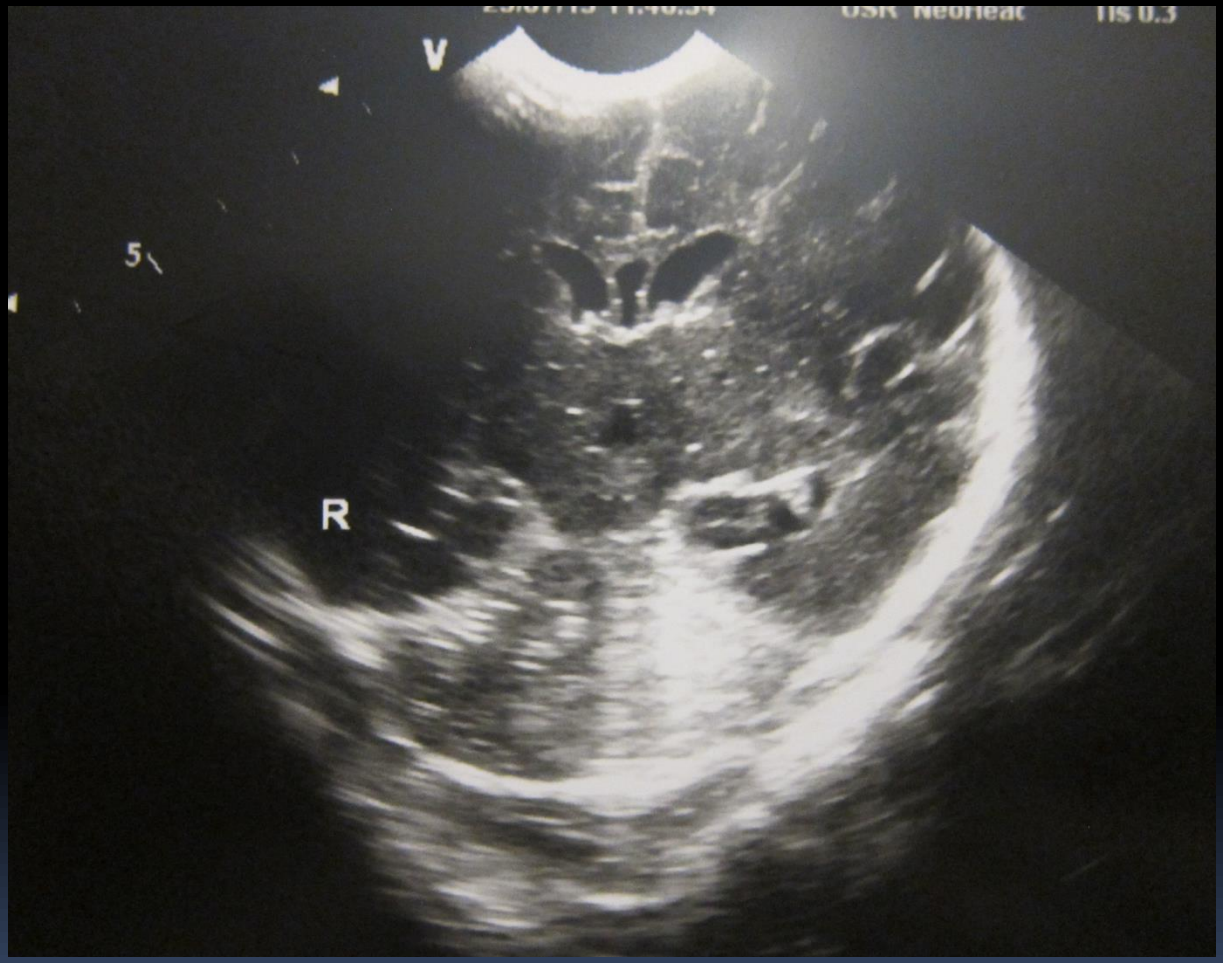
10


V
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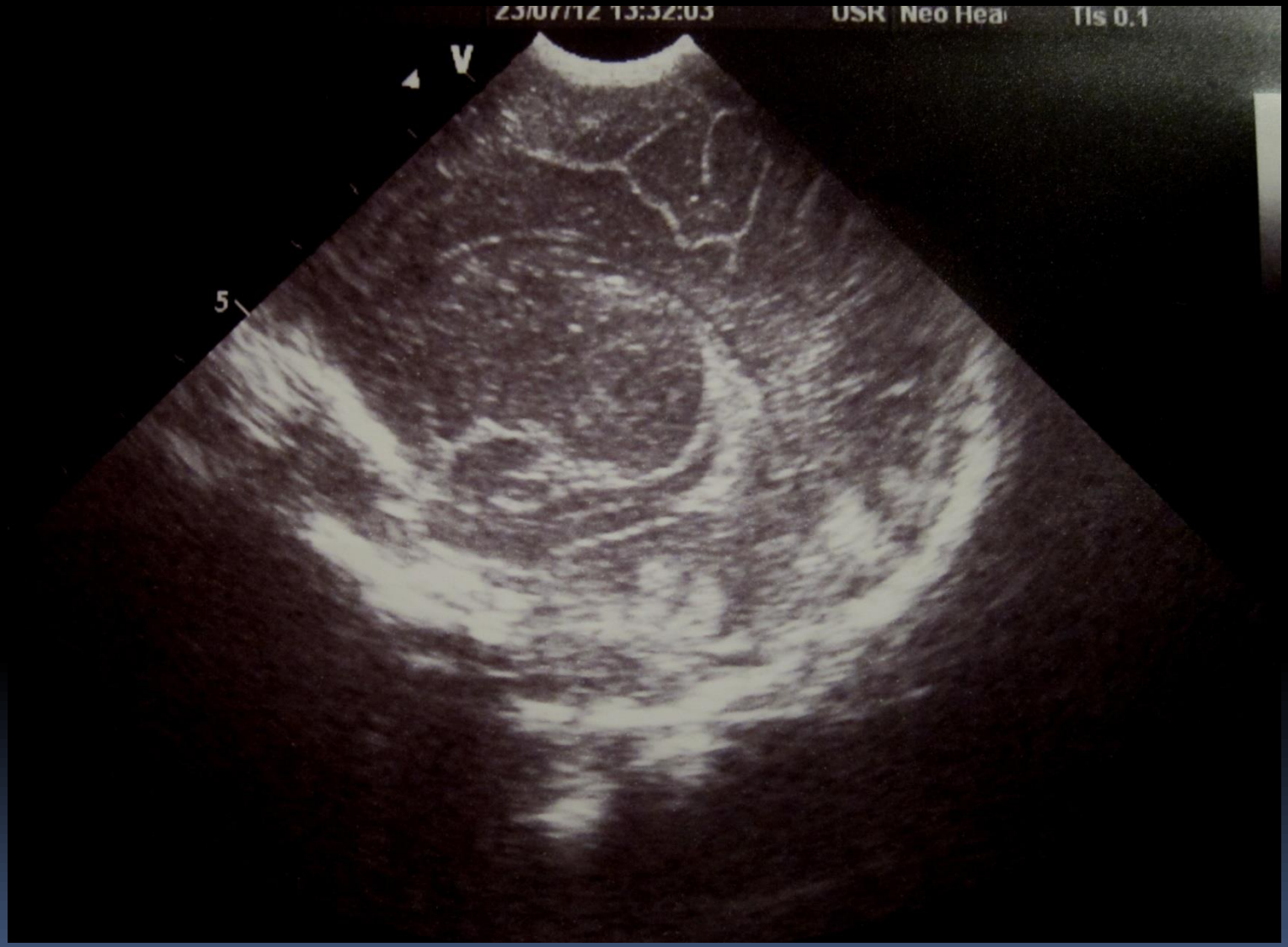
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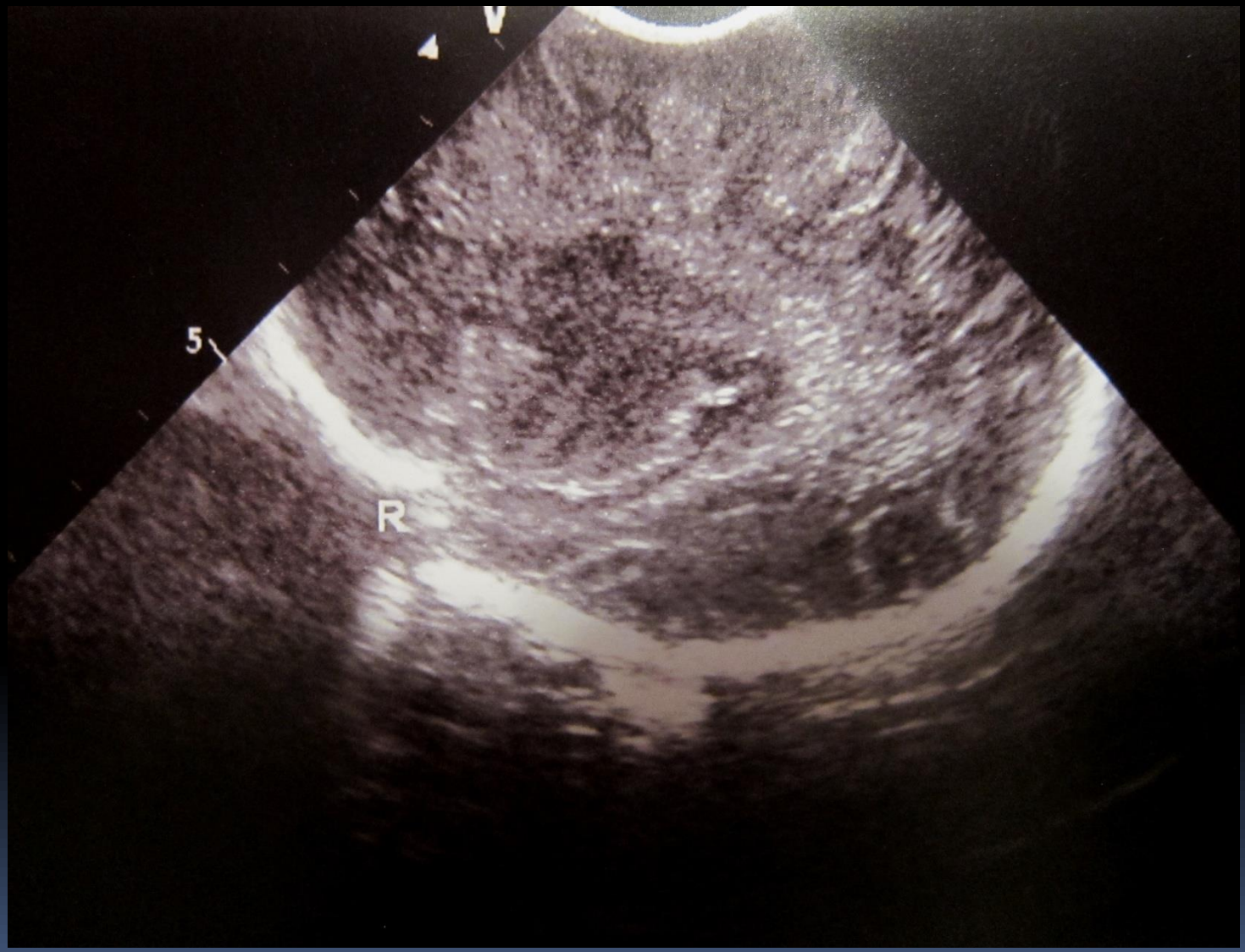
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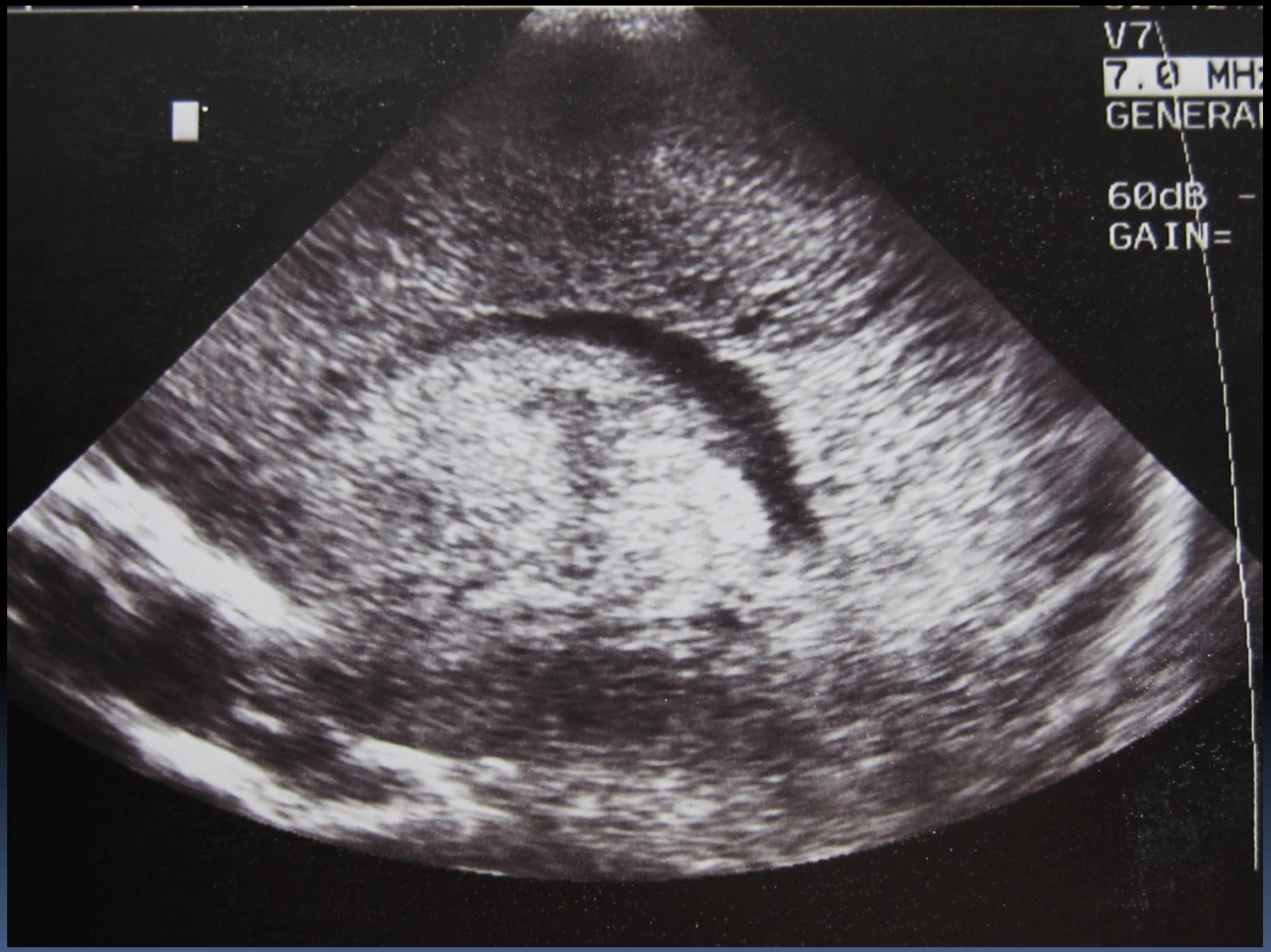
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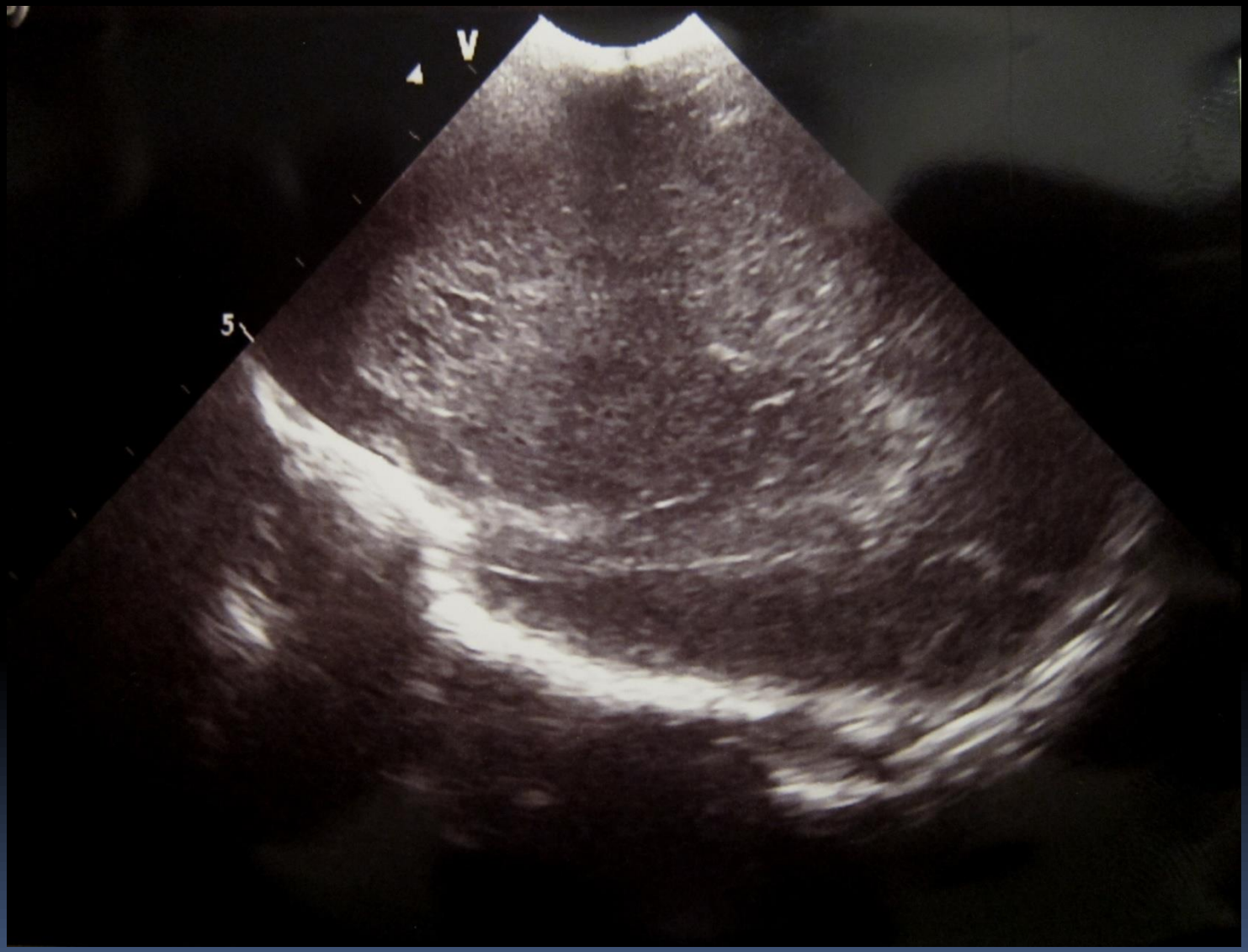
GAIN=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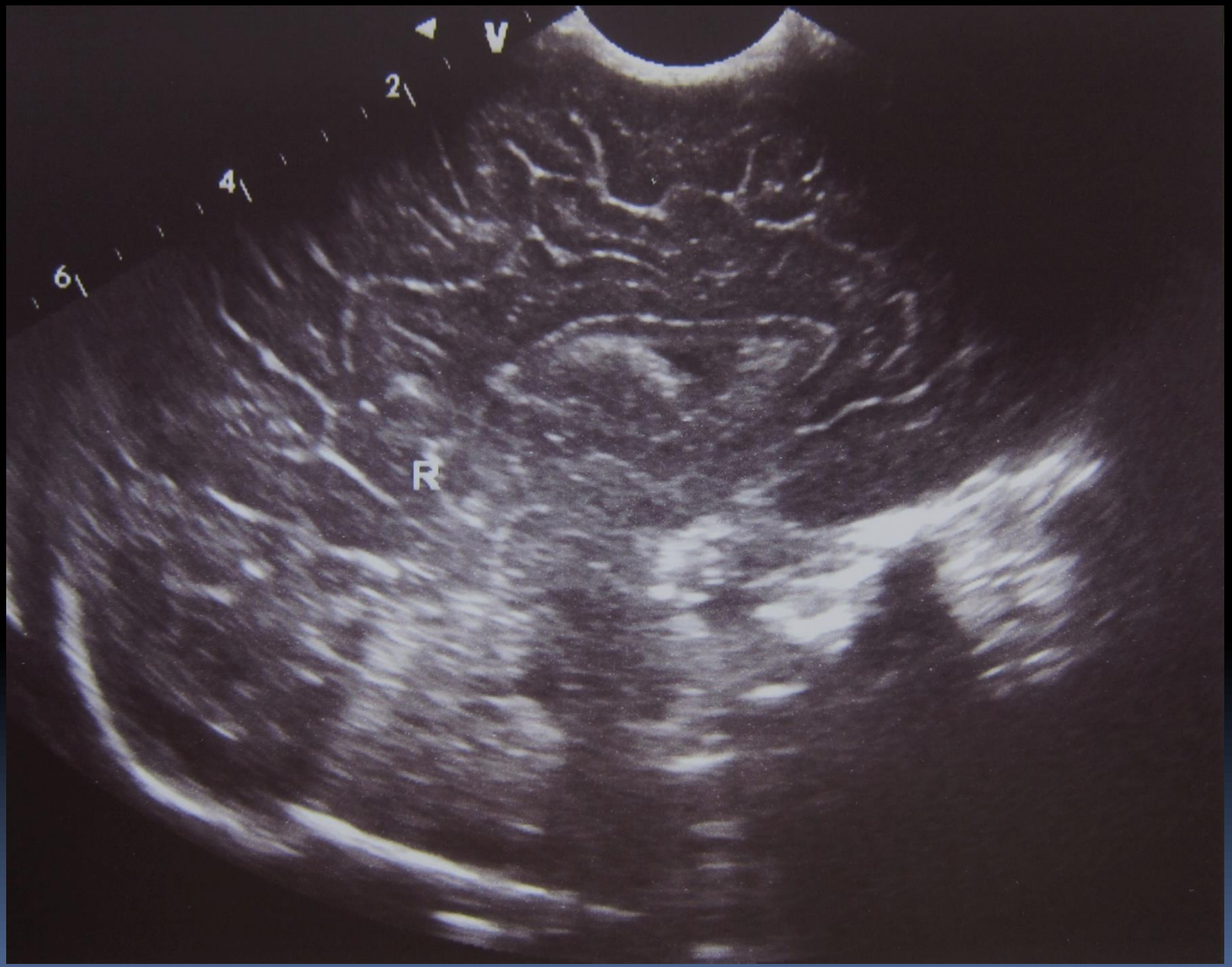
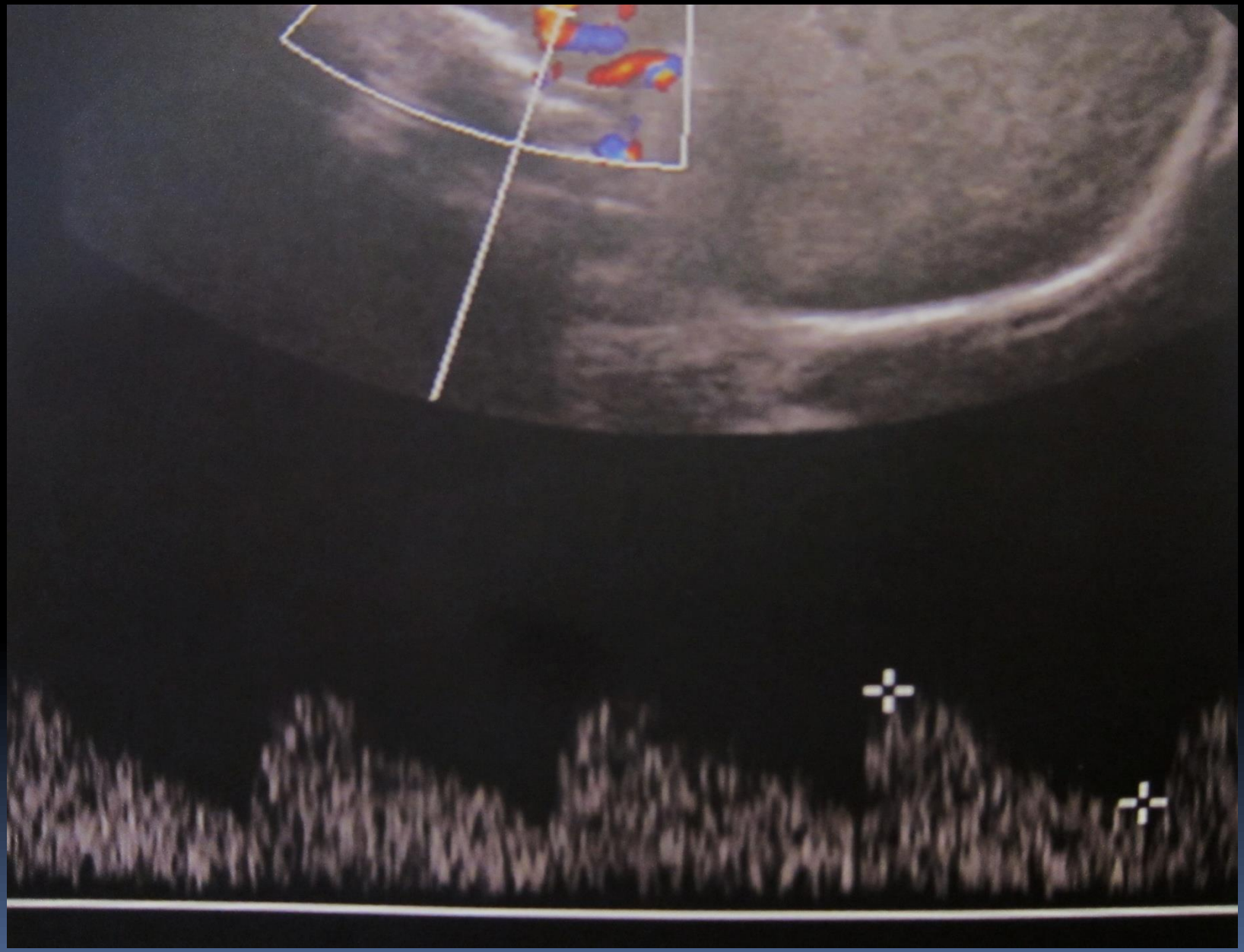
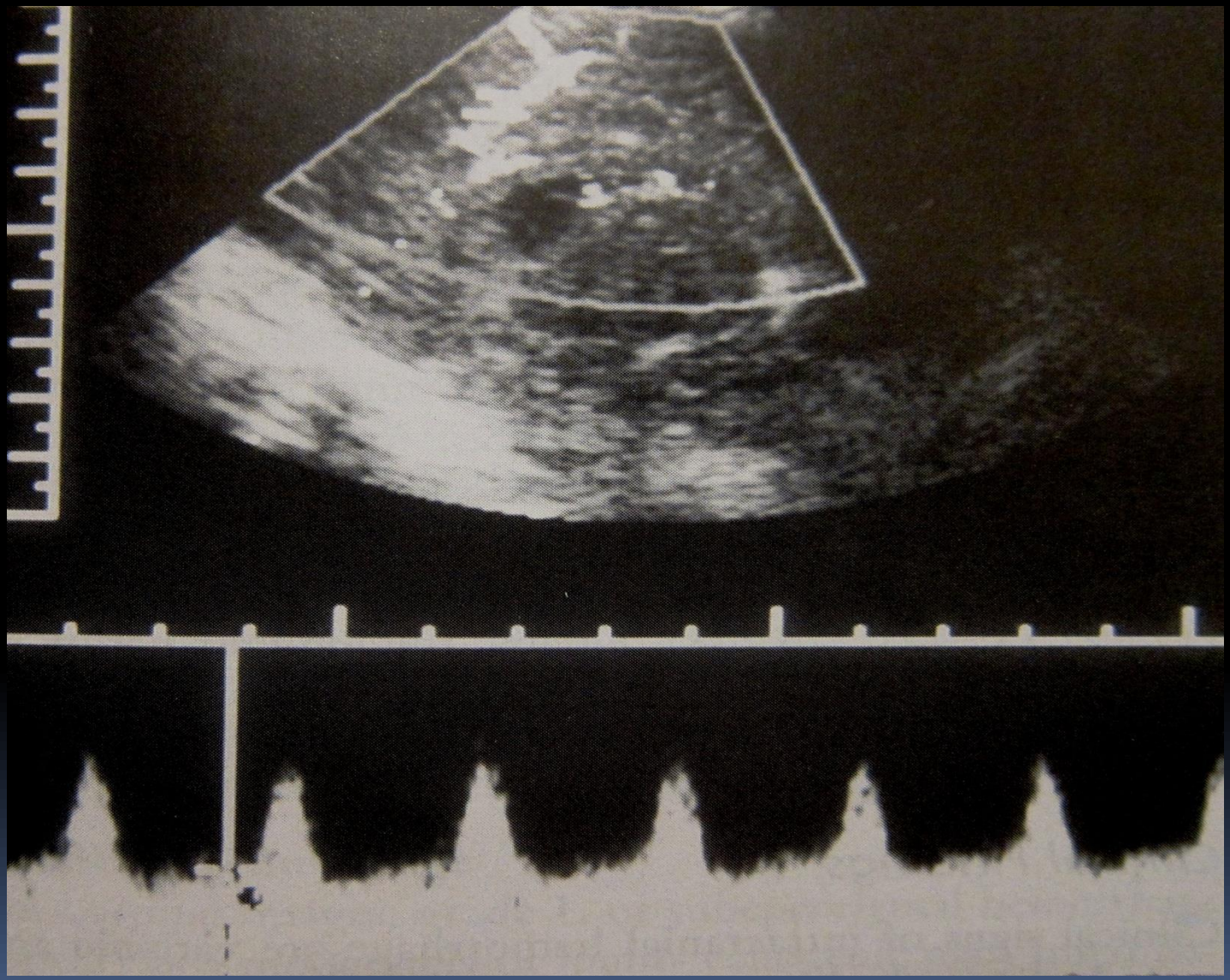
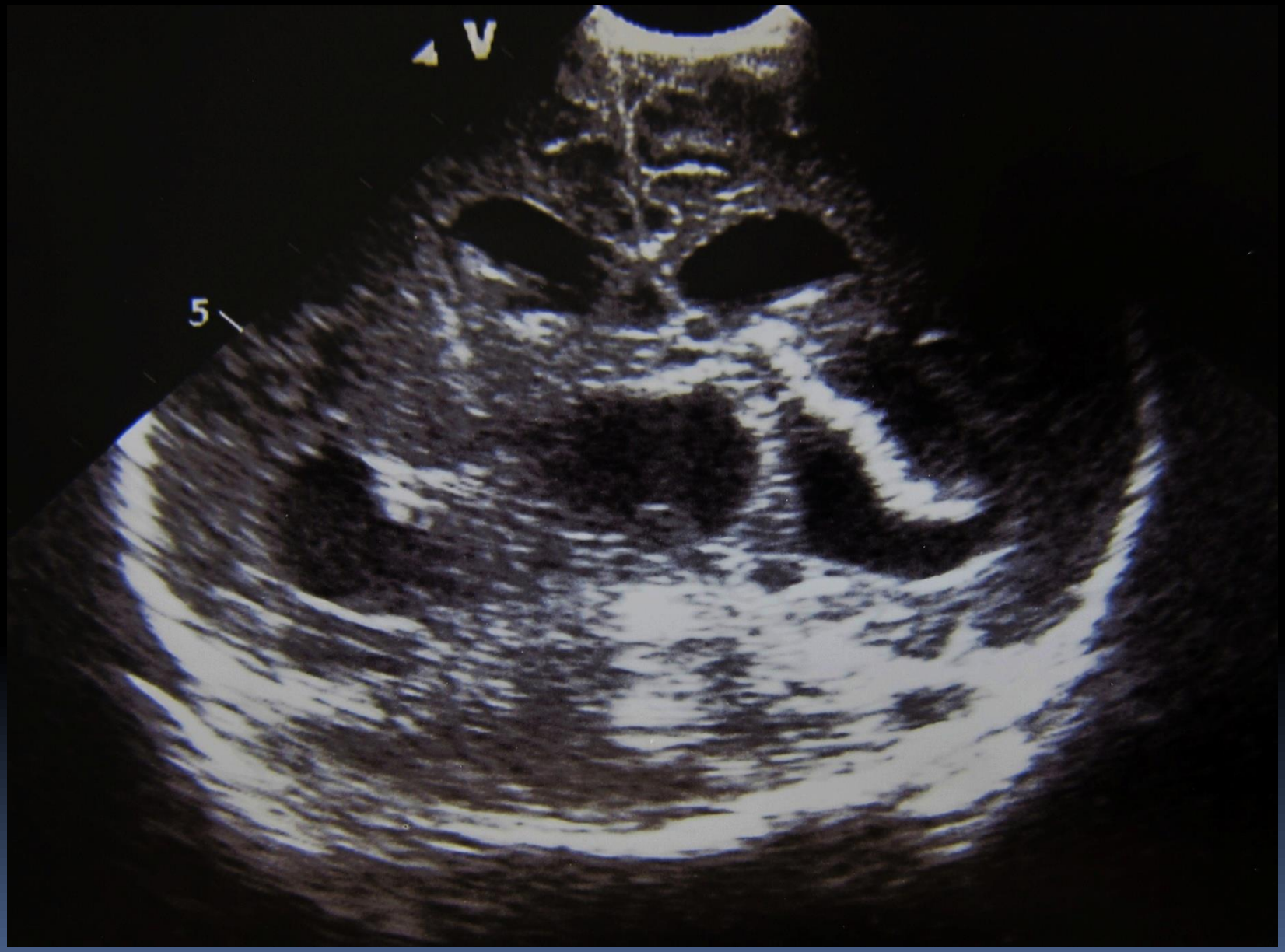

V
2
4
6
R


12/10/12 12:20:56
USR NeoHeac
TIs 0.1
V
2
4



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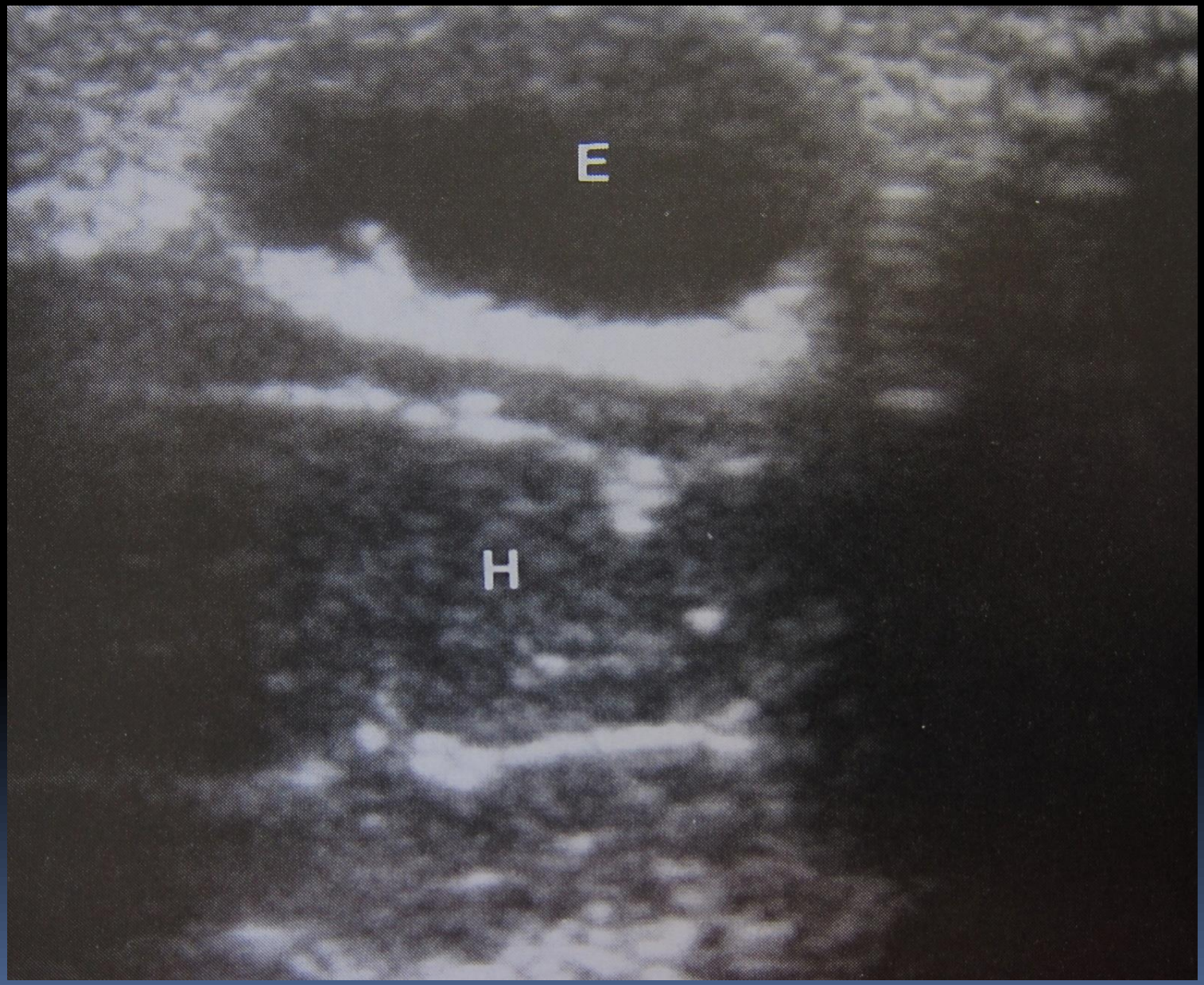
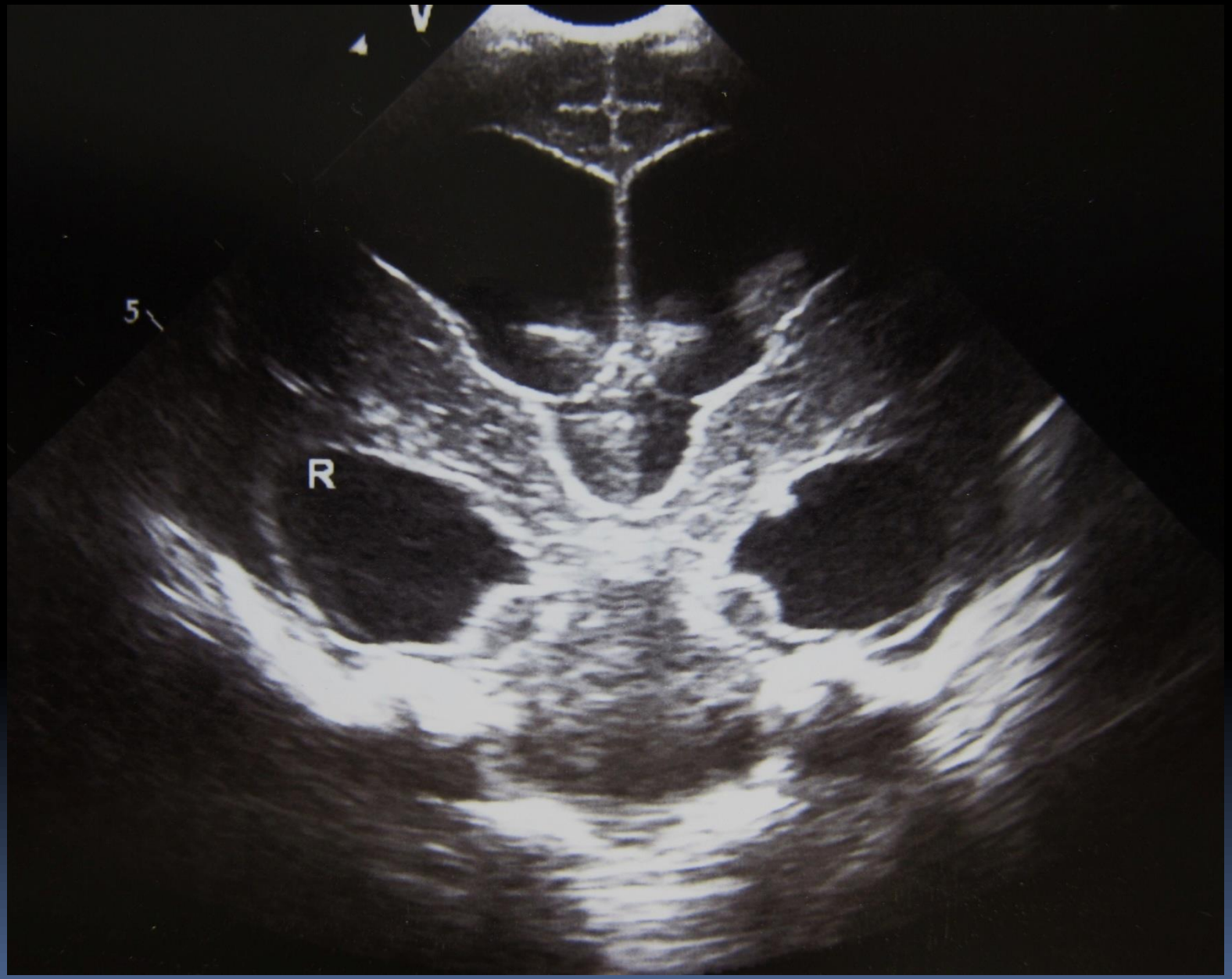

E
H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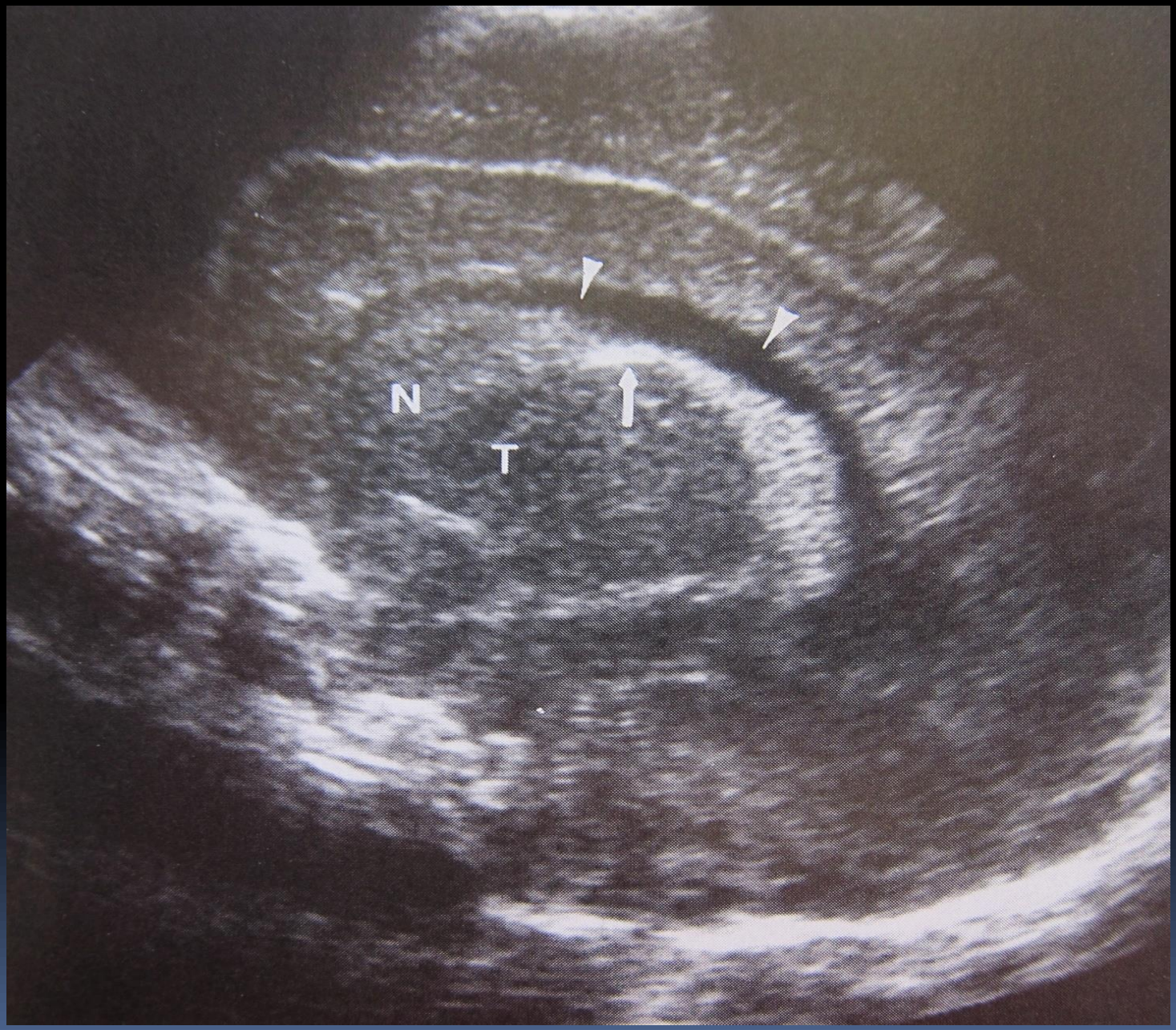
N
T