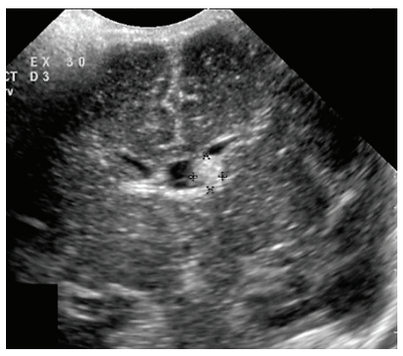
SONOGRAPHER'S QUICK REFERENCE GUIDE TO
COMMON PAEDIATRIC CRANIAL PATHOLOGY

Intraventricular haemorrhage (IVH)

GRADE 1



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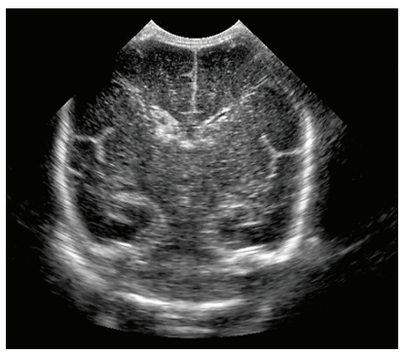

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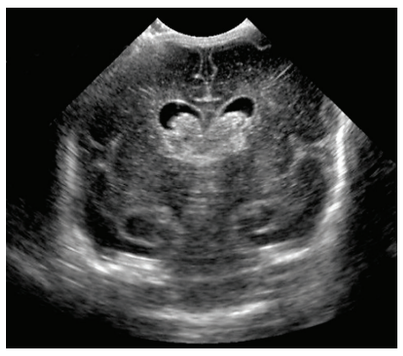

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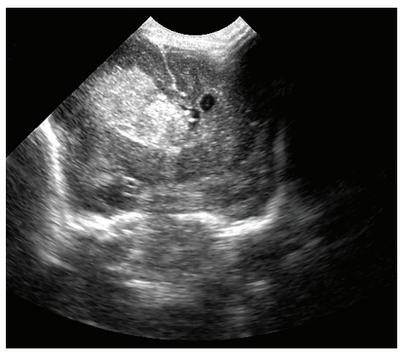


Presentation:
Seizures, hypotonia, apnea, abnormal posturing, low haematocrit.[1]

Pathophysiology:
Primarily attributed to germinal matrix fragility, disturbances in cerebral blood flow and platelet and coagulation disorders.[2]

Ultrasound findings:
Grade 1: Haemorrhage is confined to the subependymal region, i.e. germinal matrix haemorrhage.

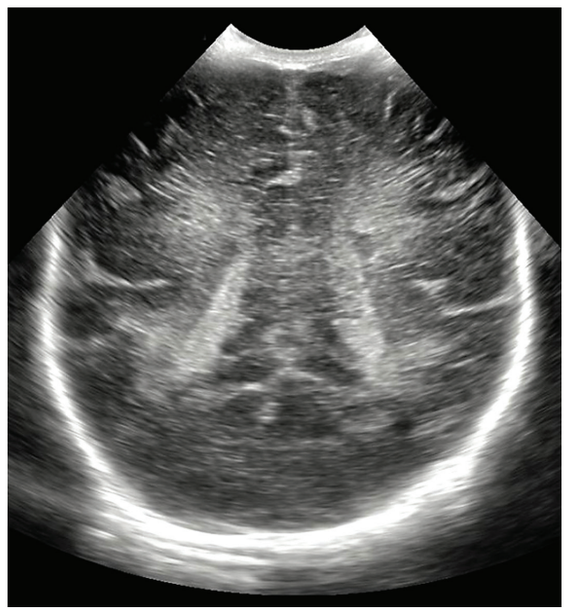
Grade 2: IVH. Haemorrhage is clearly in the ventricle, 10–50% of the ventricular area on parasagittal view.

Grade 3: IVH. >50% of the ventricular area on parasagittal view. Distension of the lateral ventricle. Concomitant periventricular echodensity.

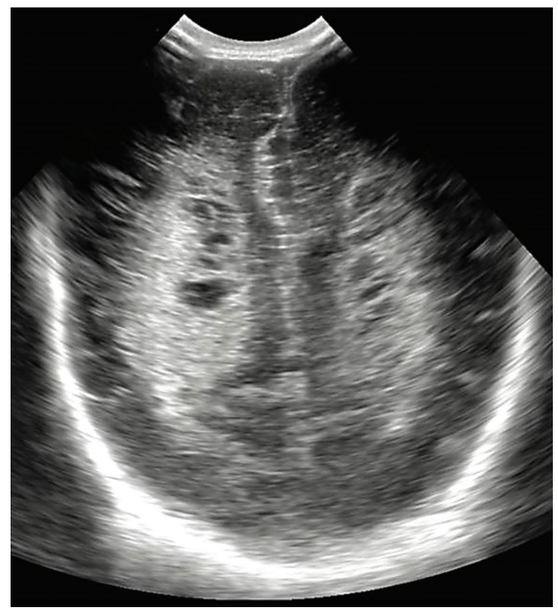
Grade 4: referred to as IPE (intraparenchymal echodensity, periventricular haemorrhagic parenchymal infarction, or venous infarction). [3,4]

Periventricular leukomalcia (PVL)

EARLY CHANGES



LATE CHANGES

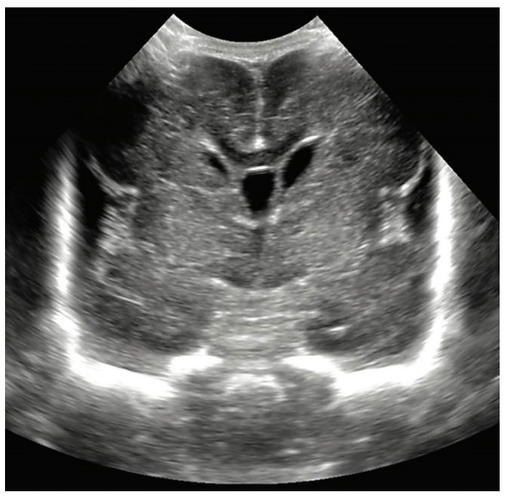


Presentation: Apnea, bradycardia, hypotonia, seizures.

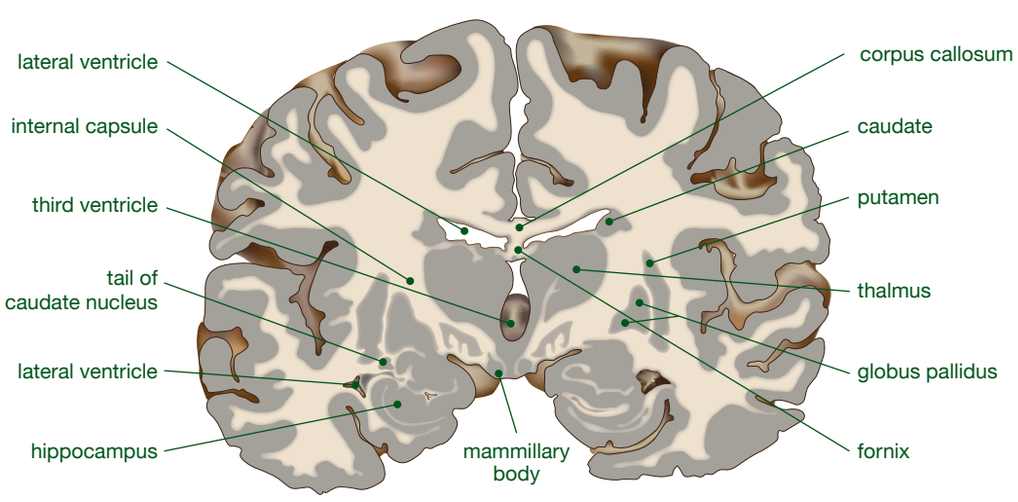
Pathophysiology: Coagulation necrosis of the periventricular deep white matter leading to macrophage and astrocyte proliferation and gradual cyst formation. Injury can occur due to the lack of cerebral autoregulation in premature infants, cardiovascular immaturity and the vulnerability of the premature white matter to hypoxia and ischemia. The cerebral cortex is generally unaffected due to the gestational meningeal intra-arterial anastamoses (involute at term).[1,5,6]

Ultrasound findings: Periventricular echogenicity greater than that of adjacent choroid. The echogenic regions undergo cavitation 2–6 weeks after the event with resultant cystic change demonstrated.[5,6]

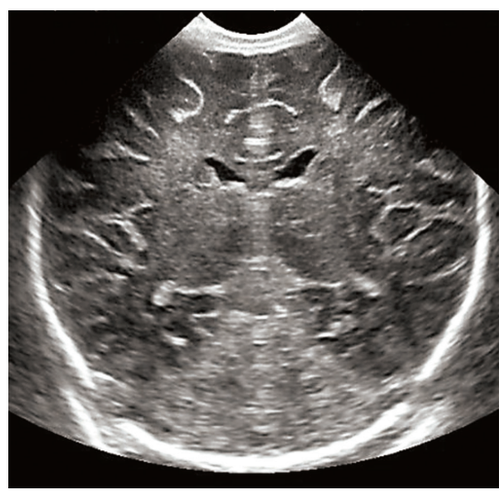
Premature sulcation



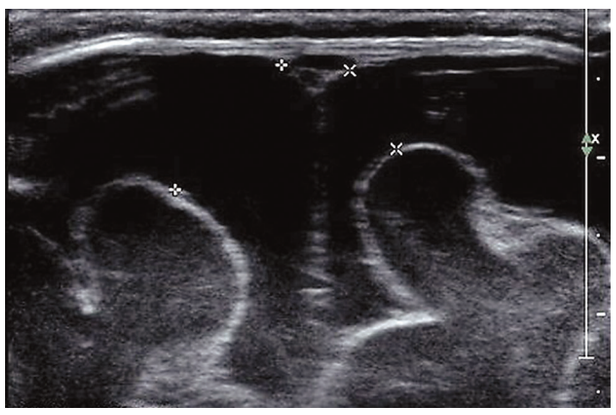
Anatomy



Term sulcation



Benign external hydrocephalus (BEH)



Presentation: Head circumference ↑95%, parental concern.

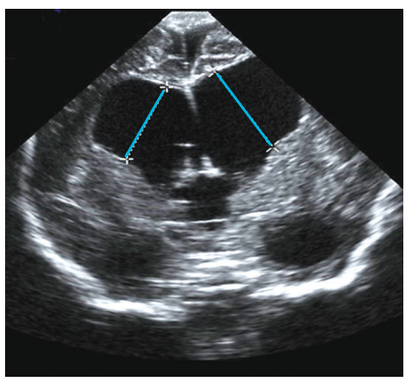
Pathophysiology: Self-limiting form of hydrocephalus in infants between 6–24 months. Most common cause of macrocephaly. Aetiology unclear – ?immature arachnoid villi in subarachnoid space causing impaired absorption for CSF.
Differential – subdural haematoma, cerebral atrophy.[7]

Ultrasound findings: Bilateral symmetric extra axial fluid collections.

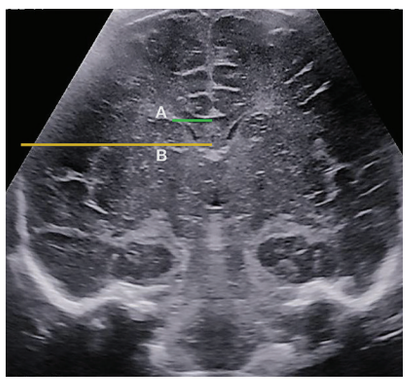
Measurement: Sinocortical width
The shortest distance between the cerebral cortex and the triangular superior sagittal sinus.
0–4 mm: normal
4–10 mm: BEH
>10 mm: abnormal requires monitoring from increased intracranial pressure. [7,8]

Hydrocephalus

HYDROCEPHALUS



NORMAL VENTRICLES WITH MEASUREMENTS



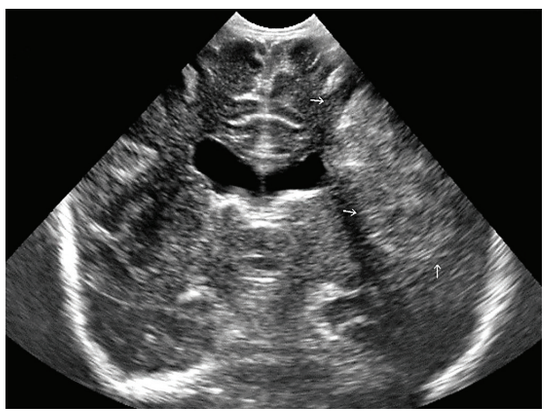
Presentation: Increasing head circumference, bulging firm fontenelles, separated sutures, lethargy, vomiting, poor feeding, seizures, eye changes, low muscle tone.

Pathophysiology: Imbalance in cerebrospinal fluid (CSF) volume resulting in ventricular dilatation.
Mechanism:
1. Obstructive hydrocephalus (secondary to haemorrhage or infection)
2. Non obstructive hydrocephalus (impaired CSF absorption).

Ultrasound findings: Ventricular enlargement, beginning in the occipital horns and trigone of the lateral ventricle and progressing to the frontal horns. In obstructive hydrocephalus the involvement of the third and fourth ventricle will be dependent on the level at which obstruction occurs.

Measurements:
1. Gestational ages 25–42 weeks: Anterior horn width (AHW) is the maximum diagonal measurement of the lateral ventricle horns at the level of the foramen of Munro. The width is measured between the medial wall and the floor of the lateral ventricle at the widest point.[9] AHW < 3 mm.
2. Gestational age > 40 weeks: Ventricular index (VI) is the distance between the falx cerebri and lateral wall of anterior horn (A) divided by the measurement of falx cerebri to inner skull (B). VI ratio < 0.3.[10]

Hypoxic ischaemic encepholopathy (HIE)



Presentation: Lethargy, seizures/epileptic activity or a low Apgar score at birth.

Pathophysiology: HIE occurs when there has been inadequate blood flow and oxygen supply to the brain, which results in focal or diffuse brain injury. This can result from the following:
1. Intrauterine asphyxia **or**
2. Perinatal asphyxia resulting from conditions causing pulmonary failure such as meconium aspirate, hyaline membrane disease of the lungs, pneumonia or congenital cardiac disease.

Ultrasound findings:
1. Diffuse cerebral oedema: global increase in cerebral echogenicity and obliteration of CSF containing spaces
2. Increased echogenicity of the cerebral parenchyma – more pronounced over time
3. Loss of deep gray matter definition
4. Mass effect resulting in ventricular effacement
5. Increase in RI of MCA in severe HIE. [11,12,13]

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1. Siegal M, et al. *Pediatric Sonography: Fourth Edition*. 2011 Lippincott Williams and Wilkins, Chapter 3: 73–4. **2.** Ballabh P. Intraventricular hemorrhage in premature infants: mechanism of disease. *Pediatr Res.*2010 Jan;67(1):1–8. **3.** Volpe JJ. Intraventricular hemorrhage and brain injury in the premature infant. Diagnosis, prognosis, and prevention. *Clin Perinatol.* 1989 Jun;16(2):387–411. **4.** Volpe JJ. Intraventricular hemorrhage in the premature infant – current concepts. Part I. *Ann Neurol.* 1989 Jan;25(1):3–11. **5.** Riccabona M. Neonatal neurosonography. *Eur J Radiol.* 2014 Sep;83(9):1495–506. **6.** Yikilmaz A, Taylor GA. Cranial sonography in term and near-term infants. *Pediatr Radiol*. 2008 Jun;38(6):605–16. **7.** Lam WW, Ai VH, Wong V, Leong LL. Ultrasonographic measurement of subarachnoid space in normal infants and children. *Pediatr Neurol.* 2001 Nov;25(5):380–4. **8.** Wiig US, Zahl SM, Egge A, Helseth E, Wester K, et al. Epidemiology of Benign External Hydrocephalus in Norway – A Population-Based Study. *Pediatr Neurol.* 2017 Aug;73:36–41. **9.** Brouwer MJ, de Vries LS, Groenendaal F, Koopman C, Pistorius LR et al. New reference values for the neonatal cerebral ventricles. *Radiology*. 2012 Jan;262(1):224–33 **10.** Gravendeel J, Rosendahl K. Cerebral biometry at birth and at 4 and 8 months of age. A prospective study using US. *Pediatr Radiol*. 2010 Oct;40(10):1651–6. **11.** Kurinczuk JJ, White-Koning M, Badawi N. Epidemiology of neonatal encephalopathy and hypoxic-ischaemic encephalopathy. *Early Hum Dev.* 2010 Jun;86(6):329–38. **12.** Bano S, Chaudhary V, Garga UC. Neonatal Hypoxic-ischemic Encephalopathy: A Radiological Review. *J Pediatr Neurosci.* 2017 Jan–Mar;12(1):1–6. **13.** Chao C, Zaleski C, Patton A. Neonatal Hypoxic-Ischemic Encephalopathy: multimodality imaging findings. *Radiographics*. 2006 Oct;26 Suppl 1:S159–72.