East of England Neonatal ODN
(Hosted by Cambridge University Hospitals)

Clinical Guideline: EAST OF ENGLAND NEONATAL NEUROPROTECTION GUIDELINE MANAGEMENT OF POST HAEMORRHAGIC VENTRICULAR DILAT

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MANAGEMENT OF POST HAEMORRHAGIC VENTRICULAR DILATATION

CONTENTS

Refer: i. Policy on Management of ventricular access devices in PHVD

1. INTRODUCTION

The major complication associated with intra-ventricular haemorrhage (IVH) in preterm infants is the development of post-haemorrhagic ventricular dilatation (PHVD), which is associated with poor neurodevelopmental outcome particularly in those requiring intervention[1].

- There is wide variation in diagnosis and management of PHVD among the units in UK[2]. Measurement commonly used is Levene's ventricular index which was established in 1981[3].
- Newer technology and resolution of Ultrasound, increased survival of premature infants and newer evidence on PHVD necessitates the need for revisiting the management of PHVD.

Thus, appropriate management of PHVD based on evidence-based guidelines is important in order to prevent its progression and decrease its impact on neurodevelopmental outcome.

2. DEFINITION

i. Levene's Criteria for PHVD:

- PHVD is defined as ventricular index (VI) above 97th percentile on the Levene's chart. Ventricular index is defined as "the horizontal measurement from the midline falx to the lateral aspect of the anterior horn of the lateral ventricle in the coronal plane obtained at the level of the third ventricle/foramen of Monro"[3].

ii. New criteria use to define PHVD[4 -5] are:

- Anterior horn width (AHW)- diagonal width of anterior horn measured at its widest point in the coronal plane (Normal ≤ 6 mm).
- Thalamo-occipital distance (TOD) – distance between the outermost part of thalamus at its junction with choroid plexus and outer most part of the occipital horn in parasagittal plane (Normal ≤ 25 mm).
  (see flow chart for AHW and TOD measurement for intervention and 24–42 weeks (https://tinyurl.com/PHVD-Measures-1) and postmenstrual age 24–29 weeks (https://tinyurl.com/PHVD-Measures-2. Appendix 1 ventricular measurement risk zones)

3. MAGNITUDE OF PROBLEM [6]

- Germinal matrix IVH seen in 20-38% of preterm born at < 28 weeks gestational age, and 15% in 28 to 32 weeks gestational age.
- According to Volpe's classification, severe IVH, grade 3 with or without periventricular infarction is seen in 10-15% of preterm born at <28 weeks gestation.
- PHVD develops in 40-50% of preterm infants with severe IVH and small percentage in grade 2.
- PHVD is associated with high risk of adverse motor and cognitive neurodevelopmental outcomes.
- 25-50% of infants with PHVD will require intervention.

4. AETIOLOGY & BRAIN INJURY

4 A. Several mechanisms are thought to result in PHVD.

1. Acute blockage of aqueduct of 4th ventricle by the blood clot due to IVH results in impaired CSF drainage and decreased CSF reabsorption.[7]
2. Hypersecretion of CSF occurs due to inflammatory response in the choroid plexus resulting in TLR-4 activation and NF-KB inflammation which affects the choroid plexus cilia.

3. CSF absorption occurs mainly via deep venous drainage system, and lymphatic channels. Therefore, ependymal and sub-ependymal damage results in destruction of ependymal cilia of choroid plexus thereby affecting CSF flow and production.[8]
4. Alternation of blood brain and CSF barriers results in increased CSF proteins (i.e. plasminogen activator inhibitor1, TGFβ1, TGFβ2, VEGF, cytokines), which results in osmotic gradient leading to ventricular inflammation and dilatation. [9]
5. Iron induced ependymal and aquaporin water channels damage have been thought to be responsible for post haemorrhagic ventricular dilatation.[10.]

4 B. Secondary brain injury [5]

The secondary brain injury due to PHVD are multifactorial.

1. Cerebral ischemia, mechanical distortion, neuro-inflammation and free radical injury are the main pathogenic factors leading to brain injury.
2. Studies indicates that at cellular level, ependymal disruption, axonal injury, microglial infiltration and impaired myelination occurs.
3. This results in disturbance in development of cerebral white matter, deep nuclear structure (thalamus)cortex and cerebellum. Nieuwets et al (2021), reported low Fractional Anisotropy value in corticospinal tract.[11]

5. SYMPTOMS AND SIGNS

The symptoms and signs of raised intracranial pressure develop several weeks following the onset of PHVD due to immature white matter and the large extradural space and higher compliance in pre-term brain. As the ventricular size increases considerably before affecting the anterior fontanelle pressure or the head circumference [12], it is therefore recommended to monitor PHVD with regular ventricular measurements (VI/AHW/TOD).

i. Recognition of raised intracranial pressure which are late signs-[13]

- Bulging fontanelle
- Splaying of the cranial sutures
- Presence of apnoea/vomiting
- Hypotonia/Hypertonia
- RI >0.85
- Increased discontinuity on EEG

ii. Guide on Monitoring of signs of raised intracranial pressure

- Twice weekly head circumference

- Head circumference enlarges by approximately 1mm/day between 26-32 weeks of gestation, 0.7mm/day between 32 -40 weeks.
- A measurement of >4mm over 2 days or 14mm over 7 days is excessive.[13,14]
- Twice weekly head scans with RI and ventricular measurements.
- Weekly neurological assessment.
- Daily neuro-observations including HR, BP, level of alertness, vomiting, apnoeas.

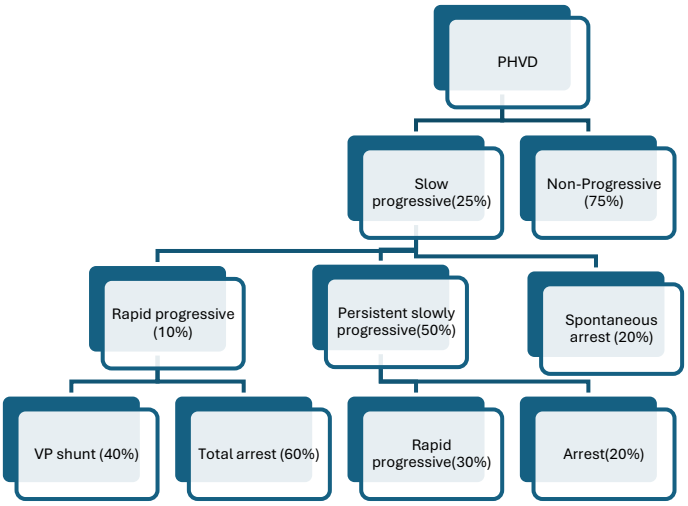
6. PROGRESSION OF PHVD and VOLPE'S CLASSIFICATION OF IVH[15]

- The natural progression of post haemorrhagic ventricular dilatation is determined by severity of intra-ventricular haemorrhage and the evolution of PHVD.
- With IVH grade 2 (Volpe's classification), slow progressive dilatation of ventricles usually occurs after 14 days whereas with grade 3 IVH ventricles will dilate more rapidly, within days[15] (See Figure 1).

Volpe's classification of IVH (2008): Grading of the severity of germinal matrix haemorrhage

Grade 1.	GMH with no or minimum intraventricular haemorrhage (<10% of ventricular area in parasagittal plane.)
Grade 2.	Intraventricular haemorrhage (10-50% of ventricular area on parasagittal view)
Grade 3	Intraventricular haemorrhage (>50% of ventricular area on parasagittal view-usually distended ventricles)
Separate	Periventricular echo-density- periventricular haemorrhagic infarction PVHI can occur along with any grade of IVH and is not caused by extension of IVH

Figure 1: Outcome of infant with PHVD
Source: Adapted from Newborn Neurology (Volpe, 2008) [15]



7. DIAGNOSIS AND MONITORING OF PHVD

7.1 Diagnosis using Levene's Ventricular Index

- To diagnose PHVD and evaluate the need for intervention, measurement of ventricular size by means of cranial ultrasonography (cuss) has been shown to be superior to measurement of head circumference or assessment of clinical symptoms of raised intracranial pressure[16-19].
- The ventricular measurement helps in diagnosis, timing of therapeutic intervention and predicting neurodevelopmental outcome [20]
- PHVD is defined as ventricular index above the 97th percentile for GA on Levene's chart[3].
- Currently, across the units in our network and in majority of centres in UK and Europe, VI is the established criterion for diagnosis of PHVD[2].
- Limitations of Levene's Index:
    a. VI increases only in severe hydrocephalus and may fail to identify PHVD with mild dilatation.
    b. In Levene's study preterm infants younger than 26 weeks gestational age were not included[3,4].

7.2 Newer ventricular measurements for PHVD (Appendix 1)

The newer ventricular measurements are AHW, TOD and others. The new references for AHW and TOD ventricular measurement have been reported for 25 to 42 weeks gestational age by Brouwer et al, 2012[4, 5].

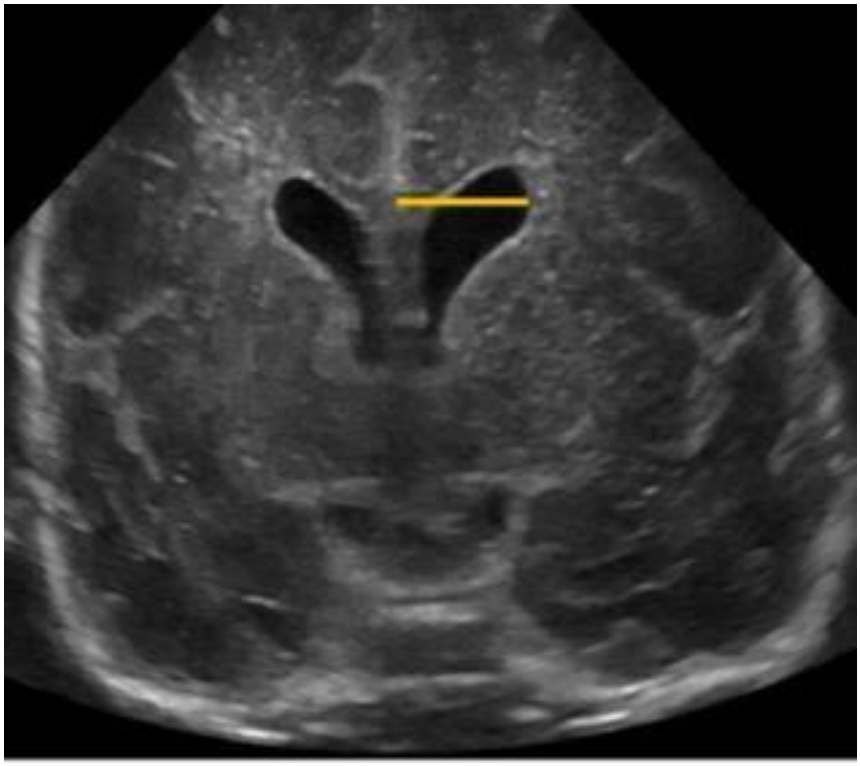


Figure 2: Ventricular index (yellow horizontal line)
Source: Brower et al 2012

7.2.1 AHW:

- Anterior horn width is defined as diagonal width of anterior horn measured at its widest point in the coronal plane.

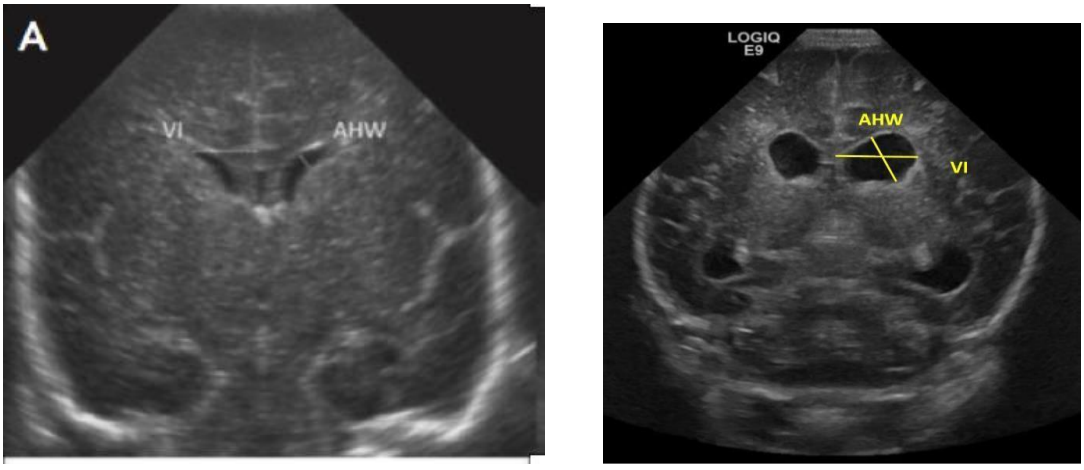


Figure 3: A) Anterior horn width (AHW)
Source: Brower et al[4], 2012, El-Dib M,2020 [5]

- The early sign of increase intracranial pressure is rounding of anterior horn width and hence AHW has been suggested as sensitive ventricular measurement for early ventricular enlargement than VI [20]. A recent study showed the inter and intra-observer reliability is best for AHW to predict development of PHVD requiring neurosurgical intervention [21]. AHW has been used to predict the trajectory of PHVD by using growth mixture model [22].

- Majority of studies have shown AHW remains constant with GA[4].

7.2.2 Thalamo-occipital distance (TOD)

- TOD is defined as distance between the outermost part of thalamus at its junction with choroid plexus and outer most part of the occipital horn in parasagittal plane[4].
- Thalamo-Occipital Distance varies with gestational age and measurement of TOD can be challenging but evaluation of TOD is of clinical value.
- TOD may show earliest and fastest increase in size in PHVD. Brouwer et al. study (2012) reported 97th percentile TOD measurement of 19 mm for preterm and 21mm for term infants[4] [5] suggests PHVD. El-Dib M et al, (2020) in their study has define ventricular measure risk zone for individual gestational age (see Appendix 1).

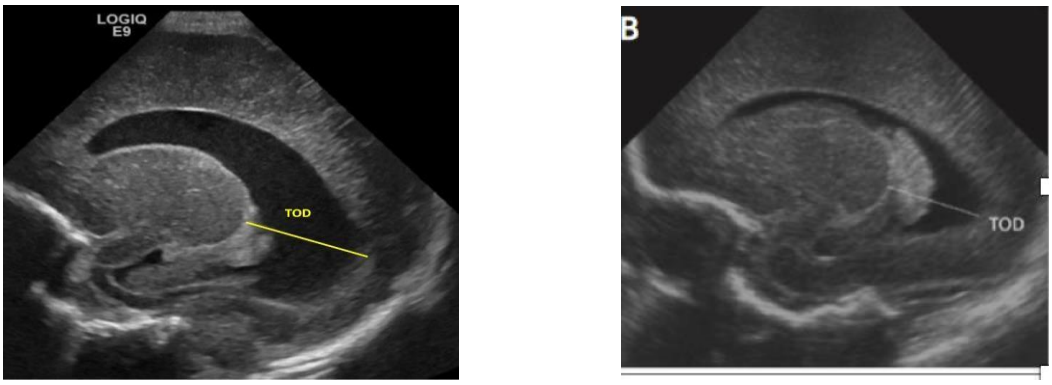


Fig 4: B) Thalamo-occipital distance (TOD)

Source: Brower et al, 2012, El-Dib M,2020

7.3 Neuromonitoring tool:[23]

Neuromonitoring tools are crucial in monitoring the progression of the PHVD.

Neuro-assessment and neuro-observation along with Ventricular indices, Resistance index (>.85) are the common neuromonitoring tools used to monitor progression of PHVD.

Another tool recommended is Near infrared spectroscopy (NIRS)

Near infrared spectroscopy (NIRS) measures tissue oxygen saturation in organs 2-3 cm below its sensor in brain.

- NIRS monitors the intensity of light passing through the brain at two wavelengths, and the changes in attenuation is converted into cerebral concentration of oxy and deoxyhaemoglobin. This provides regional mixed tissue oxygen saturation and is interpreted as rSO2%.
    - NIRS assists in determining cerebral oxygenation and cerebral autoregulation. Normal CrSo2 (cerebral saturation) ranges from 55 to 85% +/-10% in neonates. The fractional cerebral tissue oxygenation (cFTOE) which provides information on consumption of oxygen, increases when there is cerebral hypoxia. The absolute value is affected by the sensor and device type as well day to day intervention.
    - Decrease in CrS02 relative to baseline or absolute value <60%, is seen in hypocarbia, anaemia, hypotension with lack of autoregulation, PDA and GM-IVH, PHVD.
    - Increase in CrS02 relative baseline or absolute value of >90% is associated with hyperoxia, hypocarbia, or severe brain injury (El-Dib et al, 2024).

Evidence on NIRS:

- A recent prospective cohort study showed that those infants with PHVD had lower $rScO_2$ ($p < 0.001$), spent more time with $rScO_2 < 55\%$ ($p < 0.001$), and exhibited higher cFTOE ($p < 0.001$) than those without PHVD .[24]

- Also, LP and surgical intervention in PHVD maintains higher rSO2 and lower cFTOE .[25]

- Current evidence on routine used of NIRS is limited, however when used appropriately with other monitoring tool is a potential clinical tool to guide intervention [26].

- European standard of care recommends use of NIRS in high risk infants who develops PHVD [26]

7.4 Role of biomarkers

- The prediction of progression of PHVD is difficult due to clinical variability and hence timing of intervention is challenging. Also, recent evidence on the timing of treatment cannot be generalised as there are several confounding variables, limited sample size and different intervention strategies.
- Recent research is directed towards validation of biomarkers capable of early prediction of progression and development of IVH and PHVD. Biomarkers are proteins or molecules found in CNS, particularly in neurons and glial cells [27]. Some of the examples of the biomarkers for structural brain injury for PHVD are blood and CSF NiL, and GFAP protiens.TGF-B1, a growth factor, is also associated with PHVD and WMI.

- The study of genome, transcriptome, proteome and metabolome, is a model tool for identifying potential biomarkers for IVH and PHVD.
Studies have revealed change in the expression of mRNA, miRNA in peripheral blood of preterm with IVH and PHVD [27]. CSF miRNA biomarkers from the isolated extracellular vesicles has been studied as predictor biomarker for neurodevelopmental outcome in neonates with PHVD [28].

- Other investigations i.e. visual evoked potentials, amplitude integrated EEG, and near-infrared spectroscopy (NIRS) may help in optimising timing of intervention in PHVD [29].

8. TREATMENT

Infants with PHVD who requires permanent shunt are at significant risk of developing cerebral palsy and cognitive impairment [7,30]. Large multicentre observational study showed in late intervention group who required shunt had lower cognitive (p=0.002) and motor scores (p=0.03)[30]. The hypothesis for the abnormal neurodevelopmental outcome in these infants, is due to permanent

white matter injury resulting from severe and prolonged dilatation of ventricles.[30]

8.1. Treatment Modalities

8.1.1. Non- surgical management (refer flow chart management of PHVD)

- If the infant is showing excessive head growth and VI is above the 97+4th centile or is symptomatic it is recommended to perform therapeutic LP. Serial tapping of CSF by lumbar puncture is commonly used as a temporary measure to reduce intracranial pressure. (2-3times/week)
- Minimum of 10ml/kg of CSF is needed to be removed in order to have a significant effect on ventricular size. It is however wise to limit the volume of CSF removed to a maximum of 20ml/kg as larger volumes removed faster than 1ml/kg/min may be followed by apnoeas, bradycardia and desaturation.
    - Ventricular tapping has been associated with increased development of CSF infection and loculated hydrocephalus.[21]
- In those cases where repeated lumbar puncture is necessary to control excessive head enlargement and raised intracranial pressure is suspected, referral should be made to the Neurosurgical team for insertion of a temporising shunt.
- Cochrane review concluded that lumbar puncture treatment failed to reduce disability or need for the VP shunt surgery[31]. However, the study in the review, intervention with LP was carried out at VI >97+4mm.
- Pharmacological treatment with diuretics in PHVD has shown no difference in mortality or need of VP shunt but has been associated with poor neurodevelopmental outcome[32].
- Short-term outcome of randomised trial of Drainage, Irrigation and Fibrinolytic Therapy (DRIFT) for Premature Infants with Post-haemorrhagic Ventricular Dilatation was associated with secondary haemorrhage and high mortality/ severe disability [33].
- Long-term outcome of DRIFT study showed decrease in severe cognitive disability from 59% to 33%. Overall 48% were not able to walk and 20% were unable to communicate[34].

- Sensorimotor disabilities were noted to be less in DRIFT group but were not statistically significant[33].

8.1.2. Surgical Management

8.1.2 a. Temporary interventions

1. **External ventricular drainage (EVD)**

   A Catheter is introduced into frontal horn of lateral ventricle and distal end is connected to externalised closed drainage system, which is at height to control CSF drainage (10-20ml/kg/day). This drainage system is able to resolve PHVD in 30-40% of cases[35]. A recent study has concluded that placement of an EVD within the first 25 days may improve cognitive function in infants with PHVD [35]

2. **Ventricular access devices (reservoir)**

   A ventricular catheter is connected to an access device (reservoir) in the subcutaneous area, which can be used to aspirate 10-20 ml/kg /day of CSF to decrease intracranial

   pressure. This device can resolve PHVD in 30% of cases, which is marginally less than EVD. It is also associated with infections, skin defect and CSF leaks[36,37].

3. **Ventriculo-subgaleal shunts (VSGS)**

   VSGS is a continuous CSF drainage system, which drains CSF into the subgaleal space.

   It can include a subgaleal reservoir that can be used to tap in case of VSGS failure. Infection rate with VSGS reported varies with the centres and ranges between 5-15%[34].

   Recent studies have shown no significant difference in neurodevelopmental outcome and infection rate, shunt implantation or revision between VAD and VSGS [38].

4. **Endoscopic third ventriculostomy**

   This procedure has been used in cases with VAD failure[39].

5. **Neuroendoscopic lavage (NEL**) is an emerging technique that aims to directly reduce the load of intraventricular blood and its breakdown products, potentially reducing the risk of secondary brain injury.[40]

6. The recent RCT, **ENLIVEN-UK** trial aims to assess whether the addition of NEL to standard temporising device placement improves neurodevelopmental outcomes at 2 years of corrected age compared to temporising device placement alone.[41]

8.1.2 b. Permanent Intervention

The permanent surgical procedure includes Ventriculo-peritoneal shunt.

Referral for a permanent VP shunt

- VP shunting should not be carried our as the primary treatment when progressive PHVD is diagnosed.
- Approximately 50% of infants diagnosed with PHVD do not need a permanent shunt.
- Permanent shunt surgery is usually done around term in an infant who has had a reservoir and is needing repeated taps to maintain head growth or if there is persistent excessive head enlargement.

8.2. Recent evidence on Early vs. Late intervention

The optimal balance between the benefit of early versus late intervention in terms of associated risk and potential of over treatment is yet to be determined [22].

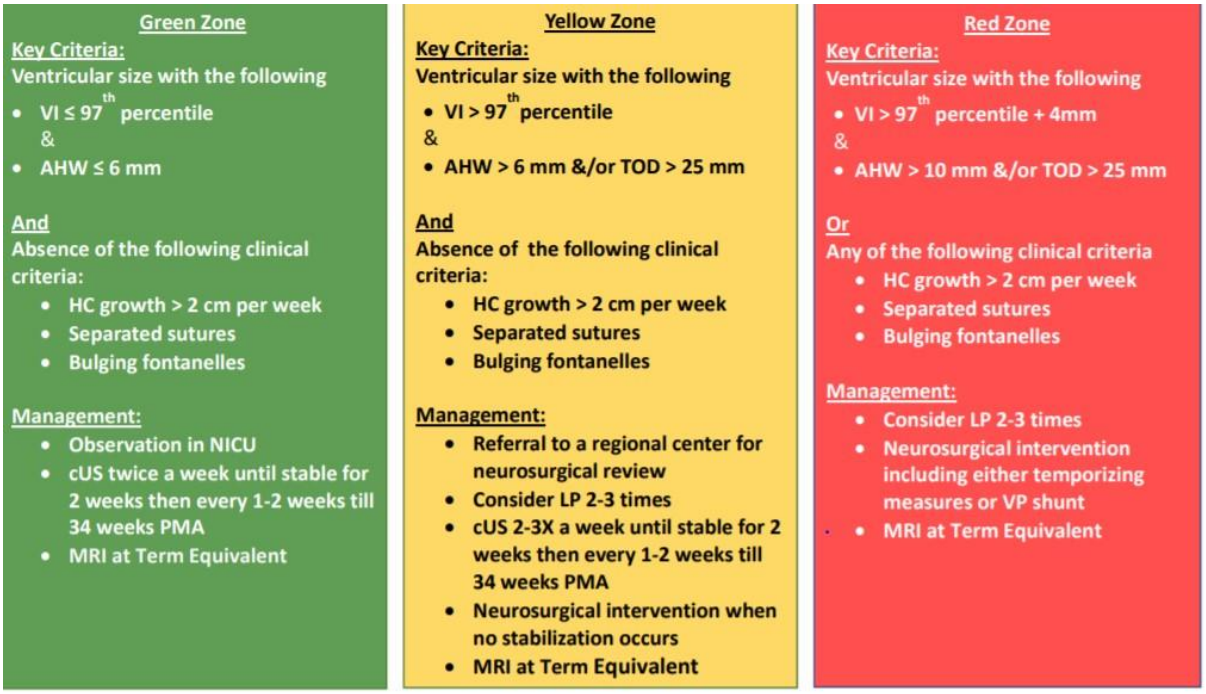
- Emerging evidence suggests early intervention for PHVD is associated with decreased white matter injury and better neurodevelopmental outcome [5,6,42,43].
- Recent RCT on early intervention observed lower odds of death and neurodevelopmental disability in preterm infants with progressive ventricular dilatation. The same study showed lower cognitive and motor scores in late intervention group [42].

- However, uncertainty of progression of PHVD results in limitation of implementing early intervention as standard treatment, in particular considering the risk benefit ratio. Hence use of biomarkers, ultrasound indices, NIRS and future blood and CSF biomarker will enable to identify robustly the neonates with PHVD who will require early intervention [44].

8.3 Referral to Neurosurgical Team at Addenbrooke's Hospital

The ventricular indices persistently remain >97+4 mm and AHW >5 mm, TOD>26 mm (see flow chart), infant should be referred to Neurosurgical Team at Addenbrooke's Hospital. All the referral should be made on the link, http://www.orioncloud.org. The Neurosurgical team would contact the referral unit on the same day with the advice on further management plan.

9. FLOW CHART: Management of PHVD[5]



(adapted from El-Dib M, Limbrick Jr. DD, Inder T, Whitelaw A, Kulkarni AV, Warf B, Volpe JJ, de Vries LS, Management of Post-hemorrhagic Ventricular Dilatation in the Preterm Infant The Journal of Pediatrics (2020), doi: https://doi.org/10.1016/j.jpeds.2020.07.079)

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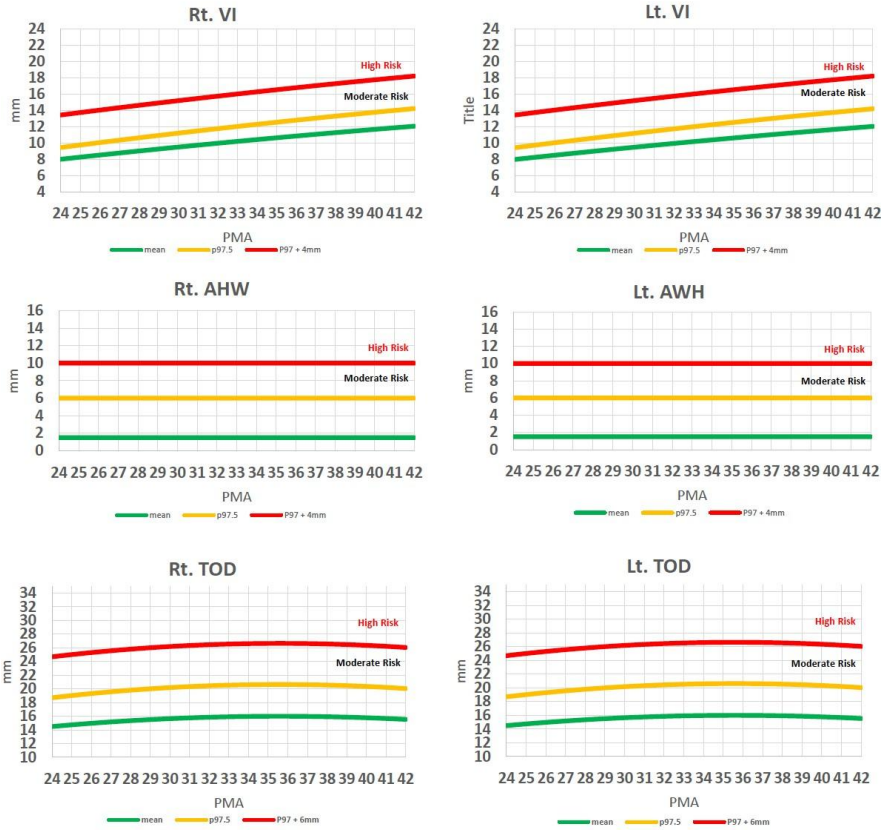
APPENDIX 1[5]

Name:
DOB:

Ventricular Measurement Risk Zones



Date	PMA	Rt. TOD	Rt. VI	Rt. AHW	Lt. TOD	Lt. VI	Lt. AHW

(ref: El-Dib M, Limbrick Jr. et al,Management of Post-hemorrhagic Ventricular Dilatation in the Preterm Infant The Journal of Pediatrics (2020), doi: https://doi.org/10.1016/j.jpeds.2020.07.079)

References

Exceptional Circumstances Form

Form to be completed in the **exceptional** circumstances that the Trust is not able to follow ODN approved guidelines.

Details of person completing the form:	
Title:	Organisation
First name:	Email contact address:
Surname:	Telephone contact number:
Title of document to be excepted from:	
Rationale why Trust is unable to adhere to the document:	
Signature of speciality Clinical Lead: Date:	Signature of Trust Nursing / Medical Director: Date:
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