

## Intensive Care Nursery House Staff Manual

# Intraventricular Hemorrhage (IVH)

**INTRODUCTION:** IVH, the most common type of neonatal intracranial hemorrhage, occurs mainly in preterm infants  $\leq 32$  weeks of gestation. The incidence ranges from 13-65% in different centers, decreases with advancing gestational age and is influenced by certain perinatal risk factors (see below).

**PATHOGENESIS** is related to:

**(1) Intra-vascular factors:**

- Impaired cerebral autoregulation
- Fluctuating cerebral blood flow (related to fluctuating arterial blood pressure)\*
- $\uparrow$  cerebral blood flow (e.g., due to hypercarbia, excess volume expansion)\*
- $\uparrow$  cerebral venous pressure (e.g., with pneumothorax, asphyxial heart failure)\*
- Hypotension and reperfusion\*
- Coagulation abnormalities

**(2) Vascular factors:**

- Germinal matrix**, a highly vascular structure with poor capillary support, is present  $< 35$  weeks and is a critical factor in pathogenesis of IVH.
- Germinal matrix capillaries are **very** vulnerable to hypoxic-ischemic injury.
- Arterial development: acute transition from large vessels to a capillary network without gradual arborization
- Venous drainage: "hairpin loop" configuration in germinal matrix is conducive to outflow obstruction and is important in pathogenesis of periventricular hemorrhagic infarction.

**(3) Extra-vascular factors:** Preterm infants have

- Increased fibrinolytic activity
- Poor vascular support in cerebral tissue
- $\uparrow$  risk of hypoxia, hypercarbia and acidosis due to immature respiratory system\*

(\*Factors that can often be prevented or alleviated with meticulous intensive care!)

**CLINICAL PRESENTATION:** 90% occur in the first 3 days after birth.

- Catastrophic:** Acute IVH with bulging fontanel, split sutures, change in level of consciousness, pupillary and cranial nerve abnormalities, decerebrate posturing, and often with rapid decrease in blood pressure and/or hematocrit.
- Saltatory:** *Gradual* deterioration in neurological status, may be subtle abnormalities in level of consciousness, movement, tone, respiration and eye position/movement.
- Asymptomatic:** 25-50% of IVH. Discovered on Ultrasound (U/S, see below). Fall in hematocrit or failure of hematocrit to rise with transfusion should cause concern.

**GRADING OF IVH** (per J. Volpe):

- Grade I:** Bleeding confined to periventricular area (germinal matrix)
- Grade II:** Intraventricular bleeding (10-50% of ventricular area on sagittal view)
- Grade III:** Intraventricular bleeding ( $> 50\%$  of ventricular area or distends ventricle)

**-Intra-parenchymal echodensity (IPE)** represents **periventricular hemorrhagic infarction** and is often referred to as **Grade IV IVH**.

**OUTCOME and PROGNOSIS:**

<u>Severity of IVH</u>	<u>Mortality (%)</u>	<u>Progressive Ventricular Dilatation (%)</u>	<u>Neurological Sequelae (%)</u>
Grade I	5	5	5
Grade II	10	20	15
Grade III	20	55	35
IPE	50	80	90

(In general, outcomes with IVH Grade I or II are similar to infants without IVH.)

**MECHANISMS of BRAIN INJURY** from IVH include:

- Preceding **hypoxic ischemic injury** that predisposes to IVH
- Increased intracranial pressure** with massive IVH which decreases cerebral perfusion
- Destruction of **germinal matrix**
- Injury to **periventricular white matter** due to infarction or to damage from blood products (K<sup>+</sup>, other vasoactive factors)
- Post-hemorrhagic hydrocephalus** due to impaired absorption of CSF by blood

**PREVENTION of IVH** is primary goal of management and important factors are:

**Prenatal:**

- Prevention of prematurity
- Improved perinatal management, including:
  - Maternal transport of women in preterm labor to regional center prior to delivery
  - Antenatal glucocorticoids: accelerate lung maturation **and** decrease IVH incidence
  - Optimal obstetrical management

**Postnatal:**

- Skilled resuscitation to avoid hypoxia and hypercarbia
- Circulatory support to avoid hypotension and fluctuating arterial blood pressure
- Correction of coagulation abnormalities

**MANAGEMENT:** Other than early diagnosis and careful supportive care (including correction of coagulopathies, circulatory and respiratory support), there is no therapy for IVH. Consider consultation with Neurology for all IVH cases except Grade I and mild Grade II. For **progressive ventricular dilatation (post-hemorrhagic hydrocephalus)**, the essential point is early recognition. Head circumference does not increase until after there has been considerable ventricular dilatation. Therefore, **do serial head U/S examinations in infants with IVH ≥grade II**. Some cases of ventricular dilatation will respond to serial lumbar punctures and/or acetazolamide (carbonic anhydrase inhibitor) or other diuretics (to decrease CSF production). Persistent, progressive ventricular dilatation requires a ventricular reservoir or ventriculo-peritoneal shunt by a neurosurgeon.

**SCHEDULE for CRANIAL ULTRASOUNDS (U/S)** for preterm infants:

<u>Age</u>	<u>Indication for U/S</u>
1 day	Perinatal asphyxia, <i>in utero</i> drug exposure
3 days	Unstable clinical course
7 days	All preterm infants $\leq 32$ weeks gestation

Then:

-If any IVH, U/S in one week (for early detection of hydrocephalus). Subsequent examinations depend on clinical course.

-If no IVH, repeat U/S at age 4 to 6 weeks, for detection of cystic PVL.

**PERIVENTRICULAR WHITE MATTER LESIONS**

Differential diagnosis:

- 1) **Periventricular hemorrhagic infarction**: usually asymmetric and grossly hemorrhagic. Presents in first few days of life.
- 2) **Periventricular leukomalacia (PVL)**: usually bilateral and small. Presents several days to weeks after birth.

Both have association with abnormal neurological outcome.