Birth asphyxia and HIE

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Birth asphyxia

- Asphyxia (definition)
  - ✓ suffocation, is characterized by reduced oxygen delivery to tissues, and accumulation of carbon dioxide in tissues.

Diagnosis of birth asphyxia

- Evidence of severe antenatal and/or intrapartum hypoxia
- Resuscitation at birth
- Features of encephalopathy
- Evidence of hypoxic damage of other organs
- No other prenatal or postnatal cause identified

APGAR SCORING SYSTEM

	0 Points	1 Point	2 Points	Points totaled
Activity (muscle tone)	Absent	Arms and legs flexed	Active movement	
Pulse	Absent	Below 100 bpm	Over 100 bpm	
Grimace (reflex irritability)	Flaccid	Some flexion of Extremities	Active motion (sneeze, cough, pull away)	
Appearance (skin color)	Blue, pale	Body pink, Extremities blue	Completely pink	
Respiration	Absent	Slow, irregular	Vigorous cry	

Birth asphyxia could not judged by Apgar score.

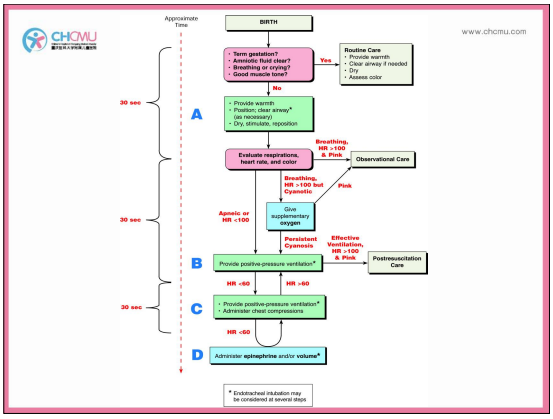
cyanosis

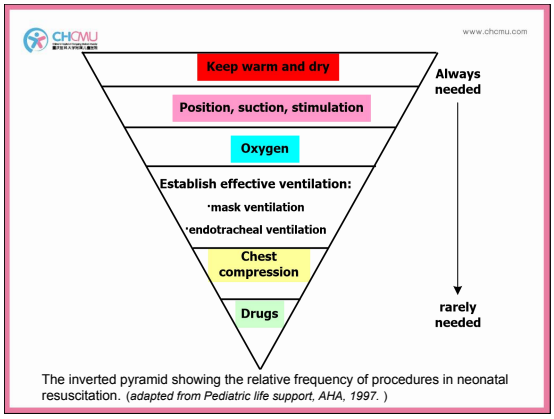
Clinical manifestation of birth asphyxia

- ➢ cyanosis
- ➢ hypotonia
- ➢ poor breathing
- ➢ bradycardia
- ➢ hypotension
- ➢ tachypnea

Active with cyanosis

hypotonia with cyanosis





The inverted pyramid showing the relative frequency of procedures in neonatal resuscitation. (*adapted from Pediatric life support, AHA, 1997.*)

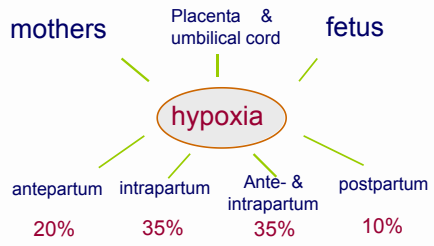
Complications of birth asphyxia

- Brain injure
  - Hypoxic-ischemic encephalopathy (HIE)
  - Intracranial hemorrhage
- Cardiovascular injury
  - heart dysfunction and failure
- Pulmonary damage
  - congestion and hemorrhage
- Renal involved
  - transient renal failure

HYPOXIC-ISCHEMIC ENCEPHALOPATHY (HIE)

- Hypoxic-ischemic encephalopathy is characterized by clinical and laboratory evidence of acute or subacute brain injury resulted from asphyxia (ie, hypoxia and acidosis).
- Most often, the exact timing and underlying cause of HIE remain unknown.

Causes of HIE



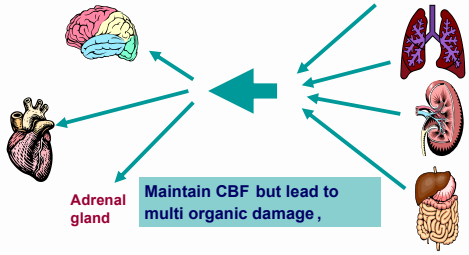
Pathogenesis of HIE

1. fail in compensation of cerebral blood supply (CBF) :

   Ⅰ. redistribution of cardiac output

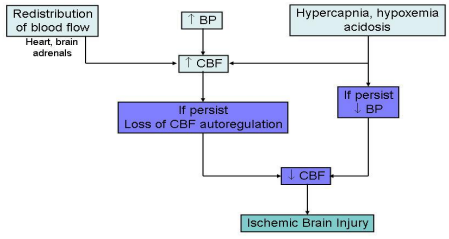
   Ⅱ. redistribution of CBF

I. initial redistribution: Diving reflex



Adrenal gland

Maintain CBF but lead to multi organic damage，



II. Redistribution of CBF

Anterior cerebral artery, ACA

middle cerebral artery,MCA

Mainly supply the blood flow of deep nuclear area:
cerebral cortex
brain stem
basal ganglion
cerebellum

Posterior cerebral artery,PCA

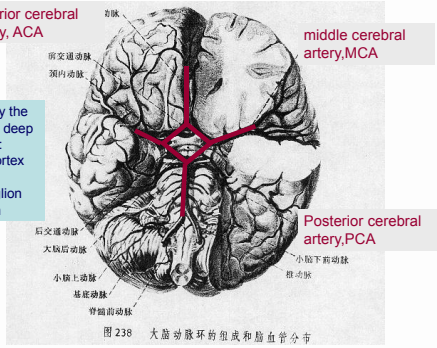
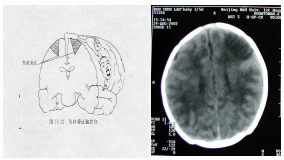


图 238 大脑动脉环的组成和脑血管分布

Whereas the areas localize on the boarder-line of the arterial circle are liable to diminished CBF, and the injury is known as watershed injury, selective brain injury.

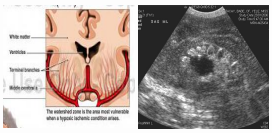
for term infant ：

Parasagittal cerebral injury



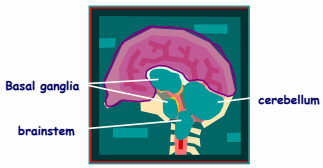
for preterm infant：

Periventricular leukomalacia (PVL)



Cerebral blood flow (CBF) ：

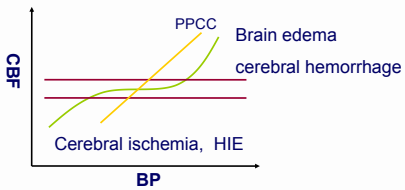
2. Non-compensation ：the deep nuclear areas with high energy demands are damaged seriously



Suddenly & completely asphyxia

Cerebral blood flow (CBF)：

3. autoregulation ↓⟶ Pressure-passive cerebral circulation (PPCC)

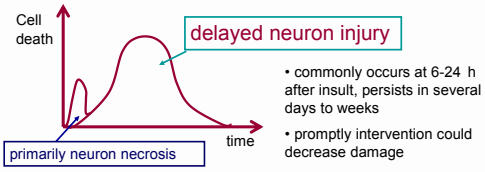


Energy failure ⇨ injury of nerve cells

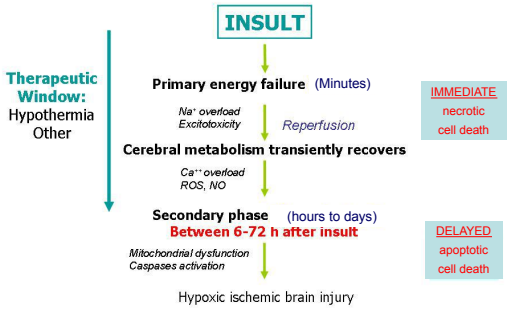
- Overload of intracellular Na^{+} and Ca^{++} due to ion pumps dysfunction.
  - Cytotoxic edema
  - Activation of phospholipase, protease, endonuclease
- Accumulation of excitatory amino acids (EAAs) and overactivation of EAAs' receptors in synaps.
- Oxygen free radicals during reperfusion.

neuropathology :

edema
necrosis } of brain cells
apoptosis



- commonly occurs at 6-24 h after insult, persists in several days to weeks
- promptly intervention could decrease damage



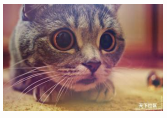
Clinical manifestation of HIE

Clinical manifestation of HIE

- Symptoms always manifests at birth or within a few hours after birth.
- Manifestations of brainstem dysfunction:
  - Unconsciousness, change of pupils
  - Refractory seizure: initially resistant to anticonvulsants, usually generalized
  - Irregularities of breathing, heart rate and blood pressure, usually life-threatening
- Features of seizure in HIE: the most frequent cause of neonatal seizures, usually subtle seizure
  - oral-buccal-lingual movements: smacking, drooling, chewing
  - grimacing; swimming, bicycling, boxing movements
- Signs of intracranial hypertension: unusual
  - Bulging fontanel

Pupils

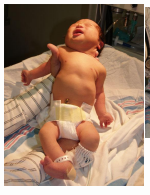
normal



mydiasis

miosis

Muscle tone



Vertical suspension

Horizontal suspension

flaccid

Investigations for HIE

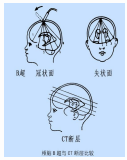
- There are nor specific tests to confirm or exclude a diagnosis of HIE because the diagnosis is made based on the history, physical and neurological examinations, and laboratory evidence.
- Many of the tests are performed to assess the severity of brain injury and to monitor the functional status of systemic organs.
- Results of the tests should be interpreted in conjunction with the clinical history and the findings from physical examination.

Investigations for HIE

- Status of inner environment
  - Serum electrolyte
  - Blood glucose
  - ABG: PaO_2, $PaCO_2$, acid-base balance
  - Others: enzymes, renal function, Coagulation
- Imaging tests
  - MRI, ultrasonograph, CT scan
- Others
  - EEG, aEEG

Investigations for HIE

- Imaging assessment:
  - cranial ultrasound

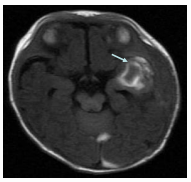


PVL

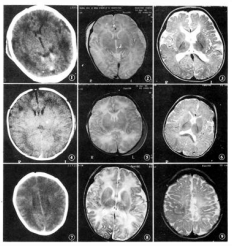
IVH (Ⅳ°)

Investigations for HIE

- Imaging assessment:
  - brain CT scan
  - MRI and spectroscopy



Intracranial hemorrhage



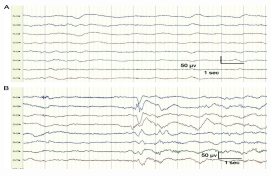
HIE in term infant

Investigations for HIE

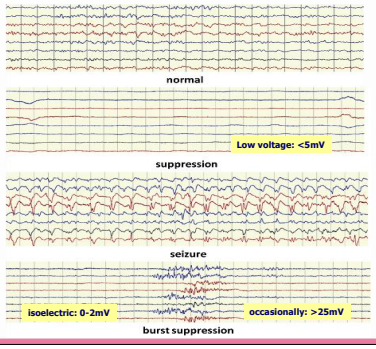
- Status of inner environment
  - Serum electrolyte
  - Blood glucose
  - ABG: PaO_2, $PaCO_2$, acid-base balance
  - Others: enzymes, renal function, Coagulation
- Imaging tests
  - MRI, ultrasonograph, CT scan
- Others
  - EEG, aEEG

Investigations for HIE

- Electroencephalogram (EEG):
  - may be normal during first few days
  - poor prognosis: suppressed or frequent seizure activity



Newborn EEG



Grades of HIE in term infants modified

(from Sarnat H, et al.)

Signs	Stage 1 mild	Stage 2 moderate	Stage 3 severe
Level of consciousness	Hyper-alert	lethargic	Stuporous, coma
Muscle tone	normal	hypotonic	flaccid
Moro reflex	strong	weak	absent
pupils	mydiasis	miosis	Unequal, poor light reflex
seizures	none	common	decerebrate rigidity
EEG	normal	Low voltage changing to seizure activity	Burst suppression to isoelectric
duration	<72hr if progresses	24hr to 14 days	Days to weeks
outcome	good	Variable	Death, severe deficit

Diagnosis of HIE

Only for term infant:

- ➢History of perinatal asphyxia
  - ✓Intrauterine, intrapartum, at birth
- ➢Symptoms of CNS: appear within 24 hours after birth and persist longer than 24 hours
- ➢Exclude the following conditions:
  - ✓Seizure related to electrolytes imbalance, intracranial hemorrhage
  - ✓Brain injury caused by congenital infection, inherited metabolic diseases and other congenital diseases.

Treatment of HIE

- therapeutic principles: avoiding further brain injury
  - – early: within 6hrs after insult
  - – combination: drugs and massage
  - – enough duration: until postnatal period
  - – confidence: the families

Treatment of HIE

- 4 stages of treatment in China
  - – within 3 day of life (key stage)
  - – 4 to 10 day of life
  - – after 10day
  - – Postneonatal period

Treatment of HIE

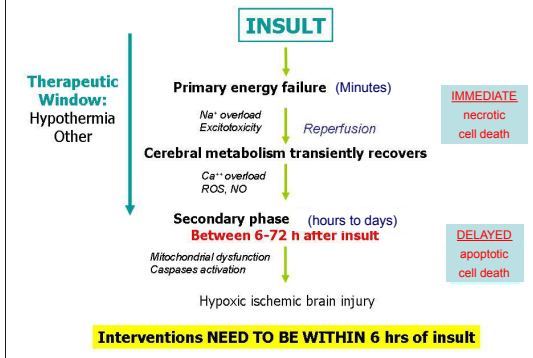
-- within 3 day of life (key stage)

- ✓purpose: stabilizing internal environment & controlling nervous symptom
- ✓3 items of supportive therapy:
  - ➢blood gas
  - ➢blood circulation
  - ➢blood sugar

Treatment of HIE

-- within 3 day of life (key point stage)

- 3 items against organ system manifestations
  - convulsions: phenobarbital
  - brain edema: Furosemide, mannitol
  - brainstem symptom: Naloxone ?
- Rescue neurocytes:
  - Hypothermia: several meta-analysis
  - Cerebrolysin, Citicoline, Salvia miltiorrhiza
  - Hyperbaric oxygen (HBO): controversial



Treatment of HIE

-- Postneonatal period period:

IF developmental quotient (DQ) <85, continue above therapies for 3-6 months

massage intervention training family members

TAECHING REQIREMENTS

1. definition of neonatal asphyxia.
2. diagnosis of neonatal asphyxia.
3. complications of birth asphyxia.
4. investigations of HIE.
5. how to grade of HIE.
6. treatment of HIE.

Case Ⅰ（1）

- ✓ Tom, male, 42 minutes old.
- ✓ Main complain: cyanosis and lethargy for 42 minutes.
- ✓ G2P1, GA: 39^{+2}wks, intrauterine asphyxia (fetal bradycardium) and low Apgar scores : 1'-4, 5'-7, 10'-9. BW=2815g, amnionic fluid stained by meconium (Ⅲ°).

Case Ⅰ（2）

- ✓ T 36.5℃，R 25-54/min，HR 158bpm，Bp 84/41 mmHg.
- ✓ Consciousness, lethargy, very weak cry. Cyanosis color of face while pale color of the extremities.
- ✓ Pupils: symmetric and round (D=3mm), very dull to light stimulation.
- ✓ irregular respiration and occasionally apnea; rhythm while blunt heart sound without murmur.
- ✓ Increased muscle tone and obviously weakened primitive reflexes.

Case Ⅰ（3）

- ✓ ABG：pH7.24，$PaCO_2$ 30mmHg，PaO_2 88mmHg，lactic acid 10.7mmol/L，HCO_3^- 12.9mmol/L。
- ✓ Hepatic and renal functions： ALT 70U/L；BUN 9.01mg/dL，Cr 92.3μmol/L。
- ✓ Serum electrolytes：calcium 1.62mmol/L。
- ✓ Cranial Ultrasound：lower echogenicity of brain.
- ✓ Cranial CT scan：decreased density of white matter, with fuzzy borderline between grey and white matter.

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Answers

- Initial diagnosis:
  - HIE
  - metabolic acidosis, hypocalcemia,
  - hepatic and renal injuries.
- Treatments:
  - Supportive and symptomatic therapy
  - Hypothermia ASAP