Hemiplegia – why, when and how?

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The Cerebral Palsies

Definition

A group of disorders characterised by abnormal control of movement or posture resulting from abnormality of brain development or an acquired non-progressive brain lesion.
Why?
Baby AH

- Preterm 33 weeks. NVD. RDS.
- 10m not sitting, adductor tone up
- 18m pulling to stand
- 21m ECOG: bilateral s-neural deafness
- 2y walking 1 hand held, “sinking gait”

Diagnosis: (spastic) diplegia, deafness
• Pregnancy: reduced fetal movements
• FTND: 16 hour labour
• Neonatal: low Apgars, ventilated minutes
• Floppy from birth, seizures for days
• Walked 4 years, wide based “ataxic” gait
• Speech problems, intellect ? OK
Diagnosis: athetoid CP
Baby JR

- FTND, B Wt 3.7 kgm
- Transposition, SABE, meningitis, NEC
- 15m sitting, L hemiplegia
- 3y severe expressive language delay
- 5y “quadriplegic”
- Diagnosis: 4 limb cerebral palsy
Baby JD

- FTND
- Normal b weight
- Home next day
- Back on day 2 with seizure, mainly L arm
- Seizures difficult to control for days
- Now well
Baby AB

- Normal pregnancy/delivery at term
- B weight 3.5 Kg
- Safe 6m
- 10m ?R handed
- 13m feeds with R hand
- 18m not yet walking, L foot adducted
- Normal school, L hemiplegia
Clinical and MRI correlates of cerebral palsy: the European Cerebral Palsy Study


585 children with CP b1996-1999; 431 clinically assessed; 351 had brain MRI

- 34.4% diplegia
- 26.2% hemiplegia
- 18.6% quadriplegia
- 14.4% dyskinesia
- 3.9% ataxia
- 2.6% other types of CP.
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Brain MRI scans
- periventricular leukomalacia 42.5%
- basal ganglia lesions 12.8%
- cortical/subcortical lesions 9.4%
- malformations 9.1%
- focal infarcts 7.4%
- miscellaneous lesions 7.1%
- Only 11.7% had normal MRI findings.
When?
New Data Show

1 in 278 Children Have Cerebral Palsy
Cerebral palsy - prevalence

• the most common physical disability in childhood
• 2-2.5% per 1000 children born
• no change in last 40 years
• some countries - increase due to increased survival of VLBW infants
Fig. 1 Cerebral-palsy prevalence per 1000 livebirths in eight surveyed populations.
Fig. 3. Spastic diplegia and other types of cerebral palsy by gestational age (from McDonald 1967, by courtesy of the Editor, Archives of Disease in Childhood).
Birthweight specific trends in cerebral palsy.
type of cerebral palsy by birthweight

- spastic bilateral
- spastic hemiplegia
- dyskinetic
- ataxic cp

frequency

birthweight
How?
Cerebral palsy - known antenatal causes

- hypoxic/ishaeemic
- vascular
- infective
- cerebral dysgenesis
- associations (IUGR, death of co-twin, prematurity)
- toxic (lead, mercury)
- metabolic (iodine deficiency)
Cerebral palsy and obstetric care

- most CP is due to birth asphyxia

- change in obstetric practice could prevent brain damage by preventing birth asphyxia

"Neither of these statements is true"

Stanley, Blair, Westaway. 1994
Cerebral palsy and obstetric care

In Western Australia despite improvements in obstetric care and a rise in the proportion of caesarian sections, cerebral palsy rates have remained constant since the mid-fifties.

Stanley, Blair, Westaway. 1994
All current research suggests that the majority of children with CP born at term were affected before labour commenced.

In Australia only about 8% of CP is associated with birth asphyxia and in the majority of cases, obstetric care could not have prevented it.

Stanley, Blair, Westaway. 1994
Antecedents of Cerebral palsy

- National Collaborative Perinatal Project

- 78% of CP infants: normal birth weight
- many had no perinatal risk factors
- those with risk factors also had antecedent risks or stigmata
- estimate - 9% of 189 CP cases were perinatally damaged

Nelson KB, Ellenberg JH. NEJM 1986;315:81-6
“Naturally” occurring cohorts

Congenital rubella syndrome

• W. Australia: 14% had major motor disorders (spastic diplegia, quadriplegia or ataxia)

• pathology due to later viral damage of developing CNS, rather than early teratogenic effect

“Naturally occurring cohorts”

Minimata disease - congenital CP in Japan

• 1953 - 1960: 6% of newborns had CP (v. 0.25%)
• mothers only mildly affected
• children: microcephaly, spasticity, ataxia, MR
• pathology: widespread in brain (cortex, cerebellum)
• ingestion of fish containing methyl mercury

Previously - thought that fetus protected by placenta
“Naturally” occurring cohorts

Papua New Guinea

- 1960 - 1966 Jimi Valley. Epidemic of “endemic” or “neurological cretins” - spasticity, deaf mutism and MR

- new source of salt from coast had no iodine

Pharoah & Hornabrook Lancet 1974;2:1038-40
Clinical and MRI correlates of cerebral palsy: the European Cerebral Palsy Study


Important findings

- high rate of infections reported by mothers during pregnancy (n = 158 [39.5%])
- 235 children (54%) born at term
- 47 children (10.9%) very preterm (<28 weeks)
- high rate of twins: 51 children (12%) multiple pregnancy.
Clinical and MRI correlates of cerebral palsy: the European Cerebral Palsy Study


CONCLUSIONS

• MRI findings suggest obstetric mishaps in a small proportion
• Maternal infections need systematic approach to identify and treat
• Multiple pregnancies should be monitored closely
• Causes of infant stroke need to be investigated further so preventive strategies can be formulated
• MRI for all children with CP to provide information on the timing and extent of the lesion
Childhood outcomes after prescription of antibiotics to pregnant women in spontaneous preterm labour

S Kenyon, K Pike, DR Jones, P Brocklehurst, N Marlow, A Salt, DJ Taylor  Lancet 2008;372

- children aet 7y born to 4221 women who completed the ORACLE study
- ORACLE II – spontaneous preterm labour and intact membranes: randomised to erythromycin, co-amoxiclav, placebo
- risk of cerebral palsy increased by either antibiotic (!)
  - erythromycin 3.3% v 1.7%, co-amoxyclyclav 3.2% v 1.9%

NB ORACLE children with CP: 28% <32w, 21.3% were <1500gm