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Covering Practice and Risk Management Issues for Physicians

# Cerebral Palsy: A Syndrome With Heterogeneous Etiology

BY DONGLI SONG, MD, PHD

**Cerebral palsy (CP) results from damage to the immature brain. CP is characterized by a set of nonprogressive motor disorders that are frequently accompanied by seizures and neurosensory and cognitive impairments. Despite advances in obstetric and neonatal care, the overall prevalence of CP has remained stable over the last 10 years, ranging from 1.0 to 2.5 per 1,000 live births. Population-based studies have identified perinatal stroke, chorioamnionitis, perinatal infection and inflammation, infertility, in vitro fertilization, multiple gestation and intrauterine growth retardation as important CP risk factors.**

While the etiology of CP is heterogeneous, extreme prematurity (< 28 weeks of gestation) is the most significant predictor as 5-15% of these infants will develop CP. However, more than half of all children with CP are born at term (even though they are at a relatively low absolute risk) because term infants constitute the majority of all births.

Recent neuroimaging evaluations of children with CP have revealed that 70-90% of affected children have identifiable underlying brain abnormalities which include perinatal stroke, congenital malformations, cerebral white matter injury, and hypoxia-ischemic encephalopathy (HIE), as well as postnatally acquired disorders. This article will focus on perinatal stroke and HIE to provide some recent advances on these issues.

## PERINATAL STROKE

Perinatal stroke has been identified as a leading cause of CP. Most perinatal strokes are focal arterial ischemic infarctions but at least 30% are due to sinovenous thrombosis. Perinatal arterial ischemic stroke (PAS) is especially notable as it is responsible for 50-70% of congenital hemiplegic CP. About half of PAS newborns present acutely with seizures without other signs of encephalopathy. Focal neurological signs, such as hemiparesis, occur in less than 25% of cases.

Although neuroimaging of symptomatic infants has identified PAS in approximately one in 4,000 term infants, the overall incidence of PAS is difficult to determine. This is because many affected infants are asymptomatic during the neonatal period with the diagnosis made retrospectively when motor deficits become apparent months or years later. A long list of maternal, placental and fetal/neonatal risk factors has been implicated for PAS, but it is important to note that newborns with PAS usually have more than one risk factor and other perinatal complications such as hypoxia-ischemia are frequently present.

Increasing awareness of perinatal stroke as a potential cause of CP should prompt obstetricians to identify infants at risk, and alert pediatricians to monitor these newborns closely.

Maternal/placental risk factors associated with neonatal stroke include inherited and acquired coagulation abnormalities (decreased levels of protein S, protein C and antithrombin

*(continued on page 2)*

## Inside PRF News

## Cerebral Palsy *(continued from page 1)*

III, elevated levels of lipoprotein (a) and homocysteine, mutation/polymorphisms of Factor V Leiden G1691A, Factor II G20210A, methylenetetrahydrofolate reductase C677T), autoimmune disease/antibodies, preeclampsia, intrauterine growth restriction, in utero cocaine exposure, acute and chronic placenta lesions (thrombosis, infection, abruption, feto-fetal, maternal hemorrhage) while fetal/neonatal risk factors include congenital heart disease, hypercoagulopathy, polycythemia/dehydration, birth asphyxia, and CNS infection.

Brain-imaging evaluation of infants with seizure or focal neurological signs should help with early diagnosis of perinatal strokes. Currently, heparin anticoagulant therapy is only recommended for neonates with a cardioembolic source. Supportive care is essential in PAS acute management to prevent infarct extension and secondary brain injury. Furthermore, proper parent consoling, close neurodevelopmental follow up and early intervention are all critical steps for a better functional outcome.

### HIE

Hypoxia-ischemic encephalopathy (HIE) is one of the most recognizable causes of neonatal encephalopathy (NE) and a major predictor of neonatal mortality and long-term neurological deficits, yet a much-debated question relates to the extent of the contribution of intrapartum asphyxia/HIE to CP. This issue has been addressed by large population-based studies conducted in Europe, Australia, Canada and the United States. The methods used in these studies involve prospective follow up of NE infants to determine what percentage of these infants develop CP later on in life, or retrospective analyses of children with CP to determine whether NE was implicated as the cause of CP. Based on these studies, the proportion of CP that may be due to perinatal events is estimated to be approximately 20%.

Studies from Sweden and Northern California Kaiser Hospitals have shown that about one third of children with CP experienced acute brain injury around the time of birth. Recent MRI evaluations of NE infants have revealed specific patterns of brain injury, timing and potential causes of NE with 80% of NE infants appearing to have acutely evolving lesions compatible with HIE, yet not all NE infants will develop permanent neurological impairment. However, as proposed by ACOG Task Force criteria, a pathway linking intrapartum asphyxia to subsequent CP must progress through NE, and other causes of NE should be carefully excluded. It is important to recognize that about 50% of infants who suffer a cerebral insult shortly before the onset of labor have recovered sufficiently to have an uncomplicated labor and delivery but will develop NE soon after birth and have MRI findings consistent with acute brain injury. Thus, all NE infants should be evaluated by MRI in the neonatal period. Although few infants develop CP due to intrapartum events, they are the ones likely to benefit from postnatal therapeutic interventions, such as hypothermia, for the reduction or prevention of brain injury. ■

*Dr. Song joined the San Francisco Neonatology Medical Group of California Pacific Medical Center in 2005.*

***Maternal/placental risk factors associated with neonatal stroke include inherited and acquired coagulation abnormalities, autoimmune disease/antibodies, preeclampsia, intrauterine growth restriction, in utero cocaine exposure, acute and chronic placenta lesions (thrombosis, infection, abruption, feto-fetal, maternal hemorrhage) while fetal/neonatal risk factors include congenital heart disease, hypercoagulopathy, polycythemia/dehydration, birth asphyxia, and CNS infection.***

# Myth Regarding Breast Bumps and Fibrocystic Change

Editor –

"The most recent issue of *PRF News* (Volume 9, Number 2) contained an important article on "Breast Evaluation and Risk Management" by R. James Brenner, MD. However, in that article Dr. Brenner may be perpetuating a bad myth. There is no combination of "lumps and bumps that define fibrocystic change" in any literature or any textbook!

It is problematic to assume that a person can decide that there is "fibrocystic change" that can be diagnosed by some combination of lumps and bumps on clinical breast examination. In fact, making such assumptions is the leading cause of delayed diagnosis of breast cancer (Goodson and Moore, *Arch Internal Med* June 2002), affecting about 5 percent of women in the Bay Area in our sample.

It is a disservice to everyone to perpetuate the misconception that fibrocystic condition is a clinical diagnosis. Fibrocystic condition is a specific pathologic change defined by the pathologist when she/he looks at biopsy material under the microscope.

While it may be appropriate to discuss cancer prevention strategies with women at sufficient risk, the criteria for use of these prevention strategies are still under development, particularly since all of them introduce their own significant risks from the treatment itself. Thus, because standard criteria do not exist at this time, having such discussions may serve to address the medical needs of the patient as well as preempt any allegation that an aggrieved patient with breast cancer would have selected such a choice had it been offered, but cannot be considered standard of care.

*William H. Goodson III, MD*
*PRF Insured*
*Risk Management &*
*Education Committee*

# 30 Years of Claims Experience

**The Physicians Reimbursement Fund has accumulated 30 years of claims experience. Our outstanding claims history when compared to national statistics supports our active "Code Green" policy and effective case review by the Patient Care and Management Committee.**

The success of Code Green can be easily seen by comparing the average total costs per case of a claim that has been resolved by negotiating a settlement as opposed to trial, arbitration or mediation. In fact, the average cost of resolving a claim by trial is nearly 50 percent greater than the average cost of resolving a claim by negotiating a settlement. The costs associated with resolving claims by arbitration and mediation are considerably higher. The Code Green principle was applied in the majority of cases where a settlement was negotiated.

We are not without our challenges, however, as we try to keep premiums competitive. Defense costs and the associated costs of arbitration continue to escalate. Including Code Green payments, we have paid out over $18 million to date on behalf of our members. The Board of Directors will continue to develop strategies to effectively manage our defense costs and risk management programs to minimize claims.

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