

Preface



Marcus C. Hermansen, MD
Guest Editor

Although cerebral palsy is rarely diagnosed in the fetus or newborn infant, the etiology is nearly always from a perinatal or neonatal cause. This issue of the *Clinics in Perinatology* brings together an impressive multinational group of authorities to review the etiologies of cerebral palsy. After analyzing each for this publication, this Guest Editor estimated the contribution of each process toward the total population of patients with cerebral palsy as follows:

Prematurity and intrauterine growth rate restriction: 40% to 50%

Birth asphyxia or birth trauma: 25% to 30%

Neonatal stroke: 5% to 10%

Toxoplasmosis, rubella, cytomegalovirus, herpes simplex, other infections:
5% to 10%

Chromosomal abnormalities: 5% to 10%

Inborn errors of metabolism: 5% to 10%

Other known causes: 5% to 10% (neonatal sepsis/meningitis, kernicterus, hypoglycemia, environmental toxins, drug and alcohol exposure, maternal thyroid disease, postnatal infections and trauma, and others)

Idiopathic: 5% to 10%

There are some obvious difficulties in producing such estimates. Many cases of cerebral palsy may be considered to have more than one cause. For example,

if a premature infant suffers severe birth asphyxia, followed by a large intraparenchymal hemorrhage, and ultimately cerebral palsy, the case can be attributed to both prematurity and birth asphyxia. If another infant with sepsis suffers protracted hypoglycemia with seizures, that case might be attributed to both sepsis and hypoglycemia. For this reason the estimates may produce a total greater than 100%.

Recently the association between chorioamnionitis and cerebral palsy has drawn much attention, but chorioamnionitis does not appear on the list of etiologies. The cases in which chorioamnionitis caused cytokine-induced periventricular leukomalacia in the preterm infant are classified as cases of prematurity in the estimates. In term babies the most likely mechanism of damage from chorioamnionitis is placenta dysfunction with subsequent birth asphyxia. These cases are classified as birth asphyxia in the estimates.

One of the more controversial estimates is birth asphyxia. The contribution attributed to asphyxia depends on the criteria used to define the condition. For example, the presence of marginally low Apgar scores alone would be considered an unacceptably liberal definition of “birth asphyxia as a cause of cerebral palsy” and would produce an excessively high estimate attributed to asphyxia. As one imposes increasingly stringent criteria for defining birth asphyxia as a cause of cerebral palsy, the contribution of asphyxia to cerebral palsy decreases accordingly. Perhaps the most stringent criteria are those proposed by the American College of Obstetricians and Gynecologists’ Task Force on Neonatal Encephalopathy and Cerebral Palsy. The use of such stringent criteria will produce an inaccurately low estimate of the risks of cerebral palsy following birth asphyxia.

It is noteworthy that the contribution attributable to an idiopathic process continues to decrease with advances in diagnostic testing. This issue of the *Clinics in Perinatology* presents the recent advances in neuroimaging, placenta pathology, chromosomal analysis, and metabolic testing that now explain cases of cerebral palsy previously classified as idiopathic.

I have been honored to work with this outstanding group of contributors. I believe you, the readers, will appreciate and benefit from every article in this collaboration.

Finally, I would like to acknowledge the support I have received from my friends, family, and colleagues in producing this work. I offer special thanks to my children and grandchildren—Sloan, Ian, Vanessa, Lauren, Caitlin, Dawson, Luke, and Quincy—for their love and support.

Marcus C. Hermansen, MD
Southern New Hampshire Medical Center
8 Prospect Street
Nashua, NH 03061-2014, USA
E-mail address: Marcus.Hermansen@SNHMC.org