CP in infants of normal birth weight and preterm infants (Hermansen and Hermansen, 2006; Shatrov, Birch, Lam, et al, 2010); however, not all term infants exposed to chorioamnionitis develop CP.

In general, infants exposed to maternal and perinatal infections are at increased risk for the development of CP as a result of the effects on the developing brain. Although CP occurs in term births, preterm birth of ELBW and VLBW infants continues to be the single most important risk factor for CP. Still, in some cases no identifiable cause is determined. Periventricular leukomalacia and intracerebral hemorrhage in low birth weight (LBW) infants are significant risk factors in the development of CP. Perinatal ischemic stroke is also associated with a later diagnosis of CP (Golomb, Saha, Garg, et al, 2007).

Additional factors that may contribute to the development of CP postnatally include bacterial meningitis, multiple births, viral encephalitis, motor vehicle crashes, and child abuse (shaken baby syndrome [traumatic brain injury]) (Krigger, 2006). One study found a higher risk of CP occurring among infants born at 42 weeks' gestation or later than among those born at 37 or 38 weeks' gestation (Moster, Wilcox, Vollset, et al, 2010). One study found that 10% to 15% of children with CP acquired the condition after birth from causes such as falls, motor vehicle crashes, and infections, such as meningitis (Centers for Disease Control and Prevention, 2013). A significant percentage (15% to 60%) of children with CP also have epilepsy. In summary, as many as 80% of the total cases of CP may be linked to a perinatal or neonatal brain lesion or brain maldevelopment, regardless of the cause (Krageloh-Mann and Cans, 2009). A number of biochemical disorders may cause motor abnormalities often seen in CP and may be initially misdiagnosed as CP (Nehring, 2010).

Pathophysiology

It is difficult to establish a precise location of neurologic lesions on the basis of etiology or clinical signs, because there is no characteristic pathologic picture. In some cases, there are gross malformations of the brain. In others, there may be evidence of vascular occlusion, atrophy, loss of neurons, and laminar degeneration that produce narrower gyri, wider sulci, and low