CANADIAN PAEDIATRIC SOCIETY STATEMENT

FN 2001-01

Routine screening cranial ultrasound examinations for the prediction of long term neurodevelopmental outcomes in preterm infants

Since the late 1970s, cranial ultrasound examinations have been performed on preterm infants to provide information about perinatal brain injury for the prediction of long term outcomes (1). Cranial ultrasound examinations and nonultrasound brain imaging techniques, such as magnetic resonance imaging, have also been used for a variety of clinical indications (2-5). Because of recent developments in the technology and process of performing cranial ultrasound examinations (6), and in the classification and understanding of the predictive value of abnormal findings (7-11), it is timely and important to consider the value and process of determining the need for routine screening cranial ultrasound examinations in neonatal intensive care units (NICUs).

OBJECTIVES

The guidelines presented in this statement were prepared to provide caregivers with answers to the questions: Should cranial ultrasound examinations be performed routinely on preterm infants for the prediction of long term neurodevelopmental outcomes? If so, when should cranial ultrasound examinations be performed? Is it possible to predict long term neurodevelopmental outcomes in preterm infants who develop hemorrhagic or ischemic lesions that are documented on routine cranial ultrasound examinations? What actions should be undertaken if infants are found to have hemorrhagic or ischemic brain lesions?

METHODS

A literature review found studies reported in English and French with any of the following keywords: cranial ultrasound, germinal matrix (GM) hemorrhage, intraventricular hemorrhage (IVH), brain injury, ventriculomegaly, echodensity, porencephaly, periventricular leukomalacia and preterm infant. The literature review consisted of a search of MEDLINE from 1976 to 2000 and a review of reference lists of literature identified by the search.

PATHOPHYSIOLOGY OF COMMON ABNORMALITIES

The fetal or preterm infant's brain is vulnerable to both hemorrhagic and ischemic injury during the late second and early third trimesters (1). This is due to vascular, cellular and anatomical features of the developing brain, and the tendency for preterm infants to experience periods of physiological instability at a time when they have limited cerebral circulatory autoregulation (12). Hemorrhagic lesions (GM and/or IVH) can be restricted to the GM, they can include bleeding into the ventricles (with or without the development of hydrocephalus) or, in the worst cases, they can be found in the brain parenchyma (13). By 32 weeks' postconceptual age, the GM is found only along the ventricular surface of the caudate

TABLE 1: Classification of hemorrhagic and/or ischemic abnormalities detected by cranial ultrasound examinations

- Isolated GM and/or IVH
- Parenchymal echodensities and/or lucencies with or without GM and/or IVH
- Ventricular enlargement with or without GM and/or IVH

GM/IVH Germinal matrix and/or intraventricular hemorrhage

nucleus and at its border with the thalamus. It normally involutes by 34 to 36 weeks' (3,14) postconceptual age. The incidence of GM and/or IVH is infrequent after that time. Hemorrhagic and ischemic injuries often occur together, even though different pathophysiological processes lead to the lesions (15). The vascular structure of the cerebral white matter in mid- to late gestation includes long penetrating arteries that originate from the anterior, middle or posterior cerebral artery (1). The end zones of these arteries are especially prone to hypoperfusion and ischemia, and, thus, there is an increased likelihood of ischemic necrotic damage along the course or end zones of the arteries, or in the periventricular area. Nonhemorrhagic cerebral infarction, ventriculomegaly or cystic lesions, such as periventricular leukomalacia or porencephaly, may evolve from white matter injuries (16-18). Ventriculomegaly that occurs in the absence of IVH is most often secondary to the loss of cerebral white matter that has been damaged or failed to develop normally (19,20).

CLASSIFICATION OF ABNORMAL CRANIAL ULTRASOUND FINDINGS

Different systems have been suggested for the classification (7,8,10,21) and grading (13) of hemorrhagic and ischemic lesions that are detected by neonatal cranial ultrasounds. Grading has traditionally been performed using the system described by Papile et al (13): grade 1 IVH is confined to the GM; grade 2 IVH involves bleeding into the ventricle; in grade 3 IVH, the blood has distended and enlarged the ventricle; and grade 4 hemorrhage refers to bleeding within the brain parenchyma. However, a drawback of this system is that it excludes certain types of hemorrhages, and lacks concordance with the pathophysiology of both severe hemorrhagic and white matter damage (22). A valuable approach to the classification of abnormalities noted by cranial ultrasounds is provided in Table 1 (10). This approach does not specifically include white matter lesions in brain locations other than periventricular areas such as the cerebellum, basal ganglia or brain stem (22); however, it does assist caregivers in interpreting the most common categories of abnormal ultrasound diagnostic findings in preterm infants.

WHICH INFANTS NEED CRANIAL ULTRASOUND EXAMINATIONS?

Because preterm infants, especially those younger than 32 weeks' gestation, are prone to both GM and/or IVH and ischemic white matter injuries, routine cranial ultrasound examinations are most valuable for this group (23). The maximum risk of GM and/or IVH is in infants born before 30 weeks' gestation (24); the incidence of IVH is less than 5% after that time (23,24). However, because occasional abnormalities are detected in infants born after 30 weeks' gestation, it seems prudent to perform routine cranial ultrasounds on infants born at or before 32 weeks' gestation.
Recommendation: Routine screening cranial ultrasound examinations are recommended for all infants born at 32 weeks' gestation or earlier.

WHEN SHOULD CRANIAL ULTRASOUND EXAMINATIONS BE PERFORMED?

Among infants who developed GM and/or IVH, at least one-third of the infants had echodensities as early as 1 h after birth, indicating an antenatal or immediate postnatal onset (25,26). Approximately 50% of GM and/or IVH occurred in the first 6 to 8 h after birth (27). Most IVHs are evident by the third day (25,28), but they can develop at any time during the first two weeks of life. It has been suggested that cranial ultrasounds performed near two weeks of age provide the most complete and reliable diagnoses of hemorrhagic lesions (9). A concern with delaying cranial ultrasounds until two weeks after birth is that early onset IVHs may be of more ominous prognostic value than later onset IVHs (29); however, if only one ultrasound examination is to be performed to predict long term neurodevelopmental outcomes, the most appropriate time is two weeks after birth.

White matter abnormalities, which may manifest as echodensities, ventriculomegaly or cystic changes, may be present at birth but they often appear later (30). Cystic periventricular leukomalacia of antenatal onset is evident by two weeks of age (31). The evolution of postnatally acquired periventricular leukomalacia has been described as a period of initial congestion (manifest as echodensities), followed by relative normalization, followed by the development of echolucency or cysts and, finally, cyst resolution occurs with the development of ventricular enlargement (16). This sequence may not be completed until three months of age or later, but most cysts are evident within 60 days of birth (30).

Caregivers of very low birth weight infants who have multiple early complications may prefer to have diagnostic cranial ultrasound examinations performed by the third day of life. An ultrasound at this time provides important input for both short term and long term clinical management (29). Repeated ultrasounds should be performed, as clinically indicated, in infants with identified brain injuries.
Recommendation: Routine cranial ultrasound examinations are recommended at about the second and the

sixth weeks of life to predict long term outcomes; early ultrasound examinations allow diagnosis of hemorrhagic lesions, and later ultrasound examinations can detect cystic lesions or ventriculomegaly.

PREDICTIVE VALUE OF ABNORMALITIES DETECTED BY CRANIAL ULTRASOUNDS

Periventricular brain damage, whether it is hemorrhagic, ischemic or both, can be associated with abnormalities in neurodevelopmental outcome (11,32,33). Some of the earlier reports focused on infant outcomes associated with isolated GM and/or IVH, but in most cases, infants with such lesions were not disabled (8). The outcome abnormalities found in GM and/or IVH with white matter damage, however, can range from subtle cognitive abnormalities to borderline or severe mental retardation (34,35). Motor abnormalities are reported often, particularly in association with disabilities such as cerebral palsy (10,21,36,37), mental retardation (2), or visual or hearing disturbances (38).

The sensitivity of cranial ultrasound examinations as predictors of later neurodevelopmental abnormalities has been reported as 16% at one and two weeks after birth, increasing to 53% at six weeks and 58% if performed when a child is at term-corrected age (39). The specificity of cranial ultrasound examinations has been 99% to 100% in all age groups (39). The presence of cysts has been reported as a predictor of cerebral palsy, with a sensitivity of 67% and a specificity of 96% (36). In most reports, estimates of the negative predictive value of cranial ultrasound examinations are consistent in that repeated normal examinations predict that an infant is unlikely to have cerebral palsy (33,36).

The specific location of cystic periventricular brain damage has been reported to be of predictive value in some but not all studies (17,40,41). Cysts are located most often at the level of the optic radiations adjacent to the trigone and at the level of the frontal white matter near the foramen of Monro. Descending fibres from the motor cortex are generally located superior and lateral to the lateral ventricles, and those fibres that are most closely related to lower extremity function are adjacent to the lateral ventricles. Therefore, leukomalacia in those areas is linked to the development of spastic diplegia. Quantitative measurements of cystic periventricular leukomalacia are difficult to accomplish by ultrasound. Quantitative measurements have included the diameters of the largest cyst or cystic region and the total cerebral mantle thickness. Parasagittal measurements of the anteroposterior dimension of cystic periventricular leukomalacia may best predict which infants will have quadriplegia, and the more severe cognitive and sensory impairments (41). Psychiatric abnormalities, including disruptive disorders, attention deficit/hyperactivity disorder, anxiety disorder or tics (42), have been reported in infants with GM and/or IVH and white matter damage.

Prenatally diagnosed isolated ventriculomegaly, in the absence of associated anomalies, is not a strong predictor of cognitive or motor abnormalities (43). However, ventricular enlargement that is detected postnatally may be more ominous in infants who develop posthemorrhagic hydrocephalus that requires shunting and in infants who develop ventriculomegaly without posthemorrhagic hydrocephalus.

In published reports, details are often lacking regarding the timing, type, severity and laterality of abnormalities noted on cranial ultrasound examinations. In addition, in some reports, long term outcome assessments are recorded in broad outcome categories that may interfere with precise interpretation. Some investigators have not reported other neonatal and postneonatal factors that can influence outcome (44,45), making it difficult to isolate the impact of the abnormalities noted on cranial ultrasounds.

Recommendation: Routine cranial ultrasound examinations can provide a relatively sensitive and highly specific means of predicting the presence or absence of later neurodevelopmental abnormalities in preterm infants.

HOW SHOULD CRANIAL ULTRASOUND EXAMINATIONS BE PERFORMED?

Advances in cranial ultrasonography have led to the improved visualization of hemorrhagic or ischemic lesions, with minimal risk to infants. The availability of real-time imaging and the use of the anterior fontanelle as an ultrasonographic 'window' were major developments in the application of neonatal ultrasonography (3). Although ultrasonographic approaches vary, ultrasound scans are done through the anterior fontanelle using sequential coronal and parasaggital projections; the posterior fontanelle is used for a detailed depiction of periventricular white matter or the presence of small amounts of blood in the lateral ventricles (15,46). It has been suggested that a scan performed through the mastoid fontanelle provides optimal visualization of the posterior fossa structures (6,47); however, a scan through the mastoid fontanelle is not performed routinely.

Pinto et al (48) studied the interobserver variability in the interpretation of abnormalities of the GM, ventricles and parenchyma; concordance among readers was the lowest for GM hemorrhages and the highest for parenchymal hemorrhages and echodensities. The American Institute of Ultrasound in Medicine and the American College of Radiology (49) have published guidelines for ultrasound examination of the brains of paediatric patients.

Recommendation: Caregivers should be aware that there will be differences in the diagnosis and interpretation of cranial ultrasound examinations according to the available ultrasound technology and expertise.

BENEFITS, HARMS AND COSTS

Cranial ultrasound examinations can provide a safe and effective screening and diagnostic test of GM and/or IVH or ischemic periventricular white matter damage in

preterm infants. However, whenever testing is performed early in life for the purpose of predicting long term neurodevelopmental outcomes, the caregiver should be aware of the likelihood of false positive and false negative results when communicating test results and the risk of later problems to the parents of each infant. If the timing of the cranial ultrasound examination is not optimal or if there are technical concerns, uncertainty increases about the predictive value of the results. In fact, uncertainty is intrinsic in all predictions that are made in relation to the long term outcome of preterm infants (50). The possibility of increasing parental anxiety or distress by presenting results that are actually false positives, or minimizing the risks of long term neurodevelopmental sequelae on the basis of false negative results, provide examples of the potential harm related to routine cranial ultrasound examinations.

The major benefits of testing are to direct families of affected infants toward the most appropriate follow-up facilities to promote early diagnosis and intervention for chronic neurodevelopmental sequelae of hemorrhagic or ischemic brain injury, and to foster ongoing research activities that are aimed at ensuring the best possible outcomes for all infants. Costs that have been considered are related to the performance of cranial ultrasounds, such as the ultrasound hardware, human resources and possible infant health risks, which are minimal, and parental anxiety associated with erroneous or delayed results. One must be cognizant of the costs related to follow-up diagnostic and treatment activities, which may occur at an earlier age than would have been the case if routine cranial ultrasound examinations were not performed. It is hoped that the recommendations in this statement encourage the most cost effective use of routine cranial ultrasound examinations in preterm infants.

Recommendation: The potential benefits and harmful consequences of misinterpreting cranial ultrasound examinations should be communicated to parents whose infants are undergoing the testing. Infants who have hemorrhagic lesions or any white matter or cystic lesions evident on cranial ultrasound examinations require close, systematic follow-up after their discharge from NICUs to facilitate the timely initiation of interventions.

SUMMARY OF RECOMMENDATIONS

- Routine screening cranial ultrasound examinations are recommended for all infants born at 32 weeks' gestation or earlier.
- Routine cranial ultrasound examinations are recommended at about the second and the sixth weeks of life to predict long term outcomes; early ultrasound examinations allow diagnosis of hemorrhagic lesions, and later ultrasound examinations can detect cystic lesions or ventriculomegaly.
- Routine cranial ultrasound examinations can provide a relatively sensitive and highly specific means of predicting the presence or absence of later neurodevelopmental abnormalities in preterm infants.
- Caregivers should be aware that there will be differences in the diagnosis and interpretation of cranial ultrasound examinations according to the available ultrasound technology and expertise.
- The potential benefits and harmful consequences of misinterpreting cranial ultrasound examinations should be communicated to parents whose infants are undergoing the testing. Infants who have hemorrhagic lesions or any white matter or cystic lesions evident on cranial ultrasound examinations require close, systematic follow-up after their discharge from NICUs to facilitate the timely initiation of interventions.

REFERENCES

1. Volpe JJ. Brain injury in the premature infant. Neuropathology, clinical aspects, pathogenesis and prevention. Clin Perinatol 1997;24:567-87.
2. Wilkinson I, Bear J, Smith J, et al. Neurological outcome of severe cystic periventricular leukomalacia. J Paediatr Child Health 1996;32:445-9.
3. Cohen HL. Neurosonography of the infant: The normal examination. In: Timor-Tritsch IE, Monteagudo A, Cohen HL, eds. Ultrasonography of the Prenatal and Neonatal Brain. Stamford: Appleton & Lange, 1996:259-85.
4. Inder T, Huppi P, Zientra GP, et al. Early detection of periventricular leukomalacia by diffusion-weighted magnetic resonance imaging techniques. J Pediatr 1999;134:631-4.
5. Stewart AL, Rifkin L, Amess PN, et al. Brain structure and neurocognitive and behavioural function in adolescents who were born very preterm. Lancet 1999;353:1653-7.
6. Taylor GA. Recent advances in neonatal cranial ultrasound and doppler techniques. Clin Perinatol 1997;24:677-91.
7. Low J. Motor and cognitive development of infants with intraventricular hemorrhage, ventriculomegaly, or periventricular parenchymal lesions. Am J Obstet Gynecol 1986;155:750-6.
8. Aziz K, Vickar DB, Sauve RS, Etches PC, Pain KS, Robertson CM. Province-based study of neurologic disability of children weighing 500 through 1249 grams at birth in relation to neonatal cerebral ultrasound findings. Pediatrics 1995;95:837-44.
9. Boal DK, Watterberg KL, Miles S, Gifford KL. Optimal cost-effective timing of cranial ultrasound screening in low birth weight infants. Pediatr Radiol 1995;25:425-8.
10. Pinto-Martin JA, Riolo S, Cnaan A, Holzman C, Susser MW, Paneth N. Cranial ultrasound prediction of disabling and nondisabling cerebral palsy at age 2 in a low birth weight population. Pediatrics 1995;95:249-54.
11. Levene MI. Is neonatal cerebral ultrasound just for the voyeur? Arch Dis Child 1988;63:1-2.
12. Pryds O. Control of cerebral circulation in the high-risk neonate. Ann Neurol 1991;30:321-9.
13. Papile L-A, Munsick-Brown G, Schaefer A. Relationship of cerebral intraventricular hemorrhage and early childhood neurologic handicaps. J Pediatr 1983;103:273-7.
14. Volpe J. Intracranial hemorrhage: germinal matrix-intraventricular hemorrhage of the premature infant. In: Volpe J, ed. Neurology of the Newborn. Philadelphia: WB Saunders Company, 1994:403-66.
15. Kuban K, Sanocka U, Leviton A, et al. White matter disorders of prematurity. J Pediatr 1999;134:539-46.
16. Dubowitz LMS, Bydder GM, Mushin J. Developmental sequence of periventricular leukomalacia. Arch Dis Child 1985;60:349-55.
17. Fawer CL, Diebold P, Calame A. Periventricular leukomalacia and neurodevelopmental outcome in preterm infants. Arch Dis Child 1987;62:30-6.
18. Fujimoto S, Yamaguchi N, Togari H, Wada Y, Yokochi K. Cerebral

palsy of cystic periventricular leukomalacia in low birth weight infants. Acta Paediatr Scand 1994;83:397-401.
19. Leviton A, Gilles F. Ventriculomegaly, delayed myelination, white matter hypoplasia, and periventricular leukomalacia: How are they related? Pediatr Neurol 1996;15:127-36.
20. Roland EH, Hill A. Intraventricular hemorrhage and posthemorrhagic hydrocephalus. Current and potential future interventions. Clin Perinatol 1997;24:589-605.
21. Hesser U, Katz-Salamon M, Mortensson W, Floodmark O, Forssberg H. Diagnosis of intracranial lesions in very low birthweight infants by ultrasound; incidence and association with potential risk factors. Acta Paediatr Suppl 1997;419:16-26.
22. Paneth N. Classifying brain damage in preterm infants. J Pediatr 1999;134:527-9.
23. Batton DG, Holtrop P, De Witte D, Pryce C, Roberts C. Current gestational age-related incidence of major IVH. J Pediatr 1994;125:623-5.
24. Harding D, Kuschel C, Evans N. Should preterm infants born after 29 weeks' gestation be screened for intraventricular hemorrhage? J Paediatr Child Health 1998;34:57-9.
25. Paneth N, Pinto-Martin J, Gardiner J, et al. Incidence and timing of germinal matrix/intraventricular hemorrhage in low birth weight infants. Am J Epidemiol 1993;137:1167-76.
26. Shaver DC, Bada HS, Korones SB, Anderson GD, Wong SP, Arheart KL. Early and late intraventricular hemorrhage: The role of obstetric factors. Obstet Gynecol 1992;80:831-7.
27. Ment LR, Oh W, Ehrenkranz RA, et al. Low-dose indomethacin and prevention of intraventricular hemorrhage: A multicenter randomized trial. Pediatrics 1994;93:543-50.
28. Dolfin T, Skidmore MB, Fong KW, Hoskins EM, Shennan AT. Incidence, severity, and timing of subependymal and intraventricular hemorrhages in preterm infants born in a perinatal unit as detected by serial real-time ultrasound. Pediatrics 1983;71:541-6.
29. Vohr B, Allan WC, Scott DT, et al. Early-onset intraventricular hemorrhage in preterm neonates: Incidence of neurodevelopmental handicap. Semin Perinatol 1999;23:212-7.
30. Goetz M, Gretebeck R, Oh K, Shaffer D, Hermansen M. Incidence, timing and follow-up of periventricular leukomalacia. Am J Perinatol 1995;12:325-7.
31. Ito T, Hashimoto K, Kadowaki K, et al. Ultrasonographic findings in the periventricular region in premature infants with antenatal periventricular leukomalacia. J Perinat Med 1997;25:180-3.
32. Tudehope DI, Masel J, Mohay H, et al. Neonatal cranial ultrasonography as predictor of 2 year outcome of very low birthweight infants. Aust Paediatr J 1989;25:66-71.
33. Levene MI. Cerebral ultrasound and neurological impairment. Arch Dis Child 1990;65:469-71.
34. Ross G, Boatright S, Auld PA, Nass R. Specific cognitive abilities in 2-year old children with subependymal and mild intraventricular hemorrhage. Brain Cognition 1996;32:1-13.
35. Whitaker AH, Feldman JF, Van Rossem R, et al. Neonatal cranial ultrasound abnormalities in low birth weight infants: Relation to cognitive outcomes at 6 years of age. Pediatrics 1996;98:719-29.
36. Graham M, Levene MI, Trounce JQ, Rutter N. Prediction of cerebral palsy in very low birthweight infants: Prospective ultrasound study. Lancet 1987;ii:593-6.
37. Doyle LW, Betheras FR, Ford GW, Davis NM, Callanan C. Survival, cranial ultrasound and cerebral palsy in very low birth weight infants: 1980s versus 1990s. J Paediatr Child Health 2000;36:7-12.
38. Jacobson L, Ek U, Fernell E, Flodmark O, Broberger U. Visual impairment in preterm children with PVL – visual, cognitive and neuropediatrics characteristics related to cerebral imaging. Dev Med Child Neurol 1996;38:724-35.
39. Nwaesei C, Allen AC, Vincer MJ, et al. Effect of timing of cerebral ultrasonography on the prediction of later neurodevelopmental outcome in high-risk preterm infants. J Pediatr 1988;112:970-5.
40. Weiss HE, Goldstein RB, Piecuch RE. A critical review of cranial ultrasounds: Is there a closer association between intraventricular blood, white matter abnormalities or cysts, and cerebral palsy? Clin Pediatr 1999;38:319-23.
41. Rogers B, Msall M, Owens T, et al. Cystic periventricular leukomalacia and type of cerebral palsy in preterm infants. J Pediatrics 1994;125(Suppl 1):S1-8.
42. Whitaker AH, Van Rossem R, Feldman JF, et al. Psychiatric outcomes in low-birth-weight children at age 6 years: Relation to neonatal cranial ultrasound abnormalities. Arch Gen Psych 1997;54:847-56.
43. Vergani P, Locatelli A, Strobelt N, et al. Clinical outcome of mild fetal ventriculomegaly. Am J Obstet Gynecol 1998;178:218-22.
44. Fowlie PW, Tarnow-Mordi WO, Gould CR, Strang D. Predicting outcome in very low birthweight infants using an objective measure of illness and cranial ultrasound scanning. Arch Dis Child Fetal Neonatal Ed 1998;78:F175-8.
45. Bendersky M, Lewis M. Effects of intraventricular hemorrhage and other medical environmental risks on multiple outcomes at age 3 years. J Dev Behav Pediatr 1995;16:89-96.
46. Anderson N, Allan R, Darlow B. Diagnosis of intraventricular hemorrhage in the newborn: Value of sonography via the posterior fontanelle. Am J Roentgenol 1994;163:893-6.
47. Cohen HL. Neurosonography of the infant: Diagnosis of abnormalities. In: Timor-Tritsch IE, Monteagudo A, Cohen HL, eds. Ultrasonography of the Prenatal and Neonatal Brain. Stamford: Appleton & Lange, 1996:259-85.
48. Pinto J, Paneth N, Kazam E, et al. Interobserver variability in neonatal cranial ultrasonography. Paediatr Perinat Epidemiol 1988;2:43-58.
49. American Institute of Ultrasound Medicine. Guidelines for the Performance of the Pediatric Neurosonology Ultrasound Examination. Rockville: American Institute of Ultrasound Medicine, 1991.
50. Eiser JR. Communication and interpretation of risk. Br Med Bull 1998;54:779-90.

FETUS AND NEWBORN COMMITTEE

Principal author: *Dr Reginald Sauve, Department of Pediatrics, University of Calgary, Calgary, Alberta*

Members: *Drs Khalid Aziz, Discipline of Paediatrics, Memorial University of Newfoundland, St John's, Newfoundland & Labrador (director responsible 2000-2001); Deborah Davis, Department of Pediatrics, Children's Hospital of Eastern Ontario, and Department of Obstetrics/Gynecology/Newborn Care, Ottawa Hospital, Ottawa, Ontario; Daniel Faucher, Department of Pediatrics, Royal Victoria Hospital, Montreal, Quebec; Shoo K Lee, Department of Pediatrics, University of British Columbia, Vancouver, British Columbia; Arne Ohlsson, Department of Pediatrics, Mount Sinai Hospital, Toronto, Ontario (chair 1999-2001); Gary Pekeles, The Montreal Children's Hospital, Montréal, Quebec (director responsible 1999-2000); John van Aerde, Department of Pediatrics, Stollery Children's Health Centre, Edmonton, Alberta; Michael Vincer, Department of Pediatrics, IWK-Grace Health Centre, Halifax Nova Scotia (1994-2000)*

Liaisons: *Drs Line Leduc, Hôpital Sainte-Justine, Montreal, Québec (Maternal-Fetal Medicine Committee, Society of Obstetricians and Gynaecologists of Canada); James Lemons, Riley Children's Hospital, Indiana University Medical Center, Indianapolis, Indiana (Committee on Fetus and Newborn, American Academy of Pediatrics); Cheryl Levitt, Hamilton Health Sciences Corporation, McMaster University Medical Centre, Hamilton, Ontario (College of Family Physicians of Canada, 1998-1999); Catherine McCourt, Bureau of Reproductive & Child Health, Health Canada, Ottawa, Ontario (Health Canada); Larry Reynolds, St Joseph's Health Care London, London, Ontario (College of Physicians and Surgeons of Canada 1999-2000); Saroj Saigal, Children's Hospital at Hamilton Health Sciences Corporation, Hamilton, Ontario (Neonatal-Perinatal Medicine Section, Canadian Paediatric Society, 1998-2000); Jill Elizabeth Boulton, Department of Pediatrics, St Joseph's Health Care London, London, Ontario (Neonatal-Perinatal Medicine Section, Canadian Paediatric Society); Ms Amanda Symington, Hamilton Health Sciences Centre – McMaster Site, Hamilton, Ontario (Neonatal Nurses)*

Consultants: *Drs Davis Elliot, Department of Diagnostic Imaging, Calgary Regional Health Authority, Calgary; Nigel Paneth, Department of Epidemiology, Michigan State University, East Lansing, Michigan*