# A New Pattern of Cerebellar Hemorrhages in Preterm Infants

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**ABSTRACT.** *Objective.* **Posterior fossa hemorrhages may be underdiagnosed in surviving neonates, with cerebellar hemorrhage discovered in 10% to 25% of autopsy specimens from very low birth weight infants. Posterior fossa lesions have been difficult to visualize by the traditional ultrasonography approach through the anterior fontanelle. Late in 1994, routine posterior fossa imaging through the posterolateral fontanelle was instituted to improve the ultrasonographic visualization of the posterior fossa in neonates.**

*Methods.* **Infants identified with posterior fossa hemorrhage by cranial ultrasonography between 1994 and 1996 were followed prospectively through discharge and their clinical courses reviewed. Infants diagnosed with posterior fossa hemorrhage between 1991 and 1994 were identified retrospectively from a comprehensive radiology database to use in comparison. All infants surviving to discharge were entered into neurodevelopmental follow-up using standard developmental assessments.**

*Results.* **Approximately 525 infants underwent cranial sonography during the study period between October 1994 and September 1996, including 250 infants weighing <1500 g. Thirteen infants were identified with posterior fossa hemorrhage using the posterolateral fontanellar approach. In contrast, only 2 infants were identified with posterior fossa hemorrhage between 1991 and 1994 using traditional anterior fontanellar views. Six very low birth weight infants were identified with cerebellar hemorrhages not associated with supratentorial, intraventricular hemorrhage. Each hemorrhage had a clinically silent presentation and, in 5 infants, was not well-appreciated by anterior fontanellar images. Magnetic resonance imaging studies were performed on 5 of the 6 infants and confirmed a cerebellar lesion in the area of previous echo density on ultrasonography. No infant is exhibiting motor abnormalities on neurologic examination, although 4 infants are demonstrating cognitive, developmental delay. Follow-up, however, is limited to a corrected age of ≤48 months.**

*Discussion.* **Cerebellar hemorrhage is an underrecognized and poorly visualized complication in preterm infants. Consistent imaging via the posterolateral fontanelle may demonstrate cerebellar hemorrhage missed by the anterior fontanellar approach. Cerebellar hemorrhage in low birth weight infants may be clinically silent and not associated with a significant supratentorial hemorrhage. These infants may survive to discharge. Long-term neurodevelopmental follow-up is necessary to establish the ultimate outcome of these infants. Future prospective study, using posterolateral fontanellar imaging, may elucidate further the pathophysiology of cerebellar hemorrhage in low birth weight infants.** *Pediatrics* **1998;102(6). URL: http://www.pediatrics.org/cgi/content/full/102/6/e62; *posterior fossa, cerebellar hemorrhage, neonate, ultrasound.***

ABBREVIATIONS. ECMO, extracorporeal membrane oxygenation, CT, computed tomography; MRI, magnetic resonance imaging; SEH, subependymal hemorrhage; MDI, Motor Development Index; PDI, Psychomotor Development Index.

Posterior fossa hemorrhage is a reported complication of a traumatic delivery,[1–6] extracorporeal membrane oxygenation (ECMO),[7,8] or coagulopathy.[2,8,9] It also has been reported in preterm infants as an extension of a severe intraventricular hemorrhage[2,10–14] or as a result of positive pressure mask ventilation with tight binding straps across the occiput.[12,15] Previous studies have identified posterior fossa hemorrhage in infants presenting with brainstem compression symptomatology, sudden shock, and disseminated intravascular coagulation.[2,3,5,6,14]

Published studies on preterm infants have suggested that cerebellar hemorrhage occurs concomitantly with an extensive supratentorial bleed, with an associated mortality approaching 100%.[2,14,16–18] It is likely, however, that posterior fossa hemorrhages have been underdiagnosed in living infants weighing <1500 g, with cerebellar hemorrhage discovered in 10% to 25% of autopsy specimens from low birth weight infants.[2,10–12,15]

Cerebellar hemorrhage has been difficult to visualize using the traditional ultrasonography approach through the anterior fontanelle, camouflaged by the highly echogenic tentorium and the cerebellar vermis. This difficulty may be exaggerated by the use of high-frequency transducers with more superficial, focal zones of penetration. Previous reports have indicated that ultrasonography through the anterior fontanelle is less sensitive than computed tomography (CT) in identifying posterior fossa hemorrhages.[1,3,5,19–21] CT remains impractical, however, for critically ill, low birth weight infants during the first few weeks of life, and CT also is limited by beam-hardening artifacts of the bony calvarium in the posterior fossa.

Recent advances in ultrasonography, including

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Parts of this work were presented at the meeting of the American Academy of Pediatrics, October 28, 1996, Boston, MA.

Received for publication Jan 14, 1998; accepted May 27, 1998.

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transducer head size and available transducer frequencies, have allowed better visualization through less used fontanelles such as the posterolateral fontanelle, present at the junction of the temporal, parietal, and occipital bones. This is a report of clinically silent cerebellar hemorrhages found in low birth weight infants.

## METHODS

Cranial sonograms were obtained, at the bedside, on neonates within the intensive care nursery at the University of California, San Francisco Medical Center, using a 5 to 7.5 MHz electronically focused sector transducer (Acuson, Mountainview, CA). All neonatal sonograms were interpreted prospectively by one of two attending radiologists specialized in ultrasonography. Before October 1994, standard intracranial images were obtained via the anterior fontanelle using coronal, sagittal, and parasagittal planes. Beginning in October 1994, this standard intracranial sonographic imaging was supplemented with transaxial views via the posterolateral fontanelle. Screening ultrasound examinations were performed on all low birth weight infants by the third day of life, with a follow-up head ultrasound examination performed by the 10th day of life. The study period for this report ranged from October 1994 to September 1996. Infants with posterior fossa hemorrhage were identified and followed prospectively. In addition, infants with posterior fossa hemorrhage born between 1991 and September 1994 were identified retrospectively from a comprehensive radiology database. Magnetic resonance imaging (MRI) studies were obtained using a GE Signa 1.5-Tesla magnet (GE Medical Systems, Mount Prospect, IL), providing sagittal, axial T1-, and spin echo T2-weighted sequences. Perinatal clinical data were obtained from retrospective chart review. All preterm infants weighing <1500 g who survived to discharge were entered into the University of California, San Francisco, high-risk neurodevelopment follow-up clinic. Standard evaluation is performed by a neonatologist trained in developmental pediatrics, and motor function is assessed at each visit. Formal neurodevelopmental testing includes the Bayley Scales of Infant Development, the Stanford–Binet Intelligence Scales, the McCarthy Scales of Children's Abilities, and the Wechsler Intelligence Scale for Children, performed at age-appropriate intervals.

## RESULTS

From October 1994 to September 1996, ~525 neonates underwent cranial sonography using the posterolateral fontanellar imaging, including ~250 infants weighing <1500 g. Thirteen of the 525 infants were identified prospectively with posterior fossa hemorrhage. Six of these 13 infants, all weighing >1500 g birth weight, had documented posterior fossa bleeding associated with risk factors reported previously, including the presence of severe birth trauma and asphyxia, ECMO, disseminated intravascular coagulation, and an arteriovenous malformation. Seven of the 13 infants with posterior fossa hemorrhages had birth weights <1500 g. Two of these infants had posterior fossa hemorrhages associated with severe, supratentorial intraventricular hemorrhage, as has been reported previously. The remaining 5 infants, however, were identified with cerebellar hemorrhage without extensive supratentorial hemorrhage.

In contrast, during the period between January 1991 and October 1994, only two posterior fossa hemorrhages were identified among all neonatal cranial sonograms obtained using standard anterior fontanellar imaging. One term infant had diffuse intracranial bleeding associated with sepsis, disseminated intravascular coagulation, and ECMO. One extremely low birth weight infant had isolated cerebellar hemorrhage that again was not associated with extensive supratentorial hemorrhage. The remainder of this report focuses on the 6 very low birth weight infants identified between 1991 and 1996 with cerebellar hemorrhagic lesions without extensive supratentorial hemorrhage.

As outlined in Table 1, birth weight ranged between 570 and 1410 g, and gestational age at birth between 25 and 30 weeks. Five of the 6 infants were born by cesarean section, and no infant experienced breech extraction, instrumentation, or birth trauma. Half of the infant group required chest compressions at birth, whereas 4 of the 6 infants required epinephrine and/or alkali therapy in the delivery room. Two infants had initial arterial blood pH <7.20, and 1 infant had an arterial blood gas base deficit >−10 at birth. Five of the infants had documented hypotension requiring single pressor infusions. Three infants were treated with high-frequency ventilation; however, the onset of high-frequency ventilation was after the identification of the posterior fossa hemorrhage in each case. Three infants had mild thrombocytopenia during the first week of life, ranging between 68 and 100 $\times 10^3/\mu$L.

All posterior fossa hemorrhages were clinically silent in presentation and discovered on routine cranial sonography. All 6 infants had intrahemispheric cerebellar lesions that were identified between day 1 and day 22 of life, with a median time to identification of 7 days. Five of the 6 cerebellar lesions identified via posterolateral fontanellar imaging were not well-appreciated by the traditional anterior fontanellar view. Five of the 6 infants had initial normal cranial sonograms, with cerebellar abnormalities identified prospectively on subsequent, follow-up studies. The abnormalities occurred in the right cerebellar hemisphere in 4 infants, the left cerebellar hemisphere in 1 infant, and in both cerebellar hemispheres in the remaining 1 infant. The hemorrhages were lentiform or crescentic and occurred in the pe-

**TABLE 1.** Profile of Cerebellar Hemorrhages of Infant Subjects

| Patient Profile | | | | | | |
|---|---|---|---|---|---|---|
| Patient | Gestational Age (wk) | Birth Weight (g) | Cerebellar Lesion | Day of Life Identified | Supratentorial Bleeding | Hydrocephalus |
| 1 | 26 | 770 | Right peripheral hemispheric | 8 | None | Y |
| 2 | 26 5/7 | 630 | Right peripheral hemispheric | 3 | None | N |
| 3 | 25 6/7 | 900 | Bilateral peripheral hemispheric | 6 | Left SEH | N |
| 4 | 30 | 1400 | Right peripheral hemispheric | 1 | None | N |
| 5 | 26 | 695 | Right peripheral hemispheric | 22 | None | N |
| 6 | 25 | 570 | Left peripheral hemispheric | 8 | Left SEH | Y |

ripheral (dorsal) aspect of the cerebellar hemisphere(s). The lesions persisted on ultrasound for 4 to 8 weeks. Four of the infants had an isolated cerebellar hemorrhage with no associated supratentorial hemorrhage, whereas the remaining 2 infants also had grade I subependymal hemorrhages (SEH). Two of the 6 infants developed transient hydrocephalus that resolved spontaneously. All of these infants survived to discharge. An MRI study was obtained in 5 of the 6 infants before discharge. The lesions seen by ultrasonography were confirmed by MRI in all cases. Examples of cerebellar hemorrhage are shown in Figures 1–3.

Corrected age at follow-up ranges between 13 and 48 months, with a median corrected age of 20.1 ± 8.73 months. Although neurologic abnormalities of cerebellar function may not present until an older age, none of the infants at the time of this report are demonstrating motor abnormalities on neurologic examination. Four infants, however, are showing signs of cognitive developmental delay, as presented in Table 2.

## DISCUSSION

The true incidence of cerebellar hemorrhage is unknown. Previous reports have suggested that posterior fossa hemorrhage usually occurs after significant trauma (breech or traumatic delivery and/or instrumentation)[1–6] or in association with catastrophic supratentorial hemorrhage.[2,10–14] Autopsy studies have documented a higher than expected incidence of cerebellar hemorrhages, reported in 10% to 25% of the very low birth weight infants, many of which were unappreciated during clinical care.[2,10–12,15] The true incidence of cerebellar hemorrhage cannot be determined from those studies, however, because of selection bias. In the current study, using routine, focused sonographic imaging of the posterior fossa, we found clinically unsuspected cerebellar hemorrhages in 6 of 250 very low birth weight infants with no or insignificant supratentorial hemorrhages. Unlike the previous reports, there was no evidence of birth trauma, breech extraction, or instrumentation with either forceps or vacuum. Each cerebellar hemorrhage had a silent presentation, and all of these infants survived to discharge. Thus, using posterolateral fontanellar imaging, we were able to identify cerebellar hemorrhages that may have eluded detection previously in surviving very low birth weight infants.

We have found that routine imaging via the posterolateral fontanelle has increased the sensitivity in identification of posterior fossa hemorrhage. Thirteen infants were identified with posterior fossa hemorrhage over a 2-year period using the posterolateral fontanellar approach. In comparison, only two cases of posterior fossa hemorrhage were identified over the 3 years preceding the change in practice, during which only anterior fontanellar views were used.

Two of 6 study infants are normal on early neurodevelopmental follow-up, whereas the remaining 4 children have shown some degree of cognitive, developmental delay. No infant with developmental delay had evidence of significant, supratentorial hemorrhage or periventricular leukomalacia, although 1 of these infants did have transient mild ventriculomegaly. No infant has exhibited neurologic deficiencies traditionally attributed to cerebellar dysfunction. The long-term outcome for these infants with intracerebellar hemorrhage is uncertain. Some persistent neurologic deficits may become apparent only as these children reach preschool years. The contribution, if any, of each cerebellar hemorrhage to cognitive developmental delay is unknown. Each infant experienced early neonatal instability complicated by acidosis, hypotension, and need for intensive resuscitation, which also may have affected later developmental outcome.

The exact pathophysiology of the intracerebellar hemorrhages seen in these infants is unclear. Posterior fossa hemorrhages associated with traumatic delivery are believed to be a result of severe distortion and disruption of the venous structures within the compliant neonatal skull, leading to laceration of the tentorium or falx or a traumatic cerebellar laceration along the vermis.[3,14,22] Cerebellar hemorrhage may relate to increases in venous pressure, as seen in infants on ECMO[7,8] or with face mask ventilation.[12,15] Cerebellar hemorrhage also may relate to impaired cerebral flow autoregulation[7,22] or coagulopathy.[7,9]

Donat and colleagues[11] postulated that cerebellar hemorrhage in preterm infants resulted from dissection of blood from either the fourth ventricle or the

**TABLE 2.** Developmental Outcome of Infant Subjects

| | Patient Developmental Follow-up | | | |
|---|---|---|---|---|
| Patient | Corrected Age at Follow-up (mo) | Outcome | Test Results | |
| 1 | 37 | Moderate delay | Battelle Development Inventory | Cognitive = 65* |
| 2 | 14 | Normal | Bayley Motor Development Index = 93 | Bayley Psychomotor Development Index = 88 |
| 3 | 19 | Moderate delay | Bayley Motor Development Index = 68 | Bayley Psychomotor Development Index = 56 |
| 4 | 22 | Mild delay | Bayley Motor Development Index = 74 | Bayley Psychomotor Development Index = score deferred |
| 5 | 18 | Moderate delay | Bayley Motor Development Index = 77 | Bayley Psychomotor Development Index = 91 |
| 6 | 13 | Normal | Bayley Motor Development Index = 99 | Bayley Psychomotor Development Index = 74 |

* School reevaluation at 48 months: "Continues with clinically significant delays"; receives full special educational services.
MDI and PDI scores have a mean score of 100 ± 15.

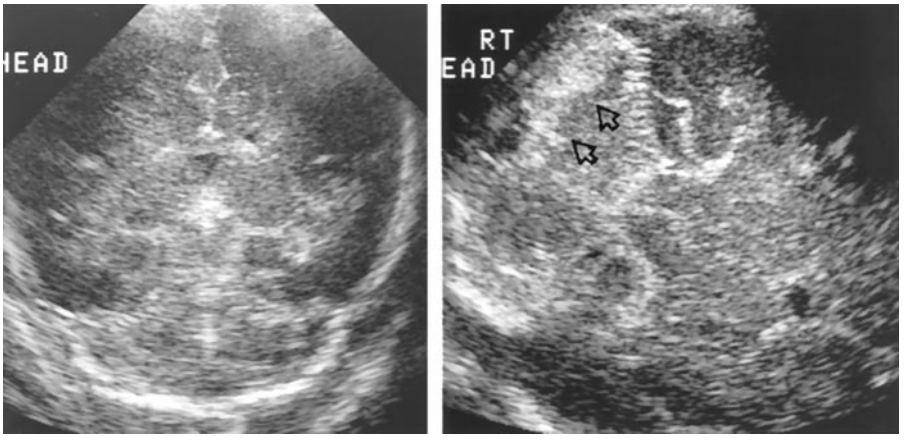


**Fig 1.** Left panel: Coronal ultrasonographic image through the anterior fontanelle, illustrating apparent normal posterior fossa. Right panel: Transaxial view of the same posterior fossa through the posterolateral fontanelle, illustrating intrahemispheric cerebellar hemorrhage, indicated by arrows.

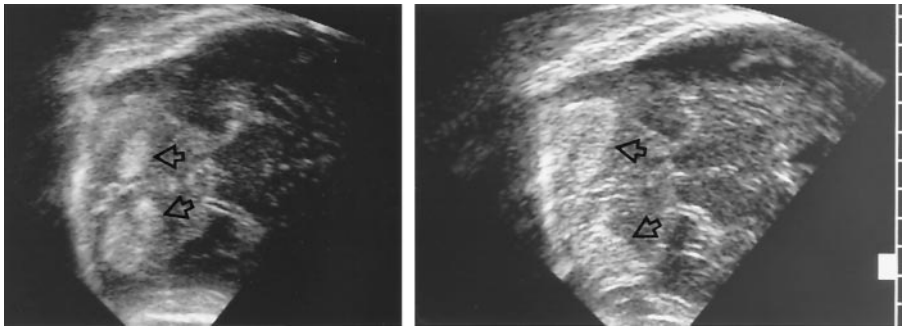

**Fig 2.** Transaxial ultrasonographic images (two planes) through the posterolateral fontanelle, illustrating bilateral cerebellar hemispheric hemorrhages, indicated by arrows.

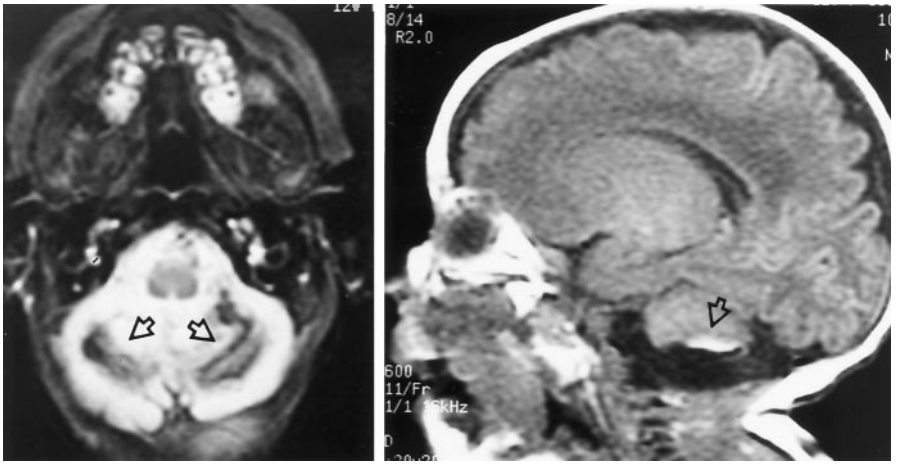


**Fig 3.** MRI images of the infant in Fig 2. Left panel: T2-weighted image illustrating bilateral cerebellar lesions, indicated by arrows. Right panel: T1-weighted image illustrating an inferior cerebellar lesion, indicated by arrow.

subarachnoid space, secondary to a concomitant severe intraventricular hemorrhage. Yet none of the 6 infants described in this report had intraventricular blood evident on ultrasonographic examination. Cerebellar hemorrhages may occur within germinal matrices located in the subependymal layer of the roof of the fourth ventricle and in the subpial external granule cell layer, the latter of which is thickest at 24 weeks' gestation and begins to involute by 30 weeks' gestation.[22–24] Subpial germinal matrix bleeding may be the source of intrahemispheric cerebellar hemorrhages identified along the outer periphery of the cerebellum, described in this report.

In summary, posterior fossa imaging is enhanced using posterolateral fontanellar views and may reveal cerebellar hemorrhages that are not evident on the traditional anterior fontanellar view. Previous reports in the literature have indicated that cerebellar hemorrhage in the preterm infant is usually associated with a devastating supratentorial bleed, with an associated mortality of nearly 100%. A new pattern of cerebellar hemorrhage has emerged in preterm infants. Cerebellar hemorrhage in low birth weight infants may be clinically silent and not associated with a significant amount of supratentorial bleeding. These infants may survive to discharge and have initial, normal neurologic examination results. Longer neurodevelopmental follow-up is necessary, however, to determine the true impact of these cerebellar lesions. Future prospective studies using posterolateral fontanellar imaging may lead to a better understanding of the pathophysiology of cerebellar hemorrhages and their long-term neurodevelopmental significance.

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