Posthemorrhagic hydrocephalus

Low incidence in very low birth weight neonates with intraventricular hemorrhage

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In addition to seizures and long-term neurodevelopmental handicaps, infants with intraventricular hemorrhage (IVH) are at risk for posthemorrhagic hydrocephalus (PHH), and the incidence of this problem in preterm infants with known IVH has been reported to vary from 25% to 74%. Over a 46-month period, 438 neonates of 1250-gm birth weight or less were admitted to this Newborn Special Care Unit, and 269 survived the first 36 postnatal hours. Of these, 265 patients underwent computerized tomography and/or cranial ultrasound scanning for evaluation of germinal matrix and/or intraventricular hemorrhage (GMH/IVH): 133 infants were found to have experienced GMH/IVH, and 27 of these died within the 1st postnatal week. Of the 95 survivors with GMH/IVH, 43 were known to have GMH only; the other 52 experienced IVH and were therefore at risk for PHH. Patients with GMH/IVH underwent repeat investigations for the development of ventriculomegaly and possible PHH. Only five patients with IVH developed PHH, defined as ventriculomegaly, elevated intracranial pressure, and increasing occipitofrontal head circumference. Serial cranial ultrasound studies of 95 other consecutively admitted patients in this birth-weight range revealed an equal incidence (45%) of low intracranial pressure ventriculomegaly in both the hemorrhage and non-hemorrhage groups, but none of them required shunting for hydrocephalus. One infant with congenital aqueductal stenosis was also identified.

KEY WORDS - preterm infant • hydrocephalus • intraventricular hemorrhage • germinal matrix hemorrhage • neonate

Intraventricular hemorrhage (IVH), or hemorrhage into the germinal matrix tissues (GMH) of the developing brain, is a major problem of preterm neonates; over 40% of infants with birth weights less than 1500 gm have been found to experience either or both disorders (GMH/IVH). In addition to seizures and long-term neurodevelopmental handicaps, infants with IVH are at risk for posthemorrhagic hydrocephalus (PHH). The hydrocephalus is generally believed secondary to obliterator posterior fossa arachnoiditis which is evoked by the presence of red blood cells and cellular debris found following IVH. Occasionally, acute PHH is attributed to a noncommunicating hydrocephalus at the level of the aqueduct; this has also been attributed to the accumulation of red cells and cellular debris along that area.

The incidence of PHH is reported to vary from 25% to 74% of low birth-weight infants with IVH, and the problem has been noted to occur with greater frequency in those infants with more severe degrees of IVH. These patients classically present with a rapidly increasing occipitofrontal head circumference (OFC), apnea, lethargy, and vomiting, and the diagnosis is made on the triad of dilated ventricular system and increased intracranial pressure (ICP) in addition to the altered clinical state.

During the past 46 months, 438 infants of 1250-gm birth weight or less have been admitted to our Newborn Special Care Unit. On the basis of computerized tomography (CT), echoencephalography, and postmortem data, all infants who survived the first 36 postnatal hours have been categorized as having GMH/IVH or no hemorrhage. Of these patients, 232 survived the first 7 postnatal days, and those who experienced GMH/
IVH were thus at risk for PHH. We reported a very low incidence of PHH in very low birth weight infants with IVH.

**Clinical Material and Methods**

**Patient Population**

Between June 22, 1979, and May 1, 1983, 438 infants of 1250-gm birth weight or less were admitted within the first 36 postnatal hours to our Newborn Special Care Unit. Of the 269 who survived the first 36 postnatal hours, 265 were evaluated for GMH/IVH. The mean birth weight of this population was 990 gm (range 630 to 1250 gm), and the mean gestational age according to the assessment of Ballard, et al., was 29.1 weeks (range 25 to 35 weeks).

**Study Method**

All neonates of 1250-gm birth weight or less who were admitted to our unit and survived the first 36 postnatal hours were examined using the standard neonatal neurological examinations of Amiel-Tison and Dargassies. In addition, all these patients underwent extensive review of the pre- and perinatal course, weekly chart reviews, and biweekly neurological evaluations during their stay in our unit.

During the first 12 months of the study period, CT scans only were performed in the first 3 postnatal weeks (mean age at time of first CT scan was 10 days), and these were interpreted by a neuroradiologist unaware of the neonate's clinical status. During the last 34 months, all of the infants in the study population were evaluated by serial echoencephalography examinations. These were routinely performed on the 3rd postnatal day and then again during the 2nd postnatal week. During the last 10 months of this study period, all infants underwent repeated serial ultrasound scanning. Cranial ultrasound studies were performed at the bedside utilizing a portable real-time sector ultrasound scanner. Studies were performed through the anterior fontanel and temporal bone in coronal, sagittal, and axial projections using a 5-mHz transducer. All ultrasound studies were reviewed by observers unaware of the infant's clinical course.

The grading system for the hemorrhages detected by CT scan was that originally described by Papile, et al., hemorrhages diagnosed by ultrasound were graded in a similar fashion, so that Grade 1 represented GMH, Grade 2 was assigned to hemorrhages with blood within the lateral ventricular system but not distending it, and Grade 3 was assigned to hemorrhages filling and distending the ventricular system. Infants with parenchymal hemorrhages were defined as experiencing Grade 4 IVH.

All infants suspected clinically of developing posthemorrhagic hydrocephalus (that is, a rapidly increasing OFC, apnea, altered level of consciousness, vomiting, or lower-extremity hypertonia and hyperreflexia) during the first 6 months of our study underwent cranial echoencephalography and/or repeat CT scanning. During the last 40 months, all infants found to have experienced any grade of IVH underwent serial repeat echoencephalography during the 2nd, 3rd, and 4th postnatal weeks, or more frequently if clinically indicated.

Infants with echoencephalographic evidence of stable GMH/IVH (that is, those with no change on ultrasound scanning for 3 or more consecutive days), in whom progressive ventricular enlargement was noted on two consecutive studies, underwent lumbar puncture for measurement of opening pressure as well as routine cerebrospinal fluid (CSF) analysis, including cell counts, and sugar and protein levels. In patients with evidence of hemorrhage, opening pressures of greater than 100 mm H2O were considered to be elevated and consistent with PHH. Infants without documented GMH/IVH who were similarly found to have ventriculomegaly on two successive echoencephalographic examinations also underwent lumbar puncture. During the phases of the study when CT scanning was not routine, those infants with blood in the CSF and no echoencephalographic evidence of GMH/IVH underwent CT scanning for investigation of possible subarachnoid hemorrhage, as this could not be determined by ultrasound.

During the last 10 months of the study period, all patients underwent serial ultrasound scanning within the first 6 postnatal hours and every 12 hours thereafter for the first 3 postnatal days. Subsequent studies were performed on the 4th, 5th, 7th, 14th, and 20th postnatal days, or more frequently as clinically indicated. This protocol was approved by the Yale University Human Investigation Committee, and written informed parental consent was obtained for all studies.

**Summary of Cases**

Of the 269 infants who survived the first 36 postnatal hours, 133 were found to have GMH/IVH. Twenty-seven of these 133 neonates died within the 1st postnatal week; none of these infants demonstrated increasing ventricular size on cranial ultrasound scanning or clinical symptoms of PHH on repeated neurological examinations. Ten patients without GMH/IVH died during the same time. Of the remaining 228 infants, 95 (42%) were found by cranial ultrasound or CT scanning to have experienced GMH/IVH. Forty-three were diagnosed as having GMH (Grade 1); 34 as having Grade 2 IVH; 12 as having Grade 3 IVH; and six as having parenchymal hemorrhages, and thus Grade 4 IVH. It is of note that the incidence of IVH has not diminished throughout the years of the study protocol, nor has the relative proportion of severe to less severe grades of GMH/IVH changed.

By definition, therefore, 52 infants had experienced Grade 2, 3, or 4 IVH and survived the 1st postnatal
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week; these patients were therefore at risk for PHH. Five infants (two with Grade 2 IVH, two with Grade 3 IVH, and one with a parenchymal hemorrhage) were found to develop increasing ventricular size and clinical signs of increased ICP. Lumbar puncture revealed elevated ICP in all five patients, and they were thus defined as suffering PHH. In one infant with Grade 3 IVH, the obstruction occurred at the level of the aqueduct and the patient was noted to experience acute neurological decompensation. A ventriculoperitoneal (VP) shunt was placed, and now, at 15 months' corrected age, the patient has been found not to be shunt-dependent. All of the other infants with increased ICP and evidence of PHH underwent serial lumbar punctures for 3 to 21 days for management of the elevated ICP. One infant died from renal failure during this course of therapy; in two other patients (one with Grade 2 and one with Grade 3 IVH), the necessity for lumbar punctures was of brief duration (less than 1 week). In the fifth patient, with parenchymal hemorrhage, serial spinal taps were required for 3 weeks.

During the last 10 months of this study, when all patients were undergoing serial echoencephalographic studies, seven of the 13 infants with GMH/IVH who survived the 1st postnatal week were found to have ventriculomegaly 3 to 14 days following stabilization of their GMH/IVH. Two of these patients were found to have elevated ICP at the time of lumbar puncture, and were thus defined as experiencing PHH. They are included among the five patients with PHH mentioned above. Five of the 11 patients without hemorrhage who survived were also found to have late ventriculomegaly. Lumbar puncture demonstrated no evidence of increased ICP or hemorrhage in any of these infants. In none of the infants with echoencephalographic evidence of ventriculomegaly with normal ICP did this finding resolve during the first 3 postnatal weeks, although almost all of the hemorrhages were noted on ultrasound scanning to be resolved by this time.

Our studies revealed one male infant with an increased OFC and CT evidence of aqueductal stenosis. This patient was never known to have experienced GMH/IVH, despite repeated CT scans and CSF examinations. Because of a rapidly increasing OFC and signs of increased ICP, he underwent VP shunting while a newborn, and, at the age of 4 years, continues to remain shunt-dependent. No other patient without GMH/IVH developed hydrocephalus.

Discussion

The recent development of sophisticated neonatal intensive care has permitted an increasing rate of survival among many very small and critically ill preterm infants.\(^7,14,36\) Unfortunately, although the mortality figures are markedly improved for this population, the incidence of overall neurodevelopmental handicaps has remained unchanged over the past 10 to 20 years, and many infants survive with significant motor and cognitive deficits.\(^11-13,19,29,32\) It has been well recognized for many years that preterm infants with IVH do not fare nearly as well as their peers without hemorrhage, when compared at the time of follow-up testing.\(^8,17,19,34\) and infants with PHH have been found to be at even greater risk for neurodevelopmental handicap than GMH/IVH patients with normal ventricular size.\(^19,26,34\)

Posthemorrhagic hydrocephalus hasclassically been described as secondary to a fibrous thickening of the meninges, particularly in the posterior fossa, with obstruction of CSF flow through the normal subarachnoid pathways. This chemical arachnoiditis has been ascribed to the presence of blood and cellular debris in the ventricular CSF. Less commonly, the shedding of cellular debris and red cells has been found to obstruct the aqueduct, causing an acute noncommunicating hydrocephalus.\(^20,23,33\)

Intraventricular hemorrhage classically occurs in asphyxiated preterm infants with respiratory distress syndrome in whom neurological deterioration is noted, although, in many patients, GMH/IVH may remain "silent."\(^38,39\) Similarly, although infants with PHH commonly present with a rapidly increasing OFC and/or the clinical symptoms of lethargy, vomiting, apnea, and hypertonia and hyperreflexia,\(^28\) Volpe, et al.,\(^39\) reported that ventricular dilatation may in fact precede rapid head growth and clinical abnormalities.

The incidence of PHH in preterm infants with IVH who have survived the 1st postnatal week has been reported to vary from 25% to 74%. We have noted a low incidence of PHH in our very low birth-weight population, and report only one patient who required a shunt for PHH. Other authors\(^1,15,19,21,27\) have not presented their data in terms of gestational age or birth weight; however, we have noticed a much higher incidence of PHH in infants of 1251-1500-gm birth weight with IVH, in whom the incidence of IVH is reported to be less than we found in our lower birth-weight infants.\(^4,9,16,25,27,31,35\) Several authors have noted that the occurrence of PHH is directly related to the degree of IVH\(^1,18,27\) and, in the series of Allan, et al.,\(^1\) and Hill and Volpe,\(^15\) over half of the patients with blood filling more than 50% of the ventricular system developed PHH. Although our incidence of PHH is quite low, our data do not support this finding.

Papile, et al.,\(^28\) reported that the presumed chemical arachnoiditis causing PHH may resolve, and that the increased ICP responsible for symptoms in these patients may be controlled by frequent lumbar punctures. Allan, et al.,\(^1\) noted that, in some infants, PHH may indeed represent a very transient phenomenon; in two of our patients with PHH, lumbar punctures were required for less than 1 week for control of increased ICP. Only one infant with Grade 4 IVH required prolonged daily spinal taps.

Several investigators have studied infants with IVH who are at risk for PHH, as we have done during the last 10 months of our study, and the incidence of ventriculomegaly in this population appears to be ap-

J. Neurosurg. / Volume 60 / February, 1984
proximately 50%,1,15,21 Allan, et al.,1 considered 46% of their patients with ventriculomegaly to have PHH, compared to 78% of the infants reported by Hill and Volpe,12 although both of these studies included infants of 34 weeks gestational age or less. During the last 10 months of our study, six of 13 surviving IVH patients and five of 11 surviving infants without hemorrhage were found to develop ventriculomegaly by serial echoencephalographic assessment. None of the infants without an IVH gave lumbar puncture evidence of increased ICP, compared with two of the IVH infants. Although Palmer, et al.,26 reported a higher incidence of neurodevelopmental handicaps in preterm neonates with IVH and low-ICP ventriculomegaly compared to those with IVH and normal ventricular size, Fitzhardinge10 demonstrated a wide prevalence of ventriculomegaly in preterm infants with IVH, and found no correlation between this finding and neurodevelopmental outcome. Our follow-up studies on this particular group of infants are still at a sufficiently early stage that we are unable to comment on this problem.

Finally, the incidence of congenital hydrocephalus has been reported to be two per 1000 live births.24 We were interested to find one case of aqueductal stenosis in our 438 very low birth-weight infants.

In summary, over the past 48 months we have surveyed a large group of very low birth-weight infants for the presence of GMH/IVH, and followed the survivors with echoencephalography for the development of ventriculomegaly and possible PHH. We have found that the incidence of this problem is quite low in this population, which is known to have a high incidence of IVH. This is a reassuring finding for those who must care for very small and critically ill preterm infants.

References
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Manuscript received May 23, 1983.

This work was supported in part by grants from the March of Dimes Birth Defects Foundation, White Plains, New York, and the Walter Scott Foundation, Newtown, Connecticut.

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