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1985

The Year Book of PEDIATRICS®

Editors

Frank A. Oski, M.D.

Professor and Chairman, Department of Pediatrics, State University of New York Upstate Medical Center

James A. Stockman, III, M.D.

Professor and Chairman, Department of Pediatrics, Northwestern University School of Medicine; Physician in Chief, The Children's Memorial Hospital, Chicago

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American Journal of Gastroenterology
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Publisher's Preface

Publication of the 1985 YEAR BOOKS marks the eighty-fifth anniversary of the original PRACTICAL MEDICINE YEAR BOOKS. To mark this milestone, the YEAR BOOKS are being issued with a more contemporary cover design, and the format for the contents has been modified to identify the article titles, authors' names, and journal citations more readily. The substance of the YEAR BOOK—the abstracts of scholarly articles with substantive editorial comments—is unchanged. What is new is the isolation of the reference information as a discrete block of copy. Other, less-visible changes will continue to be made as we strive to make the YEAR BOOKS the very best they can be.

The YEAR BOOK OF PEDIATRICS is a proud member of the original series of PRACTICAL MEDICINE YEAR BOOKS. From 1901 through 1923, pediatrics was combined with orthopedic surgery in one YEAR BOOK, but the YEAR BOOK OF PEDIATRICS has been a separate book since 1924. We are proud to hail the longevity of this outstanding member of the YEAR BOOK series.

1 The Newborn

Relationship of Cerebral Intraventricular Hemorrhage and Early Childhood Neurologic Handicaps

Lu-Ann Papile, Ginny Musick-Bruno, and Anne Schaefer (Univ. of New Mexico, Albuquerque)

J. Pediatr. 103:273–277, August 1983 1–1

To determine whether cerebral intraventricular hemorrhage (CVH) is associated with early developmental or neuromotor handicaps, a comparison was made of the outcome in 198 surviving infants with very low birth weight (VLBW) (less than 1,501 gm) with and without CVH, as determined by computed tomography scans.

Developmental assessment was normal in 61 of the infants without CVH, suspect in 43, and abnormal in 11. Neuromotor and developmental testing of these 116 infants showed that 57 were normal, 46 had a minor handicap, and 12 had a major handicap. Seven of the 12 were multihandicapped (Table 1).

Among 33 of 39 infants with a grade 1 CVH who were alive at age 1 year, 16 were not handicapped, 14 had a minor handicap, and 3 had a major handicap. Two of the last 3 were multihandicapped. Nine of 18 infants with a grade 2 CVH had no handicap, 7 had a minor handicap, and 2 had a major handicap. No child in this group was multihandicapped. Evaluation of 14 infants with a grade 3 CVH showed that 2 were normal, 7 had a minor handicap, 5 had a major handicap, and 4 were multihandicapped. Of the 17 infants with a grade 4 CVH, 2 had no handicap, 2 had a minor handicap, 13 had a major handicap, and 10 were multihandicapped.

TABLE 1.—Neurodevelopmental Outcome of Infants With VLBW Related to CVH Grade

	None	*Grade 1*	*Grade 2*	*Grade 3*	*Grade 4*
Infants (n)	147	48	23	20	22
Alive at 1 year	138	39	21	16	18
Evaluated	116*	33	18	14	17
Birth weight (kg)	1.18	1.07	1.12	1.14	1.06
Gestational age (wk)	30.2	29.2	29.4	28.9	29.1
Outcome					
Normal	57	16	9	2	2
Minor handicap	46	14	7	7	2
Major handicap	12	3	2	5	13
Multiple handicaps	7	2	0	4	10

*One infant untestable.

(Courtesy of Papile, L.-A., et al.: J. Pediatr. 103:273–277, August 1983.)

TABLE 2.—Neurodevelopmental Outcome of Infants With Grades 3 and 4 CVH Related to Development of Posthemorrhagic Hydrocephalus

Clinical status	*Number of patients*	*Grade 3 CVH*		*Grade 4 CVH*	
		Hydrocephalus	*No hydrocephalus*	*Hydrocephalus*	*No hydrocephalus*
Alive at 1 year	34	8	8	10	8
Evaluated	31	8	6	9	8
Normal	4	1	1	0	2
Minor handicap	9	4	3	2	0
Major handicap	18	3	2	7	6
Multiple handicaps	14	3	1	7	3

(Courtesy of Papile, L.-A., et al.: J. Pediatr. 103:273–277, August 1983.)

Posthemorrhagic hydrocephalus developed in 22 infants with CVH (Table 2). All had a grade 3 or grade 4 CVH. Ten of these 22 infants had a major handicap and 10 were multihandicapped. The incidence of major handicaps in these infants was similar to that in comparable infants without posthemorrhagic hydrocephalus.

Infants with VLBW who have grades 1 and 2 CVH do not have a higher incidence of major handicaps in early childhood than comparable infants without CVH. However, there is a direct relationship between grades 3 and 4 CVH and major handicaps. Posthemorrhagic hydrocephalus does not increase the risk for major handicaps, but does influence the incidence of multihandicaps.

▶ It has become traditional to solicit a comment from Dr. Joseph Volpe on the subject of cerebral hemorrhage in the low birth weight infant. Doctor Volpe, Professor of Developmental Neurology, Pediatrics, Neurology, and Biological Chemistry, Washington University School of Medicine, always provides an incisive analysis. I couldn't think of a better way to start this book than with the following remarks from Doctor Volpe:

"This report by Papile et al. is addressed to the neurologic outcome of premature infants with intraventricular hemorrhage (IVH). The authors conclude that infants with the smallest hemorrhages do not exhibit an increased incidence of subsequent neurologic deficits, whereas those with the largest hemorrhages do. However, a simple relationship between the quantity of intraventricular blood and neurologic outcome is not apparent. This large series confirms data obtained from previously reported smaller series. The lack of a linear relationship between the severity of intraventricular bleeding and the neurologic outcome has provoked debate and confusion in the medical literature, but we believe that there is no need for either. The critical determinant of neurologic outcome with IVH is the severity of the associated *cerebral* abnormality, and the relationship between this critical parenchymal involvement and the amount of intraventricular blood is by no means clear. Our task in predicting and understanding outcome in these infants is assessment of the severity of the *cerebral* involvement. In the following discussion, let us consider the major determinants of the cerebral injury in infants with IVH, the

current means of assessing this injury, and our understanding of the cause(s) of the parenchymal injury.

"First, regarding the major determinants of the cerebral injury in infants with IVH, we can identify six major factors: (1) preceding or concurrent hypoxic-ischemic insult(s), (2) increased intracranial pressure with decreased cerebral perfusion, (3) destruction of periventricular white matter by intraparenchymal blood, (4) destruction of glial precursors in the germinal matrix, (5) focal cerebral ischemia secondary to vasospasm, and (6) posthemorrhagic hydrocephalus. Concerning these factors, preceding or concurrent hypoxic-ischemic insults are not uncommon in infants with major IVH. Such insults are especially likely to cause periventricular leukomalacia, i.e., injury to periventricular white matter. Infants with severe IVH may exhibit increased intracranial pressure and, because cerebral perfusion is related to arterial blood pressure minus intracranial pressure and because arterial blood pressure may fall in infants with major IVH, impairment of cerebral perfusion may occur and cause ischemic injury to brain. Destruction of periventricular white matter by intraparenchymal blood previously has been considered the cause of the brain injury observed in infants with so-called grade IV IVH, i.e., IVH with intraparenchymal "extension." We now believe that simple intraparenchymal extension of blood from the lateral ventricle or germinal matrix into previously normal white matter is uncommon and that prior or concurrent injury to white matter is necessary to cause the intraparenchymal bleeding (see below). Destruction of the germinal matrix, of course, is a uniform feature of IVH, which emanates from this structure, the source of the glial cells that subsequently will migrate to cerebral white matter and lead to myelination. Does destruction of these glial precursors impair subsequent brain development? We can't answer this question yet, but the possibility is real. Focal cerebral ischemia, secondary to vasospasm, analogous to the vasospasm observed in older patients with subarachnoid hemorrhage, is a theoretical possibility. This possibility is supported by one study of cerebral blood velocity (Bada et al.: *J. Pediatr.* 95:775, 1979). The frequency and importance of this complication remain unclear. Finally, posthemorrhagic hydrocephalus certainly can complicate severe IVH, as shown in the article by Papile et al. Although the precise relationship between posthemorrhagic hydrocephalus and neurologic outcome is unknown, rapidly progressive hydrocephalus that is not promptly treated clearly can contribute to the brain injury in these infants.

"Second, regarding the current means of assessing the critical cerebral injury in the newborn with IVH, real-time cranial ultrasonography has been useful. The largest study of infants with IVH reported to date (McMenamin et al.: *Ann. Neurol.* 15:285, 1984) described the outcome as a function of cerebral involvement, identified on cranial ultrasonography as periventricular echodense lesions. Thus, of 177 infants with IVH, 64 had periventricular echodense lesions, and of the latter, 33 had large, unilateral, globular lesions (large IPE) and 31 had small, bilateral, linear lesions (small IPE). The outcome differed markedly in these two groups. Of the 33 infants with IVH and large IPE (grade IV IVH in most classifications), 76% died in the neonatal period and *all* of the survivors had subsequent moderate or severe neurologic deficits. Of the 31 infants with small IPE, 29% died and only 14% had subsequent moderate

neurologic deficits (none had severe neurologic deficits). The periventricular echodensities were considered to represent white matter injury, perhaps hypoxic-ischemic in basic nature and perhaps in part reversible in the infants with small IPE. Clearly, this study showed that the most critical determinant of severe parenchymal involvement and poor neurologic outcome in the infant with IVH is the presence of large IPE.

"The nature of the large intraparenchymal lesion observed on cranial ultrasonography was defined recently in a study of regional cerebral blood flow by positron emission tomography (PET) in 6 infants with this critical lesion (for reference). Thus, it was possible to adapt to the newborn the remarkably powerful technique of PET to measure regional cerebral blood flow. The essential finding was an impairment of cerebral blood flow in infants with large IPE that was much more extensive than could be accounted for by the locus of the intraparenchymal blood. The marked decrease in cerebral blood flow involved all of the cerebral white matter, although the IPE on cranial ultrasonography appeared to involve only anterior cerebral white matter. The decrease in cerebral blood flow indicated that the cerebral white matter had sustained extensive injury. Neuropathologic observations corroborated this conclusion. The topography of the cerebral involvement defined in the 6 patients by PET suggested that the intracerebral injury is ischemic in basic nature, because the lesion resided especially in those periventricular regions known to be vulnerable in the premature infant to impairment of cerebral perfusion. When and how this ischemic lesion occurs remain to be defined. It is most probable that the cerebral lesion occurs prior to the IVH and that the secondary hemorrhage into the anterior portion of the cerebral lesion occurs at the time of or shortly after the IVH. (It is of interest in this regard that 4 of the 6 infants studied by PET had sustained prior perinatal asphyxia.) The essential point is that the critical intracerebral lesion in infants with IVH and large IPE, the subset of infants that accounts for the large majority of neurologic morbidity observed with IVH, in general, is an extensive infarction, the anterior portion of which is hemorrhagic. What remains for us to accomplish is definition of the cause of this infarction, so that we can proceed to the most important goal, i.e., prevention."

Intracranial Hemorrhage in the Term Newborn

Gerald M. Fenichel, David L. Webster, and Walter K. T. Wong (Vanderbilt Univ.)

Arch. Neurol. 41:30–34, January 1984 1–2

The findings in 22 term newborn infants seen in a 5-year period with intracranial hemorrhage on computed tomography (CT) examination were reviewed. Ultrasonography also was used in the later cases. The study was partly prospective. Where both intraventricular hemorrhage (IVH) and subarachnoid hemorrhage (SAH) were present, it was assumed that the IVH was primary and was followed by extravasation of blood into the subarachnoid space.

Primary SAH was the most common form of hemorrhage in this series