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Evolution of Porencephalic Cysts from Intraparenchymal Hemorrhage in Neonates:
Sonographic Evidence

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Ninety-four low-birth-weight neonates were screened consecutively with real-time sonography for evidence of cerebroventricular hemorrhage. Among them, 13 were found to have intraparenchymal hemorrhage beyond the area of the germinal matrix. Weekly follow-up sonograms revealed progression to porencephaly in every surviving infant. Four different sonographic patterns were observed during the evolution from the original hemorrhage to the porencephalic cyst. The size of the mature porencephalic cyst correlated directly with the size of the intraparenchymal hemorrhage. After ventriculoperitoneal shunting, the porencephalic cysts became smaller or remain unchanged. One small cyst was no longer identified after successful shunting, but reappeared with shunt failure.

Porencephaly was originally described by Heschl [1] in 1859. Subsequent anatomic descriptions have been variable to the point where there is no consensus on its precise definition. Drew and Grant [2] substituted the term "benign cyst of the brain," thus avoiding the controversy. It is now felt that these cavities may be in communication with the ventricles or subarachnoid space or that they may be totally isolated [3]. The most frequent classification divides the entity into two major categories: developmental and encephaloclastic [4, 5]. The latter category incorporates cerebral cavities that result from tissue breakdown of various etiologies including cerebral ischemia [6], infection [7], previous needle puncture [8], and a host of other possibilities [9]. Cantu and Lemay [10] described the evolution of cerebral hematomas to porencephalic cysts documented by pneumoencephalography in six adult patients. The development of such posthemorrhagic porencephalic cysts was also addressed by Courville [11] and Furlow et al. [12].

Using portable real-time sonography, we observed the gradual development of porencephalic cysts in premature neonates after cerebral intraparenchymal hemorrhage. In each instance, a typical constellation of sonographic findings characterized the evolution of the porencephalic cyst. These sonographic features are described here.

Materials and Methods

During a 22 month period, 436 sonographic examinations were performed on 94 low-birth-weight neonates (below 1,700 g). All examinations were performed with an Advanced Technology Laboratories real-time sector scanner using a 5 MHz transducer system with a 90° field of vision. Examinations included angled coronal and parasagittal views, according to a recently described method [13], and they were always done in the nursery. These 94 neonates represent the entire population of premature infants admitted to Georgetown University Hospital Neonatal Intensive Care Nursery during this period.

Sonographic evaluation was performed within the first 72 hr of life on all "in-born" neonates. Transferred neonates were usually seen within 24 hr after arrival. Those diagnosed as having intraparenchymal hemorrhage were followed with sonography on a weekly

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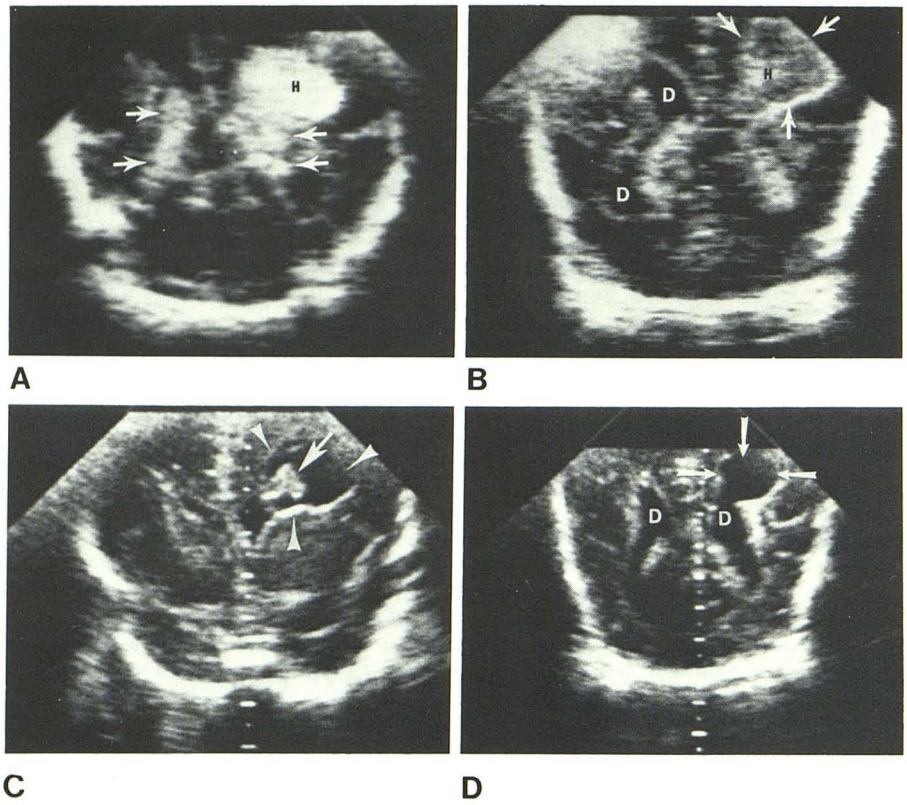

Fig. 1.—Evolution of porencephalic cyst. **A**, Coronal sonogram at 4 days of age. Intensely echogenic area in left frontoparietal region (H) represents intraparenchymal hemorrhage. Note continuity with intraventricular hemorrhage (*arrows*). **B**, "Rind" phase at 2 weeks. Echogenic border (*arrows*) now surrounds relatively hypoechoic center (H). Mild ventricular dilatation (**D**) on right. **C**, "Clot retraction" at 3 weeks. Porencephalic cyst (*arrowheads*) with small amount of echogenic clot remaining (*arrows*). **D**, 5 weeks of age. Mature porencephalic cyst (*arrows*) and moderately enlarged ventricles (D).

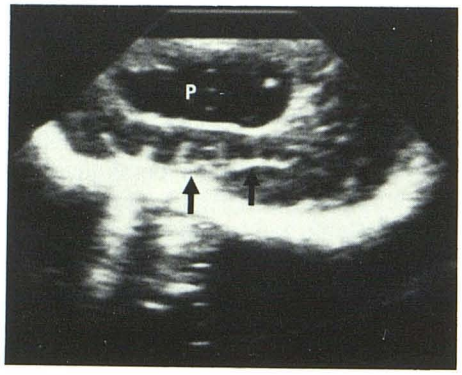

Fig. 2.—Mature porencephalic cyst (P) at 5 weeks of age. Steeply angled parasagittal sonogram. Depression of Sylvian fissure (*arrows*).

basis thereafter, usually until discharge from the Intensive Care Nursery. Although computed tomographic (CT) scans (Pfizer 0200 FS) were obtained for confirmation of the intraparenchymal hemorrhage early in the study, as experience with sonography increased, they were generally considered superfluous. Confirmation of the porencephalic cysts with CT was also not considered necessary, although CT scans were obtained in selected cases.

Results

Sonography found 13 of 94 low-birth-weight neonates to have cerebral intraparenchymal hemorrhage beyond the area of the germinal matrix. In each instance, there was associated bilateral intraventricular hemorrhage. Recent hemorrhage in the ventricles and in the brain parenchyma is highly echogenic, as is the choroid plexus. These three entities form a single continuous echogenic complex (fig. 1A).

In nine of these 13 cases, a mass effect was demonstrated, with deviation of the septum pellucidum toward the contralateral side. In addition, the Sylvian fissure on the ipsilateral side was often depressed (fig. 2). These findings of mass effect were, as expected, most pronounced in the largest of hemorrhages. In three infants, intraparenchymal hemorrhage was bilateral. In all cases of intraparenchymal hemorrhage, there appeared to be contiguity with the area of the germinal matrix, although hemorrhage occasionally extended as far as the occipital region.

Follow-up sonograms revealed a specific sonographic pattern of clot maturation that resulted in the development of a posthemorrhagic porencephalic cyst in the seven surviving infants. Over 1–3 weeks, the intraparenchymal hemorrhage changed from a homogeneously hyperechoic area to one with an echodense "rind" and a relatively hypoechoic center (fig. 1B). During this time, there was no change in the size of the clot. After the "rind" phase, a period of clot retraction was noted. The clot became smaller and was replaced by anechoic fluid (fig. 1C). This marked the beginning of actual cyst formation. Eventually, no particulate material remained in the mature cyst (fig. 1D). The time period for complete development of a porencephalic cyst was very variable, from 10 days to 8 weeks. These events

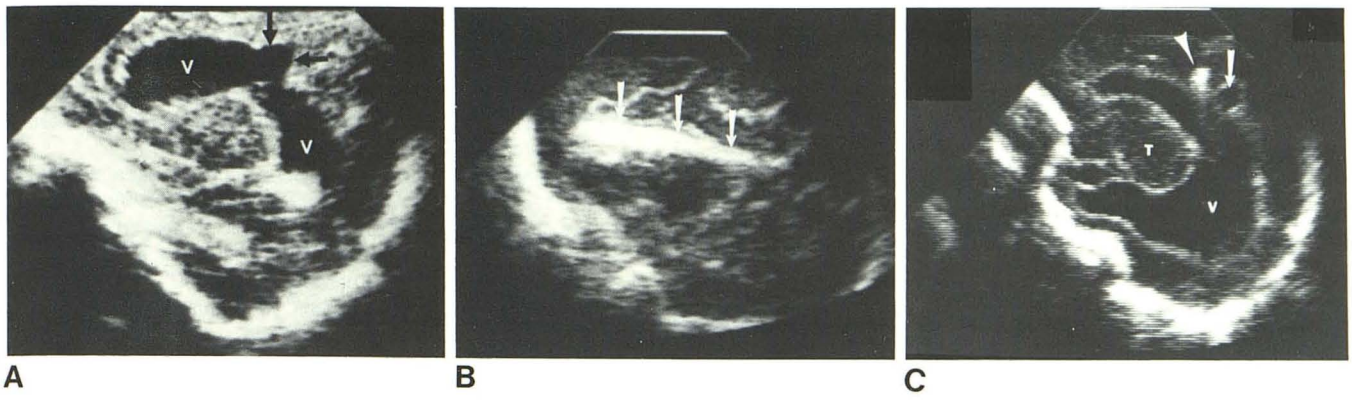

Fig. 3.—Response of cyst to shunting in 7-week-old infant, parasagittal sonograms. **A**, Small porencephalic cyst (*arrows*) and moderately dilated left ventricle (V). **B**, After ventriculoperitoneal shunt. Ventricles and porencephalic cyst no longer visible. Shunt device still present (*arrows*). **C**, Shunt failure. Markedly dilated lateral ventricle (V) and reappearance of small porencephalic cyst (*arrow*). Small piece of shunt device (*arrowhead*). T = thalamus.

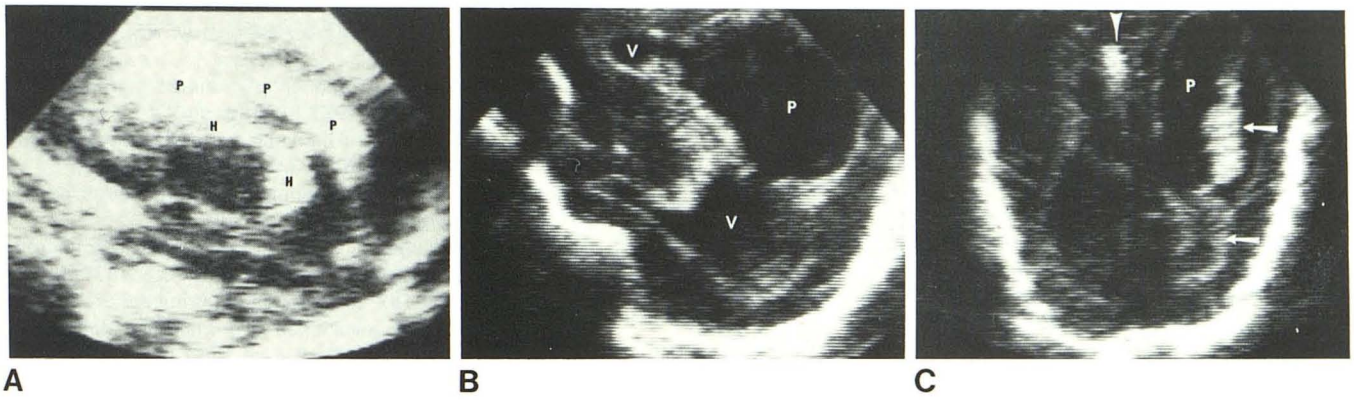

Fig. 4.—Failure of cyst response to shunting in 5-day-old infant. **A**, Parasagittal sonogram. Large intraparenchymal hemorrhage has dissected from area of germinal matrix to occipital region (P). Contiguous intraventricular hemorrhage (H). **B**, 4 weeks of age. Large porencephalic cyst (P) with moderately dilated ventricle (V). **C**, Semiaxial sonogram after ventriculoperitoneal shunt. Large porencephalic cyst (P) persists. Echogenic blood (*arrows*). Shunt tip in right ventricle (*arrowhead*).

are somewhat similar to the behavior of hemorrhage when restricted to the ventricles [9]. The size of the mature porencephalic cysts varied directly in proportion to the size of the original intraparenchymal hemorrhage.

Moderate to severe ventricular enlargement was observed in all infants who experienced intraparenchymal hemorrhage. Two of these 13 infants received ventriculoperitoneal shunts for control of severe hydrocephalus. In one child with a small porencephalic cyst, the ventricles diminished markedly and the cyst could no longer be identified sonographically, implying that the cyst communicated with the ventricles. With the onset of shunt failure and reexpansion of the ventricular system, the porencephalic cyst was again visible (fig. 3). In the other child, the shunt also diminished the ventricles, but, in this case, the large porencephalic cyst did not respond and remained unchanged (fig. 4). Follow-up sonography on a third infant without ventriculoperitoneal shunt at 1 year of age revealed minimal dilatation of the lateral ventricles, but persistence of a large right porencephalic cyst.

Discussion

Sonography has rapidly attained a prominent position in the diagnosis of neonatal intracranial pathology. Hydrocephalus [13–15] and cystic malformations [16] have been the subject of earlier research, and, more recently, cerebroventricular hemorrhage in its various forms has been investigated with sonography [13, 17–19]. Since the scans may be repeated almost as often as desired, sonography may also be used to investigate the pathophysiology of certain intracranial conditions.

Porencephaly represents but one of the several possible neuropathologic sequelae to cerebral hemorrhage. In adults, most intracerebral hematomas result in slow absorption of the extravasated blood, usually leaving an area of encephalomalacia of variable size. According to Courville [11], cerebral hematomas in adults may also lead to an enlarging posthemorrhagic cyst, and increased intracranial pressure may develop days or months after the "stroke" as a result of the space-taking cyst. He acknowledges that

more frequently the hematoma reduces itself "to a mere slit."

The development of porencephalic cysts in all of our low-birth-neonates with intraparenchymal hemorrhage beyond the area of the germinal matrix is unexpected and probably follows a different evolutionary process than that of the ordinary cerebral hematoma in adults. The cerebroventricular hemorrhages discussed here all appear to have originated from the germinal matrix, and were, therefore, close to the ventricles, if not in direct communication with them. In those cysts where there is direct ventricular communication, the cavity is continually sustained since the cyst now represents an extension of the ventricular system. Like the ventricles themselves, these cavities may respond to shunting. Other hematomas may be separated from the ventricles by an ependymal lining or by a thin tissue band. Furlow et al. [12] suggested that, in these instances, the ependyma can act as a semipermeable membrane for the passage of fluid into the original hemorrhagic cavity, which would again sustain it as a cyst.

All of our porencephalic cysts appear to have been in direct communication with the ventricles as judged by sonography. However, a small separating membrane between the juxta ventricular cyst and the ventricle itself could have been missed. The ultimate proof of isolation or communication would necessitate ventriculography, which was not performed in any of these patients.

Also, it would be desirable to study these cysts directly or during surgery to determine more precisely the configuration of the cyst wall, their exact relation to the ventricles, and their content. Since these cases are usually managed conservatively, such correlation is difficult if not impossible to obtain.

A number of studies report that increasing severity of cerebroventricular hemorrhage may lead to worsening outcome in terms of survival or morbidity [20, 21]. Although we cannot draw conclusions about the eventual significance of the porencephalic cysts at this time, these children have certainly suffered a loss of brain substance. It will be of interest to learn what inferences may be drawn from early sonographic findings when adequate long-term follow-up is available on large numbers of premature neonates. Our observations suggest that a larger than expected number of porencephalic cysts in children are the result of neonatal cerebroventricular hemorrhage.

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