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This information is current as  
of August 14, 2025.

*AJNR Am J Neuroradiol* 1986, 7 (6) 1073-1079  
<http://www.ajnr.org/content/7/6/1073>

# Focal Necrosis of the White Matter (Periventricular Leukomalacia): Sonographic, Pathologic, and Electroencephalographic Features

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Eleven preterm infants (gestational ages 27–35 weeks) with echogenic paraventricular white matter identified shortly after birth were studied with serial echoencephalograms to fully delineate the sonographic findings characterizing the pathologic stages of white-matter necrosis. Echoencephalograms were compared with autopsy findings and CT scans. Cerebral function was assessed by electroencephalograms and later by neurodevelopmental evaluations. Echogenic areas were observed in the paraventricular white matter in the acute stage. Microscopically, the echogenic white matter consisted of vascular congestion and petechial hemorrhages, but not always with foci of necrosis. Anechoic areas, which characterized the chronic stage, corresponded to cavitory lesions, and these generally appeared within 2 weeks of birth. However, six infants had anechoic lesions by day 4, suggesting that the onset of white-matter damage was antenatal. CT showed mildly decreased attenuation when paraventricular echogenic areas alone or in association with small anechoic areas were observed. Markedly decreased attenuation on CT scans corresponded to large anechoic areas. Resolution of the sonographic and CT findings did not indicate normalization of the white matter since all surviving infants were neurologically abnormal at 1 year. Electroencephalograms with central (rolandic) positive sharp waves were associated with echogenic white matter alone or with evolving anechoic areas. All patients with positive sharp waves on electroencephalograms had large anechoic areas in later studies. Early and serial echoencephalograms are necessary to evaluate white-matter necrosis in preterm infants. When echogenic white matter is identified, electroencephalography can suggest the presence of white-matter necrosis.

Cerebral white-matter necrosis, also termed periventricular leukomalacia, is associated with abnormal neurodevelopment in preterm infants. Pathologically, this lesion is divided into an acute stage, characterized by coagulative necrosis, and a chronic stage in which cystic lesions and eventual atrophy of the white matter develop [1–9]. Until recently the diagnosis of white-matter necrosis was made by autopsy examination of the brain. White-matter necrosis now may be identified clinically in the neonatal period with echoencephalography [10–17]. However, the current literature does not present a dynamic portrayal of the sonographic findings accompanying white-matter necrosis.

The purpose of this report is to describe the echoencephalographic findings associated with evolving and resolving cerebral white-matter necrosis. Serial echoencephalograms were obtained in 11 preterm infants whose paraventricular white matter was echogenic shortly after birth. The sonographic findings were compared with postmortem and CT studies to verify the interpretation of the echoencephalograms. The sonographic findings also were correlated with the infant's cerebral function as assessed by electroencephalography (EEG) and later neurodevelopmental evaluations.

## Subjects and Methods

All newborn infants with gestational ages less than 35 weeks were examined routinely

Received March 18, 1985; accepted after revision May 30, 1986.

This work was supported in part by the Burton E. Green Foundation.

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*AJNR* 7:1073–1080, November/December 1986

0195–6108/86/0706–1073

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with echoencephalograms after admission to the Infant Special Care Center at the University of California, San Diego. Eleven preterm infants with increased echogenicity in the paraventricular white matter by 3 days after birth were studied with serial echoencephalograms at least twice in the first week, and then weekly or biweekly until discharge. The echoencephalographic findings were related to post-mortem studies and CT scans. EEG and neurodevelopmental examinations also were performed to evaluate the infant's cerebral function and to relate changes to the anatomic findings. The perinatal and neonatal clinical findings associated with these 11 cases are summarized in Table 1.

Sonograms were obtained with a sector scanner (ATL, Bellevue, WA) using a 5- and 7.5-MHz transducer. The cerebral anatomy was imaged in three planes through the fontanelles and cranial sutures [18]. To better demonstrate the telencephalic white matter, modified

coronal, parasagittal, and axial sonograms were also used in this study. Modified coronal sonograms were obtained to image the cerebral white matter anterior to the frontal horns and overlying the bodies and atria of the lateral ventricles. Modified coronal sonograms were obtained by placing the transducer on the anterior fontanelle, angling it acutely anteriorly, and then sweeping posteriorly. Sonograms in the parasagittal plane showing the white matter superior and lateral to the cerebral ventricles were obtained by placing the transducer on the anterior fontanelle and angling to the right and left beyond the lateral margins of the ventricles. Axial sonograms, which provide anatomic views similar to those obtained with CT scans, were obtained by placing the transducer on the posterior fontanelle. By angling the transducer upward and downward, the cerebral white matter adjacent to the occipital and temporal horns was demonstrated.

TABLE 1: White-Matter Necrosis: Echoencephalographic (EEG) and Clinical Data

| Case No. | Perinatal Findings |    |   |       |       |    |  | Sonographic and EEG Findings |       |            |        |             | Outcome  |
|----------|--------------------|----|---|-------|-------|----|--|------------------------------|-------|------------|--------|-------------|--|
|          | BW                 | GA | Prenatal Complications                                      | Deliv | Apgar |    | Neonatal Complications   | Echogenicity                 |       | Cavitation |        | Sharp Waves | Age: Diagnosis   |
|          |                    |    |   |       | 1'    | 5' |  | Found                        | Exten | Found      | Disapp |             |  |
| 1        | 1260               | 30 | Vaginal bleeding  | Vag   | 7     | 8  | Immature lung syndrome, hypotension                                | 0.5 H                        | 7 D   | 23 D       | Unkn   | ...         | 14 M: Spastic quadripare-<br>sis, developmental de-<br>lay, microcephaly                         |
| 2        | 1360               | 29 | None  | Vag   | 6     | 7  | Immature lung syndrome   | 15 H                         | 2 D   | 15 D       | Unkn   | ...         | 18 M: Spastic quadripare-<br>sis, developmental de-<br>lay, microcephaly                         |
| 3        | 870                | 27 | Birth at home; no medical assistance                        | Vag   | Unkn  |    | None   | 12 H                         | No    | 12 H       | Unkn   | No (25 D)   | 24 M: Spastic diplegia   |
| 4        | 1720               | 33 | Rupture memb >72 hr, Streptococcus B in placenta            | Vag   | 1     | 3  | RDS, pneu-<br>monia  | 17 H                         | 3 D   | 4 D        | 4 M    | Yes (5 D)   | 16 M: Spastic quadripare-<br>sis, developmental de-<br>lay                                       |
| 5        | 1580               | 32 | Vaginal bleeding abruptio                                   | CS    | 4     | 9  | RDS  | 3 D                          | No    | 11 D       | 3 M    | Yes (23 D)  | 14 M: Spastic quadripare-<br>sis, developmental de-<br>lay                                       |
| 6        | 1940               | 32 | Vaginal bleeding abruptio, chorioamnionitis, maternal fever | Vag   | 6     | 8  | Transient tachypnea  | 19 H                         | No    | 4 D        | 4 M    | Yes (25 D)  | 23 M: Spastic quadripare-<br>sis, developmental de-<br>lay, microcephaly, sei-<br>zures          |
| 7        | 1140               | 31 | Rupture memb, vaginal bleed-<br>ing                         | CS    | 7     | 9  | Transient tachypnea  | 1 D                          | No    | 4 D        | 5 M    | Yes (5 D)   | 14 M: Spastic quadripare-<br>sis, developmental de-<br>lay                                       |
| 8        | 1200               | 30 | Vaginal bleed-<br>ing, lister-<br>iosis                     | CS    | 4     | 6  | Pneumonia, acidosis, hypoxia, hy-<br>potension                     | 12 H                         | No    | No         | ...    | Yes (1 D)   | 2 D: Died; autopsy revealed white-matter congestion, microhemorrhages, necrosis, no cysts        |
| 9        | 2700               | 35 | Congenital heart defect                                     | CS    | 5     | 9  | Respiratory failure, hy-<br>poxia, hy-<br>potension                | 1 D                          | No    | 1 D        | ...    | ...         | 5 D: Died; autopsy revealed white-matter congestion, microhemorrhages, necrosis, and cysts       |
| 10       | 1600               | 31 | Twin preg-<br>nancy, poly-<br>hydramnios                    | CS    | 0     | 0  | RDS, hy-<br>drops, aci-<br>dosis, hy-<br>poxia                     | 1 D                          | No    | 3 D        | ...    | ...         | 3 D: Died; autopsy revealed white-matter congestion, microhemorrhages, necrosis, and cysts       |
| 11       | 1300               | 32 | Toxemia   | CS    | 2     | 7  | RDS, pneu-<br>monia, aci-<br>dosis, hy-<br>poxia, hy-<br>potension | 4 H                          | 18 H  | No         | ...    | ...         | 18 H: Died; autopsy re-<br>vealed white-matter<br>congestion, microhemor-<br>rhages, no necrosis |

Note.—BW = birth weight (grams); GA = gestational age (weeks); deliv = delivery; exten = extension; vag = vaginal; H = hours; D = days; M = months; unkn = unknown; rupture memb = premature rupture of membranes; RDS = respiratory distress syndrome; CS = cesarean section.



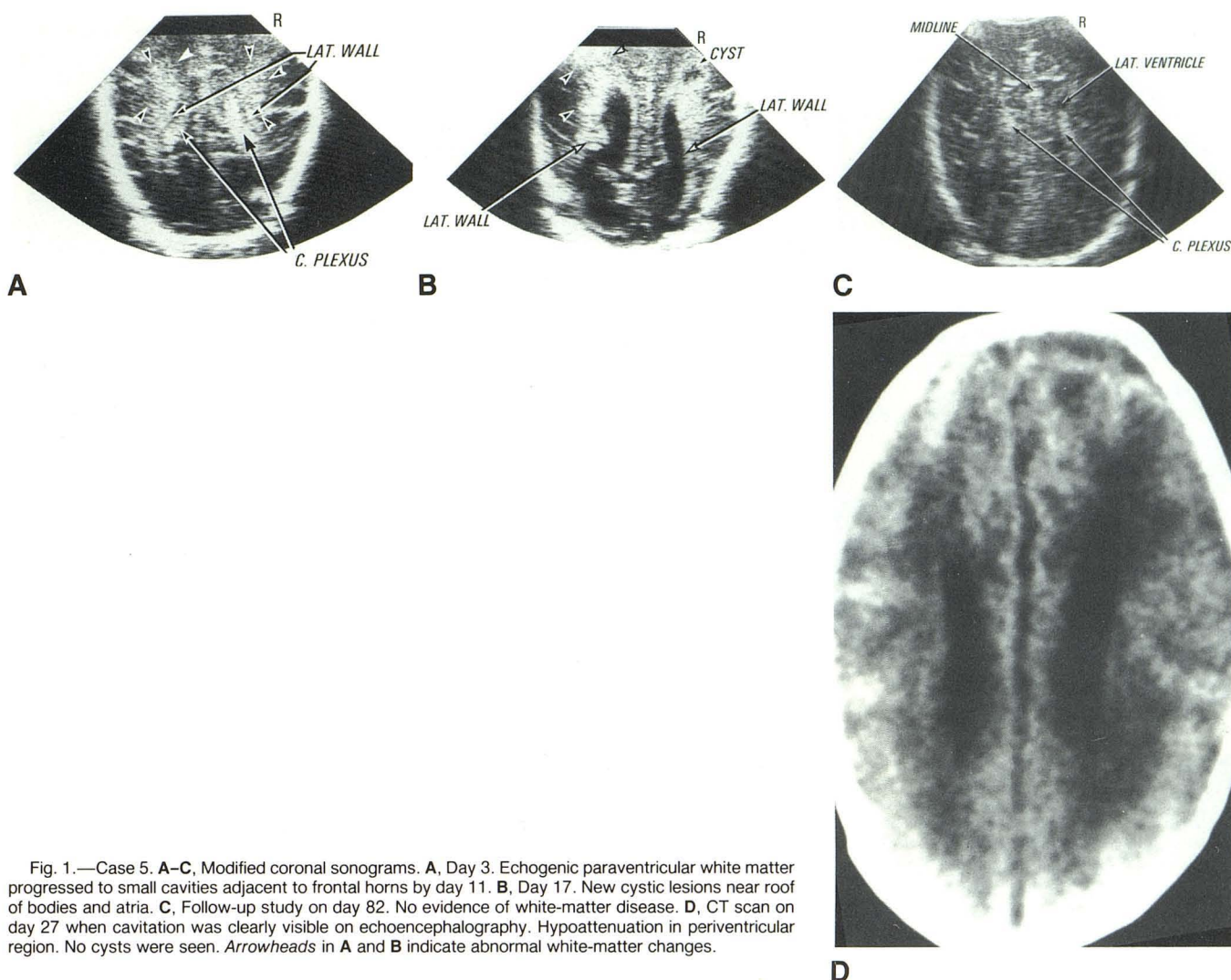


Fig. 1.—Case 5. **A–C**, Modified coronal sonograms. **A**, Day 3. Echogenic paraventricular white matter progressed to small cavities adjacent to frontal horns by day 11. **B**, Day 17. New cystic lesions near roof of bodies and atria. **C**, Follow-up study on day 82. No evidence of white-matter disease. **D**, CT scan on day 27 when cavitation was clearly visible on echoencephalography. Hypoattenuation in periventricular region. No cysts were seen. Arrowheads in **A** and **B** indicate abnormal white-matter changes.

Standard axial CT images of 10 mm thickness were obtained. Intraventricular hemorrhage (IVH) was graded according to the classification described by Papile et al. [19].

EEG findings were recorded from an anteroposterior bipolar montage on a 20-channel EEG using a chart speed of 15 mm/sec, a sensitivity setting of 7  $\mu$ V/mm, and a band pass of 1–70 cycles/sec. The EEG background activity was classified as normal or abnormal for conceptual age [20].

Pre- and postmortem echoencephalograms were compared with macro- and microscopic examinations of the brain in four patients who died in the first week. In the seven survivors, neurologic and neurodevelopmental evaluations using the Bayley scales of infant development, the modified Gesell examinations, echoencephalograms, and CT scans were obtained several times during the first year and up to 36 months of age [21, 22].

## Results

Echoencephalography was first performed in all 11 infants by day 3. Ten infants were studied initially on day 1. Grade I–II IVH occurred in eight infants. The hemorrhages enlarged to

grade III in two infants by day 5. Four infants had stable grade-III bleeds. Posthemorrhagic hydrocephalus ensued in two.

The major echoencephalographic findings associated with necrosis were areas of increased echogenicity and anechoicity in the paraventricular white matter. Highly echogenic areas superior and lateral to the bodies, atria, and occipital horns were observed in all initial studies on modified coronal and parasagittal views. These views also demonstrated echogenic areas adjacent to the frontal horns in eight patients (Figs. 1–3). Studies in the axial plane from the posterior fontanelle and the parasagittal planes from the anterior fontanelle showed echogenic areas adjacent to the occipital and temporal horns.

Nine infants had anechoic lesions. In six, they appeared by day 4; in the other three, by days 11, 21, and 23, respectively. Anechoic areas developed in the same regions as the echogenic areas. Small lucencies 2–3 mm in diameter either resolved, enlarged, or coalesced to form larger, multiloculated lesions measuring 15–30 mm (Figs. 1–3 and 5).

In four of five infants echogenic and anechoic areas re-



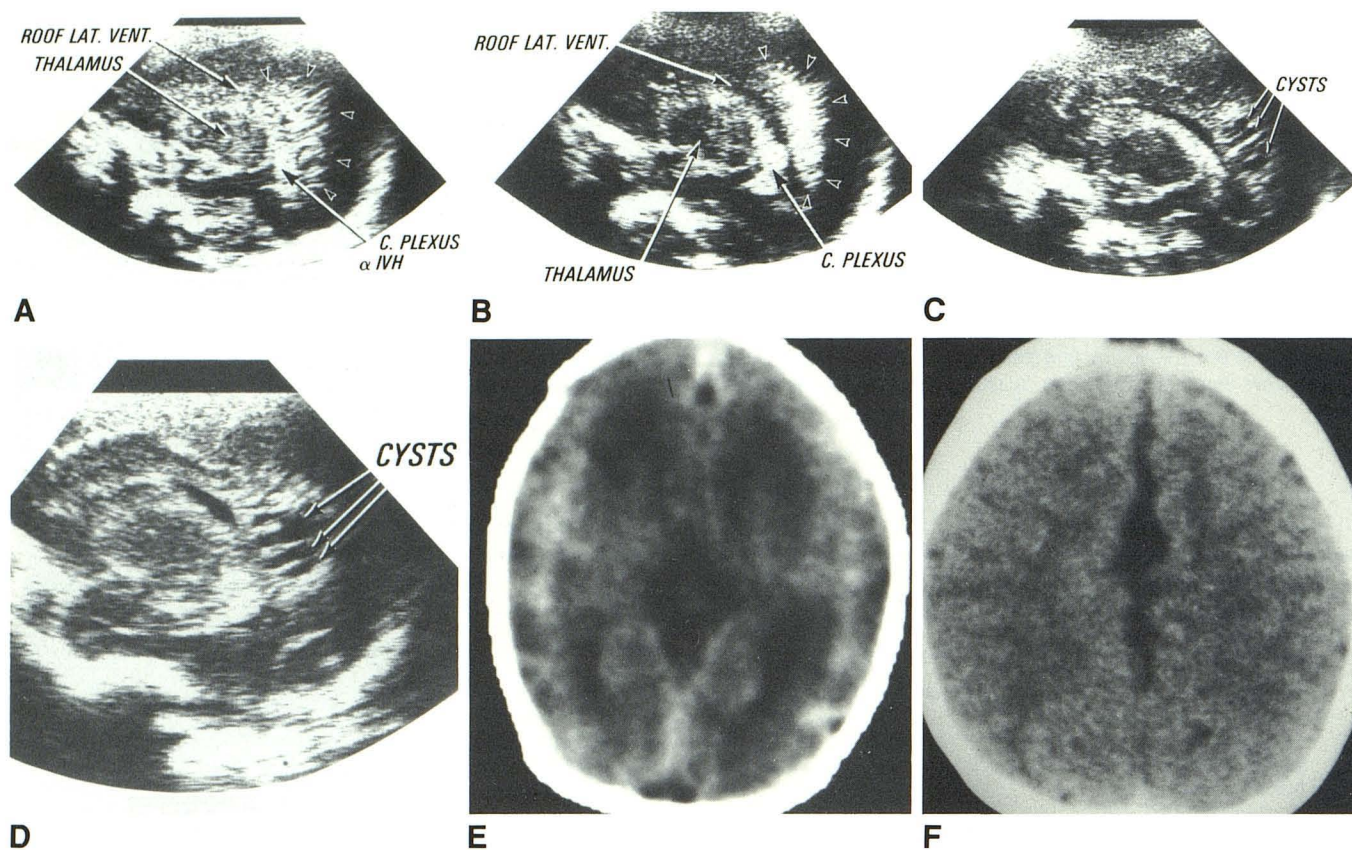


Fig. 2.—Case 1. **A**, Parasagittal sonogram ½ hr after delivery. White matter is echogenic. **B**, Day 7. Echogenicity has increased. **C**, Day 23. Cavitations are first noted. They enlarge and persist until discharge on day 51 (**D**). **E**, Unenhanced CT scan at 1 month. Extensive and markedly decreased attenuation of

paraventricular white matter in association with increased density of cortical and subcortical parenchyma. **F**, 7 months. Normal attenuation of cortex and white matter. Brain atrophy is suggested by widened interhemispheric fissure. Arrowheads in **A** and **B** indicate abnormal white-matter changes.

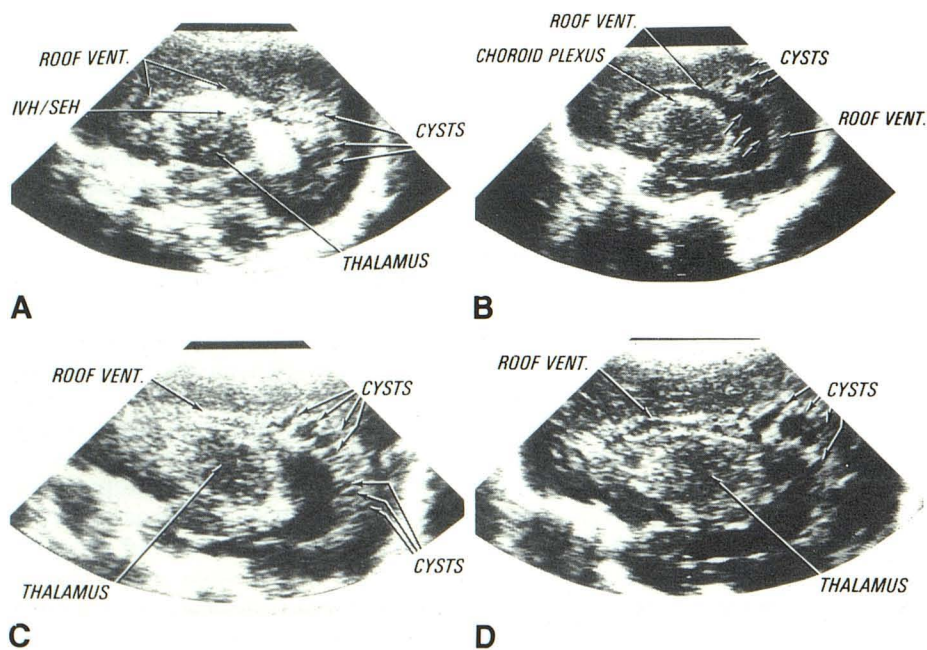


Fig. 3.—Case 3. **A**, Sagittal sonogram 12 hr after birth. Echogenic area with small anechoicities suggests that white-matter damage began antenatally. These lesions enlarged, and new lesions appeared on day 23 (**B**) and enlarged (**C**). **D**, Discharge on day 50. Cavitations are present. Loss of tissue was observed in area of trigone on day 23 (**B**, arrows inside ventricle). SEH = subependymal hemorrhage.



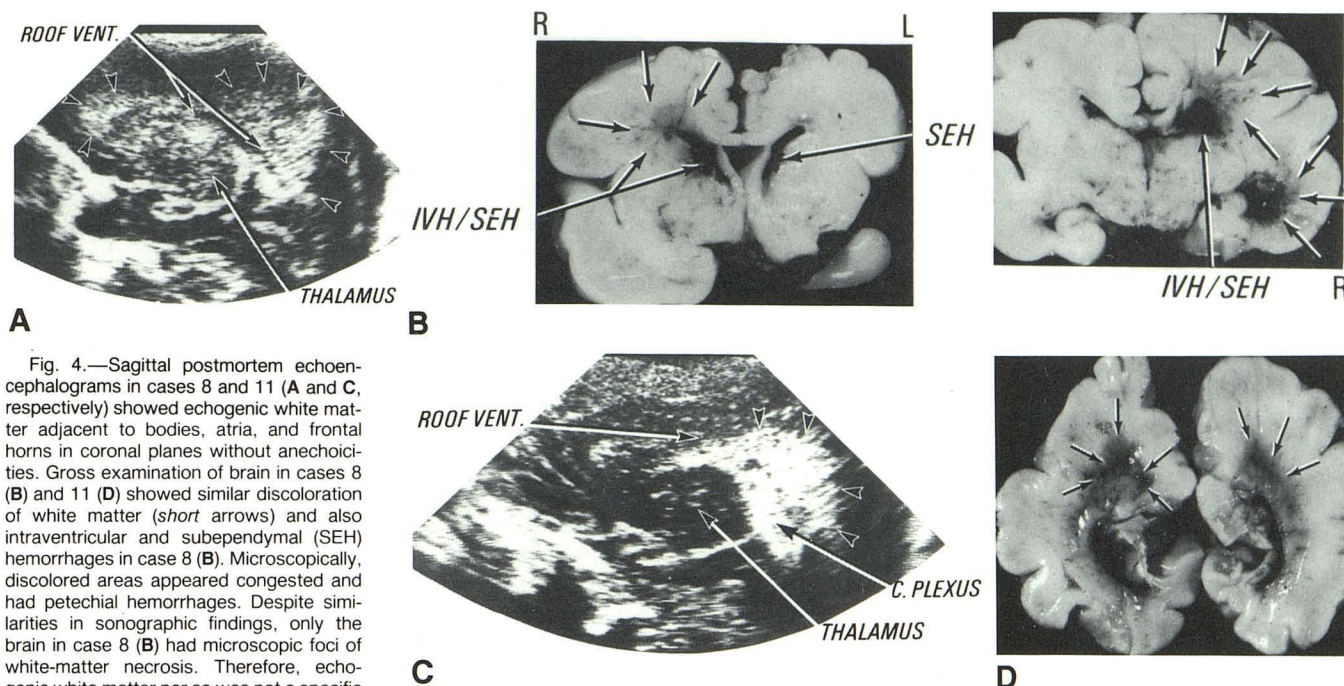


Fig. 4.—Sagittal postmortem echoencephalograms in cases 8 and 11 (A and C, respectively) showed echogenic white matter adjacent to bodies, atria, and frontal horns in coronal planes without anechoicities. Gross examination of brain in cases 8 (B) and 11 (D) showed similar discoloration of white matter (short arrows) and also intraventricular and subependymal (SEH) hemorrhages in case 8 (B). Microscopically, discolored areas appeared congested and had petechial hemorrhages. Despite similarities in sonographic findings, only the brain in case 8 (B) had microscopic foci of white-matter necrosis. Therefore, echogenic white matter per se was not a specific finding of white-matter necrosis.

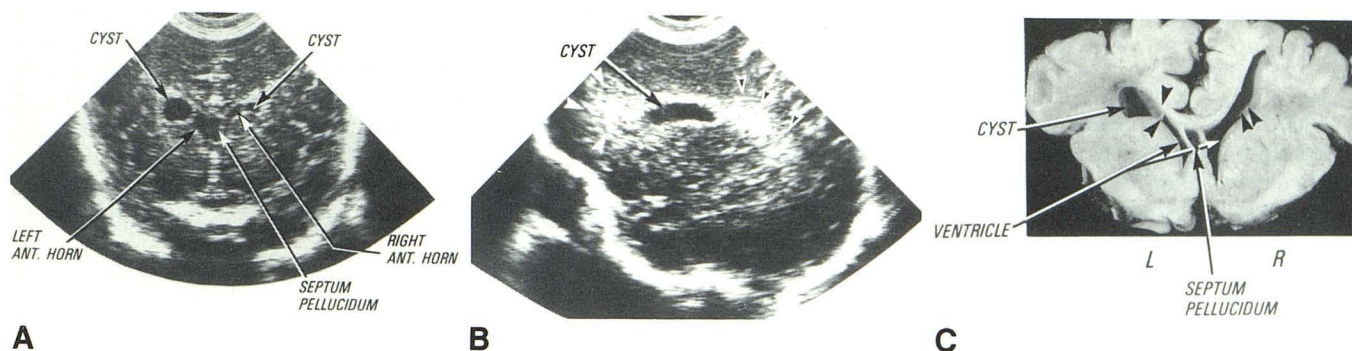


Fig. 5.—Case 9. Coronal (A) and sagittal (B) sonograms on day 1. Large anechoic lesions in white matter adjacent to anterior horns and bodies of lateral ventricles. C, Gross examination. Anechoic areas corresponded to large cysts. Arrowheads indicate septations that separate cysts from lateral ventricles. Microscopically, cysts were surrounded by extensive gliosis. The very echo-

genic white matter (arrowheads) observed on postmortem sonogram microscopically showed congestion, petechial hemorrhages, and foci of acute necrosis. Findings in this patient indicate that chronic antenatal necrosis and acute necrosis acquired postnatally may evolve simultaneously and independently.

solved by 5 months (Fig. 1). In the fifth patient the anechoic areas coalesced and formed a large porencephalic cavity. Enlargement of the interhemispheric fissure, widening of the subarachnoid spaces, and dilation of the ventricles, all suggesting brain atrophy, were observed in these five patients.

CT scans were obtained in the seven surviving infants. Initial CT studies were performed in four infants by the second week, in one during the third week, and in two by the fifth week. Four infants had follow-up CT scans between 6 months and 1 year. In five patients initial CT studies showed diffuse, minimally to moderately decreased attenuation of the white matter when echogenic areas alone or small cavities (2–4 mm) appeared on concomitant echoencephalograms. CT did not image small cysts in any of the patients. Large anechoic

areas (greater than 8–10 mm) were associated with either diffuse or circumscribed areas of markedly decreased attenuation of the paraventricular white matter in four patients (Fig. 2). Decreased attenuation of the white matter was associated with increased attenuation of the cortex in four patients (Fig. 2). Follow-up CT scans showed normal attenuation of the white matter in three patients and the unilateral porencephalic cyst that had been observed with echoencephalography in one infant. These patients also showed findings consistent with cerebral atrophy with widened interhemispheric fissures and ventricular dilatation.

Sonographic-pathologic correlations were made in four infants dying in the first week (Table 1). Figures 4 and 5 illustrate the gross anatomic findings in three patients. On microscopic



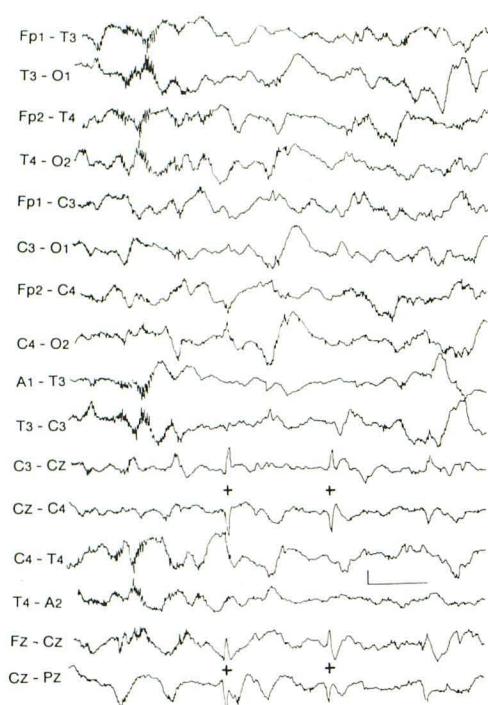


Fig. 6.—EEG shows PRS waves at central vertex location (+ signs). These were present when paraventricular cysts were forming. Calibration: 7  $\mu$ V amplitude and 2 sec interval.

examination the echogenic white matter showed vascular congestion, petechial hemorrhages, and foci of necrosis. However, in case 11 (Fig. 4), the highly echogenic areas adjacent to the atria and frontal horns had only congestion and hemorrhages without evidence of necrosis. Anechoic areas on the echoencephalograms corresponded to cystic lesions in the white matter (Fig. 5).

Six infants had EEG studies. EEG background activity was minimally abnormal in four infants, markedly abnormal in one (case 8), and normal in one (case 3) at 25 days. Five infants had positive central (rolandic) sharp (PRS) waves (Fig. 6). Cases 4 and 7 had PRS waves on day 5 when small cysts were observed with sonography. It is noteworthy that neither of these two infants had evidence of IVH. The other three infants had PRS waves when concomitant EEG studies showed only highly echogenic white matter. These three infants subsequently developed large cavitory lesions in the echogenic areas.

Follow-up neurodevelopmental information on all surviving infants is shown in Table 1. Six of the seven infants had moderate to severe spastic quadriplegia by 1 year postconceptual age. The other infant (case 3) had mild spastic diplegia. The Psychomotor Developmental Index was 50 or less in seven patients. Four of these infants had moderate to severe impairment in their Mental Developmental Index. Four had abnormal vision, three with esotropia and one with cortical blindness. Four of seven surviving infants had microcephaly.

## Discussion

The cerebral white matter adjacent to the frontal and parietooccipital regions of the lateral ventricles frequently is the site of focal infarction in preterm infants [3]. Acutely, the normal white matter is obliterated and the tissue undergoes coagulative necrosis [5]. At the onset of the chronic stage, about 2 weeks later, the necrotic areas evolve into cavitations or are replaced by glial scars. Eventually even the cavities collapse, leaving only glial scars. Some cavities persist as porencephalic cysts that communicate with the lateral ventricles. Later in the chronic stage, atrophy of the paraventricular white matter leads to dilation of the lateral ventricles and widening of the interhemispheric fissure.

The data from this study indicate that the stages of white-matter necrosis have distinct echoencephalographic findings. In the acute stage, the white matter is highly echogenic. Microscopic studies showed that the increased echogenicity is associated with vascular congestion, petechial hemorrhages, and, frequently, areas of coagulative necrosis. However, paraventricular echogenicities alone are not always diagnostic of white-matter necrosis, since the very echogenic white matter in case 11 showed only congestion and hemorrhages on microscopy.

The onset of the chronic stage is characterized by the presence of small anechoic lesions in the highly echogenic white matter. The presence of paraventricular anechoicities is evidence that necrosis has occurred in the echogenic white matter. In this study cystic lesions appeared in the echogenic white matter in three patients 11–23 days after birth. This finding is consistent with classic pathologic descriptions, which state that the chronic stage of white-matter necrosis evolves within 2 weeks of an ischemic insult. In our study six infants had cystic lesions identified by day 4. These findings are important, because they suggest that the onset of white-matter damage was antenatal.

In surviving infants, small anechoic areas either enlarged or resolved. Large cavities also resolved or formed porencephalic cysts that communicated with the lateral ventricles. Signs of white-matter necrosis were not observed on either echoencephalograms or CT scans 5 months after birth. This finding suggests that unless white-matter necrosis is diagnosed early, the lesion will be unsuspected until the infant exhibits later neurologic deficits. In addition, normal-appearing white matter on either an echoencephalogram or CT scan several months after birth is no guarantee of the absence of earlier necrosis.

In our study, echoencephalography identified the initial stage of white-matter necrosis more reliably than CT did. Similarly, others have noted that CT does not demonstrate cystic lesions as well as sonography does [13, 14]. In the acute stage, CT showed only minimally to moderately decreased attenuation, a finding that has been attributed to incomplete myelination of the white matter [23]. Large cavitory lesions were observed on CT scans as either circumscribed or diffuse areas of markedly decreased attenuation. Small cysts observed on sonography were not seen on CT studies. Given the thickness of the CT images in this study,



the small cysts may have been averaged with the normal tissue. In another recent study at our institution, CT scans with 3-mm slices showed small cavities [24].

We have no definitive explanation for the increased attenuation of the cortical parenchyma observed in four patients. This finding may be a visual artifact created by the hypoattenuation of the adjacent white matter. Alternatively, it may be explained by secondary hyperemia of the cortex, a response to the inflammatory process in the white matter.

The presence of surface PRS waves in the central region on the EEGs of infants with echogenic evolving white-matter necrosis and no IVH appears to be an important new clinical finding. Previous studies have associated PRS waves primarily with IVHs diagnosed by CT or autopsy examinations [24–26]. PRS waves also have been described in meningitis, periventricular leukomalacia, and asphyctic or hyperammonemic syndrome [27, 28]. Diffuse white-matter injury is common in these pathologic conditions. In this study, PRS waves were present in three infants with white-matter necrosis and IVH. Of particular interest was the finding of PRS waves in cases 4 and 7, since they had only echogenic white matter and no evidence of IVH. Case 3, with echogenic white matter and cavitory lesions at birth, did not show PRS waves. These findings suggest that evolving necrotic lesions rather than IVH or chronic white-matter necrosis are associated with PRS. If this finding is confirmed in a larger series, EEG studies may be extremely useful in diagnosing acute necrosis when only echogenic white matter is present.

All seven survivors with cavitory lesions in the presence or absence of IVH had abnormal neurologic development with either spasticity or paresis. Most of these infants also had abnormal vision and microcephaly. These findings suggest that the presence of cavitory lesions in areas of increased echogenicity is strongly associated with a significant neurodevelopmental handicap. Inasmuch as the sonographic and CT findings of white-matter necrosis resolve over time, it is important to identify this lesion in the perinatal period and to follow the infant's neurodevelopment closely. Furthermore, resolution of the echogenic and anechoic areas does not reflect normalization of the white matter. It merely represents the inability of sonography and CT to show paraventricular glial scars.

In summary, we believe that since the sonographic findings of white-matter necrosis change over time, it is important to identify cerebral white-matter pathology soon after birth and to follow the lesion closely with serial studies. Failure to diagnose cerebral white-matter pathology within the first 5 months may prevent future confirmation by either sonography or CT. EEG may be useful in distinguishing echogenic white matter with necrosis from that without necrosis.

#### ACKNOWLEDGMENTS

We thank George Leopold, Professor of Radiology, for manuscript review and Carol Heil, Bertha Englund, and Nancy Willsey for secretarial assistance.

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