Ashwani Kapila, M.D.
Jill Trice, M.D.
William G. Spies, M.D.
Barry A. Siegel, M.D.
Mokhtar H. Gado, M.D.

# Enlarged Cerebrospinal Fluid Spaces in Infants with Subdural Hematomas[1]

**Computed tomography in 16 infants with subdural hematomas showed enlarged basal cisterns, a wide interhemispheric fissure, prominent cortical sulci, and varying degrees of ventricular enlargement. Radionuclide cisternography in eight of the 16 patients showed findings consistent with enlargement of the subarachnoid space rather than those of communicating hydrocephalus. Clinical findings and brief follow-up showed no convincing evidence for cerebral atrophy in 13 patients. These findings suggest that the enlarged subarachnoid space, which is encountered in some infants and may be a developmental variant, predisposes such infants to subdural hematomas.**

**Index terms:** Computed tomography, in infants and children • Infants, central nervous system • Meninges, hemorrhage • (Skull, CT, 1[0].1211) • (Skull and contents, normal variation, 1[0].130) • (Skull and contents, subdural hematoma, 1[0].433) Subarachnoid space, abnormalities

**Radiology** 142: 669-672, March 1982

[1] From the Edward Mallinckrodt Institute of Radiology (A.K., W.G.S., B.A.S., M.H.G) and the Departments of Pediatrics and Neurology (J.T.), Washington University School of Medicine, St. Louis, MO. Presented at the Sixty-sixth Scientific Assembly and Annual Meeting of the Radiological Society of North America, Dallas, TX, Nov. 16-21, 1980. Received March 11, 1981; accepted and revision requested June 30; revision received Aug. 10. cd

THE computed tomographic (CT) pattern of extracerebral fluid collections associated with enlarged cerebrospinal fluid (CSF) spaces is encountered frequently in infants with large or enlarging heads (1-6). It often raises various diagnostic questions, chief of which is whether the extracerebral fluid is subdural or subarachnoid. In some cases, the extracerebral collection is clearly a subdural hematoma. The basis for the enlarged CSF-containing spaces in these cases is uncertain, although several mechanisms for this complex of findings are possible. Communicating hydrocephalus and cerebral atrophy secondarily complicating subdural hematomas are the two mechanisms usually implicated in the pathogenesis of enlarged subarachnoid spaces (1-4, 7-15). Subdural hematomas complicating cerebral atrophy and the appearance of the infantile brain with immature gyri are other possible explanations for this phenomenon. We undertook the present study of the clinical and neuroradiologic features of such patients in an attempt to determine which of these hypotheses best explains the pathogenesis of this finding.

## METHODS

We reviewed the CT scans that had been obtained in infants at our institution during the past 3 years to identify those patients with enlarged extracerebral spaces. From this large group, we selected 16 patients with associated subdural hematomas that had been documented by CT (7 patients), subdural tap (1 patient), or both (8 patients). Patients with postmeningitic subdural effusions were excluded. The CT criterion of subdural hematoma was an extracerebral fluid collection with an attenuation value greater than that of CSF. The mean age of these patients at the time of the initial CT examination was 7.6 months (range, 2-14 months).

The CT scans were reviewed with specific attention to: (*a*) the size of the cerebral sulci, basal cisterns, interhemispheric fissure, and ventricles, and (*b*) the presence of associated parenchymal abnormalities. The degree of ventricular dilatation was subjectively categorized as slight, moderate, or severe. Follow-up CT scans were available in 12 patients. These had been obtained a mean of 6.9 months after the initial study (range, 8 days-2 yrs.). The follow-up studies were reviewed for changes in the size or appearance of the subdural collection and CSF spaces. In addition, the hospital records of these patients were reviewed, with particular attention to the clinical presentation, the head circumference, and the results of subdural taps. Follow-up of the developmental and clinical status was available in 12 patients. The mean interval of follow-up was 7.6 months in these patients (range, 15 days-24 months). Follow-up

evaluation of head circumference was available in 8 patients.

In 8 of the 16 patients, radionuclide cisternography had been performed to determine whether communicating hydrocephalus might account for the enlarged ventricles and subarachnoid spaces. Cisternographic imaging had been performed with a scintillation camera 2 to 6, 24, and usually 48 hours after intrathecal injection of 70 to 100 μCi (2.59–3.7 MBq) of $^{111}$In-DTPA. The images were reviewed with attention to the rate and symmetry of tracer ascent in the subarachnoid space and the duration of ventricular activity, if present.

## RESULTS

### CT Findings

The CT diagnosis of subdural hematoma was possible in 15 of our 16 patients, in whom the attenuation of the extracerebral collection was greater than that of CSF. The remaining patient had an extracerebral collection with a density that was indistinguishable from that of CSF on the initial CT study; the diagnosis in this patient was made by subdural tap. Two weeks later, a repeat CT scan showed a subdural collection that had a density greater than CSF and that was similar in appearance to those in the other patients. In addition, the CT scans (Fig. 1) of all 16 patients demonstrated: (*a*) large basal cisterns, particularly the Sylvian and suprasellar cisterns; (*b*) a wide interhemispheric fissure, specifically its anterior one half to two thirds; (*c*) prominent cortical sulci; and (*d*) varying degrees of ventricular dilatation. The ventricles were dilated slightly in six, moderately in eight, and severely in one. This last patient had Menkes disease. The widening of the CSF-containing spaces was greatest in the two patients with known metabolic encephalopathy. The subdural collections were bilateral in all but one patient, and located anteriorly in all. An interhemispheric extension was present in 12. Two patients had a fresh hemorrhage superimposed on the chronic subdural hematoma. One presented with this finding on the initial scan and the other had an iatrogenically induced acute hemorrhage. In both instances, the sulci were effaced on the side of the fresh hemorrhage and reappeared following subsequent subdural taps.

### Clinical Findings

The most common presenting signs were macrocephaly and enlarging head circumference, which were present in 12 patients. Four of these patients had increased intracranial pressure. Two of them had retinal hemorrhages and additional associated problems (hemiparesis and focal seizures on admission in one and mild hemiparesis and a generalized seizure in the other). One of these four children was almost certainly the victim of child abuse and had a skull fracture. The remaining eight patients with macrocephaly had no overt clinical evidence of increased intracranial pressure. Associated findings in this group were: agenesis of the corpus callosum, the asymmetric crying facies syndrome, hypotonia, and severe developmental delay in one; and minimal developmental delay in two. In addition, one of the infants in this subgroup had an episode of slight trauma to the head.

Four of the 16 patients had normocephaly ($n = 3$) or microcephaly ($n = 1$). The microcephalic patient had methylmalonic aciduria, severe developmental delay, and seizures. One of the normocephalic patients had Menkes disease, also associated with severe developmental delay. Of the other two patients, one presented with a seizure and had a history of febrile seizures and a skull fracture 5 months previously. The other had hypotonia and had had a single seizure during recovery from anesthesia for drainage of a supraclavicular abscess.

Thus, of the 16 patients, severe developmental delay was noted in three; the CT findings in two of these patients were indicative of significant cerebral atrophy. The third was the patient with agenesis of the corpus callosum. Two additional patients had minimal developmental delay, and the remaining 11 patients (69%) had normal development.

### Cisternographic Findings

None of the 8 radionuclide cisternograms was totally normal. Four showed slow ascent of the radiopharmaceutical over the convexities, and one showed transient ventricular activity on early images only (2 to 6 hours). In the remaining three patients, both findings were present.

### Subdural Taps

Subdural taps, performed in nine patients, demonstrated subdural fluid in all. The fluid was frankly bloody in seven and xanthochromic with a high protein content in two. Multiple subdural taps were performed in seven patients.

### Follow-up CT Scans

Follow-up CT scans were obtained in 12 patients, eight of whom underwent subdural taps. The CT scans showed complete resolution of the subdural collection within 9 months in five of these eight patients (Fig. 2), and partial resolution in two. In the remaining patient the follow-up scan, obtained only 10 days after the initial study, showed no change. The lateral ventricles increased in size on follow-up scans in three of the eight patients. In each case the increase in ventricular size coincided with the decrease in size or the disappearance of the extracerebral collection.

Four of the patients with follow-up CT scans were not subjected to subdural taps. Three showed no change in the subdural collection, whereas one showed a spontaneous decrease in the size of the subdural hematoma in 37 days.

### Clinical Follow-up

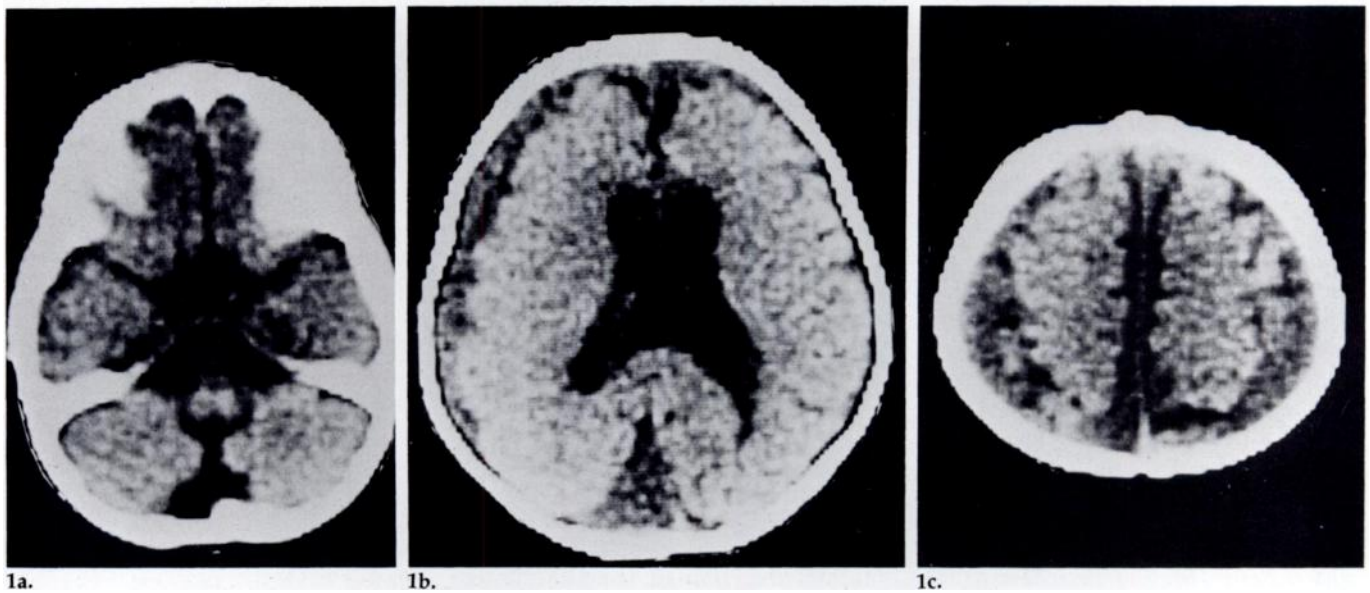
Of the nine patients who had subdural taps, the occipitofrontal circumference continued to parallel the 98th percentile in five, decreased to a lower percentile in three, and was not available in one. There was no developmental deterioration in these patients on clinical follow-up ranging in duration from 15 days to 2 years.

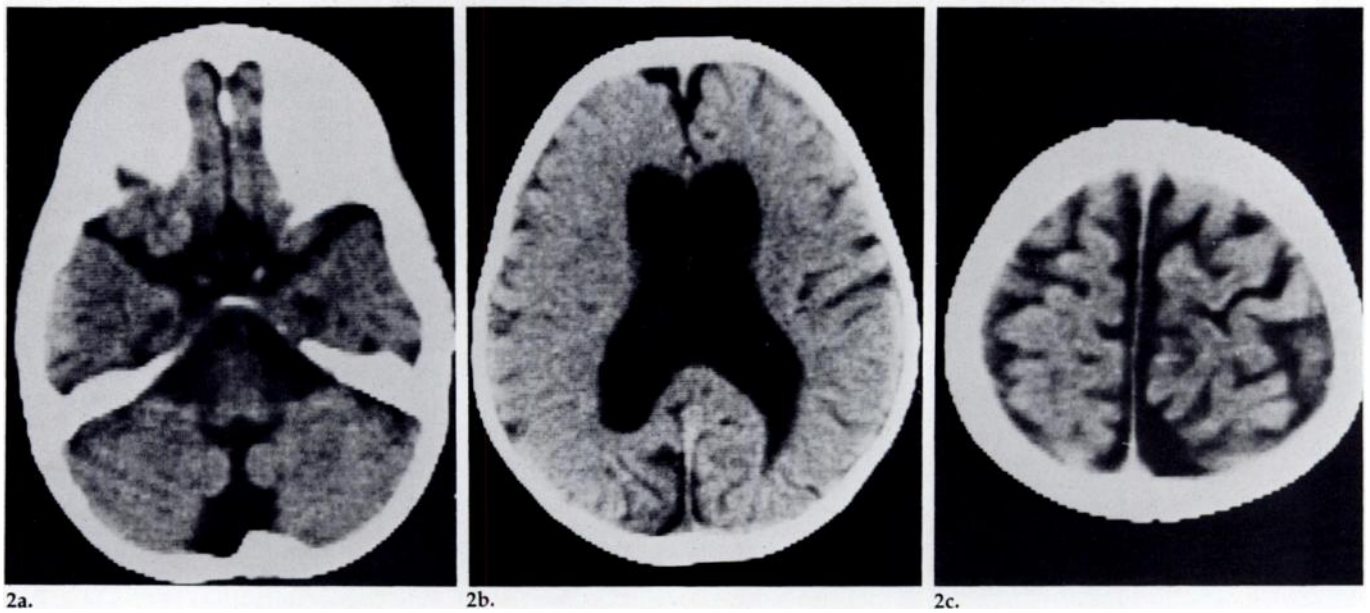
## DISCUSSION

The association in infants of large subarachnoid spaces and ventricles with subdural hematomas is well known, and has been described in both CT literature (1–4) and older neuroradiologic literature (11, 14). The significance of these enlarged, CSF-containing spaces has never been defined precisely. Several mechanisms have been proposed: communicating hydrocephalus (1–4, 7, 10–13, 15), cerebral atrophy (1, 4, 9, 11, 14), or both (8, 13) occurring as a complication of subdural hematoma. None of these mechanisms has been documented adequately.

Communicating hydrocephalus has

**Figures 1 and 2**




1. Subdural hematoma in an 11-month-old infant. The extracerebral collection is distinguished from the underlying subarachnoid space by its density, which is slightly higher than the density of CSF.
   a. At the level of the basal cisterns, there is widening of the CSF spaces.
   b. At the level of the bodies of the lateral ventricles, there is ventricular dilatation. The extracerebral collection extends into the interhemispheric fissure.
   c. At the high convexity, the interhemispheric fissure is enlarged due to a large subarachnoid space as well as extension of the extracerebral collection.
2. Follow-up CT scan obtained in the same patient as shown in Figure 1; 9 months later.
   a. Scan obtained at the level of the basal cisterns.
   b. Scan obtained at the level of the body of the lateral ventricles.
   c. Scan obtained at the high convexity. Note that the extracerebral collection has disappeared. The enlargement of the ventricles and subarachnoid space remains.

been described as a complication of chronic subdural hematomas in infants. Its pathogenesis has been related to blockage of CSF flow at a high convexity or parasagittal level by the subdural collections (1, 7) or to faulty absorption of CSF (8, 11). The latter may also be the result of arachnoid inflammation caused by concomitant subarachnoid hemorrhage originating from the same traumatic episode that caused the subdural hematoma. Communicating hydrocephalus occurring as a sequela of meningitis and associated with subdural effusions (13) is a different entity in our opinion. Accordingly, we excluded all patients with a known history of meningitis from our study population.

The results of radionuclide cisternography in eight of our patients were

significant in that none of the patients had persistent ventricular activity. Transient ventricular activity, slow flow in the subarachnoid space, or both were observed in all of our patients. These findings reflect enlarged CSF spaces, but are not indicative of communicating hydrocephalus. This conclusion is based on the observation that adults with cerebral atrophy typically have similar cisternographic findings (16, 17). Further, it has been suggested that transient ventricular activity may even be a normal finding in children under 2 years of age (18).

Raimondi (14) has emphasized that subdural hematomas may cause a secondary decrease in brain volume. Ingraham and Matson (11) related the severity of cerebral atrophy to the length of time the subdural hematoma was present. Mental retardation develops in a significant proportion of infants after treatment of subdural hematomas (7, 11, 15, 19-21). On the other hand, Rabe *et al.* (22) have stressed the significance of the antecedent event as a determining factor in the final outcome. Mealey (23) has refuted the direct injurious effect of subdural hematomas in the absence of increased intracranial pressure.

In our series, the two patients with metabolic encephalopathy and the one with agenesis of the corpus callosum had severe developmental delay, as expected. More importantly, only two of the remaining 13 patients had evidence of even minimal developmental delay. Furthermore, clinical follow-up, which was available in nine patients, showed no detectable deterioration in psychomotor development. Similarly, follow-up evaluation of head circumference indicated continued head growth at the same or a lower percentile. There was no cessation of head growth. Moreover, we have no evidence that actual brain mass was less than normal in any of these infants. These findings argue strongly against the hypothesis that the subdural hematomas led to cerebral atrophy in our patients. However, definitive proof of this contention will require longer follow-up of a greater number of patients.

It has been shown that infants up to about the age of 6 months (24) and perhaps 12 months (25, 26) may have large sulci and a space up to 5.0 mm in width between the brain and the inner table (24, 25). Minor trauma, causing displacement of the entire cranial contents, may induce bilateral subdural hematomas by tearing of bridging veins (11). It is well known that bilateral subdural hematomas may develop in geriatric patients following minimal trauma. Presumably the underlying mechanism is similar.

All of our 16 patients had concomitant enlargement of the ventricles and subarachnoid spaces on the *initial* scan; furthermore, the subdural hematomas developed in the two patients with metabolic encephalopathy and primary cerebral atrophy in the absence of a history of trauma.

Our observations suggest that the enlargement of the ventricles and subarachnoid space in this group of patients predisposed them to the development of subdural hematoma with minimal or no trauma. The underlying cause of the enlarged CSF spaces remains uncertain.

Mokhtar H. Gado, M.D.
Mallinckrodt Institute of Radiology
510 S. Kingshighway Blvd.
St. Louis, MO 63110

## References

1. Mori K, Handa H, Itoh M, Okuno T. Benign subdural effusion in infants. J Comput Assist Tomogr 1980; 4:466-471.
2. Orrison WW, Robertson WC, Sackett JF. Computerized tomography in chronic subdural hematomas (effusions) of infancy. Neuroradiology 1978; 16:79-81.
3. Robertson WC Jr, Chun RWM, Orrison WW, Sackett JF. Benign subdural collections of infancy. J Pediatr 1979; 94:382-386.
4. Rothenberger A, Brandl H. Subdural effusions in children under two years—clinical and computer-tomographical data. Neuropaediatrie 1980; 11:139-150.
5. Pettit RE, Kilroy AW, Allen JH. Macrocephaly with head growth parallel to normal growth pattern: neurological, developmental, and computerized tomography findings in full-term infants. Arch Neurol 1980; 37:518-521.
6. Robertson WC Jr, Gomez MR. External hydrocephalus. Early finding in congenital communicating hydrocephalus. Arch Neurol 1978; 35:541-544.
7. Elvidge AR, Jackson IJ. Subdural hematoma and effusion in infants. Am J Dis Child 1949; 78:635-658.
8. Entzian W, Gullotta F. Hydrocephalus, subdural effusion and sclerotic brain atrophy in infancy: case report. Neuropaediatrie 1972; 3:313-318.
9. Gega A, Utsumi S, Kyoi K, Hori Y. Neuroradiologic evaluation of the subdural pathogenesis in infants with small heads. Neuroradiology 1978; 16:36-38.
10. Gutierrez FA. Angiographic characteristics of certain subdural collections of fluid. Childs Brain 1977; 3:48-61.
11. Ingraham FD, Matson DD. Subdural hematoma in infancy. J Pediatr 1944; 24:1-37.
12. Lehrer H. Communicating hydrocephalus resembling cortical atrophy: an analysis of cases with application to the syndrome of "normal pressure hydrocephalus." AJR 1968; 104:150-161.
13. Mori K, Handa H. Subdural haematoma (effusion) and internal hydrocephalus. Neurochirurgia (Stuttg) 1977; 20:154-161.
14. Raimondi AJ. Pediatric neuroradiology. Philadelphia: Saunders, 1972:126-146.
15. Yashon D, Jane JA, White RJ, Sugar O. Traumatic subdural hematoma of infancy. Arch Neurol (Chicago) 1968; 18:370-377.
16. James AE Jr, New PFJ, Heinz ER, Hodges FJ III, DeLand FH. A cisternographic classification of hydrocephalus. AJR 1972; 115: 39-49.
17. Partain CL, Staab EV. Brain imaging of cerebrospinal fluid: computed tomography and nuclear medicine correlation. In: Sodee DB, ed. Correlations in diagnostic imaging: nuclear medicine, ultrasound, and computed tomography in medical practice. New York: Appleton-Century-Crofts, 1979:57-64.
18. Maki Y, Kokubo Y, Nose T, Yoshii Y. Some characteristic findings of isotope cisternograms in children. J Neurosurg 1976; 45: 56-59.
19. Herzberger E, Rotem Y, Braham J. Remarks on thirty-three cases of subdural effusion in infancy. Arch Dis Child 1956; 31:44-50.
20. Russell PA. Subdural haematoma in infancy. Br Med J 1965; 5459:446-448.
21. Shulman K, Ransohoff J. Subdural hematoma in children. The fate of children with retained membranes. J Neurosurg 1961; 18:175-181.
22. Rabe EF, Flynn RE, Dodge PR. Subdural collections of fluid in infants and children. A study of 62 patients with special reference to factors influencing prognosis and the efficacy of various forms of therapy. Neurology (Minneap) 1968; 18:559-570.
23. Mealey J Jr. Infantile subdural hematomas. Pediatr Clin North Am 1975; 22:433-442.
24. Harwood-Nash DC, Fitz CR. Neuroradiology in infants and children. St. Louis: Mosby, 1976:264.
25. Fukuyama Y, Miyao M, Ishizu T, Maruyama H. Developmental changes in normal cranial measurements by computed tomography. Dev Med Child Neurol 1979; 21:425-432.
26. Rosengren K, Carlsson CA. The normal encephalogram during the first two years of life. Acta Radiol [Diagn] 1972; 13:461-466.