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## **Ventricular septa in the neonatal age group: diagnosis and considerations of etiology.**

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# Ventricular Septa in the Neonatal Age Group: Diagnosis and Considerations of Etiology

Dieter Schellinger<sup>1</sup>  
Edward G. Grant<sup>1</sup>  
Herbert J. Manz<sup>2</sup>  
Nicholas J. Patronas<sup>1</sup>  
Ronald H. Uscinski<sup>3</sup>

Twenty-four patients with ventricular septa are discussed. Seventeen patients had septa acquired during the neonatal period and seven exhibited septations at birth (congenital septa). Among the acquired septa, there were true intraventricular septa and septa that originated outside the ventricles but later became part of the ventricular system (pseudosepta). Pseudosepta originate in necrotic, cavitating periventricular white matter that, in temporal sequence, becomes ventricularized. Serial use of cranial sonography provided important information about the pathologic mechanisms that govern the development of septa. Intraventricular hemorrhage and infection are the major causes of true intraventricular septa, while periventricular leukomalacia serves as primary cause of pseudosepta. Sonography is the diagnostic method of choice. Septa are associated with a high incidence (62%) of shunt failure.

Compartmentalization of ventricles by septa has not been widely recognized in the radiology literature. The discussion usually emerges in conjunction with ventriculitis [1–10]. There are isolated neurosurgical reports, which relate such septa to a high incidence of shunt failure [1–3]. The methods by which brain and/or ventricular compartmentalization have been diagnosed include air ventriculography [1–4], CT [4–6], and sonography [7–9].

Serial use of sonography in the neonatal period provides an opportunity to observe the development of septa in chronologic sequence. In this report we focus on the sonographic documentation of emerging ventricular compartmentalization by septa and wish to draw attention to causes other than infections.

## Materials and Methods

Our patient population is divided into two groups (Table 1). The larger group comprises 17 patients who developed septations or webs in the neonatal phase (acquired septa). The majority of patients in this group were premature. The second group of seven patients presented with septations at birth (congenital septa). Most were term babies. There were two deaths in the series; both had autopsies.

In the group with acquired septa, all but two patients had serial sonograms (average 3.6/patient). In the second group of congenital septa (seven patients), three had serial sonograms while four had only one sonogram, which was performed at birth.

In the entire sample of 24 patients with septa, 14 had CT scans and sonograms within 1 week, permitting comparison of the two methods. Comparison of CT and sonography was made retrospectively and was based on hard copies of CT scans and sonograms, taken in a routine fashion. The CT scans were performed on a GE 9800 or Philips 310 scanner. The cuts were 5 mm thick and contiguous. They were angled 20° to Reid's baseline. The sonograms were obtained with a real-time sector scanner (Advanced Technology Labs, Bellevue, WA) equipped with 5 MHz and 7.5 MHz transducers. The sonographic scans were sampled through the anterior fontanelle and included coronal, angled coronal, and axial images as well as sagittal and angled sagittal views.

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<sup>1</sup> Department of Radiology, Georgetown University Medical Center, 3800 Reservoir Road, N.W., Washington, D.C., 20007. Address reprint requests to D. Schellinger.

<sup>2</sup> Department of Pathology, Georgetown University Medical Center, Washington, D.C., 20007.

<sup>3</sup> Department of Surgery, Georgetown University Medical Center, Washington, D.C., 20007.

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**Results**

Septa can be present at birth (congenital septa) or acquired in the neonatal phase (acquired septa) (Table 1). The term "congenital" is used in the broadest sense, acknowledging that some of these septa may be acquired during fetal development.

In the group of acquired septa, the patients had either intraventricular hemorrhage, periventricular leukomalacia, ventriculitis, or a combination thereof (Tables 1 and 2). Five patients had purulent ventriculitis, with bacterial agents as listed in Table 1. Of these, three patients developed ventriculitis after shunting and two had primary ventriculitis.

The acquired septa may originate in the ventricles (true septa) or in the periventricular territories that later become

ventricularized (pseudosepta) (Table 2). Acquired septa and pseudosepta may also occur together (four patients).

True septa were found in eight patients. They had in common the presence of intraventricular irritants; that is, intraventricular hemorrhage or purulent exudate. True septa may span the ventricular walls or float in the lumen. They can be delicate or coarse and resemble cobwebs or thin veils. They may be rather complex, occupying the entire ventricle, or focal with only solitary strands noted (Figs. 1 and 2).

Pseudosepta occurred in 13 patients, dominating our series. Pseudosepta develop originally outside the ventricles. However, in the later and final stages, they become incorporated into the ventricle(s) and mimic true intraventricular septa (Figs. 3 and 4). They were found in neonates with cystic periventricular leukomalacia and cystic encephalopathies. Converging periventricular cysts become confluent to such an extent that they may form a uni- or multicameral cavity, separated from the lateral ventricles only by ependyma or gliotic tissue (Fig. 5A). After disruption and fragmentation of

**TABLE 1: Ventricular Septa: Distribution, Associated Cerebral Pathology and Birth Histories**

Distribution of Patients	Maturity	Infectious Agent
<b>Acquired septa</b>		
IVH, PVC	11	10 prem; 1 term
IVH	1	Prem
IVH, PVC, ITIS	2	Prem
IVH, ITIS	2	Prem
		Klebsiella; <i>E. coli</i>
		Klebsiella; Flavobact. meningosept.
ITIS	1	Term
		<i>E. coli</i>
<b>Total</b>	<b>17</b>	
<b>Congenital septa</b>		
ITIS	1	Term
ITIS (?)	1	Term
		Toxoplasmosis (?) Cytomegalovirus
Unknown etiology	2	Term
Meningomyelocele	1	Prem
Meningomyelocele	1	Term
Encephalocele	1	Term
<b>Total</b>	<b>7</b>	

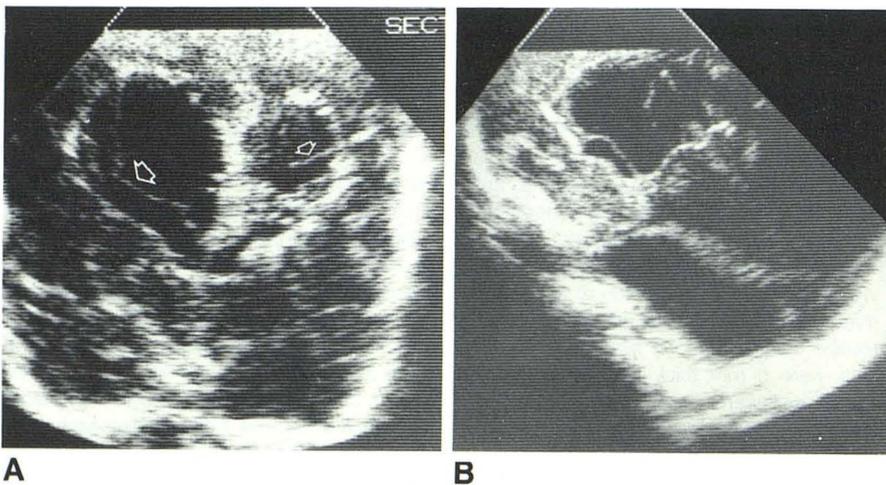
Note.—IVH = intraventricular hemorrhage; PVC = periventricular cysts; ITIS = bacterial ventriculitis.

**TABLE 2: Analysis of 17 Patients with Acquired Septa: Associated Cerebral Pathology**

<b>True septa</b>	
IVH, PVC	2
IVH, PVC, ITIS	2
IVH, ITIS	2
ITIS	1
IVH	1
<b>Total</b>	<b>8*</b>
<b>Pseudosepta</b>	
IVH, PVC	10
IVH, PVC, ITIS	2
IVH, ITIS	1
<b>Total</b>	<b>13*</b>

\* Four patients had both true and pseudosepta.

Note.—IVH = intraventricular hemorrhage; PVC = periventricular cysts; ITIS = bacterial ventriculitis.



**Fig. 1.**—Bacterial ventriculitis (*E. coli*) developed after ventriculoperitoneal shunting for slowly progressing hydrocephalus in full-term baby. **A.** Coronal section shows asymmetrically dilated lateral ventricles. The lumina of the ventricles are traversed and intersected by thin, irregular-appearing septa (arrows). **B.** Angled sagittal section through markedly dilated lateral ventricle. Appearance of septa is similar to that in part A.

Fig. 2.—Full-term neonate with congenital toxoplasmosis with hydrocephalus. **A**, Early sonogram shows thin septum (*arrow*) in frontal horn. **B**, CT scan, taken after shunt placement, documents subependymal calcifications.

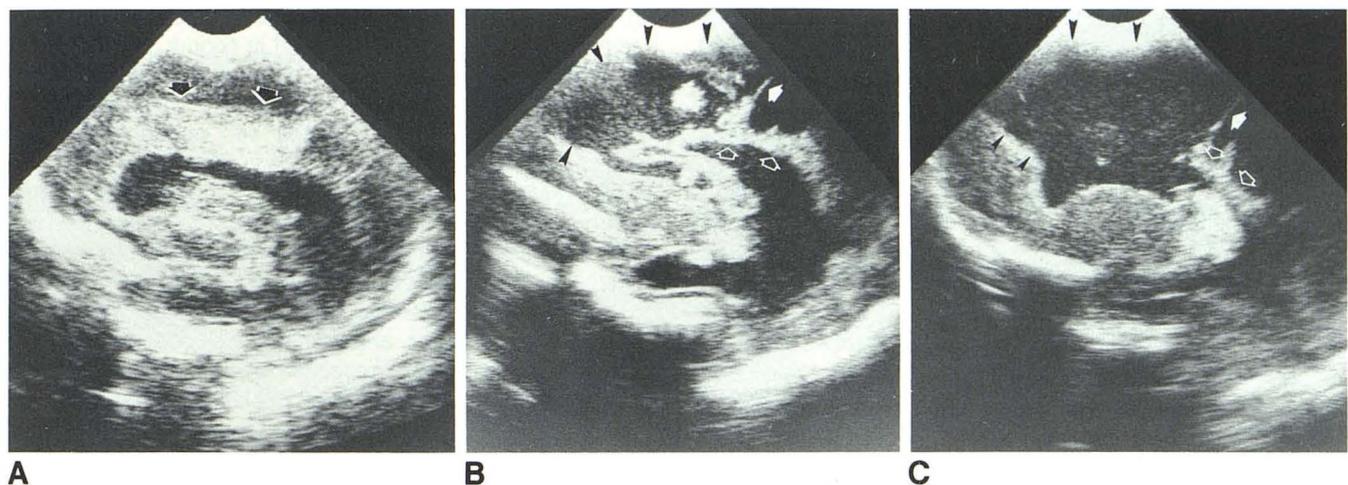
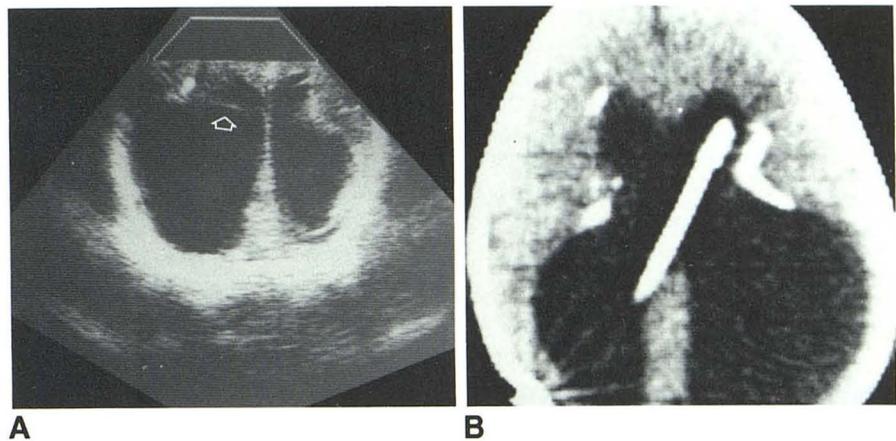


Fig. 3.—Premature infant with periventricular leukomalacia and development of pseudosepta within 17 days. **A**, Angled sagittal view showing increased echogenicity rostral to body of lateral ventricle (*arrows*). There is also increased echogenicity within ventricle, which represents blood. **B**, Confluence of periventricular cavities results in large pseudoventricle (*black arrowheads*) in area previously involved by leukomalacia. Septum is seen within pseudoventricle

(*solid white arrow*). Ependyma is the only divider between pseudoventricle and true ventricle (*open arrows*). Intraventricular blood has become coagulated. **C**, Later scan shows pseudoventricle now partially ventricularized (*black arrowheads*). Residual ependyma (*open arrows*) and adjacent septa (*solid white arrow*) appear to float within new megaventricle (*open arrows*).

the dividing ependyma, they merge with the primary ventricle. Thus, extraventricular septa are converted to intraventricular septa. Pseudosepta can appear as solid sheathlike dividers (four of 12 patients) (Fig. 6) or be fragmented and interrupted (eight of 12 patients). As may be expected, they are mostly seen in larger ventricles.

Unless observed serially during their development, pseudosepta and true septa cannot be differentiated with any degree of reliability. However, on sonograms, true septa tend to be more delicate and more interlaced (Figs. 1 and 2) while pseudosepta appear to take the form of more solid membranes (Figs. 3, 4, 6). Because of the customary association with intraventricular hemorrhage, true septa and pseudosepta may coexist (four patients). With increasing ventricular size and time, septa were observed to rarefy; eventually, they may

disappear entirely. The overall incidence of septa resolution could not be examined in this study because of lack of late follow-up in several patients.

In the congenital septa group (seven patients), the precise etiology of the septations was difficult to ascertain. In two patients, an inflammatory process was the probable cause (Table 1). One patient had congenital toxoplasmosis (Fig. 2). Another patient had periventricular calcification of unknown etiology; cytomegalovirus infection was assumed to be a likely cause. The fine septa shown in these cases should represent true septa that developed secondary to intraventricular infection. In three patients, the septations were similar to some of the pseudosepta observed in the acquired group; that is, they appeared more solid, sheathlike, less complex (Fig. 7), and had the appearance of a stripped ependyma, which can be

observed in pseudosepta (Figs. 3, 4, 6). Three patients with definite dysplastic features (myelomeningocele; encephalocele) also exhibited ventricular septa (Fig. 8).

There were 14 patients in whom CT scans and sonograms could be compared. In 10 patients, sonography was superior to CT. This includes six false-negative CT scans (Fig. 7). On sonograms, the septations were recognized on all standard images. In four patients who had unequivocal sonographic evidence of septations, the septa were faintly suggested on CT but were not initially diagnosed.

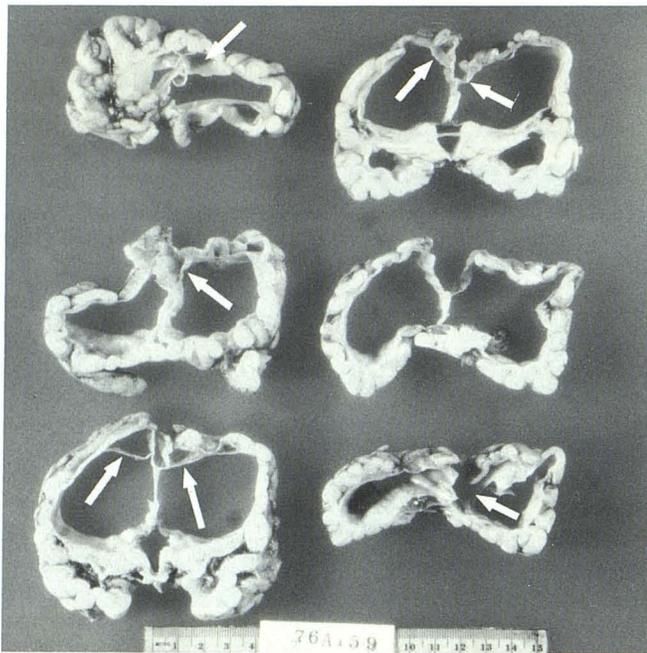


Fig. 4.—Multiple coronal sections through infant brain (not from this series). Periventricular leukomalacia has resulted in large confluent cavities (arrows). Thin pseudosepta separate these from true ventricles.

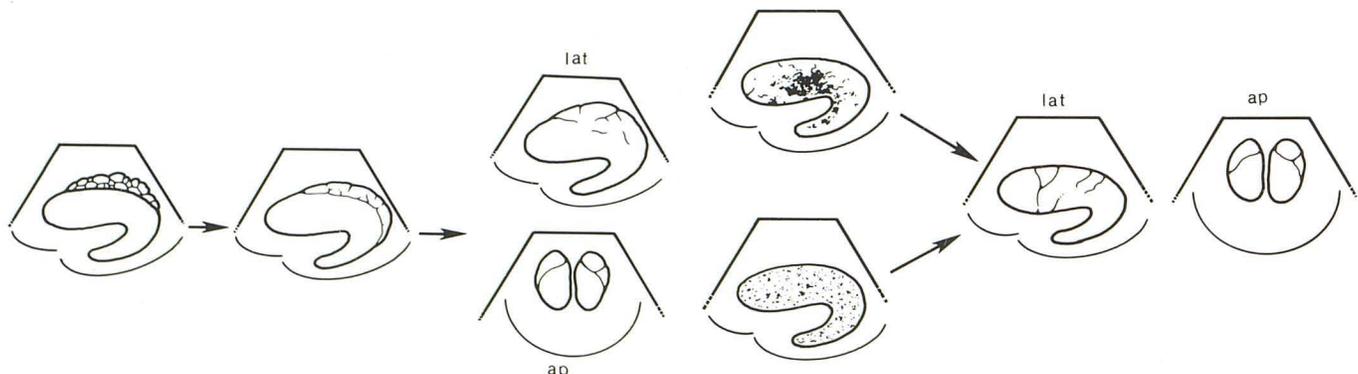


Fig. 5.—Proposed mechanism of evolution of pseudosepta (A) and true ventricular septa (B). A, Gradual ventricular incorporation of necrotic periventricular white matter (see text). B, With intraventricular hemorrhage (upper left) and bacterial ventricular infection (lower left), ventricular exudate and debris

become organized by fibroglial elements that connect with denuded ventricular wall. This results in compartmentalization of ventricles, as diagrammed on the right.

## Discussion

In this report, we categorize ventricular septa as true or false (pseudo-) septa. Among 17 patients with postnatally acquired septa, 13 had pseudosepta, eight had true septa, and four had both. In the congenital group (seven cases), a division into true septa and pseudosepta could not be achieved reliably. Only sequential intrauterine sonographic scanning would have made that distinction possible, and this was not done in our patient population.

Pseudosepta originate outside the ventricles but later become ventricularized (Fig. 5A) and may then be indistinguishable from true septa. The temporal profile, defined by preceding serial sonograms, may give the only diagnostic clue. Pseudosepta were frequently found in connection with periventricular leukomalacia and cystic encephalopathies. True intraventricular septa are those that originate within ventricles. In our series they were associated with ventriculitis and/or intraventricular hemorrhage (Fig. 5B).

In the group of patients with acquired septa, true ventricular septa were found in eight patients. Among them, four had purulent ventriculitis, seven exhibited intraventricular hemorrhage, and three had both.

Purulent ventriculitis frequently occurs in association with meningitis [6]. Berman and Banker [11] observed that it causes an inflammatory response in the ependyma with patchy or diffuse denudation of this cell layer and development of subjacent gliosis and gliotic tufts at the sites of ependymal disruption. They state further that glial tufts frequently projected into the exudate from the subependymal tissue (Fig. 9). Schultz and Leeds [3] also characterize the pathologic findings of ventriculitis as small areas of denuded ependyma, with glial tufts extending through the fragmented ependyma

Fig. 6.—Premature infant who developed intra-ventricular hemorrhage and periventricular leukomalacia on eighth day of life. Periventricular cysts appeared on 15th day of life and pseudosepta emerged on 30th day of life. **A**, Coronal scan showing large frontal horns, large blood clot in one ventricle (*solid arrow*), and pseudoseptum in other (*open arrow*). **B**, Angled sagittal view shows same lateral ventricle with pseudosepta stretching between ventricular walls (*arrows*).

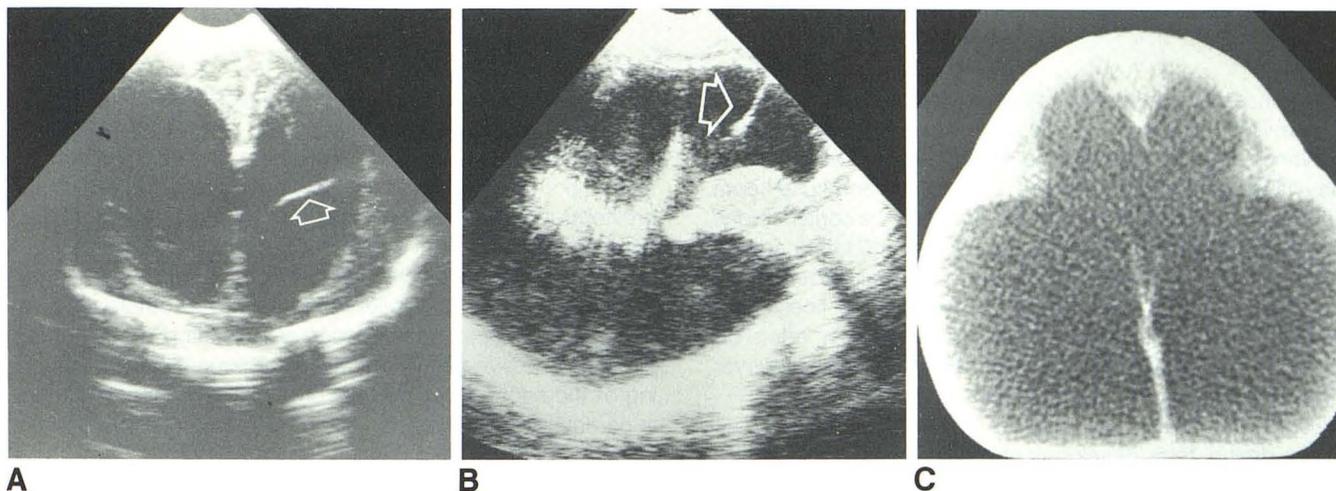
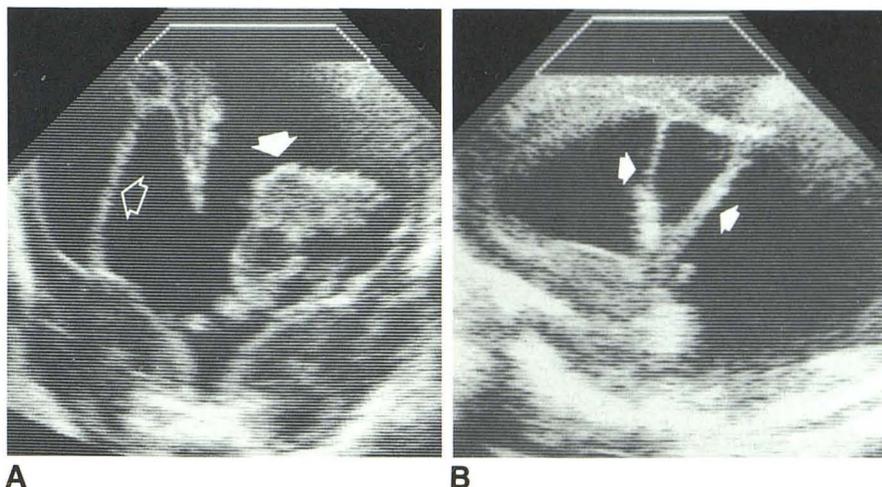


Fig. 7.—Congenital hydrocephalus in full-term baby. Axial (**A**) and angled sagittal (**B**) views show single septum in markedly dilated ventricles (*arrows*). **C**, CT scan taken within 24 hr of sonogram failed to demonstrate septum.

into the ventricular lumen. According to their description, the intraventricular septa consist of fibroglial elements with round cells and some polymorphonuclear cells (Figs. 5B and 8).

It is important to realize that bacterial ventriculitis can also lead to or coexist with severe parenchymal pathologies. This was the case in three of our patients who exhibited both true septa and pseudosepta. Among the 27 ventriculitis patients in Berman and Banker's [11] meningitis series, there were nine who also had parenchymal infarction, microabscesses, or periventricular leukomalacia. While some of their parenchymal changes (infarcts, microabscesses) were directly linked to the infectious process, it could not convincingly be determined whether leukomalacia was infection related or caused by incidentally coexisting neonatal cerebral anoxia.

Naidich et al. [10], in a study using an experimental model of *E. coli* meningitis/ventriculitis in the hy-3 mouse, showed that intraventricular septa or veils are actually ependyma,

displaced inward by subependymal cysts or sheets of pericyclic white matter. True intraventricular septa did not occur. The periventricular cavities of their experimental animals were produced by confluence of "edema-filled" extracellular spaces that formed a pseudoventricle. The septa that separated these extraventricular cavities are similar to our pseudosepta. Histologic studies [6, 12] have shown that mononuclear phagocytes, glial proliferations, and ependymal denudation also occur around zones of hemorrhage; further ependymal irritation with formation of astrocytic transependymal granulations and subsequent periventricular gliosis can be observed without associated bacterial infection [13]. Chemical irritants may be etiologic factors. With these observations in mind, it does not appear surprising that septations may develop purely on the basis of intraventricular hemorrhage.

In our series, pseudosepta were produced by converging periventricular cavities in patients with leukomalacia and/or

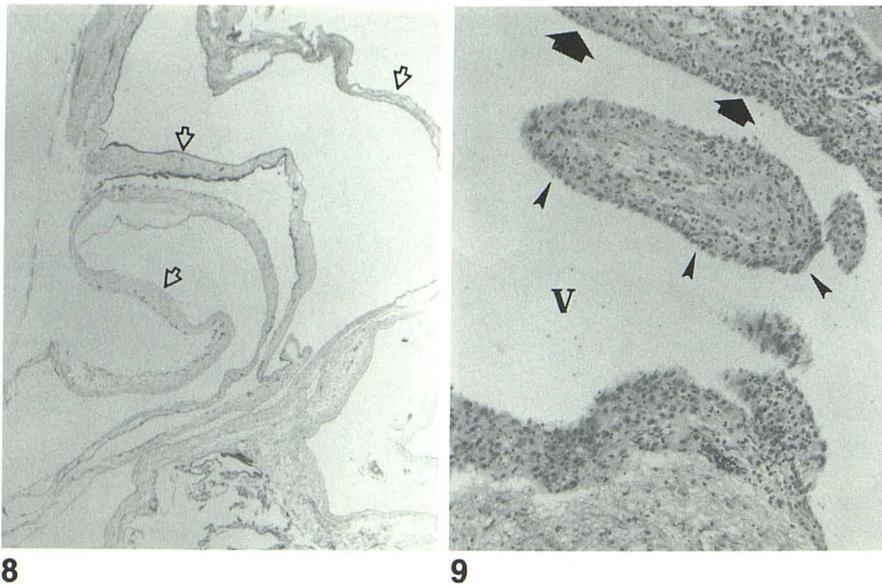


Fig. 8.—Congenital septa in full-term infant who died on second day of life (second death in series). Patient also had agenesis of corpus callosum and encephalocele. (Luxol Fast Blue H and E,  $\times 120$ .) Third and lateral ventricles are subdivided by multiple, thin glial strands (arrows) lined focally by ependyma and/or choroid plexus epithelium. These features appear more consistent with a teratogenic event causing a malformation.

Fig. 9.—Histology of ventricular denudation in premature infant (one of two deaths in our series) with perinatal anoxia and *E. coli* ventriculitis. (Luxol Fast Blue H and E,  $\times 400$ .) Tissue strands, consisting of gliotic tissue and macrophages, seen here on cross section (thin arrowheads), traverse the ventricle (V). The ventricular wall is also composed of a layer of macrophages (wide arrows).

cystic encephalopathies. We have previously reported on this mechanism [14, 15]. Pseudosepta have the same clinical relevance as true intraventricular septations. Pathologically, they have the same composition as true intraventricular septa. Both consist of gliotic strands or sheaths. In the earlier stages, the dissected ependyma itself may act as a floating septum (Figs. 3B and 3C). Sonographically, these pseudosepta tend to appear as solid membranes rather than as finely interwoven, spiderweb-shaped structures, a pattern often observed in true intraventricular septa. More typically, however, these two types of septa are difficult to distinguish.

Congenital septa are probably either teratogenic or acquired during fetal development. Histologic examination in one of the deceased patients in the congenital group showed septa that were covered with ependyma or with choroid plexus epithelium (Fig. 9). This feature appeared more consistent with a dysmorphic event. In others, intrauterine cerebral infection or anoxia may produce septa much like that observed in the neonatally acquired group.

The superiority of sonography over CT in the diagnosis of intraventricular septations is not surprising. The window widths commonly used for standard CT scans are often too large for the recognition of delicate septa. It is possible that meticulous manipulations of window widths and window levels could have improved visualization or salvaged some of the false-negative CT scans. However, in this report the diagnostic impressions were based on standard hard-copy images. The tendency of CT to miss such septa has also been commented on by others [4, 7, 8]. The shortcomings of CT can be overcome by using metrizamide to visualize ventricles [5], but this is considered invasive and is not frequently done. Sonography is the most sensitive method for detecting these septations [7, 8].

Compartmentalization of ventricles by septa can make therapeutic shunting difficult. Shunt revision is frequently re-

quired and bilateral shunting may become necessary. This has been reported previously [2, 3, 5] and is caused by collapse of shunted ventricular compartments around the shunt tip and subsequent occlusion of the shunt openings by proliferated fibrillary astrocytes. Further, loculated ventricular compartments may not communicate, which makes the shunt ineffective. The high incidence (62%) of shunt revision in our series supports these observations. We and others [7] have observed disappearance or partial resolution of ventricular septa and believe that ventricular enlargement causes stretching of the septa. In some, this process may ultimately result in partial or total disappearance of the septa. Timing of shunt placement should take this factor into account.

In summary, we have observed 24 patients with septa. Of these, 17 had acquired septa and seven had congenital septa. Cranial sonography is the diagnostic method of choice. Its serial application can provide important information regarding etiology and management. Pseudosepta are more frequent than true intraventricular septa. Ventricular compartmentalization makes diversionary shunting ineffective and increases the risk of shunt failure.

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