Cavum septi pellucidi and cavum vergae

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Introduction

This article includes discussion of cavum septi pellucidi and cavum vergae, cavum septum pellucidum, and cava septi pellucidi et vergae, and it includes information on their embryology and associated pathologies. The foregoing terms may include synonyms, similar disorders, variations in usage, and abbreviations.

Overview

The septum pellucidum is a non-neural dual membrane structure originating from the medial parts of the cerebral hemispheres. Right and left leaves of the septum are separated by a cavity in fetal life, which gradually becomes thinner and obliterated. Its posterior extension is the cavum vergae. The roof is formed by the corpus callosum and the hippocampal commissure, and posterior and inferior of the septum is formed by the fornix. The midline cavities of the brain are involved in inherited and congenital disorders. Although moderate variations in size are asymptomatic, they are easily picked up by neuroradiological means such as ultrasonography during fetal life. In this way, abnormalities of the cavities may indicate the need for further examinations. The author presents the complex association with pathology in this clinical summary. His past and present work is in the realm of congenital and inherited disorders affecting the morphology of the central nervous system.

Key points

• The cavum of the septum pellucidum is bordered by 2 leaves that fuse during the first year of life.
• The cavum vergae is a posterior extension of the former.
• The leaves of the cavum contain no vital structures, and they normally fuse before 6 months of age.
• Pathologies of the cavum, as detected by MRI, include its persistence, as well as complete absence of the septal leaflets resulting in a single cavity. Both anomalies may form part of complex congenital disorders.
• Pathology of the cava is not harmful in itself but may draw attention to associated pathology in neural structures.
• Increased width of the cavum is nonspecific but may reflect spatial compensation for lack of brain growth in the fetus or brain atrophy in adults.

Historical note and terminology

The septum pellucidum is a thin vertical membrane that connects the corpus callosum to the columns of the fornix and separates the lateral ventricles. The septum has right and left leaves, each of which is part of the respective medial hemispheric border. Sylvius first described cavum septi pellucidi in 1671 (Bruyn 1977). Cavum vergae is a posterior extension of the cavum septi pellucidi, communicating with the cavum septi pellucidi but lying posterior to the columns of the fornix (Verga 1851). In the early literature, these cavities were considered to be the fifth and sixth cerebral ventricles, respectively. They are not actually ventricles because they are not primarily part of the ventricular system, are not lined by cells of the ependyma, and do not contain choroid plexus. The anatomic boundaries of the cavum septi are the medial hemisphere walls on the lateral sides; the roof is formed by the corpus callosum. The cavum septi pellucidi is bordered posteriorly by the column of the fornix. The cavum vergae is bordered anteriorly by the posterior border of the cavum septi pellucidi, inferiorly by the body of the fornix, and superiorly and posteriorly by the corpus callosum. Anatomically, the 2 cavities are not separated. Another cavity, known as the cavum veli interpositi, is part of the leptomeningeal space and intervenes between the roof of the third ventricle and the body of the fornices. {embed="pagecomponents/media_embed" entry_id="7971"} When the corpus callosum is absent, the cavum septi becomes unroofed and, therefore, unidentifiable.

The formation of the cavities was detailed by Rakic and Yakovlev, who analyzed the brains of 113 fetuses and numerous vertebrate species. The human samples covered the crucial period of 13 to 14 weeks during which
commissuration takes place (Rakic and Yakovlev 1968). According to this study, the cavum septi is a part of the leptomeningeal space sealed off by the developing corpus callosum. The septum pellucidum is made of 2 apposed sheaths of tissue derived from the medial walls of the hemispheres. Its lower edge is bordered by the columns of the fornix (Raybaud 2010). The inferior and posterior border of the septum pellucidum is formed by the columns of the fornix, which are part of the limbic system, connecting to the hippocampi. Because of their close anatomic relationship, absence of the septum leads to ectopia of the fornices and absence of the fornical commissure. The lateral, ventricular surface of the septum pellucidum is covered with ependyma; the medial surfaces (when not fused) are poorly organized and appear glial in neonates. The cores of the pellucidal leaves contain small myelinated fibers (Raybaud 2010). Dandy first described cysts of the cavities in 1931 (Dandy 1931).

The midline cavities are essentially temporary embryonic structures that involute during late pregnancy and infancy. Persistence of these structures beyond this period does not cause any symptoms but is statistically related to malformations and psychiatric disturbances, mainly dependent on size (Table 1).

The size of persistent midline cavities is differently measured because size may be measured by antero-posterior extent or by width. The former approach is generally used in MR studies of neuropsychiatric populations, eg, with schizophrenia (Nopoulos et al 1997). The latter approach is usual in younger groups where persistence of the structure itself is rated less significant than increased transverse size. It should be stressed that progressive decrease in width of the cavities during pregnancy and infancy parallels transverse growth of the cerebral hemispheres, and, in this regard, decrease of the cavities may be seen as compensatory to the increase of brain size. A significant correlation has been found between biparietal diameter and size of the cavum septi pellucidi (Jou et al 1998).

Table 1. Midline Cavities In Normal Persons

<table>
<thead>
<tr>
<th>Measurement by US or MRI in controls</th>
<th>Size</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>Width</td>
<td>&lt;9,5 mm</td>
<td>(Mott et al 1992)</td>
</tr>
<tr>
<td>Length</td>
<td>&lt;8,0 mm</td>
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<tr>
<td>Average width</td>
<td>6.3 mm +/- 0.83 (1 sd)</td>
<td>(Jou et al 1998)</td>
</tr>
<tr>
<td>Maximal length</td>
<td>&lt; 6 mm</td>
<td>(Nopoulos et al 1998)</td>
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<tr>
<td>Maximal length (&gt; in 3- 8.7%)</td>
<td>(Born et al 2004)</td>
<td></td>
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<tr>
<td>Maximal length (&gt; in 11.5%)</td>
<td>(Takahashi et al 2008)</td>
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Clinical manifestations

Presentation and course

Persistent and enlarged midline cavities after birth. The cava septi pellucidi and vergae are seen as part of normal development in the fetus and neonate. Persistence of these structures is common throughout infancy. Typically, at 6 months gestation, the posterior half of the fusion is complete. The anterior half does not fuse until after birth, making the incidence of cavum septi pellucidi in premature infants 100% (Larroche and Baudey 1961). Generally, by 6 months of age, the process is complete (Nakano et al 1981), but the fusion is frequently not 100%, as there is often a small cavity near the genu of the corpus callosum (Shaw and Alvord 1969).

{embed="pagecomponents/media_embed" entry_id="7972"} The reported incidence of cavum septi pellucidi in normal adults varies enormously from 0.10% to 85% (Nopoulos et al 1997). Such disparity in prevalence may contribute to the uncertainty regarding the pathologic implications of cavum septi pellucidi. Speculation ranges from cavum septi pellucidi as a normal anatomic variant to a forme fruste of midline developmental anomalies such as dysgenesis of the corpus callosum (Sarwar 1989). The wide variance in reported prevalence may be due to the method of detection (autopsy vs. imaging), as well as the definition of cavum septi pellucidi based on size.

MRI studies indicate that the incidence of small cavum septi pellucidi (less than 5 mm in anterior to posterior length as seen on contiguous coronal MRI slices) is common (59% to 85%) in the normal healthy population (Nopoulos et al 1997; Kwon et al 1998; Hagino et al 2001). This suggests that small cavum septi pellucidi should be considered a normal variant of anatomy. However, enlargement of the cavum septi pellucidi (greater than 5 mm in length) was seen uncommonly in the healthy population (1% to 2.4%) and linked to developmental abnormality. Born and colleagues,
using the evaluation method of Nopoulos and colleagues, studied 3 healthy groups of patients: children, young adults, and elderly adults (Nopoulos et al 1997; Born et al 2004). Cavum septi pellucidi was detected in 80% of the cases in the pediatric group, in 68% of young adults, and in 72% of the elderly adults. A cavum vergae was noted in 22% of the children, in 39% of the young adults, and in 36% of the elderly subjects. There was no significant difference between the age-related groups.

**Association with CNS malformations.** The formation of the septum and the fusion of the septal leaflets are intimately related to the development of other midline structures such as the corpus callosum and medial temporal structures such as the hippocampus. Dysgenesis of the corpus callosum or the limbic system may be signaled by anomalies of the midline cava and septum on fetal ultrasound.

A wide range of cerebral malformations, especially those affecting the cerebral cortex, can be associated with an enlarged cavum septi. This association may be seen together with chromosome abnormalities, eg, 22q11 deletion (Shashi et al 2004; Beaton et al 2010; Schmitt et al 2014; Chaoui et al 2016) or complex congenital malformation syndromes and inborn errors that affect the fetal shaping process, ie, peroxisomal enzyme deficiencies. In an MRI study by Bodensteiner and colleagues, the incidence of enlarged cavum septi pellucidi was dramatically higher in a sample of children and adults known to have mental retardation or developmental delay compared to the incidence of cavum septi pellucidi in a healthy sample (15.3% vs. 2.4%) (Bodensteiner et al 1998). Given the variety of conditions in which enlarged cavum septi pellucidi is found, it is most appropriate to consider this anomaly as a nonspecific marker for disturbed brain development.

**Association with craniofacial disorders.** In Apert syndrome of acrocephalosyndactyly, an abnormal septum pellucidum is a regular feature. In their study on Apert syndrome, Renier and colleagues found that 55% of the cases had an anomaly of the septum (33 of a total of 60 cases) (Renier et al 1996). Agenesis was present in 18 cases and a cavum septi pellucidi in 15 cases. Nopoulos and colleagues performed a study of adult males with nonsyndromic clefts of the lip or palate (Nopoulos et al 2001). In that study, subjects with a cleft had a significantly increased incidence of enlarged cavum septi pellucidi compared to healthy controls (8.0% vs. 1.3%).

**Association with congenital syndromes due to teratogen exposure.** Enlarged cavum septi pellucidi is frequently seen in fetal alcohol syndrome (Johnson et al 1996). In an MRI series of 10 subjects with fetal alcohol syndrome, 50% had either an enlarged cavum septi pellucidi, cavum vergae, or both (Swazy et al 1997). In utero exposure to valproate has been associated with midline brain abnormalities, typically absence of the septum pellucidum (Lindhout et al 1992; McMahon and Braddock 2001); however, other reports indicate the less severe anomaly of enlarged cavum septi pellucidi (Gigantelli et al 2000).

**Association with schizophrenia.** Eight of 11 MRI studies until 2001 found increased prevalence of cavum septi pellucidi in patients with schizophrenia as compared to normal controls (Degreen et al 1992a; Degreen et al 1992b; DeLisi et al 1993; Scott et al 1993; Shioiri et al 1996; Nopoulos et al 1997; Kwon et al 1998; Rajarethinam et al 2001). The remaining 3 studies showed no increased prevalence of enlarged cavum septi pellucidi in schizophrenia (Jurjus et al 1993; Fukuzako et al 1996; Hagino et al 2001). In the study of Nopoulos and colleagues, the incidence of small-sized cava was comparable between patients and controls (Nopoulos et al 1997). However, the patient group had significantly higher incidence of large cavum septi pellucidi (ie, over 5 mm in length: 20.7% in patients vs. 3% in controls). The patients with large cavum septi pellucidi were all male. In addition, a study done on subjects with childhood-onset schizophrenia found that the incidence of enlarged cavum septi pellucidi was similar to those seen in patients with the adult onset of the disorder. However, the severity of the anomaly was worse in the childhood-onset sample; on average, the size of the cavum was much larger than those seen in the adult-onset group (Nopoulos et al 1998). These and other studies apparently support the notion that schizophrenia is a neurodevelopmental disorder with abnormal brain development as the origin of its pathology (Kasai et al 2004). Enlarged cavum septi pellucidi in neuropsychiatric adult disorders, however, may not be unique to schizophrenia, as some studies have suggested that it is also associated with affective disorder (Kwon et al 1998; Kasai et al 2004), though this population has been much less thoroughly studied. Loss of cortical grey matter has been found in several areas of the brain in both schizophrenia and bipolar disorders (Farrow et al 2005). Especially in schizophrenia, extended cortical areas are affected including lateral and medial frontal regions and bilateral posterior temporal lobe regions, with additional extensive losses over time in lateral frontotemporal regions and left anterior cingulate gyrus. Surprisingly until now, enlarged cavum septic
Migration described as part of a syndrome of bilateral full-thickness diagnosis on prenatal ultrasound investigation of the fetal brain. The absence of a septum pellucidum has also been entry_id="7975"} Holoprosencephalic brains lack a septum pellucidum, and this may serve as a marker for the midline cerebral dysgenesis (septum pellucidum, optic nerve hypoplasia, and often schizencephaly associated with absence of the septum pellucidum: Apert syndrome, septo-optic pituitary insufficiency, holoprosencephaly, holoprosencephaly, and fetal alcohol syndrome. The association of congenital absence of the septum pellucidum, optic nerve hypoplasia, and often pituitary insufficiency is known as "septo-optic dysplasia," a midline cerebral dysgenesis (De Morsier 1956; Sarnat 1992). {embed="pagecomponents/media_embed" entry_id="7975"} Holoprosencephalic brains lack a septum pellucidum, and this may serve as a marker for the diagnosis on prenatal ultrasound investigation of the fetal brain. The absence of a septum pellucidum has also been described as part of a syndrome of bilateral full-thickness porencephaly, ie, open-lip schizencephaly and neuronal migration disturbance (Aicardi and Goutieres 1981). Absence of the septum is also seen in closed-lip schizencephaly.

Association with chronic traumatic encephalopathy (CTE). Chronic traumatic encephalopathy was formerly known as "punch drunk" syndrome, or dementia pugilistica, which refers to cognitive changes seen in boxers. The syndrome is not exclusively related to boxers but may present in all sports with liability to repeated head trauma. An exhaustive literature review by Smith and colleagues gives credence to the presumed relationship between enlarged cavum septi and chronic traumatic encephalopathy (Smith et al 2013). Two mechanisms are proposed: (1) general cerebral atrophy leading to compensatory widening of the cavum septi and (2) ventricular dilation and septal changes as a consequence of repeated transient increases in intracranial pressure.

Increased size of cavum septi pellucidi on fetal ultrasound. Increased use of ultrasound in pregnancy and its steady refinement in the screening for fetal disorders has put isolated increase of cavum septi in focus and prompted the question of whether it should be given prominence in the search for broader underlying disorders. Normative data on the size of the cava in normal fetuses during the second and third trimesters are provided by Tao and colleagues (Tao et al 2013). Abele and colleagues retrospectively analyzed the width of septal cava between 18 and 40 weeks in a cohort in which chromosome analysis was done, resulting in 267 euploid and 139 aneuploid cases (Abele et al 2013). In 42% of the fetuses with trisomy 21, cavum septi pellucidi was above the 95th centile. Findings in trisomy 18 and 13 showed the same trend. The authors concluded that the finding of an enlarged cavum septi in the second and third trimesters forms an indication for a search for chromosomal abnormalities. Interestingly, the study also provided evidence that the width of the cavum is linked to the biparietal diameter. Chaoui and colleagues confirmed the increased incidence in fetuses with chromosome 22q11 microdeletion (Chaoui et al 2016).

Absence of detectable cavum septum pellucidi. Prenatal sonography may fail to detect the cavum as the first warning sign of a cerebral malformation. Intrauterine hydrocephalus may destroy the septal lamina, or its presence may be masked by an interhemispheric cyst, which is often seen in association with callosal agenesis. Absence of the cavum may also be a presenting sign of holoprosencephaly (Sundarakumar et al 2015).

Space-occupying lesions associated with cavum septi pellucidi. Hemorrhage into the cavum has been reported in preterm infants with coexisting intraventricular hemorrhage (Butt et al 1985) and in adults secondary to trauma and hypertension (Kanpolat and Mertol 1987). Tumors of the cavum can develop (French and Bucy 1948). Large cava often occur in the brains of children or adults with generalized cerebral atrophy from any cause.

Other space occupying lesions involving the cavum septi are empyema associated with purulent meningitis (Kihara and Miyata 2002; Pong et al 2003).

Recurrence of a previously closed cavum septi has been reported following the placement of an Ommaya reservoir for administration of chemotherapeutic with the tip against the septum (Sherman and Aygun 2005).

Absence of the septum pellucidum. Prenatal sonography may fail to detect the cavum as the first warning sign of a cerebral malformation. Intrauterine hydrocephalus may destroy the septal lamina, or its presence may be masked by an interhemispheric cyst, which is often seen in association with callosal agenesis. Absence of the cavum may also be a presenting sign of holoprosencephaly (Sundarakumar et al 2015). The following categories are known to be associated with absence of the septum pellucidum: Apert syndrome, septo-optic dysplasia, holoprosencephaly, schizencephaly, rhombencephalosynapsis, and fetal alcohol syndrome. The association of congenital absence of the septum pellucidum, optic nerve hypoplasia, and often pituitary insufficiency is known as "septo-optic dysplasia," a midline cerebral dysgenesis (De Morsier 1956; Sarnat 1992). {embed="pagecomponents/media_embed" entry_id="7975"} Holoprosencephalic brains lack a septum pellucidum, and this may serve as a marker for the diagnosis on prenatal ultrasound investigation of the fetal brain. The absence of a septum pellucidum has also been described as part of a syndrome of bilateral full-thickness porencephaly, ie, open-lip schizencephaly and neuronal migration disturbance (Aicardi and Goutieres 1981). Absence of the septum is also seen in closed-lip schizencephaly...
Absence of the septum, in most cases associated with fusion of the fornices, is also seen in association with rhombencephalosynapsis (Barth 2008). The fornices are normally suspended from the inferior edge of the septum. As a result of the absence of the septum, the fornices become displaced or interrupted. As the fornices form part of the limbic system, their complete absence may be expected to cause severe deficiencies. In one group study of patients with septo-optic dysplasia, hippocampal abnormalities were present in a significant proportion and related statistically to the degree of mental deficiency (Riedl et al 2008).

**Prenatal diagnosis of septal abnormalities.** Neuroimaging of intracranial structures using transabdominal ultrasonography has gained significant technological advances. As one report suggests, the cavum septi pellucidi is reliably visible and its size can accurately be quantified. In normal fetuses the cavum septi pellucidi should always be visualized between 18 and 37 weeks with a biparietal diameter of 44 to 88 mm. Failure to observe the cavum septi pellucidi in this interval, or possibly the presence of a large cavum septi pellucidi, may indicate abnormal cerebral development and demands further investigation by fetal MRI. In this way, the finding of an absent septum pellucidum may lead to the diagnosis of the causative malformations (Hosseinzadeh et al 2013). Conversely, absence of the cavum septi pellucidi prior to 18 weeks, or later than 37 weeks, is a normal finding (Falco et al 2000). In a followup study of premature infants, it was found that the time of disappearance of the cavum septi had no relationship with developmental parameters (Needelman et al 2007).

**Prognosis and complications**

Most septal cysts are harmless, and will not expand to become space occupying. Although such cysts are associated with psychiatric and developmental disorders, they rarely present problems by their size.

**Clinical vignette**

A 17-year-old male presented for psychiatric evaluation after developing psychotic symptoms of hallucinations and delusions 4 weeks prior. He was the product of full-term gestation with no complications, with the exception of maternal smoking. According to his mother, the boy met all developmental milestones “within normal limits,” but notably later than his siblings. He was also much more socially isolative and immature compared with his siblings. He did poorly in school and was held back in the first grade due to academic and social nonprogression.

On mental exam he was poorly groomed and had poor eye contact. His thoughts were disorganized, and he admitted to ongoing auditory hallucinations and persecutory delusions. On physical exam he exhibited some minor physical anomalies such as high-steepled palate and curved fifth finger. He also demonstrated some neurologic “soft signs” such as impersistence of lateral gaze and persistence of a developmental reflex (palmomental reflex).

Neuropsychologic testing indicated a full-scale IQ of 77 (borderline intellect range). MRI scan showed a combined cavum septi pellucidi and cavum vergae, indicating complete lack of fusion of the septal leaflets during development.

The patient was diagnosed as having schizophreniform disorder. He was treated with antipsychotic medications and his psychosis improved, but persisted. After 6 months his diagnosis was changed to schizophrenia, undifferentiated subtype.

**Biological basis**

**Etiology and pathogenesis**

Development of the septum pellucidum is intimately connected with development of the corpus callosum. According to the classic study of Rakic and Yakovlev, the cavum septi forms part of a section of the interhemispheric leptomeningeal space, which is sealed off by the developing corpus callosum (Rakic and Yakovlev 1968). This view, now generally accepted, holds that the corpus callosum develops extracerebrally. Raybaud provides an excellent review and summary of the work of these authors (Raybaud 2010). At the time of the outgrowth of the telencephalic hemispheres from the rostral end of the prosencephalon, the anterior midline ridge connecting the developing telencephalic outgrowths is called the lamina reuniens. The hippocampal and anterior callosal commissures and the hippocampal fornices develop within this ridge. The study by Rakic and Yakovlev ended a longstanding dispute on the mode of origin of the corpus callosum (Rakic and Yakovlev 1968). They observed that the embryonic corpus callosum develops within a separate, phylogenetically new structure, which they named the glial sling. This structure is a glial
ridge that develops from the medial parts of the hemispheres as a separate outgrowth, dorsal and anterior to but not forming part of the lamina reuniens. Fusion of these ridges across the midline creates the glial sling that bridges the interhemispheric meningeal space, creating the bed for the future corpus callosum. In this way, a closed space is created in the gap between the corpus callosum and the structures developing from the lamina reuniens. This is the cavum septum pellucidum. It follows from this reasoning that the cavum septum pellucidum is an extracerebral space. The lateral leaves of the cavum septum pellucidum are formed by the medial hemispheric walls, which fuse later in development.

In the normal development of the cava and in cysts of the cava, this space is not in communication with the ventricular system. Interestingly, cavum septi develops as a transient structure during fetal life, which disappears gradually by approximation of its lateral leaves, synchronous with the growth of the frontal lobes. Its persistence is seen in widely different prenatal-onset brain diseases, including acquired fetal disorders. Consequentially, it may be assumed that impaired transverse growth of the brain at the level of frontal lobes causes persistence of the cavum septi irrespective of the nature of its cause (genetic vs. acquired). Sometimes this relative redundancy of space causes enlargement of the interhemispheric distance.

**Epidemiology**

The leaves of the cavum septum pellucidum normally start to fuse at 34 weeks' gestation. Thirty-six percent to 50% of term babies have cavum septi pellucidi by CT and ultrasound (Nakano et al 1981), whereas up to 85% have cavum septi pellucidi by autopsy studies (Bruyn 1977). Ten percent have cavum septi pellucidi at 1 year by most imaging and pathological reports. In adulthood, incidence usually ranges from 3% to 20% (Bruyn 1977).

Cavum vergae is seen in all 6-month fetuses but in only 30% of term infants by postmortem studies (Bruyn 1977). Cavum vergae usually accompanies cavum septi pellucidi, with an incidence in adults of less than 2%. Cavum vergae without cavum septi pellucidi has also been reported (Bruyn 1977; Miller et al 1986).

**Prevention**

Cavum septi is a harmless condition in itself. It forms part of a large and divergent group of disorders with embryo-fetal onset. Prevention should be part of the approach to the respective causal disorders.

**Differential diagnosis**

By ultrasound, cavum septi pellucidi has been confused with the third ventricle and the corpus callosum. By CT, MRI, and postmortem analyses, the distinction among these structures is evident. Cysts of the cavum may appear similar to velum interpositum cysts, ependymal cysts, ventricular loculation, and neuroepithelial cysts. Cysts of the cava may be differentiated from persistence of the cava by the lateral convexity of the septal leaves, scalloping of the inferior corpus callosum, and mass effect on other adjacent structures.

**Diagnostic workup**

Cava septi pellucidi are particularly common in neonates and young infants in whom they should be considered normal. The diagnostic workup depends on clinical context, and in most cases will not be needed. In some cases, cysts of the cavum septi pellucidi or cavum vergae may compress the cerebral aqueduct or the foramina of Monro, resulting in hydrocephalus. Early in the clinical course, such patients may have papilledema and postural headache, which is characteristically bifrontal or bioccipital. Later, mental confusion and gait abnormalities also may be seen. The diagnostic workup of choice is MRI scanning, as the anatomic detail of this technique presents the clearest image of these structures. Specialized techniques to analyze the flow of cerebrospinal fluid through the foramen of Monro, third ventricle, and cerebral aqueduct may be useful in selected patients.

**Management**

Space occupying cysts should be differentiated from benign persistent fetal cava. Space occupying cysts have laterally bulging walls and tend to obstruct the foramina of Monro. Treatment of space occupying septal cysts is by surgical approach, usually by shunting procedures or by fenestration, creating an open connection between the cyst space and the ventricular system (Donauer et al 1986). A neuroendoscopic approach for fenestration has been reported by Donati and colleagues (Donati et al 2003).
**Special considerations**

**Pregnancy**

Asymptomatic septal cysts represent no risk to pregnancy.

**Anesthesia**

Asymptomatic septal cysts represent no risk for anesthesia.

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**References especially recommended by the author or editor for general reading.**

**Former authors**

James Barkovich MD, William Weiss MD (original authors), and Peg Nopoulos MD

**ICD and OMIM codes**

**ICD codes**

ICD-9:
Unspecified anomaly of brain, spinal cord, and nervous system: 742.9

**Profile**

**Age range of presentation**

0-01 month
01-23 months
02-05 years
06-12 years
13-18 years
19-44 years
45-64 years
65+ years

**Sex preponderance**

Male-female, 1:1

**Family history**

none

**Heredity**

Heredity can only be considered when its cause has been established.

**Population groups selectively affected**

none selectively affected

**Occupation groups selectively affected**

None
Differential diagnosis list

third ventricle
corpus callosum
velum interpositum cysts
ependymal cysts
ventricular loculation
neuroepithelial cysts

Associated disorders

No disorders are specifically associated with abnormalities of the midline cava. Abnormal development of the midline cava reflects the impact of disorders of cerebral growth and development.

Other topics to consider

Dementia pugilistica
Ependymal cysts
Head trauma: neurobehavioral aspects
Fetal alcohol syndrome
Septo-optic-pituitary dysplasia