Fetal cerebral ventriculomegaly

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INTRODUCTION — Fetal cerebral ventriculomegaly is a relatively common finding on second trimester obstetrical ultrasound examination. It is clinically important because it can be caused by a variety of disorders that result in neurological, motor, and/or cognitive impairment. Many cases are associated with other abnormal findings, but in some fetuses, ventriculomegaly is the only abnormality [1]. This topic will discuss the prenatal diagnosis and clinical significance of ventriculomegaly and options for management of affected pregnancies.

DEFINITION AND NOMENCLATURE — The atrium of the lateral ventricle is the portion where the body, posterior horn, and temporal (inferior) horn converge. Atrial diameter remains stable between 15 and 40 weeks of gestation [2,3]. Ventriculomegaly is defined as an atrial diameter ≥10 mm, which is 2.5 to 4 standard deviations above the mean depending on the study [2-4]. Ventriculomegaly is generally considered mild if the atrial diameter is between 10 and 15 mm and severe if >15 mm, although some authors use the categories of mild (10 to 12 mm), moderate (13 to 15 mm), and severe (≥16 mm) [1].

Ventriculomegaly is “isolated” when the fetus has no other anomalies, except those that are a direct result of the ventricular enlargement. Many cases that appear isolated prenatally are ultimately found to have other abnormalities, particularly when ventriculomegaly exceeds 15 mm [5-8]. These abnormalities include Chiari malformations, neural tube defects, Dandy Walker malformations, agenesis of the corpus callosum, and genetic syndromes.

Hydrocephalus is the correct term for pathologic dilatation of the brain’s ventricular system from increased pressure, usually due to obstruction. Ventriculomegaly is an appropriate term when dilatation is due to other causes, such as brain dysgenesis or atrophy. Because ventricular pressure cannot be measured prenatally, the two terms are sometimes used synonymously when applied to the fetus. Most commonly, the term "ventriculomegaly" is used when the ventricles are mildly enlarged, and "hydrocephalus" is used when they measure >15 mm.

Ventriculomegaly may or may not be accompanied by macrocephaly (ie, head circumference greater than two standard deviations above the mean).

PREVALENCE — The prevalence of ventriculomegaly is less than 2 percent, but reports vary widely within that range [9]. In one study, eight cases of mild idiopathic lateral ventriculomegaly were detected among 5400 routine prenatal sonographic examinations between 16 and 22 weeks of gestation (prevalence 1 in 675 [0.15 percent]) [10]. This is a reasonable assessment of the prevalence in a general obstetrical population undergoing second trimester prenatal sonography.

Ventriculomegaly is more common in males; the male-to-female sex ratio is 1.7 [11].

PRENATAL DIAGNOSIS

Atrial assessment — The preferred diagnostic technique for assessing ventriculomegaly is ultrasound measurement of the diameter of the lateral ventricles at the level of the atria. The ventricle should be measured in the axial plane, at the level of the frontal horns and cavum septi pellucidi. The calipers are placed at the level of the glomus of the choroid plexus, touching the inner edge of the ventricle wall at its widest part and aligned perpendicular to the long axis of the ventricle [12]. Incorrect placements include middle-middle, outer-outer, and placement that is too posterior in the narrower part of the ventricle or not perpendicular to the ventricle axis.

As discussed above, between 15 and 40 weeks of gestation the ventricular diameter remains stable, with reported means of 5.4 to 7.6 mm and an upper limit of normal of 10 mm [2,6,10,13,14]. The diagnosis of mild ventriculomegaly is made when the ventricular atria measures between 10 and 15 mm or between 10 and 12 mm, depending on the study (image 1). Substantial interobserver variability in interpretation can occur, and is most common at borderline ventricular diameters (ie, about 10 mm) and when other central nervous system (CNS) anomalies are thought to be present [15,16]. Severe ventriculomegaly is diagnosed when the ventricular atria is >15 mm in diameter. Diagnosis of severe isolated ventriculomegaly implies obstruction (hydrocephalus) (image 2).

Qualitative methods can also be used to assess the lateral ventricles for ventriculomegaly. Qualitatively, one looks for:

- The presence of a "dangling choroid," in which the choroid plexus appears to fall toward the dependent ventricular wall [17].
Qualitative methods are subjective and dependent upon the expertise of the sonographer or sonologist. Therefore, qualitative assessment is most useful for determining when measurement of the ventricle is needed, and then quantitative assessment is performed.

Asymmetry — Some degree of asymmetry of the lateral ventricles exists in the human fetal brain, and occasionally this can be appreciated prenatally [21]. By itself, asymmetry is not abnormal when both ventricles are of normal size.

Unilateral ventriculomegaly — Unilateral ventriculomegaly does occur and is occasionally diagnosed prenatally. The etiology and outcome of unilateral and bilateral ventriculomegaly are similar; therefore, counseling and management are generally the same [22].

Differential diagnosis — The normal fetal brain is quite sonolucent and may simulate cerebrospinal fluid, which may incorrectly lead to the diagnosis of ventriculomegaly. Therefore, careful evaluation by an experienced sonologist is important in confirming the diagnosis [23].

CNS lesions that could be misdiagnosed as ventriculomegaly by an examiner inexperienced in neurosonography include holoprosencephaly, hydranencephaly, porencephaly, and various cystic lesions, such as arachnoid cysts. (See “Prenatal diagnosis of CNS anomalies other than neural tube defects and ventriculomegaly”.)

Large isolated choroid plexus cysts may transiently dilate the fetal cerebral ventricles. While data are available on a limited number of such cases, choroid plexus cysts are typically benign and the mild ventriculomegaly in such cases is unlikely to be clinically significant [24]. (See “Prenatal diagnosis of CNS anomalies other than neural tube defects and ventriculomegaly”, section on ‘Choroid plexus cysts’ and “Sonographic findings associated with fetal aneuploidy”, section on ‘Choroid plexus cysts’.)

ETIOLOGY — Causes of ventriculomegaly include idiopathic causes, chromosomal disorders and genetic syndromes, congenital infections, aqueductal stenosis, cortical malformations, and migrational abnormalities. In congenital aqueductal stenosis, the cerebral aqueduct is narrow or consists of several minute channels (figure 1). The narrowing may be developmental (eg, X-linked hydrocephalus with stenosis of the aqueduct of Sylvius) or due to acquired changes, such as fibrosis from infection (eg, cytomegalovirus [CMV], toxoplasmosis), intraventricular hemorrhage, or a mass. Infection can also result in isolated ventriculomegaly due to cerebral atrophy, aqueductal stenosis due to ependymal fibrosis, or communicating hydrocephalus due to inflammation of arachnoid granulations. (See “Hydrocephalus in children: Physiology, pathogenesis, and etiology”.)

OBSTETRICAL ISSUES

Initial work-up — After ventriculomegaly is identified, further management involves identifying whether additional abnormalities (CNS and non-CNS) are present, diagnostic evaluation for the most common causes of ventriculomegaly, and counseling patients about the prognosis and potential pregnancy interventions.

We suggest the following approach:

Comprehensive sonographic evaluation — A physician experienced in the diagnosis of fetal anomalies should perform a comprehensive sonogram to confirm the diagnosis of ventriculomegaly and determine whether other structural abnormalities are present. Associated abnormalities have been reported in 10 to 76 percent of cases [8,25-27]. Identification of these abnormalities helps in determining the cause of ventriculomegaly and the prognosis.

A comprehensive examination requires that additional imaging of the CNS be performed, beyond that included in the basic obstetrical sonogram. Such imaging would include a detailed evaluation of the lateral, third, and fourth ventricles; corpus callosum; thalamus; germinal matrix region; and cerebellum and cerebellar vermis [12].

Because CNS infection can result in ventriculomegaly, it is important to look for characteristic sonographic findings of fetal infection, such as intracerebral and periventricular calcifications, hepatic calcifications, hepatosplenomegaly, ascites, and polyhydramnios. Such findings increase the chance that the ventriculomegaly is related to infection, but even in their absence, we suggest maternal serologic studies and/or polymerase chain reaction (PCR) analysis of amniotic fluid to aid in the diagnosis of suspected infectious etiologies. (See ‘Evaluation for infection’ below.)

In addition, a detailed fetal anatomic survey should be performed to look for non-CNS abnormalities since ventriculomegaly can be a component of several genetic syndromes (eg, trisomy 21). Because ventriculomegaly is a relatively non-specific finding, associated with numerous conditions, careful attention to all fetal anatomic structures, including nonspecific features of aneuploidy, is important. Although some experts suggest a fetal echocardiogram be performed routinely, we do not do this test if the heart appears normal on a detailed fetal sonogram.
Review of medical history — The patient’s history is reviewed for symptoms suggestive of CMV infection and exposure to sources of toxoplasmosis (eg, cats, consumption of undercooked meat) or lymphocytic choriomeningitis virus (exposure to rodents such as hamsters, mice, rats), all of which are infectious causes of ventriculomegaly [28-30]. In addition, possible exposure to Zika virus should be assessed [31,32]. (See "Zika virus infection: Evaluation and management of pregnant women", section on “Fetal ultrasonography”.)

Sporadic cases of ventriculomegaly associated with other viruses have also been reported (mumps enterovirus 71 (EV71), parainfluenza virus type 3, parvovirus B19) [33-36].

- (See "Toxoplasmosis and pregnancy".)
- (See "Cytomegalovirus infection in pregnancy".)
- (See "Aseptic meningitis in adults".)

The family history is reviewed to identify any suggestion of L1 cell adhesion molecule (L1CAM) mutations in the family. This gene plays an important role in nervous system development, including neuronal migration and differentiation. Mutations in the gene cause a variety of X-linked neurological syndromes, most including hydrocephalus. Together, these conditions have been referred to as L1 syndrome, and the phenotype can include agenesis of the corpus callosum, Hirschsprung disease and other intestinal pseudoobstruction, limb anomalies, and agenesis of the corpus callosum. If the family history is suggestive of one of these disorders, or in male fetuses with overt isolated hydrocephalus (eg, the ventricles measure >15 mm), DNA testing for a mutation in the L1 spectrum is warranted and consultation with a geneticist is recommended. We do not perform DNA testing of female fetuses or males with mild (<15 mm) ventriculomegaly because the yield of such testing is likely to be low. (See "Hydrocephalus in children: Physiology, pathogenesis, and etiology", section on ‘X-linked hydrocephalus’.)

Amniocentesis — We offer amniocentesis at ≥15 weeks for determination of the fetal karyotype. In a systematic review including over 1200 fetuses with apparently isolated mild ventriculomegaly at initial presentation, 4.7 percent were found to have an abnormal karyotype [37]. The risk is higher with severe ventriculomegaly or associated abnormalities.

Chromosomal microarray (CMA) should be offered to patients with isolated mild ventriculomegaly and recommended when additional abnormalities are detected. CMA has been recommended in the evaluation of fetal anomalies, including ventriculomegaly, and can be used as an alternative to fetal karyotype, or as a reflex test following a normal karyotype [38,39]. The yield of CMA is likely higher when ventriculomegaly is associated with other abnormalities.

The amniotic fluid is also tested for alpha-fetoprotein and acetylcholinesterase to exclude an occult open neural tube defect, which can often lead to ventriculomegaly.

Evaluation for infection — PCR for CMV and toxoplasmosis should also be obtained when amniocentesis is performed. If the patient declines amniocentesis, or karyotyping has been done previously, maternal serology is used to identify an infectious etiology. However, serology is neither as sensitive nor as specific as PCR on amniotic fluid, thus amniotic fluid PCR is the preferred method of evaluation for infection.

Many cases of ventriculomegaly due to infectious causes will have other sonographic findings, such as intraabdominal or CNS calcifications. However, in some cases, the ventriculomegaly is the first (or only) sonographic feature; therefore, testing for infectious pathogens should be offered in all cases of isolated ventriculomegaly, and strongly recommended if the fetus has other features suspicious for an infectious cause. The overall prevalence of infection is <2 percent, and toxoplasma, parvovirus, and CMV infection are the most common infections detected [40-42].

Magnetic resonance imaging — Fetal magnetic resonance imaging (MRI) can be used to identify underlying CNS abnormalities not detected by sonography [43]. We suggest considering MRI in cases of isolated ventriculomegaly (with a normal karyotype and no clear etiology) because cortical malformations and other potentially significant defects cannot be easily detected with ultrasound (eg, migrational abnormalities and porencephaly) [44] and impact patient counseling and management decisions; however, there is no consensus on the clinical value of this approach [45]. The likelihood of finding another abnormality by MRI depends, in large part, on the quality of the original sonogram and varies from 5 to 50 percent in different series [1,16,43,46-50].

In a large series of 147 fetuses with isolated ventriculomegaly on ultrasound and normal karyotype, MRI confirmed the diagnosis in 122 (83 percent) and detected additional brain abnormalities in 25 (17 percent) [1]. In 12 cases, the severity of ventriculomegaly was reclassified up (10/12) or down (2/12). It has been noted that the width of the ventricle may be slightly larger when measured by MRI as compared with ultrasound [51]. Brain abnormalities were identified in 5/90 fetuses (6 percent) with mild (10 to 12 mm) ventriculomegaly, 4/29 fetuses (14 percent) with moderate (13 to 15 mm) ventriculomegaly, and 16/28 fetuses (57 percent) with severe (≥16 mm) ventriculomegaly. The most common anomaly detected on MRI, but missed on ultrasound, was agenesis of the corpus callosum, which accounted for 11 of the 25 cases. The effects of agenesis of the corpus callosum range from subtle or mild to severe, depending on associated brain abnormalities.

Confirmation of isolated mild ventriculomegaly increases the likelihood that long-term neurodevelopment will be normal, and identification of other CNS malformations makes it more likely that the fetus will have neurologic abnormalities, including developmental delay. However, precise data on outcomes in such cases are not yet available.
Magnetic resonance imaging (MRI) is typically performed at 22 to 24 weeks of gestation because developmental milestones become more evident as gestation advances. MRI is not useful in cases of aneuploidy, when the neurologic outcome is almost certainly abnormal regardless of imaging, but may assist in determining the extent of destructive injury in fetuses with known infection, hemorrhage or ischemia, and when other sonographically evident CNS malformations, such as agenesis of the corpus callosum, are present.

Follow-up — Follow-up ultrasound examinations are obtained to look for regression or progression of ventriculomegaly, and to reevaluate for anomalies. Early isolated mild ventriculomegaly may resolve by the third trimester [25,27,52]; progression occurs in 16 percent of cases and has been associated with a worse outcome [11,50] (see ‘Outcome’ below). Follow-up ultrasounds have detected fetal abnormalities not detected on the initial scan in 13 percent of cases [11]. Therefore, at least one additional detailed ultrasound examination should be performed between 28 and 34 weeks of gestation to look for CNS and non-CNS abnormalities and regression or progression of dilatation.

Antenatal fetal testing has no proven benefit in pregnancies with isolated fetal ventriculomegaly in the absence of other findings, such as intrauterine growth restriction or oligohydramnios.

Few data exist to support a specific management plan in those patients who continue their pregnancies. When severe ventriculomegaly is present, elective preterm delivery has been proposed so a procedure can be performed to alleviate pressure on the developing brain and avoid progressive brain damage. However, we do not suggest early delivery, given there is no evidence that it is of benefit.

Intrauterine treatment with ventriculooamniotic shunting was performed in the 1980s. A series including 44 fetuses reported a procedure-related death rate of 10 percent, perinatal mortality rate of 17 percent, and moderate-to-severe handicaps in 66 percent of the survivors [53]. The expert consensus at that time was that these results did not represent an improvement in outcome over expectant management, which led to a de facto moratorium on such procedures [54]. Subsequently, some investigators have questioned whether improvements in prenatal diagnosis might allow better selection of those fetuses most likely to benefit from in utero shunting. At present, however, such procedures are investigational.

Outcome — There is wide variation in outcome of infants with prenatally diagnosed ventriculomegaly, although most children, particularly those with isolated mild (10 to 12 mm) ventriculomegaly, will have a normal outcome [22,37]. Survival rates are more guarded and the outcome among survivors is more likely to be abnormal when ventriculomegaly is more severe or other anomalies are present. For example, in a series of 253 cases of ventriculomegaly (isolated and non-isolated), survival rates were 93 percent when the ventricular measurement was 10 to 12 mm, 84 percent with measurements >12 mm, and 60 percent when other anomalies were present [55]. In a series of 78 fetuses with isolated ventriculomegaly, survival rates for mild (10 to 12 mm), moderate (12 to 15 mm), and severe (>15 mm) cases were 98, 80, and 33 percent, respectively [25]. Other series of fetuses with apparently isolated ventriculomegaly reported neonatal mortality in 3 percent with mild to moderate ventriculomegaly and in 16 percent with severe ventriculomegaly [8,27].

Neurological, motor, and cognitive impairment are more likely when associated anomalies are present (either CNS or non-CNS), with early onset, with persistence or progression of ventriculomegaly, and with more severe ventriculomegaly [25,56,57]. In one series, normal outcome rates for mild, moderate, and severe ventriculomegaly survivors were 93, 75, and 63 percent, respectively [25]. In a 2014 systematic review including 20 studies and over 700 cases of postnatally confirmed isolated ventriculomegaly between 10 and 15 mm, the overall prevalence of developmental delay was 7.9 percent (95% CI 4.7-11.1 percent) at a median age of 30 months [57]. Variations in reported rates of developmental delay are related, at least in part, to the thoroughness of neurodevelopmental evaluation, length of follow-up, and inclusion or exclusion of fetuses with other abnormalities. The authors of the meta-analysis noted that postnatal imaging showed previously undiagnosed abnormalities (false negatives) in 7.4 percent (95% CI 3.1-11.8 percent) of cases.

Prognosis has also been assessed according to progression, stability, or resolution of the ventriculomegaly. Among 106 live born infants followed in one series, ventriculomegaly increased in utero in 19, remained unchanged in 37, and improved or disappeared in 50. Normal outcomes were observed in 46 of the 50 fetuses (92 percent) in whom ventriculomegaly improved versus 13 of the 37 fetuses (35 percent) with stable ventriculomegaly and 4 of the 19 fetuses (21 percent) with worsening ventriculomegaly. Overall, in utero progression was associated with an adverse outcome in 44 percent of cases, while only 7 percent of the no-progression group had an adverse outcome [11].

Many neurodevelopmental disorders associated with mild enlargement of the lateral ventricles are thought to have origins in prenatal brain development [11]. However, relatively little is known about long-term (eg, school age) outcome of children following prenatal detection of mild ventriculomegaly. The few studies that have evaluated neurodevelopmental outcome in these children have found that about one-third have some degree of neurodevelopmental abnormalities that are generally mild. In one small series of nine children who had mild, prenatal ventriculomegaly (10 to 13 mm), 33 percent had nonverbal learning disabilities at ages 6 to 9 years [58]. Another study that evaluated children aged 24 to 77 months noted mild neurodevelopmental impairment in 9 of 25 (36 percent) children [59]. Finally, in a study evaluating postnatal MRI findings and neurodevelopmental outcomes in 28 children and 56 matched controls at ages 1 to 2 years, children with prenatal isolated mild ventriculomegaly had significantly larger lateral ventricle volumes, increased intracranial volumes, and increased gray and
white matter volumes [60]. Children with mild ventriculomegaly also had evidence of delay in fine motor and expressive language skills.

**Counseling** — The range of potential outcomes should be discussed with the family. If the etiology of ventriculomegaly has been determined (eg, trisomy, CMV) or associated malformations are identified, the parents can be given more specific information than in cases where counseling is only based on ventricular width (see 'Definition and nomenclature' above). Before viability, pregnancy termination is an option and should be offered. In those patients who elect to terminate, evaluation to confirm or determine the etiology is warranted, as identifying a cause can be helpful in determining recurrence risk in future pregnancies.

**Delivery management** — Ventriculomegaly may or may not be accompanied by macrocephaly. Most infants with ventriculomegaly have a normal head circumference; therefore, there is no increased risk of cephalopelvic disproportion and cesarean delivery is not required except for standard obstetric complications.

Cesarean delivery is indicated in cases in which the head circumference is increased and vaginal delivery is thought not to be possible. The cut-off for determining when a cesarean delivery is indicated will vary with gestational age at delivery, the absolute and relative head circumference (HC) and the size of the maternal pelvis. When the HC exceeds 40 cm, abdominal delivery should be considered. Pelvimetry is unlikely to be of benefit and is generally not used in this setting.

Cephalocentesis, which almost always results in fetal death, is rarely used to decompress the head, allow vaginal delivery, and avoid maternal morbidity from cesarean delivery. We suggest its use in cases in which the neurological prognosis is so dismal (eg, trisomy 13 or 18 or lethal co-existent anomalies) that, if the fetus does not succumb from the procedure, the outcome will not be substantially changed. From an ethical standpoint, the physician's beneficence-based obligation to the pregnant woman allows her to avoid the increased risks of cesarean delivery in cases in which such management would be of little benefit to the fetus [13].

Cephalocentesis is performed by making an incision at the base of the skull with a sharp instrument (eg, curved Mayo scissors), inserting a cannula, and then applying suction internally to collapse the calvarium [81].

**RECURRENT RISK AND GENETIC COUNSELING** — Couples with an affected child should receive genetic counseling and thorough evaluation because sometimes a generic diagnosis of congenital ventriculomegaly may indicate a more complex diagnosis with significant genetic implications. As an example, patients at risk for X-linked hydrocephalus spectrum should be offered DNA diagnosis, as the recurrence risk is high (50 percent in males). In cases of isolated ventriculomegaly in which a precise cause is not determined, the recurrence risk is in the range of 4 percent, similar to that of other multifactorial disorders [14]. For cases of mild, isolated ventriculomegaly in which the infant is normal at birth and no associated abnormalities were identified, a complete postnatal genetic work-up is not indicated in most cases.

We recommend detailed ultrasound examination at 18 to 20 weeks of gestation in a future pregnancy to look for recurrence. However, it has been noted that in some cases ventriculomegaly may develop late in gestation or after birth, so a normal mid-trimester ultrasound does not definitively rule out this diagnosis. As an example, mid-trimester sonography will not detect all fetuses with X-linked hydrocephalus [62]. For some diagnoses, such as polymicrogyria, fetal MRI is appropriate, as it may detect these relatively subtle brain abnormalities not diagnosable by sonographic examination.

**SOCIETY GUIDELINE LINKS** — Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "Society guideline links: Prenatal screening".)

**SUMMARY AND RECOMMENDATIONS**

- In the second or third trimester of pregnancy, the diagnosis of fetal ventriculomegaly is based upon ventricular dilation ≥10 mm. The diagnosis of hydrocephalus is reserved for those cases where obstruction is visualized or can be inferred. (See 'Definition and nomenclature' above.)

- We suggest the following diagnostic evaluation when fetal ventriculomegaly is detected: (see 'Obstetrical issues' above):
  - A comprehensive fetal sonogram to look for associated anomalies.
  - A detailed family and medical history to look for possible genetic or infectious causes of ventriculomegaly.
  - Fetal karyotype; chromosomal microarray should be offered to patients with isolated mild ventriculomegaly and recommended when additional abnormalities are detected.
  - Testing for infectious etiologies (cytomegalovirus [CMV] and toxoplasmosis) ideally by polymerase chain reaction (PCR) on amniotic fluid or, alternatively, by maternal serology. Testing for Zika virus or other infections may be considered if risk factors or specific exposures are present.
  - Consideration of fetal magnetic resonance imaging for isolated ventriculomegaly in which the etiology is unexplained and additional information would impact counseling and management decisions.
  - A follow-up ultrasound examination to assess progression or regression.
Most children with isolated, mild ventriculomegaly have a normal outcome. The risk of abnormal outcome increases with the severity of ventriculomegaly, progression of ventriculomegaly, and presence of other anomalies. (See 'Outcome' above.)

Fetuses with a normal head circumference may undergo vaginal delivery with cesarean delivery reserved for the usual obstetrical indications. (See 'Delivery management' above.)

Recurrence risk ranges from 4 to 50 percent, depending upon the cause. (See 'Recurrence risk and genetic counseling' above.)

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REFERENCES


Mild fetal ventriculomegaly

Image of fetal head demonstrating mild ventriculomegaly. The ventricle is measured in the axial plane, at the level of the frontal horns and cavum septi pellucidi. The calipers are positioned at the level of the internal margin of the medial and lateral walls of the atria, at the level of the glomus of the choroid plexus, on an axis perpendicular to the long axis of the lateral ventricle.

Courtesy of Mary E Norton, MD.

Graphic 68701 Version 2.0
Fetal hydrocephaly at 21 weeks of gestation

All four images were obtained using transvaginal sonography.
(A) Posterior coronal section showing the very large posterior horns of the lateral ventricles. In the posterior fossa a relatively normal appearing cerebellum (between arrows) and the cisterna magna are seen.
(B) A more anterior section than A and also showing dilation of the lateral ventricles (Lv).
(C) An axial section showing the large ventricles, the dangling choroid plexus, and the very thin cortical mantle.
(D) Oblique section showing the hydrocephaly.

T: thalamus; CP: choroid plexus.

Courtesy of Ana Monteagudo, MD.

Graphic 78382 Version 3.0
Direction of flow of cerebrospinal fluid

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