Sonographic Signs of Fetal Neural Tube and Central Nervous System Defects

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Sonography may provide prenatal diagnosis of neural tube defects and has become a routine part of prenatal care. Neural tube defects include malformations of the brain and spine. Among congenital anomalies, neural tube defects are second in incidence following cardiac defects. Laboratory tests combined with sonography are often able to diagnose the failure of the neural tube to fuse. Specific sonographic signs provide direct or indirect evidence of a neural tube defect.

Key words: neural tube, central nervous system, embryology, anencephaly, encephalocele, spina bifida, prenatal laboratory values, microcephaly, Arnold-Chiari, hydrocephalus, holoprosencephaly, folic acid

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The failure of the neural tube to fuse in the third and fourth weeks of gestation results in several malformations, with the most common manifestations in anencephaly, encephalocele, and spina bifida. An essential part of the obstetric sonogram is the evaluation of the spine and brain structures. This literature review includes etiology, normal formation of the spine and brain structure, and specific abnormalities and their sonographic appearance.

The central nervous system (CNS) was one of the first systems evaluated with sonography, with anencephaly being the first defect diagnosed prenatally. Among congenital defects, neural tube defects are second only to cardiac defects in incidence. Approximately half of infants with neural tube defects abort before diagnosis. One-quarter of the remainder are stillborn and one quarter result in live births. This high mortality rate underscores the importance of early detection of neural tube defects to provide prospective parents time to plan their pregnancy.

TABLE 1
Central Nervous System (CNS) Sonographic Features of Selected Syndromes

Syndrome	Sonographically Detectable CNS Defects
CHARGE	Dandy-Walker malformation, holoprosencephaly
HARD	Encephalocele, hydrocephalus
Jaracho-Levin	Myelomeningocele
Mekel-Gruber	Occipital encephalocele, anencephaly, Arnold-Chiari malformation, hydrocephalus, Dandy-Walker malformation, cerebral hypoplasia, cerebellar hypoplasia, absence of corpus callosum
Median cleft facial	Anterior encephalocele
Trisomy 13	Ventriculomegaly, abnormal posterior fossa, holoprosencephaly, Dandy-Walker malformation
Trisomy 18	Choroid plexus cysts, cerebellar hypoplasia, enlarged cisterna magna, myelomeningocele, strawberry-shaped head, Dandy-Walker malformation
Trisomy 21	Choroid plexus cysts, ventriculomegaly, Dandy-Walker malformation
VATER/VACTERL	Spinal dysraphia, occipital encephalocele, hydrocephalus

This collection of defects lists only the CNS malformation. Many syndromes encompass multiple organ systems, including genitourinary, cardiac, skeletal, lymphatic, facial, gastrointestinal, and respiratory.

Teratology of the Spine and Brain

Sonography is a valuable part of modern prenatal care. Most examinations performed are normal; however, population groups have an increased incidence of birth defects. Individuals predisposed to chromosomal abnormalities or exposed to teratogens have an increased risk of CNS malformations. Teratology, the study of abnormal development and causes of congenital malformations, helps establish the genetic, environmental, and multifactorial causes of birth defects.

Chromosomal abnormalities begin at the basic building block of the cell, the DNA. Each individual has 22 homologous pairs of chromosomes, with a paired XX in a female and a paired XY in a male. Changes in this number result in chromosomal abnormalities of aneuploidy or polyploidy. Table 1 lists some known syndromes that have associated CNS defects.

A teratogen is any agent that causes or raises the statistical average of a given defect. Environmental factors include drug-induced, maternal, and viral causes. The link between drugs and neural tube defects has been solidly established through multiple studies. Other factors that increase the risk of malformations include diabetes, vitamin deficiencies, and maternal family history of neural tube defects. One factor to always remember is that less than 10% of neural tube defects link to a family history. For a partial list of environmental and maternal teratogenic causes, see Table 2.

Multifactorial etiology includes both environmental and genetic components. The multifactorial premise is that a birth defect occurs when some environmental component influences the genetics of the individual causing a malformation. The defect does not occur until an environmental threshold is reached and surpassed. Multifactorial etiology includes those in which the defect occurs as an isolated malformation and those presenting with the defect as part of a syndrome.

Studies of multifactorial inheritance patterns highlight the increased incidence of certain defects within a population. Geography plays some role in the incidence of neural tube defects, with the highest incidence in the United Kingdom. These studies resulted in the connection between folate levels and neural tube defects. Mothers taking a daily multivitamin supplement with folic acid showed a sharp decrease in the incidence of neural tube defects.[1] This resulted in the recommendation of adding supplements and folic acid–enriched food to the maternal diet at least one month before becoming pregnant.[2] Timing is an important component in the efficacy of folate supplements as neural tube development occurs within the first few weeks after fertilization.

TABLE 2
Central Nervous System (CNS) Sonographic Features for Selected Teratogens

Teratogens	Sonographically Detectable CNS Defects
AIDS	Microcephaly, lateral skull bossing
Alcohol	Microcephaly, Dandy-Walker malformation
Coumadin	Dandy-Walker malformation, agenesis of corpus callosum
Cytomegalovirus	Mild ventriculomegaly, microcephaly, brain atrophy, Dandy-Walker malformation
Fifth disease (Parvoviris B19)	Hydrocephalus
Maternal diabetes	Sacral agenesis, absence of vertebrae (spina bifida), Dandy-Walker malformation
Radiation—high levels	Microcephaly
Rubella	Microcephaly, Dandy-Walker malformation
Thalidomide—rare	Anencephaly, myelomeningocele
Toxoplasmosis	Microcephaly, hydrocephalus, encephalomyelitis, intracranial calcifications, cerebral atrophy
Valproic acid	Spina bifida, microcephaly
Varicella	Ventriculomegaly or atrophy, microcephaly

This collection of defects lists only the CNS malformation. Many teratogens encompass multiple organ systems, including genitourinary, cardiac, skeletal, lymphatic, facial, gastrointestinal, and respiratory.

Embryology

Determination of the age and developmental stage of an embryo became the life's work of embryologist Franklin P. Mall. Before imaging techniques, the only method to determine embryonic age was documentation of the last menstrual period (LMP) or the size of the aborted products of conception. These did not prove accurate because the time of fertilization could not be determined, and preserving fluid caused a specimen to shrink to as much as half of the original size.

Mall began collecting embryos in 1887, usually from aborted fetuses; however, some were the result of routine autopsy findings done at the Johns Hopkins Hospital. Mall focused his research on embryology and the resulting organogenesis and physiology in the adult after being appointed as the first professor of anatomy at the school of medicine at Johns Hopkins University. His focus on the first eight weeks of development caught the attention of the Carnegie Institute of Washington, and he received a grant in 1913 to continue embryonic studies.

Because dates and size were inaccurate predictions of embryo age, Mall developed a classification based on the development of organ systems. This classification became the 23 Carnegie stages that divide embryonic development by the appearance of specific anatomy such as the neural tube. The process of documenting the various stages of embryonic development required many diverse talents. Not only were slides prepared of specimens, but photographs, drawings, and wax models were developed. Currently, the Carnegie Collection of Human Embryos containing Mall's specimens is housed at the National Museum of Health and Medicine in Washington, D.C. Carnegie stages became the international standard to classify and describe embryos.[3,4]

Neuralization, the formation of the neural tube, begins early in Carnegie stage 10 (22-23 days) and fusion is complete within 3 to 4 days (Fig. 1). The brain development begins in stage 13 (day 28) and continues throughout gestation. This early development often occurs before the mother is aware of the pregnancy, underscoring the importance of preemptive nutrition, vitamin supplementation, and avoidance of teratogenic substances.

The anatomic variations seen with spina bifida occur early in development. Failure of the caudal neuropore to fuse at the end of Carnegie stage 13 (fourth week) results in a characteristic skin dimple seen in the sacral area. This separation is due to the neural tube and surface ectoderm failing to fuse. This indication of a minor developmental disruption often occurs with spina bifida occulta. Many degrees of spina bifida occur, with the most severe including the spinal cord due to the failure of neural folds to fuse.

Brain abnormalities are the result of the rostral neuropore failing to fuse. Exencephaly and

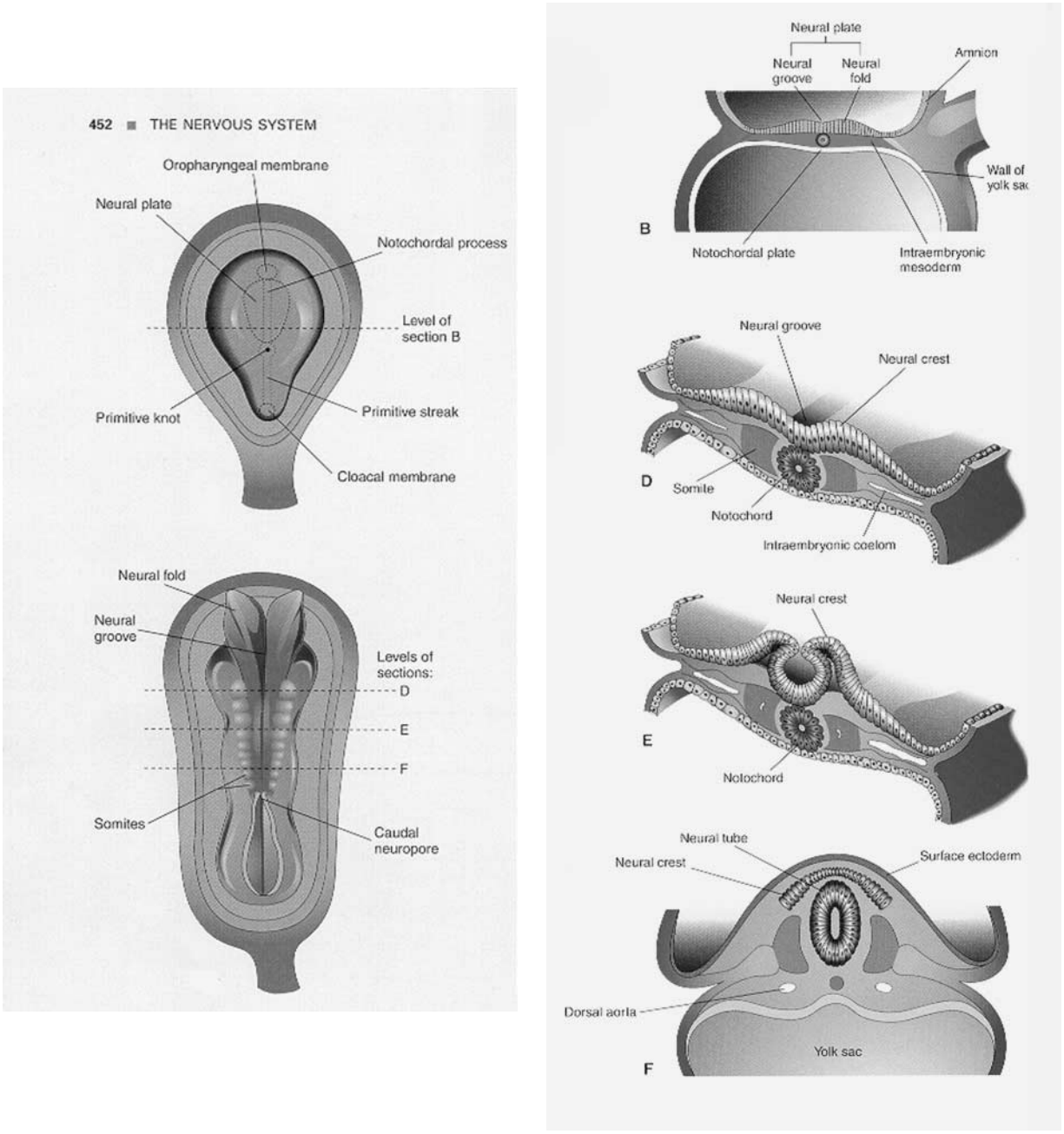

FIG. 1. During Carnegie stage 10 (approximately 22 days), the neural groove begins to fuse. Transverse views of the embryo (diagrams D, E, F) demonstrate the fusion, with the caudal end fused. Neural crest cells (level E) surround the neural groove (level D), resulting in a normal neural tube. Failure to fuse at this stage results in a neural tube defect. Brain anomalies occur, with failure of the rostral neuropore to close (located opposite the caudal neuropore within the neural groove). From Moore K, Persaud T: *The Developing Human: Clinically Oriented Anatomy*, 6th ed., New York, Elsevier, 1998, p 452. Reprinted with permission from Elsevier.

meroanencephaly are the anatomic manifestation of this fusion failure within the fourth week of development. The resultant defective forebrain is outside the skull, resulting in degeneration. Holoprosencephaly, microcephaly, or hydrocephalus, on the other hand, is due to the disruption of development due to teratogen exposure in the third week of gestation (Carnegie stage 7).

What the Lab Values Mean

Many times, the indication for an initial second trimester sonogram is an abnormal triple screen. This indication alone does not necessarily aid the sonographer in performing the screening examination. The most common cause for abnormal results is inaccurate dates followed by the possibility of a multiple gestation and placental hemorrhage. What exactly do the laboratory values mean, and why do we need to know which portion is abnormal?

Routine triple screening has become a regular part of prenatal care. This biochemical test assigns a value to α fetoprotein (AFP), human chorionic gonadotropin (hCG), and unconjugated estriol levels found in maternal blood. These laboratory tests, performed in the 16th to 18th weeks of gestation, include factors such as maternal weight, ethnicity, and diabetes. The use of these three laboratory values increases recognition of trisomy 21 when the hCG has a high value and AFP and estriol levels are below normal. On the other end of the spectrum, a low hCG, AFP, and estriol level raises suspicion for the presence of trisomy 18 and 13. The triple screen itself does not diagnose birth defects, but abnormal values raise suspicion for the possibility of specific groups of malformations.

Pregnancy-associated plasma protein A (PAPP-A) is a serum marker used in the first trimester to detect fetal malformations. This glycoprotein begins with the trophoblastic tissues of the developing placenta and becomes evident in maternal blood in the first trimester. Normal values increase throughout the pregnancy, but low or decreased levels indicate the possibility of aneuploidy such as trisomy 13, 18, and 21.

The AFP levels are of greatest interest when determining the risk of fetal neural tube malformations. Although not exclusive to defects such as anencephaly or spina bifida, neural tube defects are the leading cause of an abnormal AFP value. This fetal protein begins with its production in the yolk sac and fetal liver, with the spine, gastrointestinal tract, and kidneys also containing AFP. Normal production and circulation of AFP begin through secretion by the liver, and then it is secreted into the amniotic fluid via the kidneys, swallowed by the fetus, and filtered by the placenta and released into the maternal circulatory system. The resulting maternal AFP values are the familiar MSAFP values used in early screening.

Different laboratory techniques and manufacturers make it difficult to establish an absolute abnormal value for MSAFP levels. This resulted in the routine reporting of the AFP levels in multiples of the median (MoM). The median is the midpoint of an established laboratory value, and in the case of MSAFP, a result 2.0 to 2.5 times higher than the median is a red flag for exposure of spinal or brain tissues to amniotic fluid. MSAFP levels are directly related to the amount of neural tissue exposed to amniotic fluid and higher levels are associated with more severe defects.[3,5–8]

Selected Congenital Malformations of the Spinal Cord and Brain

Birth defects rarely occur as a single entity and encompass multiple organ systems. The easily identified brain and spinal cord constitutes a routine portion of the sonographic obstetric examination. Because sonography can image malformations of these structures, clinicians and patients are able to plan the course of medical care during a high-risk pregnancy. The following are some samples of the more common forms of neural tube defects imaged by the sonographer.

SPINA BIFIDA OCCULTA TO MYELOSCHISIS

Varying degrees of spinal cord and/or meningocele protrusion through a spinal defect are known by the general term *spina bifida*. This absence or hypoplasia of one or more vertebrae develops with an associated cyst-like sac occurring in 1:1000 live births. Areas of the British Isles have an increased incidence (4.1:1000) of this spinal defect, underscoring the importance of geography in the presentation of spina bifida.

The mildest form of this type of spinal defect is spina bifida occulta. Fusion failure of the posterior arches of the vertebral arches does not result in bulging membranes and spinal cord. Most individuals are asymptomatic, with a possible dimple or tuft of hair over the area of nonfusion.

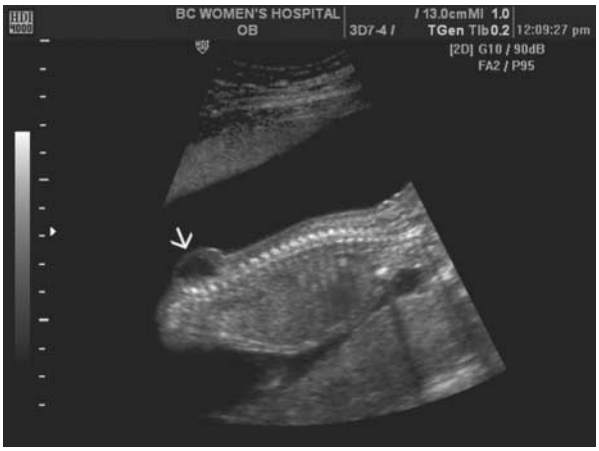

FIG. 2. Spina bifida with meningocele images with a sac containing cerebrospinal fluid and meninges (arrow) projecting through a sacral defect.

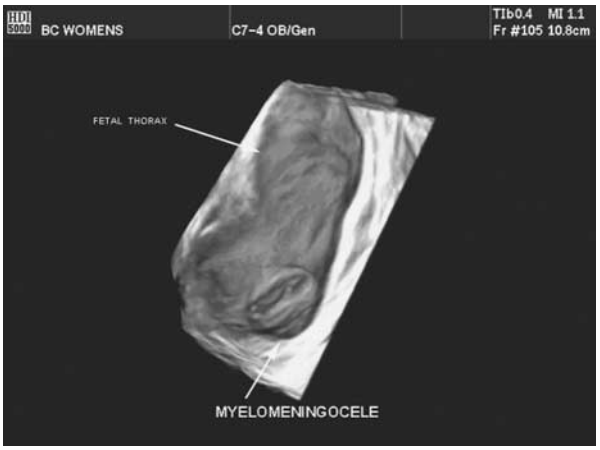

FIG. 3. New technologies, such as 3D, aid in diagnosing the myelomeningocele containing the spinal cord and/or nerve roots within the sac by allowing removal of obstructing information.

Establishing the level of the spinal defect determines the type of function inferior to the malformation. Nerve fiber impairment, due to nervous tissue incorporation into the sac, results in neurological deficits of varying degrees. The most common site, the lumbar and sacral region, results in sphincter paralysis, saddle anesthesia, and decreased limb movement.

Total failure of the neural tube to fuse results in the most severe type of spina bifida. Myeloceles exhibit a flattened mass of nervous tissue in place of the spinal cord. This defect is due to the failure of the caudal neuropore to fuse in the fourth week of gestation (Carnegie stage 23).

Sonography images the spinal defect easily if the fetus is in a position with amniotic fluid surrounding the protruding mass (Figs. 2-3). Scanning the spine requires longitudinal images to document the parallel spine and transverse to check for fusion of the vertebral arches. A sagittal view of a spine with dysraphism images a widened area indicating the defect. A transverse image shows splaying of the vertebral arches due to lack of fusion. Often, associated brain and cranial abnormalities give clues to the possibility of a spina bifida malformation. Coexisting malformations include the lemon head sign of flattened frontal bones, cisterna magna obliteration, a banana-shaped cerebellum, and ventriculomegaly.

ARNOLD-CHIARI MALFORMATION AND SPINA BIFIDA

Spina bifida is often difficult to image due to the fetal position, oligohydraminos, or size of the spinal defect. Due to these limitations, indirect cranial signs either raise suspicion or establish normalcy of the fetal spine. The most consistent sign is a thinned or obliterated cisterna magna due to posterior fossa compression seen with Arnold-Chiari malformations. Confirmation of a normal cisterna magna suggests the absence of a spina bifida. Rarely does an Arnold-Chiari malformation exist without a spinal defect.

The Arnold-Chiari malformation encompasses the herniation of the cerebellar vemis through the foramen magnum. The fourth ventricle and tentorium is displaced inferior into the neural canal. Due to the low position of the brain structures, foramina of Magendie, distenmatomelia, and syringomyelia, obstructive hydrocephalus develops. Three types of the Arnold-Chiari malformation have classifications depending on the severity of brain displacement. Type I is often an incidental finding on a computed tomography (CT) or magnetic resonance imaging (MRI) scan with only downward displacement of the cerebellum. The fourth ventricle remains in the posterior fossa in the normal position. Type II is the malformation seen

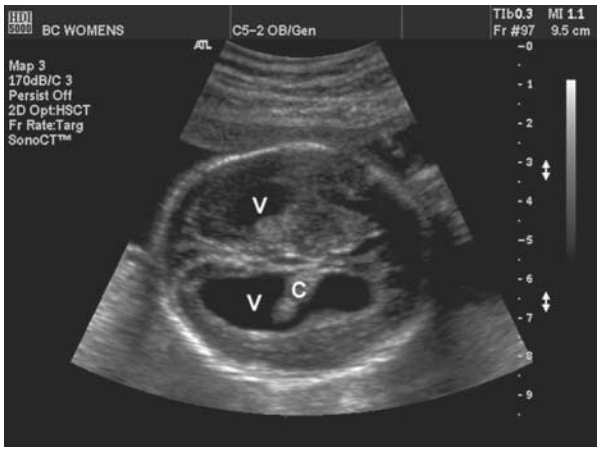

FIG. 4. Ventricular enlargement (V) seen in Arnold-Chiari type II malformation results in a thinned cortex of the brain. A dangling choroid (C) images in the dependant ventricle.

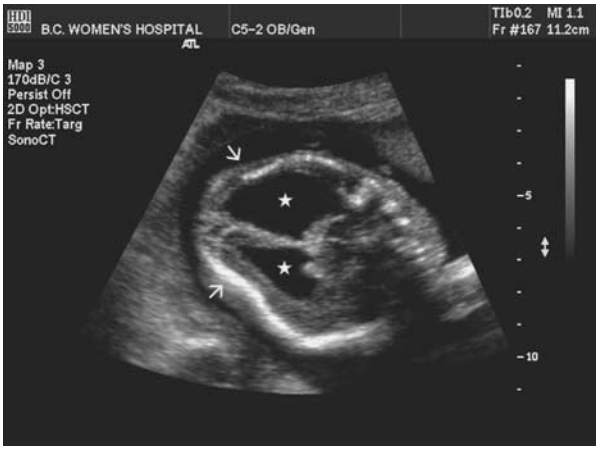

FIG. 5. The misshapen temporal region has a lemon shape, known as the lemon sign (arrows), in this fetus with dilated anterior horns (stars).

prenatally and associated with myelomeningocele, hydrocephalus, and displacement of brain structures (Fig. 4). The most severe form, type III, includes herniation of the posterior fossa contents, hydrocephalus, and a myelomeningocele.

The sonographic examination images several "signs" that raise suspicion for this brain malformation. One of the most important is the appearance of the cerebellum. Due to the downward displacement of brain structures, the cerebellum becomes lumpy, loses the normal vermian notch, and becomes banana shaped (banana sign). Another sign of an Arnold-Chiari type III malformation is the presence of lemon-shaped cranium due to temporal bone malformations (Fig. 5).

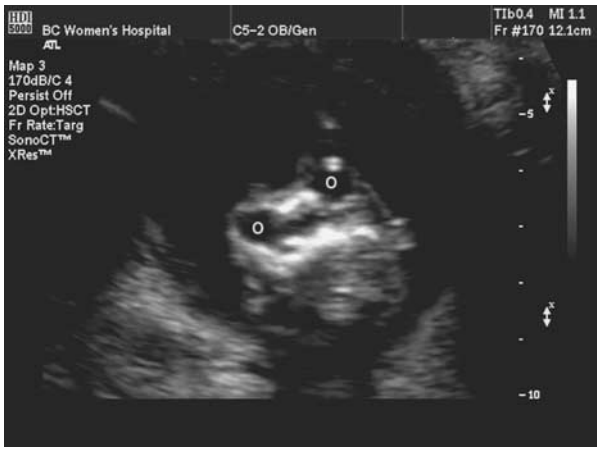

FIG. 6. Degeneration of the fetal brain occurs in several stages, leading to the renaming of the imaged anomaly as meroanencephaly. Due to the brain remnants that remain in the fetus, anencephaly incorrectly describes the malformation.[3] Notice the lack of cranium and brain tissue above the orbits (O).

EXENCEPHALY VERSUS ANENCEPHALY

Anencephaly, the absence of brain tissue above the brain stem, was the first neural tube defect diagnosed with abdominal sonography. Research throughout the years has resulted in the production of this abnormality in laboratory animals with high doses of vitamin A and other teratogens. The developmental sequence of events that occurs with the resultant anencephaly is a three-part process. Early in development, the failure of the rostral end of the neural groove to fuse results in dysraphia occurring within the first two weeks after implantation (Carnegie stage 5). Exencephaly in the embryonic period is the exposure of the brain to the amniotic fluid due to a lack of cranial development. If imaging occurs while the brain is still intact, the head will have a characteristic bilobed appearance termed the "Mickey Mouse" head.[9] Images of this stage are rare due to the quick disintegration of brain tissue after exposure to amniotic fluid. The living infant diagnosed with anencephaly will retain a rudimentary brain stem and has a life expectancy of a few days.

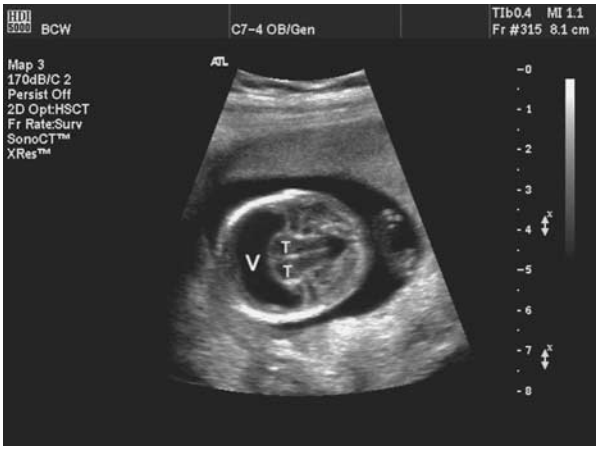

FIG. 7. Alobar holoprosencephaly results in a single large ventricle (V) seen on the midline. The thalami (T) are small and at the base of the large ventricle.

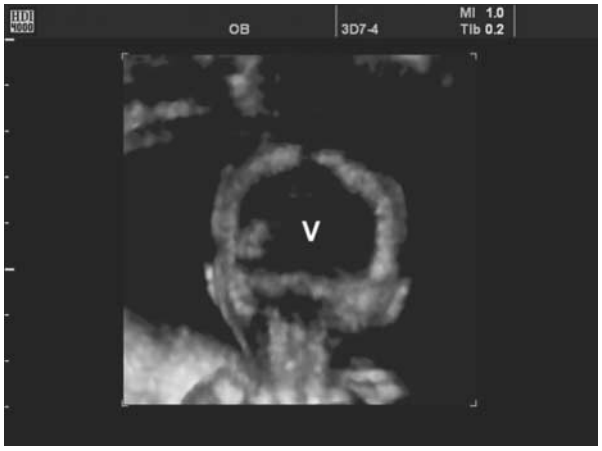

FIG. 8. Live 3D images help identify the type of holoprosencephaly present. The large ventricle (V) with the lack of superior brain tissue raises suspicion for the pancake-type alobar holoprosencephaly.

Sonography of the anencephalic fetus has a dramatic appearance (Fig. 6). The cranium and brain tissue above the orbits is absent. Due to the lack of brain structures, a biparietal diameter (BPD) measurement cannot be performed. Alternate measurements include ocular distances combined with the femur and abdominal circumference.

HOLOPROSENCEPHALY

Failure of the brain to divide into lobes results in the formation of abnormal cerebral hemispheres. This defect occurs early, during the third week of gestation, forming alobar, semilobar, and lobar categories.

The most severe, alobar, has the defining characteristic of a single large midline ventricle (Figs. 7-8). This large monoventricle has a cup, pancake, or ball formation and is accompanied by a fused thalamus and agenesis of the cavum septum pellucidum, corpus callosum, optic tracts, and olfactory bulbs.

Semilobar holoprosencephaly is a less severe form of this midline malformation. There is still the single ventricle, but partial formation of the occipital horns with a rudimentary falx and interhemispheric fissures present. The thalamus is either partially or completely fused, and the corpus callosum, cavum septum pellucidum, and olfactory bulbs fail to form. Both the semilobar and alobar forms have a high mortality rate because these malformations are not compatible with life.

Lobar holoprosencephaly exhibits the absence of the cavum septum pellucidum with normal separation of the ventricles and thalami. The corpus callosum is normal, hypoplastic, or absent, and the ventricles show close to a complete division.

Sonography of the fetus with holoprosencephaly allows for imaging of the large C-shaped ventricle, brain tissue formed in a horseshoe shape, the degree of fusion of the thalamus, and the absence of the third ventricle. The corpus callosum and cavum septum pellucidum are absent, as is the interhemispheric fissure. These developmental abnormalities link to chromosomal variations (usually trisomy 13) and facial malformations.

AGENESIS OF THE CORPUS CALLOSUM (ACC)

The corpus callosum is a flat structure that connects the two hemispheres of the brain. This late-developing structure forms in the 12th to 18th weeks superior to the cavum septum pellucidum (CSP). Abnormalities in development range from partial to complete agenesis, with the posterior portion affected most often. Although corpus agenesis may be due to lack of development, it is also seen with several chromosomal abnormalities, specifically trisomy 8, 13, and 18.

Sonography images the corpus callosum as an echogenic area superior to the CSP (Fig. 9). Mid-

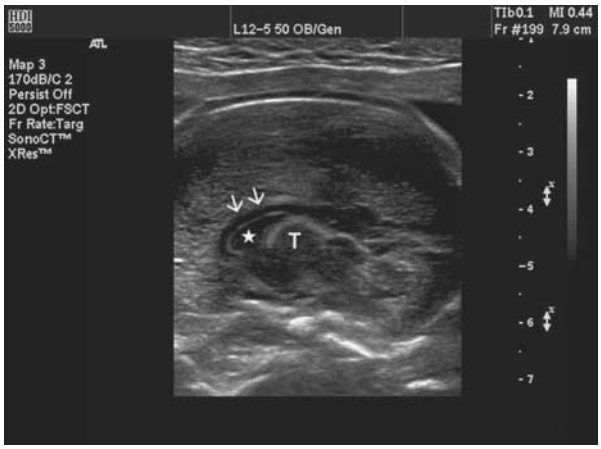

FIG. 9. The normal corpus callosum images as a hypoechoic linear structure (arrows) superior to the cavum septum pellucidum (star). This image only shows the portion anterior to the thalamus (T), but the corpus callosum extends posteriorly over the echogenic vermis.

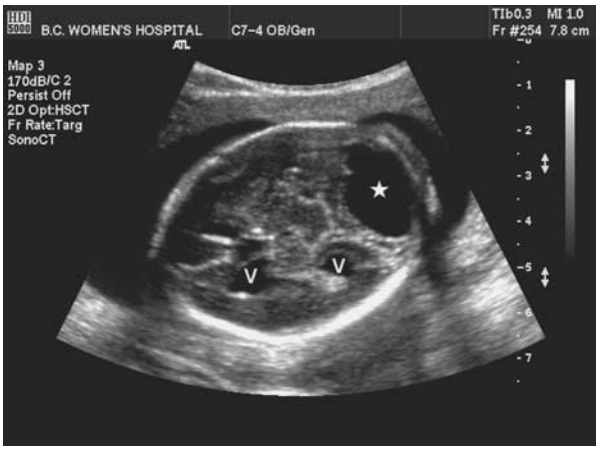

FIG. 11. The classic Dandy-Walker malformation images with a large cisterna magna (star). Ventriculomegaly may not become evident after birth, but the frontal and occipital horns of the ventricles are seen in this image (V).

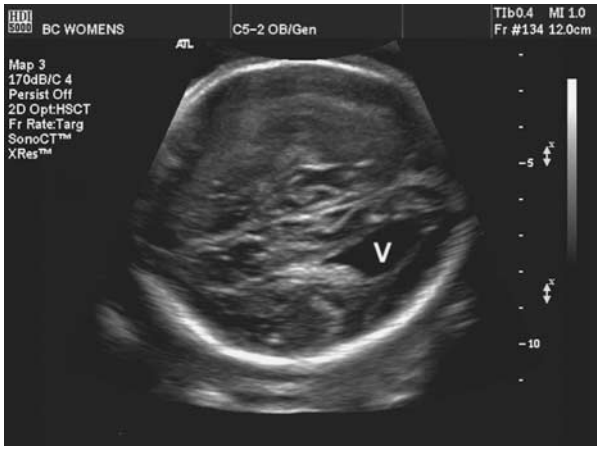

FIG. 10. The teardrop-shaped ventricle (V), characteristic of agenesis of the corpus callosum (ACC), appears on the axial scan plane.

sagittal and coronal scanning planes allow visualization of the corpus, but these difficult views are not always available due to fetal position. This results in the diagnosis of corpus callosum agenesis based on indirect sonographic findings. These include the frontal horns of the lateral ventricles having a greater than normal separation, third ventricle elevation and dilatation, and the characteristic teardrop shape of the dilated occipital horn of the lateral ventricle (Fig. 10).[9]

DANDY-WALKER COMPLEX

Several different terms describe degrees of malformation within the nonspecific grouping of Dandy-Walker complex. These include Dandy-Walker syndrome (DWS), Dandy-Walker malformation (DWM), and the Dandy-Walker variant (DWV). DWS includes ventriculomegaly, a large cisterna magna, and a communication with the fourth ventricle through a defect of the cerebellar vermis. This is the form most commonly seen during the prenatal sonographic examination and will be discussed further.

Determination of normalcy for the posterior fossa is the first step in identifying DWS. The normal cisterna magna measures 3 to 11 mm from the vermis to the inner skull table of the occipital bone. Imaged dural folds appear as linear paired echoes running parallel to the midline of the brain. There is normal variation in size with gestational age, but measurements do not exceed the upper limits of normal.

As early as the 18th week of gestation, the sonographic examination of the fetus with a posterior fossa cyst, splaying of cerebellar hemispheres, enlarged cisterna magna, and ventriculomegaly raises concern for the presence of DWS (Fig. 11).[10] Associated neural defects include ACC, holoprosencephaly, and encephalocele.

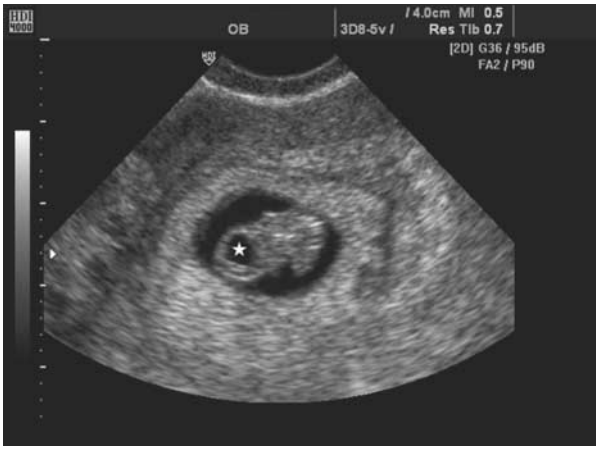

FIG. 12. The rhombencephalon (star) images during Carnegie stage 20 mimic the Dandy-Walker malformation or hydrocephalus, which does not occur until after the 18th week of gestation.

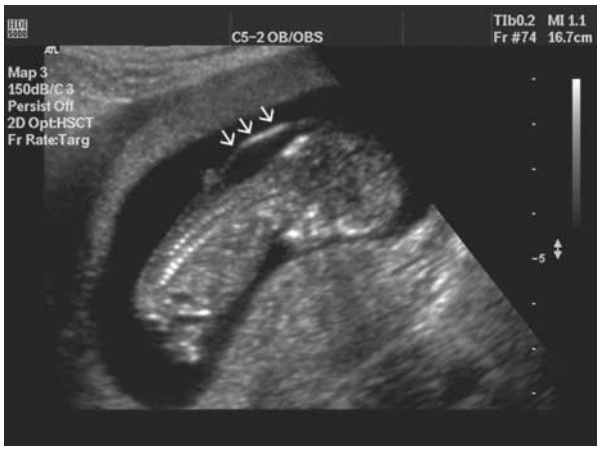

FIG. 13. Diagnosis of the abnormal nuchal area depends on the presence of a bulging membrane (arrows) and is an important finding in the first trimester. Careful longitudinal and transverse scanning of the cervical area rules out other structural defects such as spina bifida.

Findings That Can Fool Us

The developmental detail imaged during pregnancy continues to increase, resulting in findings, both normal and abnormal, that mimic pathologies. The sonographer familiar with early development can decrease the possibility of a false-positive finding for a CNS anomaly if aware of false, indirect, or coexisting signs such as those that follow. The anxiety resultant from an incorrect diagnosis is a great source of stress for new parents.

RHOMBENCEPHALON

The rhombencephalon develops between the 7th and 9th weeks of gestation. This fluid-filled area appears as a hypoechoic area within the cranium in the first trimester exam (Fig. 12). The posterior portion of the brain develops into the brain stem, fourth ventricle, and cerebellum.

ABNORMAL NUCHAL LUCENCY

The first trimester examination includes inspection of the nuchal lucency found on the posterior portion of the fetal neck. An increased dimension ($\geq$ 3 mm) raises suspicion for aneuploidy. Careful scanning of the nuchal lucency decreases the possibility of mistaking the finding for a spinal defect or encephalocele. Other normal findings seen posterior to the fetal neck and cranium are cervical fat pads and fetal hair floating in amniotic fluid (Fig. 13).

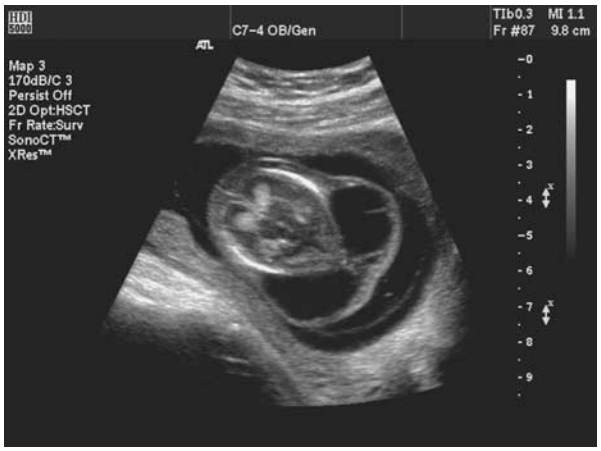

FIG. 14. The cystic hygroma has a characteristic cystic appearance in the cervical region. These should not be mistaken for encephalocele because this fetus appears to have normal brain anatomy.

CYSTIC HYGROMA

A thick-walled cystic area arising from the cervical region raises suspicion for the presence of a hygroma (Fig. 14). Coexisting signs that aid in confirmation of a hygroma include internal septations, generalized edema, and fetal hydrops. Differentiat-

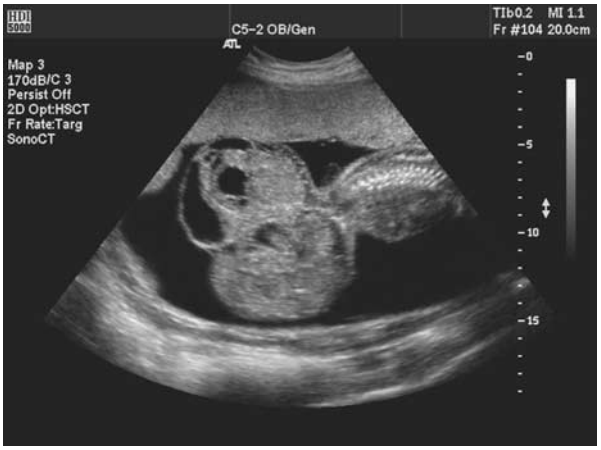

FIG. 15. A large complex mass extending off the sacrum raises suspicion for a teratoma. Due to the similarity to other malformations, other sonographic findings such as a large placenta, polyhydraminos, and urinary tract obstruction aid in differentiating the teratoma from a myelomeningocele.

ing the cystic hygroma from an encephalocele is accomplished through careful scanning for the presence of a cranial defect and fetal brain tissue within the cystic mass. Due to similar echo properties, it may be impossible to distinguish between a cranial meningocele, edema, and a cystic hygroma. The finding of a cystic neck mass raises concern for trisomy 21, thanatophoric dysplasia, hydrops fetalis, Saldino-Noonan, and Turner syndrome.

SACROCOCCYGEAL TERATOMA

The most common neoplasm seen in the fetus is the sacrococcygeal teratoma (SCT) (Fig. 15). Located in any area of the spine, the majority (80%) develops in the sacral area, followed by the cervical area. The composition and size of the teratoma determine the prognosis. The greater the solid component combined with a high degree of vascularization, the greater the malignancy potential. A cancerous lesion is rare, however; there are other risks to the fetus with a large mass. Due to arteriovenous shunting within the sacrum or venous compression of the mass, cardiac output increases. This results in the development of nonimmune fetal hydrops.

The teratoma could be initially confused with a sacral myelomeningocele due to the complex sonographic presentation. Careful scanning of the posterior vertebral arches confirming completion of neural tube closure rules out a spinal defect. Another differentiating factor is the size of the mass extending off the sacrum. The teratoma tends to be large, as great as 25 cm, whereas the myelomeningocele has a compact mass close to the defect.

Conclusion

Embryonic growth occurs quickly, with important organ system development complete before the mother becomes aware of the pregnancy. The central nervous system is one of the first systems to complete development. Failure of the neural tube and the rostral neuropore to close during the third and fourth weeks (Carnegie stages 7-12) results in central nervous system malformations.

The spine and brain are two of the most important areas for the sonographer to image and examine. Defects in these areas are easily located, but they may be difficult to diagnose. A spinal defect is often difficult to image due to fetal position or lack of amniotic fluid. This underscores the need to be familiar with coexisting, indirect, and false signs of possible defects.

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