# *Spina Bifida: A Review of the Importance of Sonography's Role in Its Detection*

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Spina bifida is a serious birth defect that involves an incomplete closure of the spine, spinal cord, brain, and/or protective covering of the fetal spine. Even though the causes of spina bifida are still uncertain, diabetes, obesity, severe hyperthermia in early pregnancies, and poor nutrition are believed to be causes of neural tube defects (NTDs). Because spina bifida may go undetected by α-fetoprotein screening and amniocentesis, sonography becomes a valuable tool in its identification. Spina bifida can also affect the fetal brain, so sonography is important in detecting any abnormal fetal head signs associated with Arnold-Chiari (type II) malformation. Surgical planning is another important part of sonography's role when detecting NTDs. Although still in experimental stages, in utero repair has proven beneficial in some cases of spina bifida after careful sonographic assessment as part of the treatment considerations.

*Key words:* neural-tube defects, sonography, spina bifida, spina bifida occulta, spina bifida cystica, meningocele, myelomeningocele, Arnold-Chiari (type II) malformation

Each year in the world, 400,000 fetuses are affected with a neural-tube defect (NTD).[1] There are many types of neural-tube defects. In addition to spina bifida, other NTDs include anencephaly, cephalocele, and encephalocele, along with a host of other fetal anomalies. Spina bifida is the second most common NTD, being a serious birth defect that involves an incomplete closure of the spine, spinal cord, brain, and/or protective covering of the fetal spine. Because spina bifida can be missed by α-fetoprotein (AFP) screening and amniocentesis, sonography becomes a valuable tool in its identification. In addition, abnormal findings within the cranium in association with a spinal defect, known as Arnold-Chiari type II malformation, can readily be detected through sonographic techniques, as ev-

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idenced in a case study that will be presented, followed by a discussion of the various diagnostic and treatment considerations of spina bifida.

## Case Study

In the maternal-fetal medicine department of a metropolitan hospital, a woman discovered, as a result of in vitro fertilization, that she was pregnant with an intrauterine dichorionic-diamniotic twin pregnancy. At this time, Twin A was measured, using sonographic techniques, at 10 weeks, 2 days of gestation, and Twin B was measured to be 10 weeks and 0 days of gestation. No anatomic anomalies were evident, with the uterus and the gestational and yolk sacs also appearing normal at this time. Four weeks later, the patient returned for a follow-up sonogram. With appropriate interval fetal growth, Twin A measured 14 weeks, 1 day of gestation, whereas Twin B measured only 13 weeks, 3 days.

Another sonography appointment was scheduled at an additional 4-week interval. At this time, the stomach, bladder, and heart motions of both fetuses were visualized and were within normal limits. During this examination, Twin A's biparietal diameter (BPD) measured 18 weeks, 2 days; the head circumference (HC) measured 17 weeks, 3 days; the abdominal circumference (AC) was 18 weeks, 0 days; femur length (FL) was 18 weeks, 1 day; and humeral length (HL) was 18 weeks, 1 day. However, the measurements of Twin B were 1 to 2 weeks less. Twin B's BPD measured 16 weeks, 5 days; HC was 16 weeks, 5 days; AC measured 18 weeks, 0 days; FL measured 17 weeks, 0 days; and HL was 17 weeks, 4 days. An enlargement of the lateral ventricles, a posterior pulling of the cerebellum consistent with the Arnold-Chiari (type II) malformation, and the "lemon sign" head contour were obvious within Twin B at this time. Also, an opening of Twin B's spine was demonstrated to extend from the lower thoracic region to the sacrum.

The following month, the patient returned for another examination. The amniotic fluid volume was normal for both twins. The stomach, bladder, renals, and heart motion of both fetuses were documented to be within normal limits. The intracranial structures of Twin A were normal; however, Twin B had enlarged lateral ventricles, with an atrial measurement of 13.5 mm, greater than the normal threshold standard measurement of 10 mm. Twin B's choroid plexus appeared to "dangle" within each enlarged ventricle. The cerebellum was low-lying, displaying the "banana sign," and the posterior fossa was obliterated. The "lemon sign" was also again demonstrated. A severe curvature in the lumbar region, due to a myelomeningocele, was noticed in Twin B, and Twin B also displayed bilateral clubbed feet (Fig. 1). The BPD, HC, AC, FL, and HL measurements of Twin B measured 1 to 2 weeks behind Twin A on this examination. However, Twin B measured 24 weeks and 4 days, only a 2-day difference from Twin A, on the next month's follow-up scan, demonstrating appropriate interval fetal growth during this time.

The physician explained to the patient that Twin B had a neural-tube defect, which would cause the fetus to have limited neurological distributions, problems of uncontrolled bladder and bowel functions, and difficulties of ambulation. The physician was less concerned at this time of the possibility of mental retardation. The patient was informed that, at the time of her delivery, pediatric neurosurgeons would be present. The patient was also provided with the advantages and disadvantages of undergoing intrauterine fetal surgery versus postnatal repair. In this particular case, there was an obvious disadvantage to considering intrauterine surgery due to the presence of a coexistent healthy twin. The detection and follow-up of the anomaly through sonography did allow appropriate preparations to be made in advance of delivery, however.

Although the original estimated date of confinement (EDC) was still 5 weeks away, twin girls were delivered via C-section at approximately 35 weeks due to two consecutive failed biophysical profiles (BPPs) by Twin B. Just prior to delivery, Twin A measured 35.5 weeks and Twin B measured 33.5 weeks by sonographic technique. Following delivery, Twin A appeared to have no complications, but due to the early delivery and because the mother had been receiving steroids to assist with development of fetal lung maturity (FLM), Twin A remained in the neonatal intensive care unit

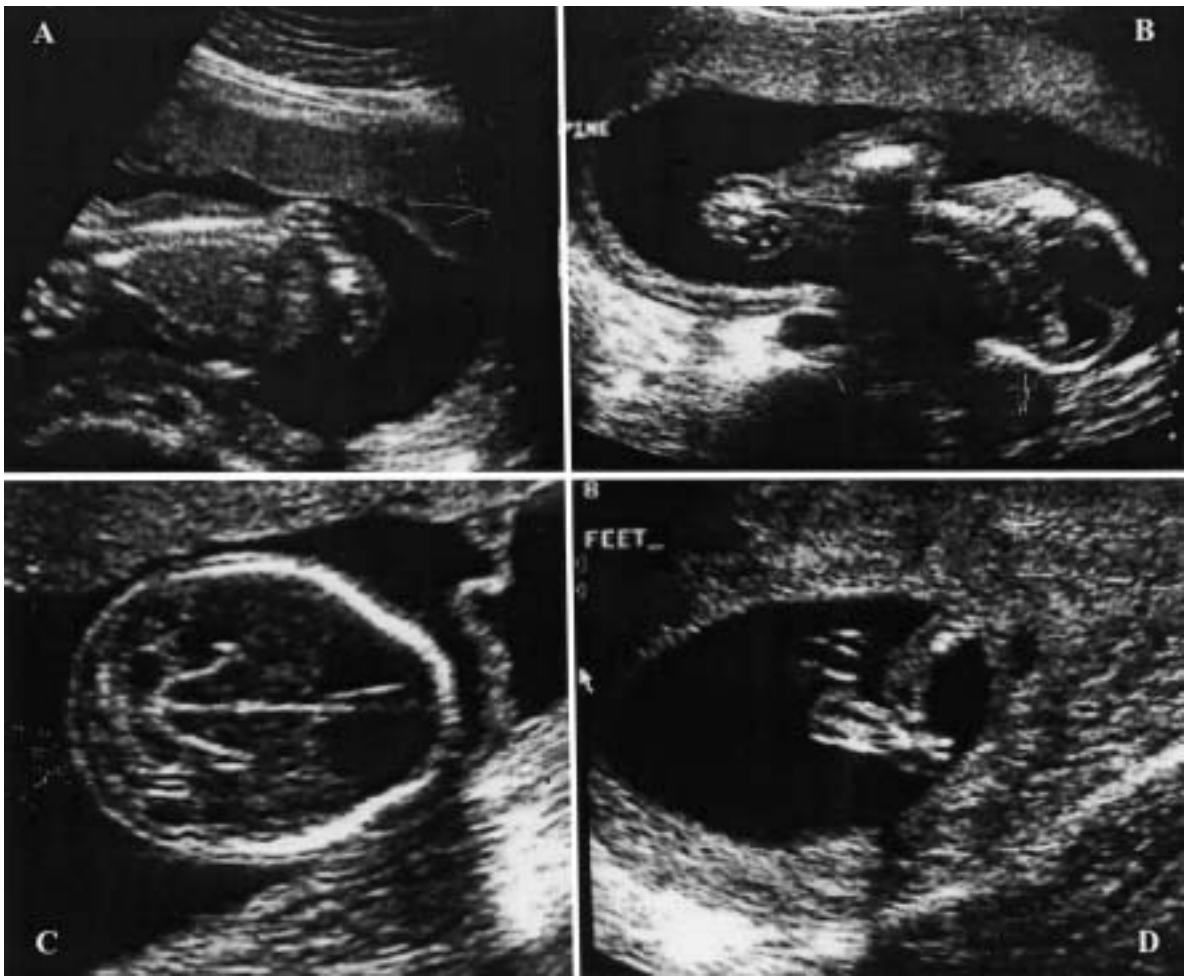


**FIG. 1.** Twin B: A sagittal plane of Twin B's spine with a disruption in the curvilinear line is demonstrated (A). A coronal plane demonstrating a hypoechoic myelomeningocele from Twin B's back (B). The Arnold-Chiari (type II) malformation is pictured by the "banana" and "lemon" signs in Twin B's head (C). Twin B also has a clubbed foot (D).

(NICU) for 2 weeks of observation and then was allowed to leave the hospital.

As was already demonstrated multiple times sonographically, Twin B was born with an open spinal lesion. This twin was taken to the operating room (OR) approximately 18 hours following delivery for surgical closure of the myelomeningocele. The surgery was successful, with the infant being given several weeks of recovery time prior to the next surgical procedure. Five weeks later, Twin B returned to surgery for placement of a ventriculoperitoneal (VP) shunt for her hydrocephalus. Unfortunately, this shunt had to be revised two months later; however, the second shunt was successful and remains intact today. Another complication that was discovered at eight weeks of life was failure of Twin B to demonstrate hearing capabilities during administration of testing in the NICU. A developmental ear deformity was discovered, and the infant has since begun speech therapy and sign language, with the possibility of a cochlear implant in the future. Twin B was released to go home five months following delivery, and both twins are doing well at this time.

## *Spina Bifida and Its Types*

Spina bifida is a neural-tube defect of the spine in which the dorsal vertebral arches fail to fuse together, thus allowing the meninges and/or spinal cord to protrude. The lumbar and/or sacral regions of the spine are the most common locations of

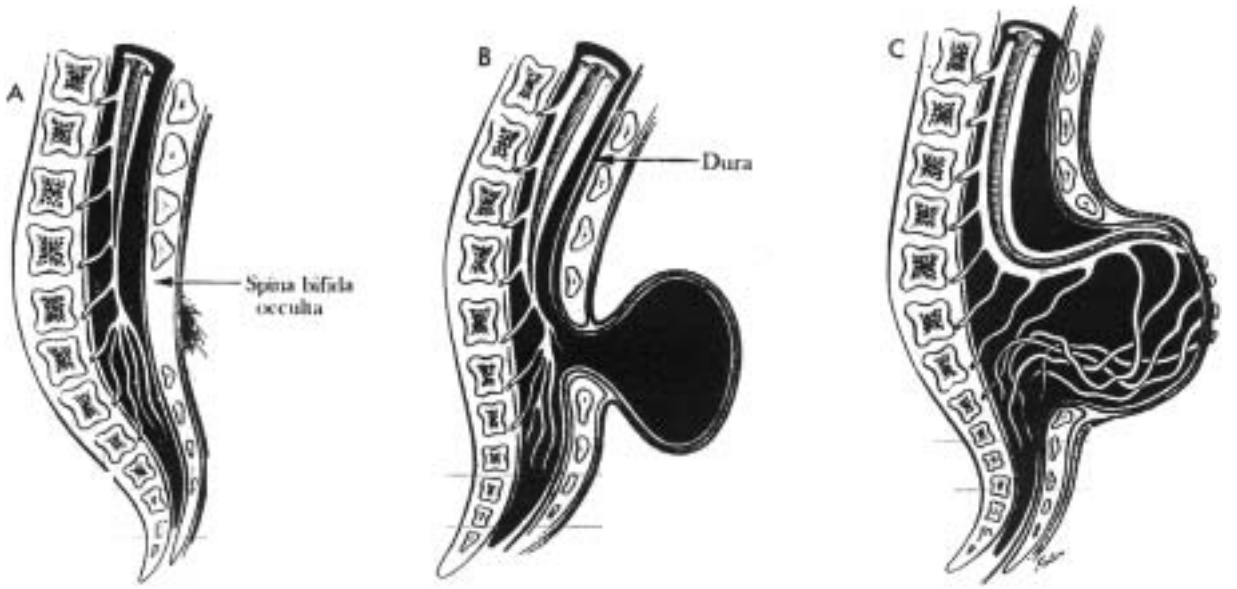


**FIG. 2.** Demonstration of spina bifida occulta. The skin, spinal cord, and nerves are normal; there are no protrusions from the skin (A). Meningocele is demonstrated by a protrusion of spinal fluid (B). Protrusion of meninges and spinal cord is demonstrated in a myelomeningocele (C).[3] Copyright 1999, Massachusetts Medical Society. Reprinted with permission.

spina bifida. *Spina bifida occulta* and *spina bifida cystica* are the two general classifications of spina bifida.

Spina bifida occulta, the closed type, is the mildest form of spina bifida. In this condition, the skin, spinal cord, and nerves are normal; however, the spine has a small defect. Even though there is a defect, there are no protrusions of the meninges or spinal cord, and the neurological developments are normal (Fig. 2A). Therefore, spina bifida occulta can be extremely difficult to detect sonographically in the fetus due to the skin covering the defect, particularly without a mass protrusion. AFP screenings and amniocentesis would be unable to detect a closed spina bifida. Because there are no openings in the skin, AFPs would not leak into the amniotic sac, therefore resulting in a "normal" test. About 10% to 15% of spina bifida include the closed types.[2]

The second classification of spina bifida is spina bifida cystica. This type of spina bifida is an open spinal defect. A sac protrudes with either meninges and/or spinal cord, differentiating meningocele and myelomeningocele as the two types of spina bifida cystica. *Meningocele*, the rarest form of spina bifida, consists of an open spinal defect characterized by protrusion of the spinal meninges. The meninges are made up of dura mater (external), arachnoid (middle), and pia mater (internal) (Fig. 2B). The *myelomeningocele* is also an open spinal defect characterized by the protrusion of meninges and the spinal cord (Fig. 2C). Either form of spina bifida cystica is much more readily recognizable on sonography due to these protrusions.

Around 80% to 90% of fetuses with myelomeningocele often have cranial defects associated with Arnold-Chiari (type II) malformation, which are also readily recognized upon close sonographic inspection.[2] Because there are openings in both meningocele and myelomeningocele, AFPs will leak into the amniotic sac, causing a great increase of protein. Unlike spina bifida occulta, AFP screening and amniocentesis should be able to detect this abnormal increase associated with meningocele and myelomeningocele. However, because there are other causes of increased AFP values, such as multiple gestation, sonography became a particularly important tool in the twin case study by demonstrating not only the spinal defect but also the corresponding cranial defects in determining a myelomeningocele in Twin B.

**TABLE 1.**
**Nervous System Malformations Resulting From Interruption of Development at Specific Times in the Gestation**

| Days of Gestation | Developmental Event | Potential Outcome |
|---|---|---|
| 0-18 | The three germ layers and neural plate form | Fetal death likely |
| 18 | Neural plate and groove form | Anterior midline defect |
| 22-23 | Optic vessels appear | Ventriculomegaly, hydrocephalus (likelihood of development extending beyond this period) |
| 24-26 | Cranial neuropores close | Anencephaly |
| 26-28 | Caudal neuropores close | Spina bifida cystica, spina bifida occulta |
| 32 | Vascular circulation begins | Microcephaly (likelihood of development extending beyond this period) |
| 33-35 | Prosencephalon splits—paired telencephalon | Holoprosencephaly |
| 70-100 | Corpus callosum forms | Agenesis of corpus callosum |

## *Embryology of the Spine*

The central nervous system (CNS) develops from the thick ectodermal neural plate of the central nervous system, which occurs by the 18th gestational day.[2] The edges of the neural plate become elevated to form into neural folds. These folds then begin to approach each other, fusing together to form the neural tube. The fusion of the neural tube begins in the cervical region and then continues to fuse in cephalic and caudal directions. The fusion, however, is delayed at the cranial and caudal ends. These two openings communicate between the lumen of the neural tube and the amniotic fluid. At approximately the 25th day after conception, the cranial neuropores close, and approximately 2 days later, the caudal neuropores follow.[4] Within just 28 days of conception, the fetal spine and brain have developed.[3] When an opening does not close within the third or fourth week of conception, a congenital malformation, such as spina bifida, can occur. This opening can occur anywhere from the brain to the end of the spinal cord; however, 80% of spina bifidas occur in the thoracolumbar, lumbar, or lumbosacral regions,[5] with the majority of these occurring in the lumbar and lumbosacral regions.[6] Obviously, then, those portions of the CNS in the middle segment—the cervical and thoracic regions, where early fusion occurs—are the least likely areas of occurrence. Figure 3 demonstrates both complete and incomplete closure of the neural tube. Table 1 provides various timing sequences in the development of the central nervous system, along with some of the potential outcomes of disruption during these specific time periods. Note, most specifically, consequences from days 24 through 28, during the time of closure of these neuropores.

"The primitive spinal cord divides into two regions. The alar plate region matures into the sensory region of the cord, and the basal plate region develops into the motor region of the cord. These regions further subdivide into specialized functions."[2] At 6 weeks of gestation, neural functions begin. At this time, simple involuntary facial and neck movements begin to occur. Except for the back and the top of the fetal head, sensitivity has spread across the body of the fetus by the 12th week. By week 16, the fetus begins to have periods of active and inactive movements. Fetuses are capable of producing gripping actions and demonstrating shallow respiratory movements by weeks 16 to 20. At 24 weeks, the fetus can suck its thumb, and at 28 weeks, the normal fetal brain demonstrates significant brain wave patterns.

## *Sonographic Features of the Spine and Spina Bifida*

It is imperative for the sonographer to scan the fetal spine thoroughly while having a complete understanding of the normal developmental appearance of the fetal spine and head because small defects may be difficult to detect. The fetal spine should be scanned in the sagittal, coronal, and transverse planes. Sonographically, the spine demonstrates three hyperechoic bony structures. These structures are the anterior centra and the two poste-

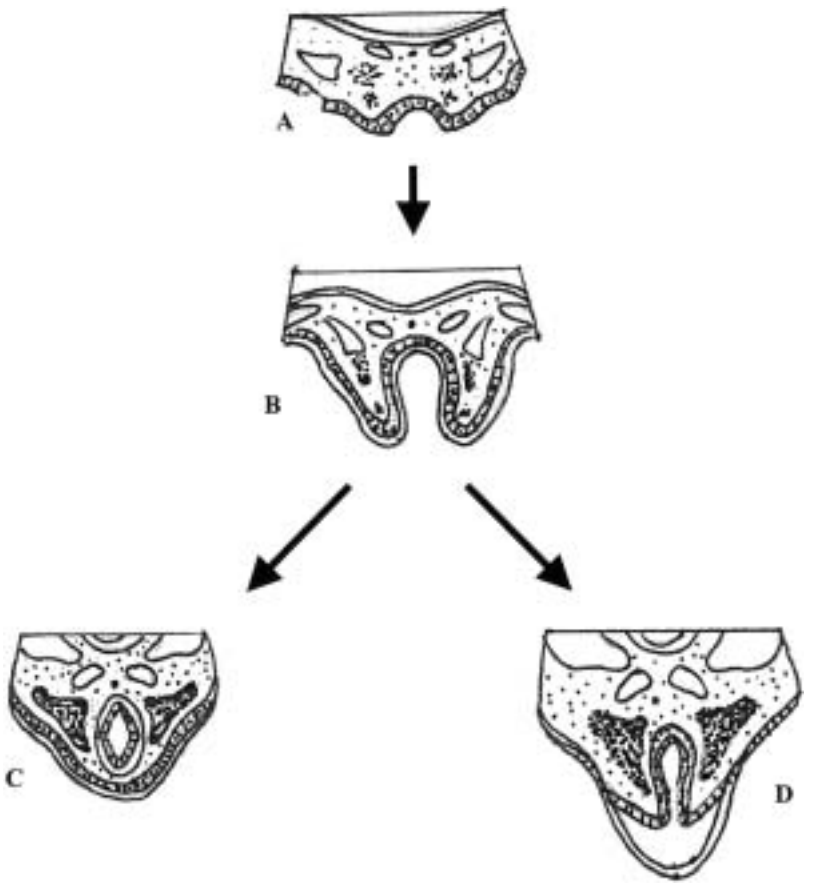


**FIG. 3.** The central nervous system begins development around the 18th to 20th days after conception (A). The edges of the neural plate elevate to form neural folds, which begin to approach one another (B). At approximately 28 days of development, a complete closure occurs when the folds fuse together to form the neural tube (C). If closure does not occur by the 28th day, a neural-tube defect (NTD) develops (D). Illustrations courtesy of Thomas E. Hancock.

rior elements (laminae), overlaid with soft tissue. In the longitudinal planes, this appearance resembles "railroad tracks" or the "railway sign" by the echoes from the anterior and posterior laminae and spinal cord (Fig. 4A). The spinal cord can be visualized between the anterior and the posterior laminae. A coronal plane is a cross section of both laminae with equal amounts of tissue on either side of the spinal echoes (Fig. 4B). In the transverse plane, the spine should appear as a closed circle, made up of the central vertebral body and the posterior elements. This normal circle is a closure of the neural tube. Also, a smooth contour of skin should appear posteriorly to verify no breaks or protrusion of the back.

The optimal time for detection of NTDs is between 16 and 18 weeks, but screening can occur between 15 and 20 weeks. With spina bifida, there is a splaying (spreading out) of the posterior elements and abnormality of the overlying of the skin. Sonographic features of spina bifida are identified by the posterior ossification center with a U (or markedly V) configuration. Protrusions may appear as either anechoic (meningocele) or contain hypoechoic neural elements (myelomeningocele). The sagittal view of spina bifida will demonstrate irregularities of the bony spine as bulging within the posterior contour of the fetal back or obvious disruption of the contour of the fetal skin (Fig. 5A). Within the transverse plane, spina bifida is revealed as an open vertebra with a U shape (Fig. 5B). A divergent configuration replaces the normal parallel lines of the normal vertebral arches in the coronal view (Fig. 5C). The soft tissue can be a flat

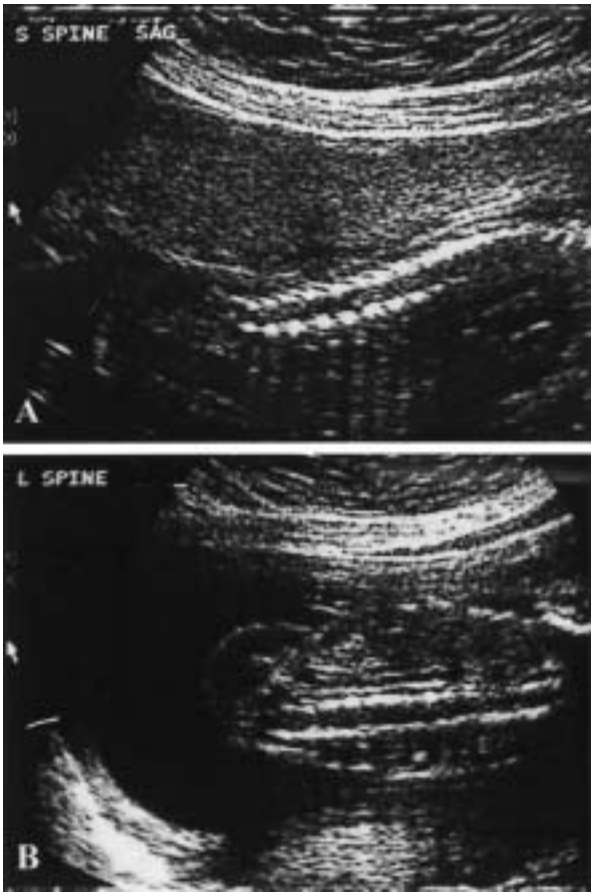


**FIG. 4.** Parallel, curvilinear, sagittal spine resembles the "railway sign" (A). Both posterior laminae are visualized in the coronal scan plane with equal amounts of tissue on either side of spine (B).

defect (spina bifida occulta), cystic (meningocele), or hypoechoic to complex (myelomeningocele) in its appearance.

## *Sonographic Features of the Head Associated With Spina Bifida*

Spina bifida affects not only the spine but also the fetal head. With defects in both the fetal spine and the head, Arnold-Chiari (type II) malformation has occurred. "Fetuses with myelomeningoceles often present with the cranial defects associated with the Type II malformation which is identified in 90% of patients."[2] Characteristics of this Arnold-Chiari malformation include displacement of the fourth ventricle and the upper medulla, displacement of the inferior parts of the cerebellum through the foramen magnum, and defects in the calvarium and spinal column, with the development of secondary hydrocephalus.[7] Arnold-Chiari (type II) malformation is present in almost every case of thoracolumbar, lumbar, and lumbosacral myelomeningocele.[8]

The fetal head should be assessed for the "banana sign," the "lemon sign," and ventriculomegaly or hydrocephalus if an NTD is either suspected or has been identified. The "banana sign," which is specific for spina bifida, is a downward pulling of the cerebellum through the cisterna magna, which is located posterior to the cerebellum, and the foramen magnum. The normal appearance of the cerebellum should be shaped as a dumbbell. A small distorted or displaced cerebellum, often appearing banana shaped, may signify an open spina bifida (Fig. 1C).

Once the cerebellum has the "banana" appearance, the resultant pressure of this restructuring in shape will lead to disruption or obliteration of the cisterna magna, which will then lead to caudal displacement of the frontal bones, creating a collapse and curvature. This collapsing of the frontal bones is called the "lemon sign." Although the "lemon sign" may be one of the sonographic clues to spina bifida, the sonographer should realize that it is not specific only for this disorder.

Ventriculomegaly is dilatation of the ventricles of the brain and is associated with these findings of spina bifida. Hydrocephalus is an increased amount of cerebrospinal fluid and an associated increase of intraventricular pressure that, ultimately, creates head enlargement, which will only occur in the most severe cases of ventriculomegaly (a head circumference of 18 mm or greater than the abdominal circumference or a biparietal diameter greater than the abdominal diameter by at least 6 mm).[4] The sonographer will more likely carefully assess for ventriculomegaly, as the fetal head size usually remains normal and proportional to the fetal body when the diagnosis of spina bifida is made. The most consistent measurement to detect ventriculomegaly is within the atrium of the ventricle, where the width measurement should not exceed 10 mm. Sonographically, hydrocephalus can be diagnosed if at least one of the following additional findings is present: a disrupted falx echo (appears as a wavy line within the dilated ventricles, instead of a

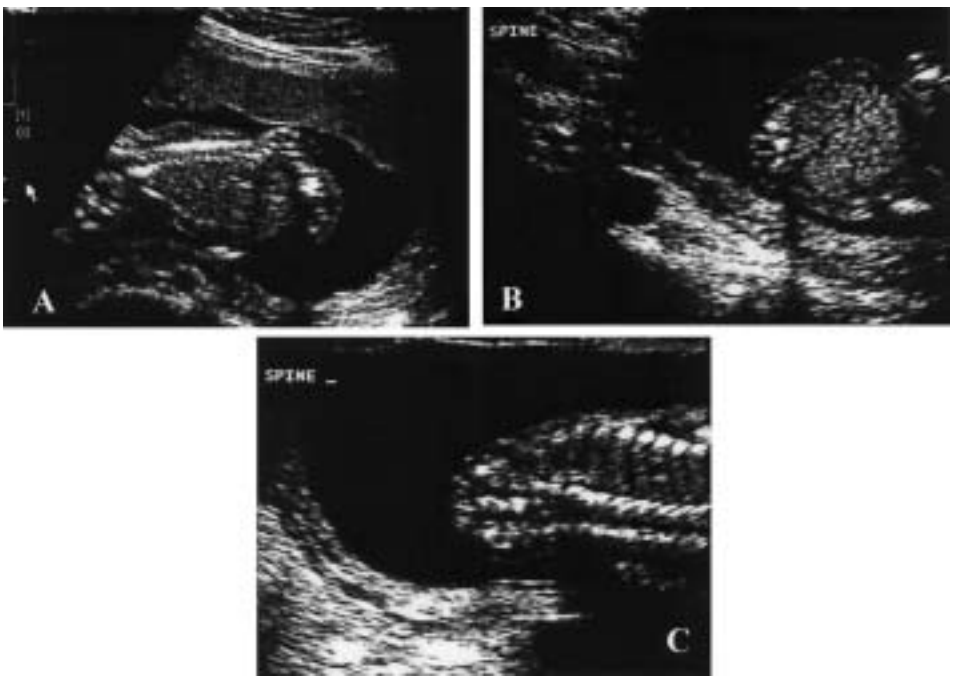


**FIG. 5.** Sagittal view of a posterior bulging in the fetal spine (A). Transverse view demonstrates a disruption of the contour of the fetal skin (B). Coronal image demonstrates divergent configuration of spine (C).

smooth straight echo), third ventricular dilatation (greater than 2 mm in diameter), specific posterior fossa abnormalities, increased ventriculomegaly, and head enlargement.

## *Detection Methods for Spina Bifida*

Spina bifida can be found before a fetus is born by using maternal serum α-fetoprotein (MSAFP) screening, sonography, and amniocentesis. "AFP is a normal fetal protein that is synthesized throughout fetal life, first in the yolk sac and then by the liver. Because it is excreted by the fetal kidneys, it can be identified in the amniotic fluid."[4] The highest concentration of AFP is in the first trimester and then should decrease during the pregnancy. An AFP level between 2.0 and 2.5 multiples of the median (MOMs) is within normal limits; however, levels greater than this are abnormal, provided that the gestational age has been properly correlated. Elevated levels occur when there is an open spinal defect due to the leakage of AFP from the cerebrospinal fluid into the amniotic fluid, which circulates in the maternal blood or if the values are taken too early in the gestation. Sonography can readily serve a very useful role in determining if such a discrepancy exists. Once levels are determined to be elevated, sonography is used for further testing and aids in corresponding fetal age to AFP levels.

Even though MSAFP can detect spina bifida and false negatives are very uncommon, AFP testing is not specific for spina bifida because an increased AFP can also occur with other birth abnormalities. Errors may also occur when levels are not adjusted for other factors, such as multiple gestations or obesity of the mother. As previously discussed, the major downfall of using AFP screening alone is that it does not detect closed spina bifida. Therefore, sonography becomes very important in verifying the presence, location, and severity of the spina bifida. A sonographic study becomes particularly important in that it can demonstrate both spinal and brain defects. "The sensitivity, specificity, and positive and negative predictive values of ultrasound evaluation for the detection of NTDs [have been demonstrated to be] 99%."[5] In most pregnancies, the spine can be identified by 16 weeks of gestation, but almost all spines can be identified accurately by 18 weeks.[4] Sonography is

also considered to be a safe procedure for both mother and fetus.

Amniocentesis can also be used to check for spina bifida, from which amniotic fluid is withdrawn from the womb and tested for AFP levels. This can provide more specific information than does MSAFP. However, there is an increased rate of threatened abortions from this procedure, averaging an estimated fetal loss of 1:200.[4] "[Many studies have demonstrated] that the rate of detection of NTDs by sonography alone has prevented the need for amniocentesis."[2] In any event, sonography becomes an important tool for guiding the amniocentesis, so again it proves its usefulness in the diagnosis of spina bifida and other NTDs.

### *Treatment of Spina Bifida*

In spina bifida, the normal passage of electrical impulses and neural tissue is interrupted and damaged due partly to chronic mechanical injury and chemical trauma induced by exposure to amniotic fluid.[9] Termination can be offered if the pregnancy is less than 24 weeks; if the pregnancy is over 24 weeks of gestation, a Cesarean procedure is planned to decrease shock-like trauma to the neural tissue that occurs during birth. In addition, an earlier term delivery date can be planned to reduce continued exposure of the neural components to the amniotic fluid. It is greatly recommended that within 24 to 48 hours of fetal life, surgical closure of the spine be performed to decrease morbidity and mortality. Surgical care is important to preserve neural tissue, restore anatomy, and minimize infection. Many of those who have had an operation for NTD after birth have lived to the third decade of life.[3]

Although in utero repair has proven beneficial in some cases, it is still in the experimental stage and not as widely used. Some physicians have, however, surgically treated spina bifida in utero around 22 weeks of gestation, even though most anomalies are best managed postnatally.[3] Because in utero surgery affects both mother and fetus, when the mother decides to treat her fetus in this manner, "it is [considered] ethically acceptable to place the mother at some risk for the benefit of her fetus."[10] There are a number of prerequisites for fetal intervention, including accurate prenatal diagnosis, no associated anomalies, defined natural history, correctable lesion, and technical feasibility. For fetal surgery to be performed, it must be either life saving or able to prevent irreversible disease, injury, or handicap to the fetus. It must also involve the least risk of death to the fetus, as well as the mother.[11] Finally, there are certain selection criteria for mother and fetus. A team of experts must fully evaluate the mother and fetus, and this evaluation includes detailed sonographic studies, fetal magnetic resonance imaging (MRI), fetal echocardiography, and fetal karyotyping.[12]

The benefits of in utero surgery simply involve the repair of a fetal anomaly to allow continuation of pregnancy and the best life possible for the fetus. Specifically, the repair of a myelomeningocele (MMC) can be beneficial due to its associated concerns. MMC causes such complications as paraplegia, mental retardation, sexual dysfunction, bowel and bladder dysfunction, skeletal deformities, and hydrocephalus, beyond speculation surrounding the earlier discussion that exposure of the spinal cord to amniotic fluid can cause neural damage as well as the known trauma during birth.[9,13] Proof of this last complication has been supported through the findings that "fetuses with myelomeningocele have leg movement early in gestation that is often absent after birth at term, suggesting loss of function in-utero."[14] "The rationale behind fetal intervention in the treatment of MMC is to correct the structural defect at a time when significant neuronal damage has either not yet occurred or still can potentially be reversed or arrested."[10] If surgical intervention can be performed in utero, the time for the fetus to remain in utero might also be extended, thus allowing time for FLM and other developmental concerns associated with premature deliveries. Perhaps most important to current treatment capabilities are the results of a study that demonstrated that "NTD patients who underwent in utero surgery experienced a lower incidence of hydrocephalus than [did] the control group (59% versus 91%)."[6] The in utero intervention that is described could decrease the rate of lifelong, postnatal VP shunt dependency, a common treatment that has previously been demonstrated in the case study of Twin B.

Also of importance to note when considering treatment options is that the lower the defect, the better the prognosis. For instance, motor function

is needed to at least the third lumbar level to stand erect. To walk, motor function from the fourth and fifth lumbar is needed, and to function sexually, motor function is needed to at least the second to fourth levels. Whether considering surgical treatment in utero or postnatally, "the degree of handicap and survival rate depends on the level of spinal segments, the severity of the lesion, the treatment program, and the associated anomalies."[1]

## Causes and Prevention of Spina Bifida

Even though the causes of spina bifida are unknown, it is believed that a primary cause of NTD is due to mothers who are either obese or diabetic or have had hyperthermia early in the pregnancy. The highest incidences of NTDs are found in Great Britain, Ireland, Pakistan, Northern India, Egypt, and Arab countries. Finland, Japan, and Israel have the lowest incidences of spina bifida. Within the United States, the highest are in the East and the South, and the lowest is in the West. "There is [also] an increased incidence of neural-tube defects in Hispanics, especially if the mother was born in Mexico."[1] An increased incidence of NTDs has occurred more frequently in monozygotic twins, in females than males, in Trisomy karyotypes, and with other chromosomal abnormalities. In the United States, the recurrence risk for any NTD was 1.5% to 3% when there was one affected sibling and 5.7% with two affected siblings.[5]

Folic acid supplements, food fortified with folic acid, and foods naturally rich in folates have been identified as intervention factors for NTDs. Folic acid has been shown to decrease the risk of NTDs, even though the theory is not well understood. "The US Public Health Service recommends that all women of reproductive age consume 0.4 mg of folic acid daily in order to reduce the risk of having a child with an NTD, since over 50% of pregnancies in the United States are unplanned."[5] Research studies report that women who have had children with an NTD decreased the incidence of recurrence when folic acid was taken around the time of conception and through the first trimester. Because of this, women who are planning a pregnancy and are at higher risk for spina bifida are advised to consume 4 mg of folic acid one to three months prior to becoming pregnant.[15] Certain foods in the United States are fortified with folic acid, as mandated by the U.S. Food and Drug Administration (FDA), which requires that "enriched grain products be fortified with 140 micrograms of folic acid per 100 grams of product."[5] In addition, fruits, dark-green leafy vegetables, dried beans, and peas are a few types of food that are natural sources of folate. Unfortunately, at least 30% of NTDs are not prevented by the consumption of folic acid supplements,[3] as other risk factors are still involved.

## Conclusion

Even though the causes of spina bifida cannot completely be ascertained, studies have shown that folate and folic acid supplements help prevent spina bifida. And even though AFP screening and amniocentesis can detect spina bifida cystica, they cannot effectively identify closed spina bifida, whereas careful sonographic screening often can. For increased accuracy, it is extremely important for the sonographer to accurately evaluate the fetal spine in all three scanning planes—sagittal, transverse, and coronal—to increase the likelihood of detecting difficult to define defects, as well as to carefully evaluate the fetal head and intracranial structures for associated findings.

The effects of spina bifida result in a different lifestyle for both parents and the affected child, so without the valuable tool of sonography in identifying spina bifida, parents would not be informed and prepared for this change, and specific follow-up and treatment for the fetus could likely never occur. Although the case study presented and the description of spina bifida treatments may not seem like ideal happy endings, many lives have been saved and improved through new techniques such as those that have been described in this report, and progression continues to occur in the prevention, identification, and treatment of this disorder. Beyond Twin B's life improvement in the described case study, the researchers of this article were informed that Twin A is very close to Twin B and does not like to be far from her. This could not have been possible without appropriate intervention, which was greatly assisted by the initial detection and follow-up of the sonographic technique.

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