A woman with a seizure disorder can carry a pregnancy safely. Her seizure treatment must be managed effectively to minimize the risk of a seizure that could harm the fetus. Seizures can harm the developing fetus by reducing the blood supply to the placenta. The treatment can be managed by the patient’s neurologist and obstetrician. Anti-convulsant medications should NOT be discontinued during pregnancy.

It is recommended that women discuss the management of their seizure disorder and Anti-convulsant medication(s) with their neurologist and obstetrician prior to pregnancy. If you are thinking of becoming pregnant your medications may be switched to those that are known to be the safest to the developing fetus. For women considering pregnancy it is also recommended that 4 mg of folic acid be taken daily starting two to three months prior to pregnancy and be continued through the first trimester. This will reduce the risk for neural tube defects like spina bifida.

The chance for any pregnancy to result in a baby with a birth defect is 3-5%, regardless of family history. This data comes from the pregnancy outcomes among the entire population. For the population of pregnant women with seizures, the risk for birth defects is two to three times this risk (6-15%), particularly for neural tube defects, heart defects and cleft lip/palate.

Some of the risk for developmental and physical problems in exposed babies may be attributed to the seizure disorder itself but many anti-convulsants are known to be associated with increased risks. There is a good volume of data in the medical literature about the developmental risks associated with anti-convulsants; each will be briefly summarized below.

**Depakote (Valproic Acid)**
Neural tube defects like spina bifida (open spine) are the main malformation associated with the use of depakote in the first trimester of pregnancy. In the general population, about 1 per 1000 babies is born with spina bifida. However, the risk among the children of women treated with depakote during the first trimester of pregnancy is 1-2 per 100 babies (1-2%). In addition, depakote has been associated with a distinctive pattern of anomalies called “fetal valproate syndrome”. This involves poor growth, small head circumference, characteristic facial features, heart defects, cleft lip/palate, and limb anomalies (particularly absent radius). The risk for a birth defect or valproate syndrome in infants exposed prenatally to depakote is reported in the range of 6-18%. A variety of developmental issues have also been reported, including developmental delay, decreased IQ scores, hyper-excitability, behavior problems, autism spectrum disorders and neurological dysfunction.

**Dilantin (Phenytoin)**
The use of dilantin during pregnancy has been associated with a 2-3 fold increased risk (6-15%) for malformations, including heart defects, small head circumference and cleft lip and palate. A characteristic pattern of abnormalities, called “fetal hydantoin syndrome”, has been reported in about 10% of infants born to women who took dilantin (phenytoin) during pregnancy. Features of this syndrome include a unique facial appearance, abnormalities of fingers, toes and nails, growth delay and developmental delay/disability.

**Diazepam (Valium)**
Diazepam is prescribed as a tranquilizer, muscle relaxant, preoperative medication, and as an adjunct to anticonvulsant therapy. There is a no consistent pattern of malformations observed in infants exposed to diazepam. The risk for birth defects is described as minimal. Some neonatal behavior problems have been noted in exposed infants.
Phenobarbital
The frequency of birth defects appears to be increased among children of women who take phenobarbital during pregnancy for treatment of seizure disorders as compared to women taking the medication for other medical indications. The risk for physical, motor and intellectual impairments is estimated in the range of 6-18%. Chronic use of phenobarbital late in pregnancy has been associated with transient neonatal sedation or withdrawal symptoms in infants. Features seen in these infants include hyperactivity, irritability and tremors. Perinatal or neonatal hemorrhage has been observed occasionally in infants of women who took phenobarbital late in pregnancy. This has been attributed to drug-induced suppression of the synthesis of vitamin K-dependent clotting factors.

Tegretol (Carbamazapine)
The use of tegretol during pregnancy has been associated with an increased risk for spina bifida of up to 1% (1/100), as compared to the general population risk of 1/1000. In addition, tegretol use during pregnancy has been associated with a unique facial appearance and the underdevelopment of the fingers, toes, and nails. The range of risk for non-specific developmental abnormalities is in the range of 6-15%.

Lamictal (Lamotrigine)
Data on the effects of this treatment are limited. A number of birth registries of exposed children have found no increased rate of major birth defects among registrants. One study found an increase in the rate of oral clefting; however this has yet to be observed in other registries. The data is too limited to exclude any small risks.

Neurontin (Gabapentin)
The data available on the effects of this anti-convulsant are insufficient to determine risks to fetal development. There are case reports of normal and abnormal outcomes. The few cases reported do not show a consistent pattern of birth defects.

Additional findings:
Studies have shown that children whose mothers were taking anti-convulsant drugs during pregnancy were more likely to have “additional educational needs”. Valproate and combination anti-convulsant therapies may be associated with a 2 to 3 fold increase for developmental delay. Tegretol was not associated with an increased risk for developmental delay.

Using a combination of two or more anti-convulsant medications may cause a further increase in the risk for birth defects due to the possible synergistic (interactive) effects of the medications.

Prenatal Surveillance for Birth Defects
Fetal imaging by ultrasound is recommended to screen for neural tube defects. Severe types of neural tube defects such as anencephaly may be detected as early as 11-12 weeks of gestation. Smaller neural tube defects like spina bifida are typically detectable by the 18th week of gestation. Routine maternal blood screening for alpha-fetoprotein at 16 weeks detects 85% of neural tube defects. Congenital heart defects, cleft lip and other major malformations may be screened for by ultrasound at 18-20 weeks. Minor findings such as unusual facial features or digit anomalies are not likely to be detected by ultrasound. Ultrasound imaging by a fetal ultrasonography specialist is recommended. Genetic counseling to discuss risks and surveillance plan is recommended.