HHS Public Access

Author manuscript

J Midwifery Womens Health. Author manuscript; available in PMC 2020 November 03.

Published in final edited form as:

J Midwifery Womens Health. 2018 January ; 63(1): 23–32. doi:10.1111/jmwh.12720.

PREGNANCY-ASSOCIATED STROKE

Bethany Sanders, CNM, MSN,
clinical practices at Vanderbilt faculty midwifery practice in Nashville, TN.

Melissa Davis, CNM, FNP, DNP,
director for the Vanderbilt School of Nursing Faculty Nurse-Midwife Practice and teaches in Vanderbilt University School of Nursing's midwifery and family nurse-practitioner programs.

Sharon Holley, CNM, DNP,
Chief of the Division of Midwifery at Baystate Medical Center, Springfield, Massachusetts.

Julia Phillippi, CNM, PhD
Assistant Professor of Nursing at Vanderbilt University School of Nursing in Nashville where she provides intrapartum care, performs midwifery research, and teaches doctoral students.

Abstract

Cerebrovascular accident, or 'stroke,' is the fourth leading cause of death for all women, and the eighth leading cause of pregnancy-associated death. The physiologic changes of pregnancy increase the risk of cerebrovascular accident for women. With current incidence rates, a facility with 3300 births per year can anticipate caring for a woman with a pregnancy-related stroke at least every 2 years. All maternity care providers must be able to assess women experiencing stroke-like symptoms and initiate timely care to mitigate brain tissue damage, decrease long-term morbidity, and prevent mortality. The 2 main types of stroke, ischemic and hemorrhagic, have similar presenting symptoms but very different pathophysiology and treatment. This article reviews assessment and initial treatment of pregnant and postpartum women experiencing stroke and provides guidance for subsequent maternity and primary care to assist front-line perinatal care providers who may be the first to treat affected women or may resume primary-care following diagnosis.

Precis

Physiologic changes in pregnancy can increase women's risk of stroke. Prompt recognition and differentiation of ischemic and hemorrhagic strokes improves outcomes.

Keywords

stroke; pregnancy; hypertension; cerebrovascular accident; ischemic stroke; hemorrhagic stroke; transient ischemic attack; hematologic changes of pregnancy; preeclampsia; gestational hypertension; venous thromboembolism

CORRESPONDING AUTHOR Melissa Davis, CNM, FNP, DNP, 461 21st Ave. S, Nashville TN, 37240, Melissa.g.davis@vanderbilt.edu.

CONFLICT OF INTEREST
The authors have no conflicts of interest to disclose

INTRODUCTION

Cerebrovascular accident, or "stroke," is the fifth leading cause of death in the United States, the fourth leading cause of death for all women, and the eighth leading cause of pregnancy-associated death.[1] Pregnancy-associated stroke is a cerebrovascular accident that occurs antepartum, intrapartum, or up to 6 weeks postpartum and causes damage to brain tissue. Physiologic changes in pregnancy increase the risk of stroke, especially for women with existing cardiovascular compromise or risk factors.[2] Strokes occur in non-pregnant women ages 15–49 years at a rate of 21 per 100,000 women. Pregnancy-associated strokes occur at a rate of approximately 34 per 100,000 births.[3] Rates of pregnancy-associated strokes have been increasing, mirroring the rising prevalence of obesity, cardiac disease, and hypertension in childbearing-age women.[4] There is significant maternal morbidity and mortality associated with pregnancy related stroke, 15% of affected women die and most stroke survivors experience residual weakness and cognitive dysfunction.[3,5] In addition, maternal stroke in pregnancy can affect fetal and neonatal health and well-being.[6]

This article explores pregnancy-associated stroke including types, pathophysiology, risk factors, symptoms, diagnosis, and initial clinical management. With current incidence rates, a facility with 3300 births per year can anticipate caring for one woman with a pregnancy-associated stroke every 9 months to 2 years.[4] All perinatal care providers must be able to understand risk factors for and symptoms of pregnancy-associated stroke to initiate timely care.

INCIDENCE OF PREGNANCY-ASSOCIATED STROKE

The incidence of stroke in pregnancy is difficult to determine as the terminology used for diagnosis and billing is not uniform, especially for pregnant women. Furthermore stroke mimics other conditions and may be misdiagnosed or assigned an incorrect billing code. It is estimated that stroke occurs in the perinatal period at a rate of 34.2 per 100,000 births in the United States,[4] and the rate appears to be increasing. Between 1994–1995 and 2010–2011, the rate of stroke in women with hypertensive disorders of pregnancy increased 103%.[7] Pregnancy-associated stroke can occur at any point during the perinatal period. Between 11% and 32% of all pregnancy-associated strokes occur during the antepartum period, 34% to 41% present during labor or birth, and 34% to 48% occur postpartum, within 6 weeks of birth.[7,8] While risk of stroke is elevated above non-pregnant levels until 12 weeks postpartum,[9] women who experience stroke during the postpartum period present on average at approximately 8 days postpartum,[4,10]

PATHOPHYSIOLOGY OF STROKE

The brain relies on a continuous flow of oxygenated blood to function. When blood flow is interrupted, levels of glucose, oxygen, sodium, and calcium become disrupted. Brain cells do not store glucose and therefore cannot function well following even small changes in the levels of nutrients and cell waste products. Temporary interruptions in blood flow and/or

exposure of the cells to blood that has leaked out of the vessels can induce inflammatory processes that further damages the cells.

The resulting lack of brain cell function leads to motor and sensory symptoms such as headaches, weakness, partial paralysis, speech difficulties, confusion, and loss of consciousness. Symptoms vary by the location and extent of the insult within the brain. If proper blood flow to and away from the brain can be re-established quickly, the cells can regain function. If the interruption in blood flow is prolonged, the cells will die. Since brain cells lack the ability to regenerate, loss of brain cells leads to morbidity such as long-term motor and cognitive deficits or even mortality.

While all cerebrovascular accidents involve disruptions in brain cell function, there are many types of strokes with differing pathophysiology. Strokes are categorized by the underlying cause of the brain injury. The 2 main types of strokes seen in the perinatal period are ischemic and hemorrhagic strokes. Women with either type will present with similar symptoms, but accurate diagnosis of the type of insult is essential to begin treatment and preserve long-term function.

Transient Ischemic Attack

A transient ischemic attack (TIA) is an episode of temporary and focal dysfunction of vascular origin. Transient ischemic attack is not a type of pregnancy-associated stroke per se but TIAs precede strokes in up to 15% of cases.[11] Transient ischemic attacks are variable in duration and usually lasts from 2–15 minutes but can persist for up to 24 hours. Most importantly, there is no permanent neurologic deficit associated with a TIA, unlike with a stroke.

Ischemic Stroke

An ischemic stroke occurs when blood flow to an area of the brain is blocked or interrupted, causing cell death.[12] Eighty percent of all strokes and the majority of pregnancy-associated strokes are ischemic.[8,11] The most common cause of blockage is a thrombus, a blood clot that has been dislodged from an original location that comes to rest in a vessel within the central nervous system, blocking blood flow. Other emboli, such as atherosclerotic plaques, can also block cerebral blood flow, but these etiologies of ischemic stroke are less commonly associated with strokes that occur during the perinatal period.

Ischemic strokes occur in pregnancy at a rate of 18 per 100,000 births[13] and are often associated with maternal conditions such as coagulation disorders that increase the likelihood of clot formation. Other maternal conditions associated with increased risk of ischemic stroke include cardiac abnormalities and abnormal blood vessel shape or integrity such as occurs during carotid dissection. In these women, blood does not flow smoothly and becomes stagnant in the area around the anatomic defect, which increases the risk of clot formation. These clots can then dislodge and circulate until they stop in a small brain vessel. If the clot can be lysed and blood flow restored rapidly enough, then cell death and permanent morbidity can be avoided. Clots can dissolve or dislodge spontaneously, as occurs in a temporary ischemic attack, or medical treatments can be used to lyse the thrombus.

A rare cause of ischemic stroke is a cerebral venous thrombosis. This occurs when thrombi originate in the cerebral or dural sinus veins within the brain.[14] Cerebral venous thrombosis accounts for approximately 2% of pregnancy-associated strokes, with the highest risk of occurrence during the third trimester and postpartum.[15] Factors that can contribute to the development of cerebral venous thrombosis include: maternal vessel wall injury, infection, dehydration, smoking, advanced age, and recent cesarean.[4,16]

Hemorrhagic Stroke

Hemorrhagic strokes occur when one or more blood vessels break that disrupts flow of nutrients and waste products and results in blood coming into direct contact with brain tissues which initiates an inflammatory cascade. The lack of circulating blood supply along with the mechanical forces of the blood, edema, and inflammatory response result in damage to the brain tissue.[14] The amount of blood and the inflammatory response in the brain tissue can increase the intracranial pressure, which causes some of the early symptoms.

Hemorrhagic strokes are less common than ischemic strokes but associated with higher maternal mortality. A population-based study in the United Kingdom conducted between 2007 and 2010 found the incidence of hemorrhagic stroke in pregnancy to be 0.6 per 100,000 births.[17] According to US data, intracranial hemorrhage has the highest risk of maternal mortality of all pregnancy-associated strokes and is responsible for 5% to 12% of all maternal deaths.[15,18]

Underlying risk factors for hemorrhagic stroke include blood vessel abnormalities that lead to weakening of the vessel wall in response to stress. Integrity of the vessel wall can be affected by congenital defects, such as arteriovenous malformations, where the vessels or vessel walls were abnormally formed, or connective tissue disorders that impair formation of strong vasculature.[8] Vessel defects can also develop over time such as would occur with an aneurysm.[19] Vasculature structure can also be weakened through hypertension or arteriosclerosis as vessel walls become rigid an inflexible.[19] Compromised vessels are prone to injury and rupture when stressed by the increased blood volume of pregnancy or conditions such as pregnancy-induced hypertension.[19]

PHYSIOLOGIC CHANGES OF PREGNANCY RELATED TO RISK OF STROKE

The hormones of pregnancy cause wide-ranging physiologic changes. The increase in blood volume, changes in vasculature distensibility, and hypercoagulable state, increase a woman’s risk of stroke, especially if she has underlying abnormalities.

Blood Volume Changes

Blood volume begins to increase during the first weeks of pregnancy and, in women with normal pregnancies, the volume of whole blood continues to increase until it reaches levels 45% higher than pre-pregnancy levels.[20] The increase in blood volume can cause strain on the vessel walls. This strain, combined with underlying vessel damage present when a woman has hypertension, increases the risk of hemorrhagic stroke.[14,19] Blood volume changes rapidly following birth related to postpartum blood loss and physiologic decreases in total body water. However, the risk of stroke remains elevated until approximately 12

weeks postpartum in part related to cardiovascular changes such as alterations in clotting factors and increased vessel capacity.[9,11]

Cardiovascular Changes

The cardiovascular system adjusts to accommodate and transport the additional blood volume by relaxing the vessels and increasing cardiac output. Estrogen, progesterone, and increases in relaxin, contribute to vasodilation.[21] Systemic vascular resistance falls during pregnancy beginning as early as 5 weeks gestation.[20,22] This hormonally-mediated vasodilation contributes to lower systolic blood pressure in the second and third trimesters in normal pregnancy.[20] While these vascular changes are needed to support blood flow to the placenta and accommodate increased blood volume, if there is an existing abnormality in the woman's vasculature, the stress of the increased blood volume or the increased distensibility of the vessel wall also increases the risk of hemorrhagic stroke.[11]

Cardiac output also increases during pregnancy. Beginning in the first trimester, cardiac output rises to 45% above the non-pregnant state by the 24th week of gestation.[20] During labor and birth, cardiac output further increases to 60% to 80% above pre-pregnant levels.[20,22] This increased cardiac output may exacerbate or cause structural changes, resulting in turbulent blood flow around cardiac or major vessel abnormalities, increasing the risk of formation of clots that can dislodge and cause ischemic strokes.

The cardiovascular system is also affected by the growing weight of the uterus. The pressure of the uterus can inhibit or slow blood return from the lower extremities, especially in the third trimester. Decreased physical movement during late pregnancy, labor, and postpartum may also contribute to venous pooling in lower extremities and increased clot formation. Labor and birth may also cause tissue and vessel injuries that can trigger clot formation.[19] For example, as the fetus distends the vagina and perineum, tissues and vessels can be damaged.

Dramatic changes to maternal cardiovascular load occur immediately postpartum. Blood volume decreases, and maternal cardiac output initially increases. Once blood volume is stabilized with adequate tissue perfusion, cardiac output decreases to pre-labor levels after 1–2 hours postpartum.[21] Although the maternal blood volume returns to pre-pregnant levels quickly, often within the week,[23] vessel resistance is slower and typically returns to pre-pregnant levels by 2 weeks postpartum.[23] During the time vessel capacity is still increased and blood volume is decreasing, there may be areas within the vascular system where blood can become stagnant and form clots. While the vast majority of women can accommodate the increased vascular load in pregnancy and the rapid postpartum resolution, those with cardiovascular conditions are at increased risk for clot formation or vascular compromise resulting in stroke.[22]

Coagulation Changes

Pregnancy is a hypercoagulable state in which blood factors that induce coagulation are increased and factors that prevent or lyse clots are present in lower amounts.[21,23] The formation of clots involves a cascade of factors. As a result, pregnant women are more likely to form and retain clots, especially around new or existing vasculature defects and in the

lower extremities.[23] Beginning at 11 weeks of pregnancy, there is an increase in factors I, VII, VIII, IX, X, XII, XIII, and von Willebrand factor; all of which promote coagulation.[21] Factors that inhibit clot formation, including protein S and protein C, fall.[14,24] Factors involved in the breakdown of clots, known as fibrinolysis, also decrease.[14,24] This decrease in fibrinolytic activity in pregnancy increases the likelihood that clots circulate around the body.[24]

The changes to the clotting cascade toward coagulation and decreased fibrinolysis is most pronounced in pregnancy and in the immediate postpartum period. The ability to form and retain clots reverts to non-pregnant levels usually within 4 weeks postpartum.[11,25] Women are at increased risk of pregnancy-related stroke until the hypercoagulable state resolves.

If women have a genetic pre-disposition for increased clotting or decreased fibrinolysis their ability to form and maintain clots may be further enhanced. If these women also have factors that contribute to clot formation, such as vascular defects or injuries, their risk of a thromboembolism is greatly increased.

MATERNAL RISK FACTORS FOR PREGNANCY-ASSOCIATED STROKE

The risk factors associated with pregnancy-associated stroke include: smoking, race, age younger than 20 years or older than 35 years, obesity, hypertension, sickle cell disease, autoimmune disorders, migraine headaches with aura, patent foramen ovale, and vascular malformations. Nearly two-thirds of the women who experience a pregnancy-associated stroke are smokers.[4,16,26] Elevated body mass index (BMI) is associated with increased risk of ischemic stroke in women of childbearing age.[27]

Race and ethnicity are linked with both the incidence and the severity of strokes. These linkages are thought to be due to genetic factors, health behaviors, as well as access to preventative and emergency services. African American women older than 35 years are at increased risk for stroke when compared to Hispanic and Caucasian women of the same age group.[3] However, Hispanic women have an increased risk of death if a stroke occurs during pregnancy.[4,14]

Women with a genetic predisposition to clot formation, known as a thrombophilia, are at increased risk for ischemic stroke. Women with known clotting disorders may benefit from a consult to have an individualized determination if anticoagulation therapy would be valuable and to develop a medication plan if needed.[28] Women with a family history of venous thromboembolism or a first-degree relative with a history of a high-risk thrombophilia should be assessed to determine if screening for clotting disorders would affect their care.[28] Screening is controversial as prophylaxis has risks and may not improve pregnancy outcomes. In addition, not all diagnostic tests are reliable in pregnancy or in the presence of an obstructive clot.[29]

Vascular malformation also increases the risk of both hemorrhagic and ischemic stroke. Vessel abnormalities, such as aneurysm and arterio-venous malformations, are a risk factor for hemorrhagic stroke.[30] Cardiovascular disease also affects vessel integrity and damaged or abnormally formed vessels are more prone to development of clots. Traditional vascular

risk factors for stroke, such as hyperlipidemia, do not seem to contribute to risk for pregnancy-associated stroke.[31]

The stresses related to pregnancy can also accentuate pre-existing cardiovascular abnormalities. For example, in 25% of adults the foramen ovale that divides the left and right atrium remains closed only due to the pressure differential between the right (low pressure) and the left (high pressure) atria. Changes in pregnancy, and especially during periods of high intrathoracic pressure during pushing, can allow the foramen ovale to open, allowing blood, and potentially clots, to spill over from the right into the left atrium.[4,32] A patent foramen ovale increases the chance that a thrombus will come to rest in the brain rather than in the lungs. In addition, a patent foramen ovale disrupts blood flow within the heart, increasing the risk of clot formation. Percutaneous device closure of patent foramen ovale may be used in pregnant women with ischemic stroke to reduce risk of recurrence.[33]

PREGNANCY-RELATED DISORDERS THAT INCREASE STROKE RISK

Certain pregnancy-related disorders increase the risk for stroke. Hypertensive disorders of pregnancy are risk factors for all types of pregnancy-associated stroke.[3,4,7,8] These disorders include gestational hypertension, preeclampsia, and HELLP (hemolysis, elevated liver enzymes, and low platelets syndrome).[3,4,7,8]

Preeclampsia is the single greatest risk factor for pregnancy-associated stroke. Even women with moderately elevated blood pressures are at risk.[34] While the pathophysiology of preeclampsia and eclampsia is not thoroughly understood, the underlying changes to the cardiovascular system compound the pregnancy-related changes that increase women's risk for stroke.[19] Women with severe preeclampsia and eclampsia are at the greatest risk of stroke.[34] Eclampsia can result in posterior reversible encephalopathy, which is associated with hemorrhagic stroke in 10% to 15% of women who develop eclampsia.[35]

Postpartum hemorrhage, fluid and electrolyte imbalance, blood transfusion, anemia, and infection are also risk factors for stroke.[8] Postpartum hemorrhage is often treated with methylergonovine (Methergine), which has a side effect of transient hypertension and increases stroke risk. Anemia following postpartum hemorrhage can lead to cerebral hypoperfusion, increasing the risk of ischemic stroke. The process of storing and preserving red blood cells for transfusion increases the cell aggregability that increases the risk of ischemic stroke due to thrombus formation following transfusion.[8]

Cesarean birth increases the likelihood of stroke.[3,4] After adjusting for other risk factors, one retrospective population-based study conducted in Taiwan evaluating ICD-9 codes, reviewed the charts of 987,010 singleton births. A 44.7% higher incidence of stroke was reported in the 3 months following cesarean birth when compared to women who had a vaginal birth, and the increased incidence persisted even out to 12 months postpartum.[36] Using national representative sampling of US data from 1993–1994, researchers reported a risk of 34.3 peripartum strokes per 100,000 births for women who have a cesarean birth compared to a risk of 7.1 peripartum strokes per 100,000 births in women who have a vaginal birth ($P<0.001$).[5] Explanations for this increase include surgery-related hemodynamic changes,

changes in protein C levels related to stress response, anesthesia-related hemodynamic changes and underlying conditions for which cesarean birth was indicated.[5,36]

Hemodynamic changes during cesarean birth include increased maternal stroke volume and cardiac output following delivery of the placenta.[36] In addition, tissue damage from surgery increases a woman's tendency toward clot formation.[5] The combination of surgical and pregnancy-induced changes in coagulation may further increase the risk of thrombosis development. The use of spinal anesthesia during cesarean birth affects maternal hemodynamics and may further compound the risk of pregnancy-associated stroke in women undergoing operative birth.[36]

DIAGNOSIS OF CEREBROVASCULAR ACCIDENT

When women present with symptoms consistent with stroke, rapid assessment, including history, vital signs, physical assessment, laboratory tests, and cardiac monitoring is needed. (Table 1) This initial information is needed to determine the differential diagnoses as stroke symptoms mimic many other, more common, conditions. Following these first steps, if stroke remains as a likely cause, immediate brain imaging is warranted. While both major types of stroke present in a similar manner, treatment differs significantly.

Signs and Symptoms

The signs and symptoms of ischemic and hemorrhagic stroke are similar. There is considerable overlap in initial presentation and examination findings. Headache is the most common presenting symptom for women experiencing pregnancy-associated stroke. Often described as the "worst headache of my life," the pain is severe. Characteristics of headache onset vary. In some women it begins in a mild form and gets progressively worse.[16] In others, especially those experiencing cerebral venous thrombosis, the severe pain begins suddenly and without warning, known as a 'thunderclap' headache.[11]

Women may also present with alterations in neurologic function such as confusion, disturbed consciousness, or psychosis-like symptoms depending on the portion of the brain affected.[14] Motor function on one side of the body may also be affected.[14] Women may also present with symptoms of increased intracranial pressure, such as vomiting and seizures.[9–11,14] Women experiencing a hemorrhagic stroke may have symptoms of intracranial inflammation such as neck stiffness.[14]

Differential Diagnoses

The differential diagnoses of women who present with severe and unremitting headache include migraines and preeclampsia. Neurological alterations are also seen in women with hypoglycemia, Bell's palsy and postpartum psychosis.[37]A rapid clinical assessment, including a thorough neurologic examination is helpful in ruling in or out these conditions. Many alternative diagnoses do not need immediate treatment to prevent mortality and long-term morbidity. Stroke, however, requires immediate intervention to preserve brain function. Therefore, unless the alternative diagnosis is certain, testing and imaging to rule out stroke is indicated.

Vital Signs

Immediately on presentation, vital signs should be assessed, including pulse, blood pressure, respirations, and oxygen saturation. Pulse oximetry is useful as maternal hypoperfusion can impair fetal oxygenation and exacerbate maternal cerebral inflammation.[37]

Neurological Examination

A thorough neurologic examination focuses on the gross motor examination. A brief cranial nerve assessment should be conducted, and extremity strength assessed for unilateral weakness. If altered level of consciousness is suspected, rapid initiation of assistance from an interprofessional team is indicated.

While being careful to not prematurely rule out stroke, Bell's palsy is a more common diagnosis in pregnant women.[38] Bell's palsy involves only peripheral facial nerve weakness, while a stroke involves central, or cortical, nerve involvement. Hallmark features of Bell's palsy include sudden onset of a drooping eye, the absence of a nasolabial fold on the affected side, and a drooping corner of the mouth.[38] Preservation of upper facial movement is a good indicator that there is cortical nerve involvement.[38] Individuals with Bell's palsy remain lucid and deny other neurologic symptoms such as headache. Women with a definitive diagnosis of Bell's palsy do not need further laboratory tests or imaging.

Laboratory Tests

Women experiencing stroke-like symptoms need blood tests including levels of glucose, electrolytes, renal and liver function, complete blood count with platelets, a prothrombin time/international normalized ratio, an activated partial thromboplastin time, and baseline markers of cardiac ischemia.[37] When evaluating stroke symptoms, a troponin level is preferred over the other cardiac markers. Other laboratory tests, such as HbgA1C and a toxicology screening can be useful.[37]

Cardiac Monitoring

Women experiencing symptoms of stroke also benefit from 24 hours of cardiac monitoring because arrhythmias, such as atrial fibrillation, can contribute to emboli formation.[39,40] A 12-lead electrocardiogram is appropriate to monitor cardiac rhythm throughout assessment, diagnosis, and initial treatment.

Brain Imaging

If rapid assessment supports cerebrovascular compromise as the possible cause of the woman's symptoms, immediate imaging is indicated to make an accurate diagnosis.[39] Both computed tomography (CT) and magnetic resonance imaging (MRI) without the use of contrast are safe for pregnant and breastfeeding women. Computed tomography of the woman's head exposes the fetus to an estimated 50 mrad of radiation, which is below the 500 mrad considered safe.[41] Magnetic resonance imaging does not use ionizing radiation and is therefore safe for the fetus. Of the 2 modalities, CT without contrast is generally the imaging of choice.[19]

Contrast is used frequently in diagnostic imaging of the brain to visualize vascular structures; however, contrast agents are not safe in pregnancy. Computed tomography contrast with iodinated agents should be avoided during pregnancy due to association with neonatal hypothyroidism, but may be safely used when the woman is postpartum and breastfeeding.[19] Magnetic resonance imaging contrast with gadolinium agents should be avoided during pregnancy due to concern for developmental abnormalities and miscarriage or stillbirth, but is safe for use postpartum and in breastfeeding women.[19]

CLINICAL MANAGEMENT OF STROKE

For all individuals experiencing a cerebrovascular accident, the most critical time is the hyperacute phase, within the first 24 hours. The acute phase begins at 24 hours and lasts until one week after the event.[40] Rapid intervention preserves brain function and decreases long-term morbidity and mortality. While women need a multi-disciplinary team including neurological specialists to provide care, front-line perinatal healthcare workers may need to initiate treatment while awaiting referral to other facilities or providers. Consultation with specialists across the regional perinatal care network can improve long-term maternal and fetal prognosis. All front-line perinatal providers should be familiar with clinical management steps to improve timeliness of essential care. In addition, knowledge of common treatments is useful for when women return to community-based care.[39]

Ischemic Stroke

Initial treatment of ischemic stroke is focused on removing the blockage of cerebral blood flow. This can be accomplished using medications or through physical removal of the thrombus or embolus. Once a diagnosis of ischemic stroke is made, all women should receive intravenous normal saline, supplemental oxygen as needed, and blood glucose and electrolyte correction if necessary.[37] Medications to correct blood pressure are not usually provided as they can further decrease cerebral perfusion.[37]

Medications—There are 2 major medications for treatment of clots, recombinant tissue plasminogen activator (rtPA) and unfractionated heparin or subcutaneous low-molecular weight heparin (LMWH). Specialist consultation is needed to determine the type of treatment, but front-line maternity care providers may be administering these drugs to preserve brain function while awaiting transfer. Therefore, an understanding of these medications is important to ensure informed consent and proper monitoring following administration.

Recombinant tissue plasminogen activator is administered either intravenously or intra-arterially and lyses clots to restore blood flow.[39,42] Administration of rtPA within 3 hours of ischemic stroke symptom onset is associated with improved functional outcomes at 24 hours and 3 months after ischemic stroke.[43] Current guidelines allow for the use of rtPA up to 4.5 hours after ischemic stroke symptom onset in select patients.[39] While rtPA is useful in lysing clots, the risk of intracranial hemorrhage is approximately 6% higher after administration.[43]

Clinical studies of rtPA excluded pregnant women; current drug labelling encourages providers to explore the risk and benefits to the woman and her fetus prior to use.[39,44] The

molecular size of rtPA is too large to cross the placenta and animal studies have shown neither teratogenicity nor effects on postnatal development.[44] A number of case studies have been published detailing the use of rtPA in pregnant women and analysis of these case reports found that failure and complication rates of thrombolysis with rtPA in pregnant women was similar to those in the general population.[44]

Administration of rtPA increases the risk of bleeding and in pregnant women this raises concerns about antepartum, fetal, or postpartum hemorrhage.[14] Of the 28 women with rtPA use during pregnancy that were evaluated in the collected analysis of case reports, there were 2 fetal deaths in which a causal relationship to rtPA was determined.[44] In the surviving infants, no adverse effects were noted.[44] Another analysis of thrombolysis specifically for pregnancy-associated ischemic stroke concluded that treatment should not be withheld from pregnant women but risks and benefits to the woman and her fetus must be carefully weighed in each individual case.[45]

Vascular blockages from clots can also be treated with LMWH, especially if the diagnosis of ischemic stroke is made outside of the time-sensitive window for rtPA treatment.[4] Heparin has a large molecular weight and does not cross the placenta. LMWH is considered safe in pregnancy and has been in widespread use for clotting prophylaxis for many years.[4] Bleeding is a potential side effect of LMWH; women are usually instructed to stop LMWH 24 hours before planned birth or with the onset of labor symptoms.[46] However, use is not associated with significant obstetric complications, including postpartum hemorrhage. Once the clots causing the cerebral blockage are dissolved, the woman is often placed on a lower dose of LMWH designed to prevent further clot formation.[47]

Mechanical treatments—Endovascular thrombolysis or mechanical thrombectomy are other treatments for ischemic stroke, particularly when the woman is not a candidate for rtPA. Decisions regarding treatment are highly individualized and require consultation with a multidisciplinary team; however, pregnancy is not an absolute contraindication to endovascular thrombolysis or mechanical thrombectomy.[4] Risks and benefits to both the woman and fetus must be carefully considered when making decisions regarding treatment modalities.

Hemorrhagic Stroke

Treatment of hemorrhagic strokes focuses on stopping intracranial bleeding, alleviating pressure on brain tissues, and preventing future vascular compromise.[48] Detailed imaging will be used to determine the type and location of the bleeding. The plan of care will depend on many factors, including the potential for recovery and the risk of future injury from abnormal vasculature. Less invasive surgical correction using instruments threaded through vessels to the site of injury may be possible or surgeons may need full access to the brain tissues.[48]

CARE OF WOMEN WITH A HISTORY OF STROKE

Primary Care

Primary care and preventative appointments are excellent opportunities to discuss risk factors for stroke, both general and pregnancy specific. The diagnosis of preeclampsia has lifelong implications and is associated with a 2-fold increase in the risk of cerebrovascular accident (CVA) later in life.[3] It is recommended that women report a history of preeclampsia to all primary care providers, and the diagnosis should be noted in her medical record.

A woman with a history of preeclampsia or stroke during pregnancy can benefit from information about lifestyle changes that may reduce her risk of future stroke. Lifestyle modifications that reduce the risk of stroke include maintaining normal BMI, blood pressure, glucose, and cholesterol levels.[49] Smoking cessation can decrease stroke risk as well.[49] By reducing modifiable risk factors, it is estimated that the cardiovascular risk of a woman who had preeclampsia can be reduced by 4% to 13%.[49] One of the barriers to educating women regarding the risk of stroke is lack of health care provider knowledge that preeclampsia is a risk factor for later vascular diseases.[50]

Women who have experienced a stroke also need information to help them in reproductive life planning. The most commonly reported reason for not conceiving is concern about recurrent risk of stroke.[51] However, the risk of recurrence is 1% within the first year of the initial stroke and 2.3% within the first 5 years. Thus, a history of pregnancy-associated stroke not an absolute contraindication to becoming pregnant again.[51]

When a woman with a history of stroke desires pregnancy, a preconception or early pregnancy consultation with a maternal fetal medicine specialist can be beneficial to plan her care. If she has a thrombophilia, use of prophylactic LMWH during pregnancy may be recommended.[28] There is some evidence that women with a history of cerebral venous thrombosis have a higher rate of miscarriage during subsequent pregnancies (21.1% compared to 10%−15% of the general population).[41]

Many contraception options are available for women who have a history of stroke, but others are contraindicated. Even women with severe stroke-related disabilities retain their fertility and can be sexually active and at risk for pregnancy. Sterilization is an option for women who are certain they do not want to conceive in the future. Long-acting contraception methods are preferred for women who want to retain their fertility but do not currently desire pregnancy.[52]

If a woman prefers shorter-acting contraceptives, barrier methods can be used, even though they have higher failure rates.[52] Progestin-only pills can also be used, but require careful attention to timely administration, and their safety in women with a history of stroke has not been established.[53] Estrogen-containing contraceptives, including pills, patches and rings, are contraindicated as they increase the risk of thromboembolism.[54,55]

Maternity Care

Women with a history of stroke benefit from collaborative management during pregnancy. Care is highly dependent on their comorbidities and functional status. For example, a woman with a history of a previous pregnancy affected by preeclampsia can benefit from aspirin as a form of pre-eclampsia prophylaxis in subsequent pregnancies. [29] The rate of recurrent hypertensive disease of pregnancy in future pregnancies is 20%.[56] New research supports that preeclampsia can be prevented or the severity lessened through the use of aspirin. The American College of Obstetricians and Gynecologists (ACOG) recommends low dose aspirin (60–80 mg daily) starting in late first trimester for women who had early onset preeclampsia and preterm delivery at less than 34 0/7 weeks gestation or for women who developed preeclampsia in more than one prior pregnancy.[29] ACOG also states that calcium supplementation may reduce the severity of preeclampsia in populations with inadequate calcium intake. The American Heart Association and American Stroke Association guidelines recommend daily low dose aspirin starting after 12 weeks' gestation and continuing until birth for women with chronic primary or secondary hypertension or previous pregnancy-related hypertension.[3]

Labor and birth present a risk of damage to pelvic floor blood vessels, thereby increasing the risk of venous thrombosis.[41] Operative vaginal delivery is associated with an increased risk of venous thrombosis compared to spontaneous vaginal birth.[46] The safety of vaginal birth in women with a history of pregnancy related stroke has not been extensively studied; however, extant research indicates vaginal birth is not contraindicated in women with pregnancy-associated stroke.[57] This recommendation was based on the low absolute risk of intracranial hemorrhage in pregnant women, despite an increase in intracranial pressure associated with second stage pushing efforts. Pregnant women with subarachnoid hemorrhage resulting from aneurysm have improved maternal and fetal outcomes after neurosurgical intervention, and mode of delivery does not negatively affect maternal complication rates.[48] Cesarean delivery may be recommended if an aneurysm is diagnosed at term or if neurosurgery has been performed within a week of giving birth.[41] Intrapartum care will need to be personalized to the health of the woman and her fetus as well as the location and availability of advanced medical care.

Women who have experienced a pregnancy-associated stroke are likely to have ongoing morbidity postpartum and continue to require an interprofessional team of healthcare workers including social work, home health, and mental health professionals. Common postpartum difficulties may be even more challenging for affected women. Mobility restriction can complicate infant care and activities of daily living and women may require ongoing help. Comprehensive lactation support can assist the woman in breastfeeding or pumping, if needed, while she receives medical care and ensure that maternal medications are compatible with breastfeeding. Women affected by pregnancy-associated stroke may need referral for mental health services in addition to routine screening for postpartum depression.

The woman's need for medications for treatment and stroke prophylaxis can change postpartum. Anticipatory guidance on the value of medications for prevention of maternal

morbidity are important. With clear instructions on medication continuation, 93% of women are compliant with postpartum heparin administration.[47]

CONCLUSION

While hypertension and preeclampsia are risk factors, pregnancy-associated stroke is difficult to predict but associated with major maternal morbidity and mortality. The 2 major types of pregnancy-associated stroke, ischemic and hemorrhagic strokes have similar presenting symptoms, but their underlying pathophysiology and treatment are very different. All perinatal care providers should be able to assess women presenting with symptoms of stroke and be familiar with initial steps in treatment, especially if they do not work directly in tertiary care centers with specialist help nearby. While specialist consultation is indicated, prompt assessment can allow for timely treatment even as referral plans are being made. As first-line providers in a levelled care system, all maternity care providers are likely to encounter women in the acute phase of stroke; a time when prompt diagnosis and treatment can greatly affect outcomes. In addition, maternity care providers may be involved in the primary, antepartum, intrapartum, or postpartum care of women with a recent or distant history of stroke. More research is needed on stroke in pregnant women to reduce the morbidity and mortality associated with this potentially catastrophic event.

ACKNOWLEDGEMENTS

During manuscript production, Dr. Julia C. Phillippi was supported by grant number K08HS024733 from the Agency for Healthcare Research and Quality. The content is solely the responsibility of the authors and does not necessarily represent the official views of the Agency for Healthcare Research and Quality.

References

1. Kochanek K, Xu J, Murphy S, Arias E. Mortality in the United States, 2013. NCHS Data Brief, No. 178. Hyattsville, MD: National Center for Health Statistics, Centers for Disease Control and Prevention, Department of Health and Human Services 2014 https://www.cdc.gov/nchs/products/databriefs/db178.htm. Accessed September 2016.
2. Darlington A, McBroom K, Warwick S. A northwest collaborative practice model. Obstet Gynecol. 2011;118(3):673–677. [PubMed: 21860299]
3. Bushnell C, McCullough LD, Awad IA, et al. Guidelines for the prevention of stroke in women: a statement for healthcare professionals from the American Heart Association/American Stroke Association. Stroke. 2014;45(5):1545–1588. [PubMed: 24503673]
4. Moatti Z, Gupta M, Yadava R, Thamban S. A review of stroke and pregnancy: incidence, management and prevention. Eur J Obstet Gynecol Reprod Biol. 2014;181:20–27. [PubMed: 25124706]
5. Lanska DJ, Kryscio RJ. Risk factors for peripartum and postpartum stroke and intracranial venous thrombosis. Stroke. 2000;31(6):1274–1282. [PubMed: 10835444]
6. Murphy SL, Kochanek KD, Xu JQ, Arias E. Mortality in the United States, 2014. NCHS Data Brief, No. 229. Hyattsville, MD: National Center for Health Statistics, Centers for Disease Control and Prevention, Department of Health and Human Services 2015 https://www.cdc.gov/nchs/products/databriefs/db229.htm.
7. Leffert LR, Clancy CR, Bateman BT, Bryant AS, Kuklina EV. Hypertensive disorders and pregnancy-related stroke: frequency, trends, risk factors, and outcomes. Obstet Gynecol. 2015;125(1):124–131. [PubMed: 25560114]
8. James AH, Bushnell CD, Jamison MG, Myers ER. Incidence and risk factors for stroke in pregnancy and the puerperium. Obstet Gynecol. 2005;106(3):509–516. [PubMed: 16135580]

9. Kamel H, Navi BB, Sriram N, et al. Risk of a thrombotic event after the 6-week postpartum period. N Engl J Med. 2014;370(14):1307–1315. [PubMed: 24524551]
10. Witlin AG, Mattar F, Sibai BM. Postpartum stroke: a twenty-year experience. Am J Obstet Gynecol. 2000;183(1):83–88. [PubMed: 10920313]
11. Grear KE, Bushnell CD. Stroke and pregnancy: clinical presentation, evaluation, treatment, and epidemiology. Clin Obstet Gynecol. 2013;56(2):350–359. [PubMed: 23632643]
12. Adams HP Jr., Bendixen BH, Kappelle LJ, et al. Classification of subtype of acute ischemic stroke: definitions for use in a multicenter clinical trial. Stroke. 1993;24(1):35–41. [PubMed: 7678184]
13. Jaigobin C, Silver FL. Stroke and pregnancy. Stroke. 2000;31(12):2948–2951. [PubMed: 11108754]
14. Beal CC, Faucher MA. Stroke and pregnancy: an integrative review with implications for neuroscience nurses. J Neurosci Nurs. 2015;47(2):76–84; quiz E71. [PubMed: 25700192]
15. Tate J, Bushnell C. Pregnancy and stroke risk in women. Womens Health (Lond). 2011;7(3):363–374. [PubMed: 21612356]
16. Bashiri A, Lazer T, Burstein E, et al. Maternal and neonatal outcome following cerebrovascular accidents during pregnancy. J Matern Fetal Neonatal Med. 2007;20(3):241–247. [PubMed: 17437226]
17. Scott CA, Bewley S, Rudd A, et al. Incidence, risk factors, management, and outcomes of stroke in pregnancy. Obstet Gynecol. 2012;120(2 Pt 1):318–324. [PubMed: 22825091]
18. Dias MS, Sekhar LN. Intracranial hemorrhage from aneurysms and arteriovenous malformations during pregnancy and the puerperium. Neurosurgery. 1990;27(6):855–865; discussion 865–856. [PubMed: 2274125]
19. Razmara A, Bakhadirov K, Batra A, Feske SK. Cerebrovascular complications of pregnancy and the postpartum period. Curr Cardiol Rep. 2014;16(10):532. [PubMed: 25239155]
20. Sanghavi M, Rutherford JD. Cardiovascular physiology of pregnancy. Circulation. 2014;130(12):1003–1008. [PubMed: 25223771]
21. Blackburn ST. Maternal, Fetal, & Neonatal Physiology: A clinical perspective, 4th ed. Maryland Heights, MO: Elsevier; 2013.
22. Tan EK, Tan EL. Alterations in physiology and anatomy during pregnancy. Best Pract Res Clin Obstet Gynaecol. 2013;27(6):791–802. [PubMed: 24012425]
23. Monga M Maternal cardiovascular, respiratory, and renal adaptation to pregnancy In: Creasy RK, Resnik R, Iams JD, Lockwood CJ, Moore T, Greene MF, eds. Creasy and Resnik's Maternal-Fetal Medicine: Principles and practice. 7th ed. Philadelphia: Elsevier; 2013.
24. Brenner B Haemostatic changes in pregnancy. Thromb Res. 2004;114(5–6):409–414. [PubMed: 15507271]
25. Saha P, Stott D, Atalla R. Haemostatic changes in the puerperium '6 weeks postpartum' (HIP Study) - implication for maternal thromboembolism. BJOG. 2009;116(12):1602–1612. [PubMed: 19681851]
26. Savitz DA, Danilack VA, Elston B, Lipkind HS. Pregnancy-induced hypertension and diabetes and the risk of cardiovascular disease, stroke, and diabetes hospitalization in the year following delivery. Am J Epidemiol. 2014;180(1):41–44. [PubMed: 24879314]
27. Dehlendorff C, Andersen KK, Olsen TS. Body mass index and death by stroke: no obesity paradox. JAMA Neurol. 2014;71(8):978–984. [PubMed: 24886975]
28. American College of Obstetricians Gynecologists. ACOG Practice Bulletin No. 138. Inherited thrombophilias in pregnancy. Obstet Gynecol. 2013;122(3):706–717. [PubMed: 23963422]
29. American College of Obstetricians and Gynecologists Committee on Obstetric Practice, The Society for Maternal-Fetal Medicine. ACOG ommittee Opinion No. 560: Medically indicated late-preterm and early-term deliveries. Obstet Gynecol. 2013;121(4):908–910. [PubMed: 23635709]
30. Ogilvy CS, Stieg PE, Awad I, et al. Recommendations for the management of intracranial arteriovenous malformations. A statement for healthcare professionals from a special writing group of the Stroke Council, American Stroke Association. Stroke. 2001;32(6):1458–1471. [PubMed: 11387517]

31. Miller EC, Yaghi S, Boehme AK, et al. Mechanisms and outcomes of stroke during pregnancy and the postpartum period: a cross-sectional study. Neurol Clin Pract. 2016;6(1):29–39. [PubMed: 26918201]
32. Chen L, Deng W, Palacios I, et al. Patent foramen ovale (PFO), stroke and pregnancy. J Investig Med. 2016;64(5):992–1000.
33. Dark L, Loiselle A, Hatton R, Bhagwandeen R, Collins N. Stroke during pregnancy: therapeutic options and role of percutaneous device closure. Heart Lung Circ. 2011;20(8):538–542. [PubMed: 21459671]
34. Miller EC, Gatollari HJ, Too G, et al. Risk factors for pregnancy-associated stroke in women with preeclampsia. Stroke. 2017;48(7):1752–1759. [PubMed: 28546324]
35. Martin JN Jr., Thigpen BD, Moore RC, et al. Stroke and severe preeclampsia and eclampsia: a paradigm shift focusing on systolic blood pressure. Obstet Gynecol. 2005;105(2):246–254. [PubMed: 15684147]
36. Lin SY, Hu CJ, Lin HC. Increased risk of stroke in patients who undergo cesarean section delivery: a nationwide population-based study. Am J Obstet Gynecol. 2008;198(4):391.e391–397.
37. Zweifler RM. Initial assessment and triage of the stroke patient. Prog Cardiovasc Dis. 2017.
38. Zandian A, Osiro S, Hudson R, et al. The neurologist's dilemma: a comprehensive clinical review of Bell's palsy, with emphasis on current management trends. Med Sci Monit. 2014;20:83. [PubMed: 24441932]
39. Jauch EC, Saver JL, Adams HP Jr., et al. Guidelines for the early management of patients with acute ischemic stroke: a guideline for healthcare professionals from the American Heart Association/American Stroke Association. Stroke. 2013;44(3):870–947. [PubMed: 23370205]
40. Del Zoppo GJ, Saver JL, Jauch EC, Adams HP Jr., American Heart Association Stroke Council. Expansion of the time window for treatment of acute ischemic stroke with intravenous tissue plasminogen activator: a science advisory from the American Heart Association/American Stroke Association. Stroke. 2009;40(8):2945–2948. [PubMed: 19478221]
41. Treadwell SD, Thanvi B, Robinson TG. Stroke in pregnancy and the puerperium. Postgrad Med J. 2008;84(991):238–245. [PubMed: 18508980]
42. Gravanis I, Tsirka SE. Tissue-type plasminogen activator as a therapeutic target in stroke. Expert Opin Ther Targets. 2008;12(2):159–170. [PubMed: 18208365]
43. The National Institute of Neurological Disorders and Stroke rt-PA Stroke Study Group. Tissue plasminogen activator for acute ischemic stroke. N Engl J Med. 1995;333(24):1581–1587. [PubMed: 7477192]
44. Leonhardt G, Gaul C, Nietsch HH, Buerke M, Schleussner E. Thrombolytic therapy in pregnancy. J Thromb Thrombolysis. 2006;21(3):271–276. [PubMed: 16683220]
45. Del Zotto E, Giossi A, Volonghi I, et al. Ischemic stroke during pregnancy and puerperium. Stroke Res Treat. 2011;2011:606780.
46. Guimicheva B, Czuprynska J, Arya R. The prevention of pregnancy-related venous thromboembolism. Br J Haematol. 2015;168(2):163–174. [PubMed: 25312482]
47. Patel JP, Auyeung V, Patel RK, et al. Women's views on and adherence to low-molecular-weight heparin therapy during pregnancy and the puerperium. J Thromb Haemost. 2012;10(12):2526–2534. [PubMed: 23039905]
48. Khan M, Wasay M. Haemorrhagic strokes in pregnancy and puerperium. Int J Stroke. 2013;8(4):265–272. [PubMed: 22863273]
49. Berks D, Hoedjes M, Raat H, et al. Risk of cardiovascular disease after pre-eclampsia and the effect of lifestyle interventions: a literature-based study. BJOG. 2013;120(8):924–931. [PubMed: 23530583]
50. Wilkins-Haug L, Celi A, Thomas A, Frolkis J, Seely EW. Recognition by women's health care providers of long-term cardiovascular disease risk after preeclampsia. Obstet Gynecol. 2015;125(6):1287–1292. [PubMed: 26000498]
51. Lamy C, Hamon JB, Coste J, Mas JL. Ischemic stroke in young women: risk of recurrence during subsequent pregnancies. French Study Group on Stroke in Pregnancy. Neurology. 2000;55(2):269–274. [PubMed: 10908903]

52. Curtis KM. US selected practice recommendations for contraceptive use, 2016. MMWR Recommendations and Reports. 2016;65.

53. Chakhtoura Z, Canonico M, Gompel A, et al. Progestogen-only contraceptives and the risk of stroke: a meta-analysis. Stroke. 2009;40(4):1059–1062. [PubMed: 19211491]

54. Lidegaard O, Lokkegaard E, Jensen A, Skovlund CW, Keiding N. Thrombotic stroke and myocardial infarction with hormonal contraception. N Engl J Med. 2012;366(24):2257–2266. [PubMed: 22693997]

55. Roach RE, Helmerhorst FM, Lijfering WM, et al. Combined oral contraceptives: the risk of myocardial infarction and ischemic stroke. Cochrane Database Syst Rev. 2015(8):CD011054.

56. van Oostwaard MF, Langenveld J, Schuit E, et al. Recurrence of hypertensive disorders of pregnancy: an individual patient data metaanalysis. Am J Obstet Gynecol. 2015;212(5):624.e621–617.

57. Cohen-Gadol AA, Friedman JA, Friedman JD, et al. Neurosurgical management of intracranial lesions in the pregnant patient: a 36-year institutional experience and review of the literature. J Neurosurg. 2009;111(6):1150–1157. [PubMed: 19408979]

QUICK POINTS

1. Risk factors for pregnancy-associated stroke include maternal pre-existing conditions such as obesity, hypertension, and cardiac abnormalities as well as intrapartum factors such as cesarean birth.
2. There are 2 main types of stroke, ischemic and hemorrhagic. While presenting symptoms are similar, their pathophysiology and treatment differ. Pregnant and postpartum women with symptoms of stroke should have brain imaging and laboratory tests to determine etiology as this information is crucial to treatment.
3. Rapid diagnosis and treatment of strokes can prevent further brain tissue death that contributes to long-term morbidity. Since rates of pregnancy-associated stroke are increasing, all perinatal providers should be prepared to begin initial diagnostic and management steps to improve perinatal outcomes.
4. Women with a history of pregnancy-associated stroke have a 1% to 2.3% risk of recurrence within the first 5 years, but this is not a contraindication to future pregnancy.

Table 1.

Evaluation of Pregnancy-Related Stroke

Assessment	Components
History	Symptom timeline
	Current medications supplements and last dose
	Pertinent history including: trauma or physical abuse, metal implants, drug or alcohol use, seizures, diabetes, anticoagulant use
Vital Signs	Temperature, pulse, respirations, blood pressure
	Oxygen saturation
Physical Assessment	Examine skin for: discoloration, bruising, petechiae, or edema
	Examine head for: signs of trauma, eye nystagmus, pupil dilation, tongue lacerations
	Palpate bilaterally for pulses in neck, arm, legs
	Auscultate for carotid bruits
	Auscultate the heart and lungs
	Neurological evaluation including cranial nerve function and bilateral gross motor and sensory function
Laboratory Test	Complete blood count including platelets
	Cardiac enzymes and troponin
	Serum electrolytes
	Prothrombin time (PT), partial thromboplastin time (PTT) & international normalized ratio (INR)
	Additional labs for consideration may include:
	Labs to evaluate for pulmonary embolism, sepsis, blood sugar, substance abuse, or x-ray
Cardiac Monitoring	12-lead electro-cardiac monitoring
Brain Imaging	Computed tomography (CT) or magnetic resonance imaging (MRI)
	If a CT scan or MRI is ordered, radiology will need to be notified that she is
	pregnant

Sources: Jauch et al.[39]