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Gerard W. Ostheimer "What's New in Obstetric Anesthesia" Lecture

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IF physicians would read 2 articles per day of the 6 million medical articles published annually, in 1 yr, they would fall 82 centuries behind.[1]

Since 1975, the "What's New in Obstetric Anesthesia" Lecture has been an integral part of the Society for Obstetric Anesthesia and Perinatology Annual Meeting. The Society for Obstetric Anesthesia and Perinatology was founded in 1968 to provide a forum for discussion of problems unique to the peripartum period. The society is comprised of anesthesiologists, obstetricians, pediatricians, and basic scientists who share an interest in the care of pregnant patients and newborns. After the death of Gerard W. Ostheimer, M.D., in 1995, the lecture was renamed the "Ostheimer What's New in Obstetric Anesthesia Lecture" to celebrate the life and important contributions to regional and obstetric anesthesia of Dr. Ostheimer, former Professor of Anesthesiology at Brigham and Women's Hospital (Boston, Massachusetts). Each year, the lecture provides a critical appraisal of the literature from the previous year with contributions from obstetric anesthesia, obstetrics, and neonatology. Nine hundred ninety references were selected and included in the 2005 36th Annual Meeting program syllabus. Although the lecture syllabus was not intended to be exhaustive, it represented less than 10% of the references published in those areas during 2004. This article focuses on four specific areas relevant to anesthesiologists who practice obstetric anesthesia: cardiac disease during pregnancy, preeclampsia, morbidity and mortality in pregnant patients, and risk management in obstetric anesthesia. These topics were selected for their clinical relevance as well as to provide the most novel and recent information about obstetrics and obstetric anesthesia complications.

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Cardiac Disease during Pregnancy

Today, more women with complex coexisting medical diseases are choosing to become pregnant or continue pregnancies that had been considered too risky in the past. In the most recent Report of the Confidential Enquiries into Maternal Deaths in the United Kingdom 2000–2002, the number of women dying from diseases exacerbated by pregnancy (*e.g.*, cardiac disease) exceeded those directly caused by pregnancy (*e.g.*, thromboembolism).[2] After psychiatric causes, cardiac disease was the second most frequent indirect (*i.e.*, death from a preexisting condition aggravated by pregnancy) cause of maternal death. Peripartum cardiomyopathy, myocardial infarction, aortic dissection, and secondary pulmonary hypertension were identified as leading causes of maternal death due to cardiac disease.[3] In these cases, deviation from standard practice resulted from failure of communication between members of multidisciplinary teams, lack of clear policies for management of cardiac problems, and failure of individual clinicians to diagnose cardiac problems accurately or to appreciate the severity of these conditions. Patients with cardiac disease benefit from careful preoperative evaluation, medical optimization, and planned elective delivery. In most cases, vacuum-assisted or forceps-assisted vaginal delivery are used in the anesthetized parturient to avoid excessive pushing and reduce cardiovascular stress during delivery. Because second-stage labor results in a greater than 100% increase in cardiac output from the prepregnancy state, excessive pushing increases venous return, increasing risk for atrial irritability and elevated atrial pressures. Judicious dosing of neuraxial anesthesia is considered safe for many of these patients in addition to multidisciplinary consultation and invasive monitoring, if indicated. Cesarean delivery is usually reserved for obstetric and not maternal indications because of increased risk for blood loss, postoperative pain, endometritis, and pulmonary embolus.

Of the acquired cardiac diseases, the cardiomyopathies (*i.e.*, peripartum, hypertrophic, dilated) account for

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many cases of maternal mortality reported in the United Kingdom.[4] Peripartum cardiomyopathy is a rare type of heart failure with left ventricular ejection fraction of less than 45% occurring in the last month of pregnancy or within 5 months postpartum and without an identifiable cause. Although this is a rare disorder, it is associated with mortality rates of 20–85%. One case report describes administration of levosimendan to a patient with peripartum cardiomyopathy after dobutamine failed to improve the hemodynamics associated with acute cardiac failure during delivery.[5] Levosimendan administration resulted in decreased pulmonary capillary wedge pressure measurements, improved stroke volume, and mixed venous oxygen saturation. Ten weeks postpartum, the patient demonstrated a left ventricular ejection fraction of 60%. Levosimendan is a calcium-sensitizing drug with vasodilating properties that reduces cardiac workload by opening adenosine triphosphate–dependent potassium channels, resulting in vasodilation and enhanced cardiac contractility. Although the risk of death is less in patients with hypertrophic cardiomyopathy compared with dilated cardiomyopathy, patients with hypertrophic disease who develop symptoms (*e.g.*, shortness of breath, chest pain) usually respond to β-blocker administration to improve coronary perfusion and ventricular filling. Atrial fibrillation is not well tolerated because of reduced ventricular filling, and prompt conversion to sinus rhythm is needed. Regardless of the type of cardiomyopathy, patients with cardiac failure often tolerate cardiac changes associated with pregnancy poorly, resulting in increased maternal risk.

Primary pulmonary hypertension, especially when severe, is poorly tolerated during pregnancy because of increased cardiovascular demands, inadequate adaptation of the right heart, and poor compliance of the pulmonary vasculature. Mortality rates approach 50%.[6] Both intravenous and inhaled prostacyclin have been shown to improve pulmonary endothelial function and right heart hemodynamics in patients with primary pulmonary hypertension unresponsive to calcium channel blockers. Prostacyclin is a naturally occurring vasodilator that is produced by the vascular endothelium. It produces vasodilation by binding to a G protein–coupled receptor, activating adenylate cyclase and increasing the production of cyclic adenosine monophosphate. Inhaled prostacyclin administration was described in two case reports for management of primary pulmonary hypertension during labor and delivery.[7,8] Both patients responded favorably to intravenous prostacyclin, but systemic absorption of prostacyclin is known to interfere with platelet aggregation, increasing the potential risk for bleeding and hematoma formation after epidural placement. These patients were weaned from intravenous prostacyclin, and inhaled prostacyclin was initiated because of its efficacy, limited systemic absorption, and lack of effect on platelet aggregation. Because there have been no clinical reports of platelet dysfunction or excessive bleeding after inhaled use at normal pH, epidural placement was considered safe. Both patients had uneventful postpartum courses after forceps-assisted vaginal deliveries. The reports suggest that inhaled prostacyclin may be a safe alternative to intravenous prostacyclin during care of these high-risk patients.

Before the development of sophisticated cardiac surgical procedures, less than 20% of children with congenital heart disease survived to adulthood.[9] Now, with an 85% survival rate in the developed world, congenital heart disease has replaced valvular disease as the major cause of heart disease during pregnancy. Because pregnancy increases the cardiovascular workload by increasing plasma volume and cardiac output (approximately 50% of baseline),[10] patients with and without surgical repair are at risk for cardiac decompensation during pregnancy. In a review of pregnancy outcomes of patients with corrected or uncorrected tetralogy of Fallot, adverse maternal outcomes were rare, but when they did occur, they were associated with left ventricular dysfunction, pulmonary hypertension, and severe pulmonic regurgitation with right ventricular dysfunction.[11] Although congenital heart disease is an infrequent cause of maternal death, it may be associated with significant morbidity.[4] Many of these patients tolerate pregnancy well, but others decompensate, particularly patients with cyanosis or congestive heart failure.[12] Increased cardiac work and oxygen consumption as well as increased maternal catecholamines due to pain during labor and delivery contribute to patient risk. Patients with congestive heart failure may be unable to withstand the cardiac demands imposed by the relative hypervolemia and increased venous return after vaginal delivery. In patients with cyanotic heart disease, increased concentrations of maternal catecholamines result in increased systemic vasculature resistance and left-to-right shunting. Like most parturients with cardiac lesions, these patients benefit from supplemental oxygen administration and judicious fluid management. In addition, many tolerate the hemodynamics of carefully conducted neuraxial analgesia better than those without effective analgesia during labor and delivery.

Preeclampsia

Preeclampsia is a multisystem disorder affecting up to 8% of pregnancies. It is a pregnancy-specific disease occurring after 20 weeks gestation and is characterized by hypertension and proteinuria. The disease is often described as a "disease of theories" because over several decades, many hypotheses have been proposed and later refuted. Although the cause remains unknown, the understanding of the pathophysiology has dramatically increased. Atypical placentation and placental vascular in-

sufficiency are characteristic of preeclampsia, but the source of these alterations is still a mystery. Some theories that remain under consideration include flawed placental vascular remodeling resulting from impaired trophoblast invasion, immunologic intolerance, genetic factors (*e.g.*, polymorphisms), increased oxidative stress, vascular cell activation, prostaglandin imbalances, and exaggerated inflammatory processes.

Although the specific factors that initiate endothelial damage are largely unknown, recent studies demonstrate a clearer understanding of the role of inflammatory system activation in preeclampsia. Inflammatory changes are known to be induced in peripheral blood leukocytes during normal pregnancy.[13] To examine potential inflammatory marker changes during preeclampsia, Freeman *et al.*[14] conducted a prospective study comparing the short- and long-term pregnancy-induced changes in plasma inflammatory markers in women with healthy pregnancies compared with women with preeclampsia. In that study, both short- and long-term changes in inflammatory status were identified in patients with preeclampsia. To test the hypothesis that leukocytes from preeclamptic patients are in an excessive inflammatory state, Holthe *et al.*[15] used flow cytometry to compare the expression of leukocyte adhesion molecules, intracellular reactive oxygen species, and vasoactive substances. These authors determined that there are both qualitative and quantitative differences in activation of maternal blood leukocytes in patients with preeclampsia compared with patients with normal pregnancies and that oxidative stress is a contributing factor in the pathophysiology of preeclampsia. Because increased oxidative stress has been implicated in the etiology of preeclampsia, Moretti *et al.*[16] used a breath test to measure the oxidative stress in nonpregnant women and women with normal pregnancies as well as preeclampsia. During oxidative stress, lipid peroxidation of polyunsaturated fatty acids generates alkanes that can be measured in the breath as volatile organic compounds. Breath tests have been used previously to identify increased oxidative stress in several diseases (*e.g.*, breast cancer, rheumatoid arthritis, acute myocardial infarction). In the current study, the breath test accurately identified women with established preeclampsia and demonstrated significantly greater oxidative stress in these women. Further studies are needed to determine whether breath tests will predict the onset of preeclampsia or whether antioxidants can prevent the disease.

Epidemiologic factors associated with increased risk of preeclampsia include diabetes mellitus, chronic hypertension, previous history of preeclampsia, multiple gestations, and increased body mass indices. Unfortunately, these risk factors lack both sensitivity and specificity. Despite years of research and suggestions of several markers predicting the onset of disease, there are currently no screening tests for preeclampsia. However, a recent study demonstrated that soluble fms-like tyrosine kinase 1 (sFlt-1) induced endothelial dysfunction by adhering to receptor binding areas of placental growth and vascular endothelial growth factors resulting in impaired interaction with endothelial receptors on cell surfaces.[17] This nested case–control study within the Calcium for Preeclampsia Prevention Trial identified increased serum levels of circulating soluble fms-like tyrosine kinase 1 and reduced levels of placental growth factor in patients up to 5 weeks before the onset of preeclampsia. These findings should be approached with caution, however, because preeclampsia did not develop in all women with increased soluble fms-like tyrosine kinase 1 and low placental growth factor. Large longitudinal studies are needed to determine this study's relevance and whether therapies aimed at reducing the level of soluble fms-like tyrosine kinase 1 or blocking its effects will have a therapeutic effect.

Despite the fact that treatment of preeclampsia continues to elude practitioners, therapies are currently directed at reducing maternal and perinatal morbidity and mortality, especially preventing or reducing the rate of eclampsia and its complications. Several observational and randomized trials have compared anticonvulsant regimens used to prevent or reduce the rate of seizures and complications in preeclampsia. However, magnesium sulfate remains the mainstay of seizure prophylaxis in preeclampsia and for prevention of recurrent seizures in women with eclampsia. Several of the comparisons evaluated the efficacy of magnesium sulfate compared with diazepam or phenytoin. Although only one of the trials was multicenter with an adequate sample size, collectively, the studies suggest that magnesium sulfate administration is associated with a significantly lower rate of recurrent seizures (risk ratio, 0.41; 95% confidence interval, 0.32–0.51) and rate of maternal death (risk ratio, 0.62; 95% confidence interval, 0.39–0.99) compared with other anticonvulsants.[18] Although the benefits of magnesium administration are unmistakable in patients with eclampsia and severe disease, magnesium sulfate's role in the mild preeclamptic is less clear. There are only two double-blind, placebo-controlled trials evaluating magnesium sulfate use in mild preeclampsia. In these studies, there were no cases of eclampsia or differences in the number of women who progressed to severe disease (risk ratio, 0.90; 95% confidence interval, 0.52–1.54), but the number of patients enrolled in these studies is too limited to draw conclusions.[19] Despite studies suggesting that magnesium sulfate is currently the "best" anticonvulsant for patients with eclampsia and severe preeclampsia, magnesium sulfate administration does not reduce overall perinatal morbidity and mortality and is associated with increased risk of maternal respiratory depression (risk ratio, 2.06; 95% confidence interval, 1.33–3.18).[19]

Preeclampsia has also been associated with stroke re-

sulting from cerebral infarction and intracerebral hemorrhage[20]; however, the pathophysiology of the neurologic disorders associated with eclampsia is poorly understood. Several studies have been undertaken to better understand the mechanisms of these disturbances using angiography, computed tomography, magnetic resonance imaging, and Doppler velocimetry. From these evaluations, two theories have emerged to explain development of cerebral lesions (*e.g.*, edema, petechial hemorrhages, infarction) and convulsions. First, ischemia, cytotoxic edema, and infarction result from cerebrovascular "overregulation" with vasospasm. Second, lesions may result from a loss of cerebral autoregulation producing hyperperfusion and subsequent vasogenic edema. Both vasogenic and cytotoxic edema have been described in these patients with evidence that vasogenic edema is reversible, whereas cytotoxic edema implies infarction. To better characterize the incidence of vasogenic and cytotoxic edema in patients with eclampsia, Zeeman *et al.*[21] used diffusion-weighted magnetic resonance imaging and coefficient mapping to characterize the neuroimaging findings of cerebral edema associated with eclamptic seizures. Of 27 women with eclamptic seizures, 93% had reversible vasogenic edema. Six of the 27 also had areas consistent with cytotoxic edema and infarction. Of the women with cytotoxic edema, 5 had persistent findings of infarction but were without neurologic deficits 6–8 weeks postpartum. Although eclampsia rarely causes permanent neurologic sequelae and the long-term implications are unknown, symptomatic cerebral edema may be preceded by sudden or severe hypertension that exceeds the limits of autoregulation.[22] Collectively, these findings emphasize the importance of blood pressure control in eclampsia, especially when general anesthesia is necessary, and radiologic evaluation in women with focal neurologic deficits, atypical seizure activity, or prolonged unconsciousness.

Labor- and Delivery-related Mortality

Most studies of maternal morbidity and mortality evaluate each independently, not allowing for examination of potential relations between the two. To evaluate this relation, Geller *et al.*[23] performed a case–control study of pregnancy-related deaths, women with "near-miss" morbidity, and those with severe but not life-threatening morbidity to determine whether sociodemographic, clinical, and other service-related factors, as well as preventability issues, contribute to the progression from morbidity to mortality. In the past, intensive care unit admissions have been the single variable most often used to identify near-miss obstetric morbidity. Because these admissions vary greatly across hospitals, Geller *et al.*[24] developed a reproducible scoring system evaluating multiple variables (*e.g.*, diseases/conditions, morbid events, procedures/interventions) to reliably define near-miss and severe morbidity. Incorporating the scoring system into their most recent study, Geller *et al.*[23] determined that 41% (n = 37) of deaths, 46% (n = 33) of near misses, and 17% (n = 101) of severe morbidities were preventable. Of all preventable cases, approximately 90% were provider-related issues that were associated with incomplete or inappropriate management. After controlling for sociodemographic characteristics, clinical diagnosis and preventable events were associated with progression along the continuum from morbidity to mortality. However, system and patient factors were not related with progression except insurance status. Based on their findings, the authors concluded that the more severe the morbidity, the less opportunity there is for successful clinical intervention and the more difficult it is to impact the outcome of a woman who has a life-threatening condition.

Since 1952, the United Kingdom has published Reports of the Confidential Enquiries into Maternal Deaths every 3 yr. Because these reports are published regularly and provide detailed information on all maternal deaths occurring during each triennial period, they serve as a measure of obstetric anesthesia care. Since the early 1980s, there has been a dramatic reduction in anesthesia-related maternal deaths. Increased use of regional anesthesia, administration of effective aspiration prophylaxis, and improvement in training and education have all contributed to this decrease despite a progressive increase in the cesarean delivery rate. However, in the most recent triennium, there were an increased number of deaths attributed to anesthesia. Of the 6 reported cases, all were related to general anesthesia and suboptimal airway management. These cases represent an increase over the 4 cases reported in the last triennium. Although this is not a statistically significant difference, these deaths are concerning because of the decreased use of general anesthesia for cesarean delivery. It represents a risk of 1 death per 20,000 maternal general anesthetics, which is a rate similar to the 1982–1984 report. In addition, the report identified an additional 20 maternal deaths in which suboptimal anesthesia care was contributory. Poor interdisciplinary cooperation and communication, delay in the recognition of the severity of illness, and inadequate management of intractable hemorrhage were all contributory factors in these maternal deaths.

In the United States, there has been a significant decline in the number of maternal deaths related to regional anesthesia that were reported in the American Society of Anesthesiologists Closed Claims Database from the 1970s to the 1990s.[25] This project is a structured evaluation of adverse anesthetic outcomes collected from closed anesthesia malpractice insurance claims of professional liability companies. In a subset comparison between obstetric and nonobstetric

neuraxial anesthesia claims from 1980 to 1999, obstetrics (n = 42) had a lower proportion of claims with death or brain damage compared with nonobstetric (n = 143) claims. Thirty-two percent of these obstetric neuraxial claims were associated with cardiac arrest. Cases of death or brain damage were more often associated with less than appropriate care, a greater number of claims with payment, and highest median payment compared with all other regional anesthetic injuries.

Other reports have been consistent in identifying similar service-related factors associated with labor- and delivery-related morbidity and mortality. In the Editor's Note to Sentinel Event Alert Issue #30, 47 cases of perinatal death or permanent disability were reported to the Joint Commission on Accreditation of Healthcare Organizations for review under the Sentinel Event Policy since 1996.† Cases that were considered reviewable under the Policy included "any perinatal death or major permanent loss of function unrelated to a congenital condition in an infant having a birth weight greater than 2500 g." Root cause analyses determined that communication issues, staff competency, inadequate fetal monitoring, credentialing/privileging/supervision issues for physicians and nurse midwives, staffing issues, and unavailable or delayed physicians all contributed to infant death or permanent disability. Joint Commission recommendations included team training to improve communication, clinical drills and debriefings to evaluate performance and identification of areas for improvement, review and application of the American College of Obstetricians and Gynecologists Practice Bulletins and Guidelines, Association of Women's Heath, Obstetric and Neonatal Nurses Standards and Guidelines, and American Academy of Pediatrics and American College of Obstetricians and Gynecologists Guidelines for Perinatal Care.

Labor- and Delivery-related Morbidity

In the United States, maternal morbidity affects nearly 1.7 million women each year.[26] Research suggests that the extent of the morbidity is often underappreciated, with approximately 40% of women experiencing morbidity associated with labor and delivery. Maternal neurologic injury is a well-known complication of labor and delivery. Injuries may be physiologic and result from labor and delivery or be directly or indirectly related to obstetric and/or anesthetic interventions. Anesthesiologists are often asked to evaluate neurologic symptoms because of the close association of neuraxial anesthetic placement and labor and delivery. After review of the literature, Wong[27] concluded that permanent neurologic injury after labor and delivery is rare. However, transient injuries are more common and can be particularly debilitating when there is motor weakness. Most injuries are not related to neuraxial analgesia, but when they do occur, serious complications (*e.g.*, spinal epidural hematoma, spinal infections, vascular complications) must be diagnosed without delay to prevent permanent neurologic damage.

In 2004, two reports evaluated the complications associated with regional anesthesia and obstetrics.[25,28] The American Society of Anesthesiologists Closed Claims Analysis reported on 260 temporary injuries after neuraxial anesthesia. The most common obstetric injuries included headache (32%), back pain (22%), nerve damage (17%), inadequate anesthesia (17%), and emotional distress (13%). The total obstetric claims (n = 368) were more often associated with lumbar epidural anesthetics (70%) than subarachnoid anesthetics (25%). Limitations of the database have been described previously and include the lack of denominator data on how many anesthetics are performed per year as well as an inability to include claims on all adverse events. However, the results are useful because the data could not otherwise be prospectively collected by a single institution. The second report evaluated severe neurologic complications after central neuraxial blockade in Sweden from 1990 to 1999. Moen *et al.*[28] reported eight complications in obstetric patients, including epidural abscess, cord lesion, subdural hematoma, permanent abducens paresis, and Horner syndrome with facial pain. Two of the eight patients developed spinal hematoma after central neuraxial blockade. These patients had HELLP (hemolysis, elevated liver enzymes, and low platelets) syndrome but were without any apparent signs of coagulopathy before block placement. This report was consistent with the Closed Claims study in that epidural-related complications were also more common than spinal complications in obstetrics patients (70% *vs.* 25%). Temporary and low-severity injury from neuraxial blocks were also more common in obstetric than in nonobstetric patients (71% *vs.* 38%). In addition, obstetric patients had a lower incidence of serious complications.

Risk Management of Neuraxial Anesthesia/Analgesia

During the past three decades, use of neuraxial anesthesia has dramatically increased in labor and delivery, resulting in greater patient satisfaction, fewer emergent general anesthetics, and a reduction in maternal mortality.[29] Despite the clear benefits of regional anesthesia, the techniques are not without complications. Both obstetrics and anesthesiology are specialties with great

† Editor's Note to Sentinel Event Alert Issue #30. Joint Commission on Accreditation of Healthcare Organizations. Available at: http://www.jcaho.org/About+Us/News+Letters/Sentinel+Event+Alert/sea_30_reference.pdf. Accessed June 15, 2005.

medicolegal risk. Patients receiving analgesia/anesthesia for labor and delivery represent a different patient population than those receiving anesthesia for other procedures. Two patients instead of one, anatomic and physiologic changes of pregnancy, and the belief that childbirth is a joyous occasion without serious complications all contribute to differences in obstetric and nonobstetric surgical patients. All of these factors likely contribute to a larger number of claims related to minor injuries in obstetric compared with nonobstetric patients.[25]

A recent survey by Wang *et al.*[30] estimated the severity of low back pain during pregnancy and its impact on daily living. Of 950 surveys returned (84%), 69% of patients reported low back pain during their current pregnancy. Back pain was more common in younger women, but prevalence was not affected by gestational age. Although back pain impaired activities of daily living in 58% of patients and produced sleep disturbances in 57%, only 32% of the patients informed their care providers. Because back pain was one of the most common temporary injuries reported in the most recent Closed Claims Analysis, the high incidence of back pain during pregnancy underscores the importance of careful documentation of preoperative history and physical to reduce risk of possible claims.

Many factors increase the risk of nerve injury during labor and delivery. Because potential nerve injury is intrinsic to neuraxial anesthesia and many women receive regional techniques for labor and delivery, nerve injury is more common in these patients. In addition, labor and delivery itself can be associated with nerve injury. Although permanent nerve injuries related to neuraxial anesthesia are rare in obstetrics, temporary injuries constitute a larger percentage of obstetric neuraxial anesthesia claims.[25] Before block placement, a neurologic history should be obtained. A history of lower extremity weakness or numbness will usually identify preexisting neurologic conditions and prompt a carefully documented neurologic examination.

Cases of postpartum nerve injury may also be complicated by obstetric-related nerve injuries. Regardless of the cause, it is important to differentiate between central and peripheral lesions in patients with suspected injury. Although early expert consultation is imperative, one of the simplest methods to rule out a central lesion is examination of the paraspinous musculature and skin over the lower back.[27] Central neurologic injuries are associated with weakness of the paraspinous muscles and abnormal sensation of the lower back because these structures are innervated by nerves of the posterior rami. Patients with central lesions also commonly report back pain. Although rare, these life- or limb-threatening complications must be diagnosed early to avoid adverse outcomes. Such injuries are best diagnosed with magnetic resonance imaging.

In cases of peripheral injury, electrophysiologic testing is used to determine the site of the lesion and to define the neurogenic basis of the complication.[31] Although testing will assist in determining the severity of the injury and prognosis, it will not establish a cause. When testing is indicated, electromyography is the most commonly used test in obstetric patients. Testing involves measurement of electrical activity of specific muscles that are stimulated by a needle electrode inserted within the muscle. Because electromyography only measures changes in large nerve fibers, changes related to peripheral nerve injuries may not be apparent for several weeks after injury. If the electromyogram is abnormal within the first week, it suggests a preexisting injury. Electromyography is helpful in determining injury prognosis because voluntary motor activity reappears before clinical recovery in patients with incomplete lesions. However, serial studies are generally not indicated because progress can often be followed clinically.

Conclusion

During 2004, there have been significant contributions to the literature in the areas of obstetric anesthesia and obstetrics. The pregnant patient with coexisting disease, preeclampsia, labor- and delivery-related morbidity and mortality, and risk management of neuraxial anesthesia are areas of particular importance.

The accompanying Web Enhancement represents the complete reference list presented in part at the 2005 Annual Meeting of the Society for Obstetric Anesthesia and Perinatology and is available on the ANESTHESIOLOGY Web site at http://www.anesthesiology.org.

References

1. Miser WF: Critical appraisal of the literature. J Am Board Fam Pract 1999; 12:315–33
2. Confidential Enquiry into Maternal and Child Health: Why Mothers Die 2000-2002: The Sixth Report of the Confidential Enquiries into Maternal Death in the United Kingdom. London, RCOG Press, 2004
3. Ray P, Murphy GJ, Shutt LE: Recognition and management of maternal cardiac disease in pregnancy. Br J Anaesth 2004; 93:428–39
4. Thorne SA: Pregnancy in heart disease. Heart 2004; 90:450–6
5. Benlolo S, Lefoll C, Katchatouryan V, Payen D, Mebazaa A: Successful use of levosimendan in a patient with peripartum cardiomyopathy. Anesth Analg 2004; 98:822–4
6. Weiss BM, Zemp L, Seifert B, Hess OM: Outcome of pulmonary vascular disease in pregnancy: A systematic overview from 1978 through 1996. J Am Coll Cardiol 1998; 31:1650–7
7. Bildirici I, Shumway JB: Intravenous and inhaled epoprostenol for primary pulmonary hypertension during pregnancy and delivery. Obstet Gynecol 2004; 103:1102–5
8. Hill LL, De Wet CJ, Jacobsohn E, Leighton BL, Tymkew H: Peripartum substitution of inhaled for intravenous prostacyclin in a patient with primary pulmonary hypertension. ANESTHESIOLOGY 2004; 100:1603–5
9. Macmahon B, McKeown T, Record RG: The incidence and life expectation of children with congenital heart disease. Br Heart J 1953; 15:121–9
10. Desai DK, Moodley J, Naidoo DP: Echocardiographic assessment of cardiovascular hemodynamics in normal pregnancy. Obstet Gynecol 2004; 104:20–9
11. Veldtman GR, Connolly HM, Grogan M, Ammash NM, Warnes CA: Out-

comes of pregnancy in women with tetralogy of Fallot. J Am Coll Cardiol 2004; 44:174–80

12. Lovell AT: Anaesthetic implications of grown-up congenital heart disease. Br J Anaesth 2004; 93:129–39

13. Redman CW, Sacks GP, Sargent IL: Preeclampsia: An excessive maternal inflammatory response to pregnancy. Am J Obstet Gynecol 1999; 180:499–506

14. Freeman DJ, McManus F, Brown EA, Cherry L, Norrie J, Ramsay JE, Clark P, Walker ID, Sattar N, Greer IA: Short- and long-term changes in plasma inflammatory markers associated with preeclampsia. Hypertension 2004; 44:708–14

15. Holthe MR, Staff AC, Berge LN, Lyberg T: Leukocyte adhesion molecules and reactive oxygen species in preeclampsia. Obstet Gynecol 2004; 103:913–22

16. Moretti M, Phillips M, Abouzeid A, Cataneo RN, Greenberg J: Increased breath markers of oxidative stress in normal pregnancy and in preeclampsia. Am J Obstet Gynecol 2004; 190:1184–90

17. Levine RJ, Maynard SE, Qian C, Lim KH, England LJ, Yu KF, Schisterman EF, Thadhani R, Sachs BP, Epstein FH, Sibai BM, Sukhatme VP, Karumanchi SA: Circulating angiogenic factors and the risk of preeclampsia. N Engl J Med 2004; 350:672–83

18. Witlin AG, Sibai BM: Randomized trials for prevention and treatment of eclamptic convulsions, Hypertensive Disorders in Women. Edited by Sibai BM. Philadelphia, Saunders, 2001, pp 221–7

19. Sibai BM: Magnesium sulfate prophylaxis in preeclampsia: Lessons learned from recent trials. Am J Obstet Gynecol 2004; 190:1520–6

20. Sharshar T, Lamy C, Mas JL: Incidence and causes of strokes associated with pregnancy and puerperium: A study in public hospitals of Ile de France. Stroke in Pregnancy Study Group. Stroke 1995; 26:930–6

21. Zeeman GG, Fleckenstein JL, Twickler DM, Cunningham FG: Cerebral infarction in eclampsia. Am J Obstet Gynecol 2004; 190:714–20

22. Cunningham FG, Twickler D: Cerebral edema complicating eclampsia. Am J Obstet Gynecol 2000; 182:94–100

23. Geller SE, Rosenberg D, Cox SM, Brown ML, Simonson L, Driscoll CA, Kilpatrick SJ: The continuum of maternal morbidity and mortality: factors associated with severity. Am J Obstet Gynecol 2004; 191:939–44

24. Geller SE, Rosenberg D, Cox S, Brown M, Simonson L, Kilpatrick S: A scoring system identified near-miss maternal morbidity during pregnancy. J Clin Epidemiol 2004; 57:716–20

25. Lee LA, Posner KL, Domino KB, Caplan RA, Cheney FW: Injuries associated with regional anesthesia in the 1980s and 1990s: A closed claims analysis. ANESTHESIOLOGY 2004; 101:143–52

26. Danel I, Berg C, Johnson CH, Atrash H: Magnitude of maternal morbidity during labor and delivery: United States, 1993-1997. Am J Public Health 2003; 93:631–4

27. Wong CA: Neurologic deficits and labor analgesia. Reg Anesth Pain Med 2004; 29:341–51

28. Moen V, Dahlgren N, Irestedt L: Severe neurological complications after central neuraxial blockades in Sweden 1990-1999. ANESTHESIOLOGY 2004; 101: 950–9

29. Hawkins JL, Koonin LM, Palmer SK, Gibbs CP: Anesthesia-related deaths during obstetric delivery in the United States, 1979–1990. ANESTHESIOLOGY 1997; 86:277–84

30. Wang SM, Dezinno P, Maranets I, Berman MR, Caldwell-Andrews AA, Kain ZN: Low back pain during pregnancy: Prevalence, risk factors, and outcomes. Obstet Gynecol 2004; 104:65–70

31. Aminoff MJ: Electrophysiologic testing for the diagnosis of peripheral nerve injuries. ANESTHESIOLOGY 2004; 100:1298–303