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Maternal Morbidity, Mortality, and Risk Assessment

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Maternal mortality is the tip of the maternal morbidity iceberg; several obstetric, anesthetic, and social challenges impact morbidity and mortality in women. Maternal mortality is the yardstick to measure when health care personnel fail to recognize risks, lack interdisciplinary communication, or provide substandard care, thus resulting in complications during pregnancy, labor, or delivery.

Pregnancy-related death is defined by the International Classification of Diseases, 10th Revision (ICD-10) as the death of a woman while pregnant or within 42 days of termination of pregnancy, despite the cause of death. Although the risk for death from complications of pregnancy decreased dramatically during the 20th century in the United States, the Centers for Disease Control and Prevention (CDC) reports a fairly static maternal mortality ratio (MMR), of approximately 7.5 maternal deaths per 100,000 live births. In the year 2000, a collaborative effort involving World Health Organization (WHO), United Nations Children's Fund (UNICEF), and United Nations Population Fund (UNFPA) estimated 660 maternal deaths, thus averaging 11 maternal deaths per 100,000 live births, placing the MMR above the statistics reported by the CDC. These surveys on maternal mortality surveillances are limited in scope because the information is obtained from death certificates, and various states or academic institutions could be underreporting. Accurate statistics are lacking, thus resulting in only a snapshot of the actual maternal morbidity and mortality. The recent WHO estimate in the United States show that maternal mortality is approximately 17 in 100,000 pregnancies. This estimate is significantly higher than the goal set by the U.S. Department of Health and Human Services in *Healthy People 2010*, which sets the target for maternal mortality at less

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than 3.3 in 100,000 live births. Some regional reports document ratios as high as 22.8 per 100,000 live births, which is an unacceptably high rate.

In United States, the most common causes of maternal deaths, although they vary among states, include thromboembolism; amniotic fluid embolism; hemorrhage; complications of hypertension, including preeclampsia and eclampsia; and infection. Pulmonary disease, anesthesia-related deaths, and cardiomyopathy are also significant contributors to maternal morbidity and mortality.

In United Kingdom, the regularly conducted Confidential Enquiries into Maternal Deaths (CEMD) has the longest and most successful history of conducting and publishing findings triennially. The latest publication on "Why Mothers Die 2000–2002," was the first report of the new millennium and marks the 50th anniversary of the first triennial report [1]. In United Kingdom, maternal deaths are classified as direct (death caused by pregnancy occurring within 42 days of delivery), indirect (death from a preexisting condition aggravated by pregnancy), coincidental (death unrelated to pregnancy), and late (death occurring between 42 days and 1 year after delivery). In the triennium 2000 to 2002, 391 deaths were reported, of which 106 were classified as direct. The most common cause of direct death was thromboembolism, which remained unchanged since the previous triennium, 1997 to 1999. An increase in mortality rate from hemorrhage occurred, with it being the second leading cause of direct deaths and hypertensive disease as the fourth leading cause. Anesthesia ranked seventh among the top leading causes. Of the total deaths, 155 were classified as indirect. Cardiac disease remains a leading cause of indirect maternal deaths (Figs. 1 and 2).

The Confidential Enquiry into Maternal and Child Health (CEMACH) report (2000–2002) [1] is generally targeted toward health care professionals in the United Kingdom, and was originally criticized for its limited international scope. However, the lessons learned from the disciplined approach to rigorously collecting and analyzing data have had a far-reaching global impact, and the information is being disseminated in peer-reviewed journals in the United States [2]. Similarities are seen in the leading causes of maternal deaths in the United States and United Kingdom.

Thromboembolism

Maternal morbidity and mortality statistics

Thromboembolism remains the most common cause of direct pregnancy-related maternal death during live births in developed countries [3,4]. Because pregnancy is a hypercoagulable state, the risk for venous thromboembolism is 5 to 10 times higher in the pregnant population than the nonpregnant population [5,6]. The latest CDC data showed that thromboembolism accounted for 21.4% of pregnancy-related maternal mortality in

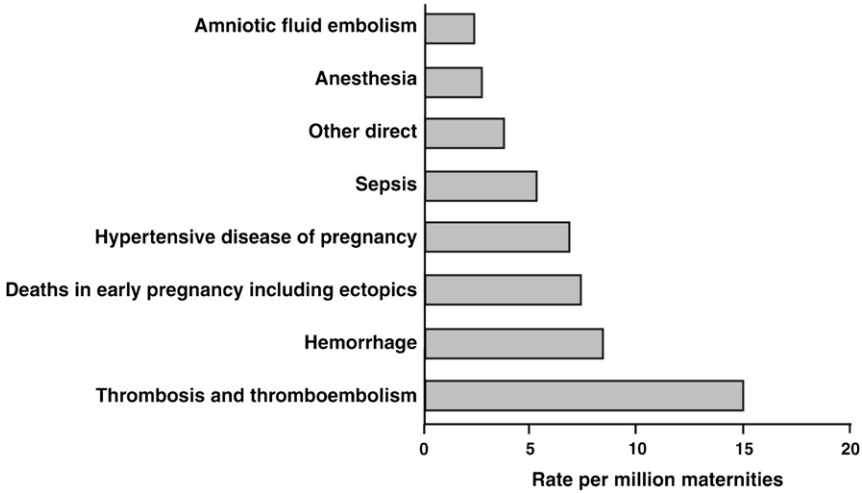

Fig. 1. Mortality rates per million maternities of leading causes of direct deaths as reported to the Confidential Enquiry into Maternal and Child Health in the United Kingdom, 2002–2002. (*From* Lewis G. Introduction and key findings 2002–2002. In: Confidential Enquiry into Maternal and Child Health. Why mothers die 2000–2002: the sixth report of the Confidential Enquiries into Maternal Death in the United Kingdom. London: RCOG Press; 2004. p. 32; with permission.)

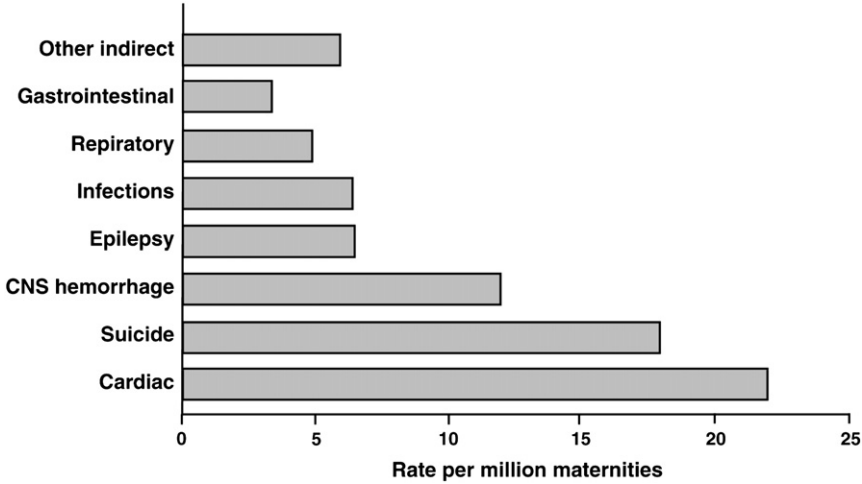

Fig. 2. Maternal mortality rate from leading causes of indirect deaths per million maternities as reported to the Confidential Enquiry into Maternal and Child Health in the United Kingdom, 2000–2002. (*From* Lewis G. Introduction and key findings 2002–2002. In: Confidential Enquiry into Maternal and 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. p. 32; with permission.)

the United States [3,4]. According to the CEMACH report (2000–2002), thromboembolism was the leading cause of direct maternal death in the United Kingdom, with two thirds occurring in the postpartum period. The overall incidence has continued to decline, probably because of improved thromboprophylaxis [7]. However, substandard practice from either failure to recognize risk factors and slow introduction or inadequate dosing of thromboprophylaxis and treatment was implicated in the report [7].

Deep vein thrombosis during pregnancy accounts for most pulmonary thromboembolism [8] occurring in the antepartum period [9]. If untreated, 13% to 24% parturients will have pulmonary embolism, with a high mortality rate of 12% to 15% [10]. Adequate treatment has helped reduce the incidence to 0.7% to 4.5% and the mortality to 0.7% [10].

Risk assessment

Risk factors for the increased incidence of thromboembolism during pregnancy include age older than 35 years, increased venous stasis, hypercoagulability, vascular injury from vaginal delivery or cesarean section, obstetric conditions (eg, preeclampsia, grand multiparity, multiple gestation) [11], and other comorbidities (eg, previous history of thromboembolism, smoking, obesity [BMI >30 kg/m^2], gross varicose veins, antiphospholipid syndrome, protein C and S deficiency, antithrombin III deficiency, hyperhomocysteinemia, factor V Leiden mutation) [10].

Strategies to minimize risk and recommendations

Prepregnancy counseling should occur and include strategies designed to reduce thrombotic risk before, during, and after pregnancy. These strategies may include reduction of body weight [12] and cessation of smoking [13]. Use of elastic compression stockings (ECS) during pregnancy, intraoperative use of pneumatic stockings during cesarean section, and ECS use during puerperium have been shown to be effective for thromboprophylaxis [11,14,15]. Drugs commonly used to prevent and treat venous thromboembolism include heparin, low molecular weight heparin (LMWH), coumadin, and antiplatelet drugs.

Heparin is ideally suited for the parturient because it does not cross the placenta due to its large molecular size, strong polarity, and lipid insolubility [11]. Because of increased heparin requirements during pregnancy, dose adjustments must be made continuously to maintain the activated partial thromboplastin time (aPTT) ratio between 1.5 and 2.5 [16] or factor Xa level between 0.5 and 1.0 IU/mL [17]. However, large-dose heparin therapy has been associated with heparin-induced thrombocytopenia (HIT) [18] and osteoporosis [19]. Furthermore, the short half-life of intravenous heparin (60 minutes) requires administration by continuous infusion.

LMWH has a longer half-life, and doses are administered based on body weight. It has a lower risk for HIT [18] and osteoporosis [19] and is predominantly excreted by the kidney [11]. Because of the increased renal blood flow of pregnancy and associated increased renal clearance of LMWH in the parturient, it is administered as a twice-daily dose in contrast to the once-daily dose in the nonpregnant patient [11]. Factor Xa level monitoring is used in certain centers to adjust dosing, even though its efficacy is not well proven [20]. LMWH seems to be the preferred thromboprophylactic agent in pregnancy.

Warfarin has a low molecular weight, crosses the placenta, and is associated with teratogenicity, and is therefore contraindicated in pregnancy. Case reports exist of successful and safe use during pregnancy of antiplatelet drugs, such as aspirin [21], clopidogrel [22], and ticlopidine [23], as adjuvants to heparin or LMWH.

According to the American College of Chest Physicians Consensus Conference on Antithrombotic Therapy [24], no medication is necessary in women who have temporary risk factors, although they should remain under strict surveillance. However, thromboprophylaxis should be administered to women who have a previous history of venous thromboembolism if they are pregnant, using oral contraceptives, or have a thrombophilia.

Based on risk–benefit assessment, LMWH is the preferred treatment of venous thrombosis and established thromboembolism [11]. The typical dose for enoxaparin is 1 mg/kg twice daily; it has a predictable and adequate anticoagulant effect and [25] does not require anti–factor Xa monitoring [11]. Anticoagulated parturients are at special risk for developing neuraxial hematoma (epidural or spinal) during regional anesthesia. Individual risk evaluation based on The American Society for Regional Anesthesia Guidelines should be performed before neuraxial block placement [26].

Case reports exist of other successful treatment modalities, including thrombectomy [27] and use of inferior vena cava filters [28], in parturients who have a contraindication to anticoagulants.

Hemorrhage

Maternal morbidity and mortality statistics

Obstetric hemorrhage is one of the leading causes of maternal morbidity and mortality worldwide. In the United States, hemorrhage is the third most common cause of maternal mortality [4,29], with 17 % of maternal deaths attributed to obstetric hemorrhage [4]. Significant racial differences were seen in mortality rates between African American and Caucasian women in the United States [4,30].

The CEMACH (2000–2002) report from the United Kingdom showed that hemorrhage was the second most common cause of direct maternal death [1]. According to the CEMD, advanced maternal age older than

35 years seemed to increase the risk for maternal morbidity and mortality from obstetric hemorrhage in the United Kingdom from 1997–1999. Based on worldwide estimates, one woman dies of postpartum hemorrhage every 4 minutes, accounting for 140,000 deaths per year [31]. Maternal morbidity, from severe obstetric hemorrhage, was seen in 6.7 per 1000 deliveries in the United Kingdom [32].

WHO estimates show that postpartum hemorrhage alone has been incriminated in 25% of maternal mortality [33]. However, application of a temporal qualifier in obstetrics can be problematic, because exsanguinating hemorrhage in the late antepartum, intrapartum, and immediate postpartum periods is often related [34]. Patients at greatest risk for severe, life-threatening blood loss [35] include those who have abruptio placentae, placenta previa, placenta accreta, and uterine rupture.

Risk assessment

Risk factors for antepartum hemorrhage include placental abruption, trauma, uterine rupture, genetic, exposure to cocaine, methadone, and tobacco, hypertensive disorders of pregnancy, and presence of a uterine fibroid [34]. Risk factors for placenta previa include advanced maternal parity, advanced maternal age, prior placenta previa, and prior cesarean section [34].

The approximately 40% reduction in maternal mortality rates is attributed to improved prenatal care, improved blood banking techniques, better appreciation for the risks for hemorrhage, and improved anesthetic and obstetric management of excessive blood loss during pregnancy [34].

The most common causes of postpartum hemorrhage include uterine atony, genital trauma, retained placenta [36], placenta accreta (0%–5% in women who have an unscarred uterus [37], increasing to 67% in those who have a scarred uterus [38]), and uterine inversion (1/5000–1/10,000 pregnancies) [39].

Uterine atony leading to postpartum hemorrhage [39] is the most common indication for peripartum blood transfusion [40,41]. Risk factors for uterine atony include protracted labor requiring oxytocin augmentation, chorioamnionitis, obstructed labor, fetal macrosomia, polyhydramnios, placental abruption, placenta previa, grand multiparity, family history, uterine laceration, and prolonged tocolytic therapy [34].

Strategies to minimize risk and recommendations

In the antepartum period, vaginal delivery is pursued if no fetal or maternal compromise is present. Cesarean section is reserved for severe maternal hemorrhage or worsening coagulopathy. Blood product availability and continuous electronic fetal monitoring are essential for a successful and safe outcome.

Anesthetic management begins with careful preparation of patients. Preparation involves a multidisciplinary team effort. Open communication

and frequent consultation should occur among anesthesiology, obstetric, blood banking, hematology, urology, neonatology, and nursing services. Preparation for adequate resuscitation requires placement of large-bore intravenous catheters. Monitoring and fluid management are managed with invasive arterial blood pressure, central venous pressure, and urine output catheter. A level-one rapid infuser is essential in severe hemorrhage requiring massive blood resuscitation. If time and hemodynamics permit, preoperative bilateral ureteric stents should be placed, particularly in patients who have placenta accreta/percreta, by the urology team to facilitate identification of ureters during intraoperative dissection by the obstetric team [34].

Patients presenting with severe ongoing obstetric hemorrhage, severe anticipated hemorrhage, or acute fetal compromise should receive general anesthesia for cesarean section. Regional anesthesia is contraindicated in hemorrhaging parturients with acute fetal distress, coagulopathy, or hypovolemia. Loss of compensatory mechanisms caused by the sympathectomy from regional anesthesia puts these patients at further risk. Hypotension is exacerbated by sympathectomy from regional anesthesia, ongoing hemorrhage, loss of compensatory mechanisms from sympathectomy, and use of magnesium for preeclampsia and preterm labor. If a patient is bleeding excessively under regional anesthesia, the airway should be secured electively. The anesthesia care team can then focus on massive volume resuscitation rather than dealing with airway management emergently during surgery. Massive volume resuscitation may result in airway edema, which can make tracheal intubation difficult if the surgery is performed under regional anesthesia, or may make tracheal extubation difficult at the end of surgery. Postoperatively, patients who have undergone massive volume resuscitation should be cared for in an intensive care unit.

Novel treatment options in severe hemorrhage

Uterine atony is a common cause of, or complication during, intractable obstetric hemorrhage. The uterine smooth muscle fails to contract after delivery of the fetus, resulting in hemorrhage from the dilated venous and arterial bleeders within the placenta. Pharmacologic treatment includes use of intravenous oxytocin, intramuscular methylergonovine, intramuscular 15-methyl prostaglandin $F_{2\alpha}$, and rectal misoprostol. If pharmacologic treatment fails, surgical therapy may be necessary, such as B-Lynch procedure, Bakri balloon placement, or hysterectomy.

The B-Lynch procedure involves suturing the uterus with a single, long, absorbable suture to avoid hysterectomy. The suture is run over the uterus to fold the uterus over itself, while compressing uterine blood vessels [42]. Bakri balloon, a fluid-filled balloon, inserted inside the uterine cavity to achieve hemostasis in cases of postpartum hemorrhage because of placenta previa/accreta, causes a tamponade effect and has been found useful in providing hemostasis [43]. Recently, the uterine sandwich technique, a combination of B-Lynch compression suture and Bakri balloon for patients who

have uterine atony presenting with postpartum hemorrhage, has shown excellent results [44]. Hysterectomy may be the only option to control bleeding in uncontrolled hemorrhage.

Coagulation monitoring during massive obstetric hemorrhage is crucial to successful management. Thromboelastography has been used to detect coagulation defects associated with intraoperative blood loss in parturients and may help in monitoring coagulation parameters and reducing use of blood and blood components in hemorrhaging parturients [34,45].

Some blood conservation techniques that can be used rapidly and safely during intractable obstetric hemorrhage include acceptance of a lower hematocrit as a trigger for transfusion, erythrocyte salvage, rectal misoprostol, intravenous desmopressin, and intravenous recombinant factor VIIa. Additional techniques for hemorrhage prophylaxis, if time and hemodynamics allow, include use of preoperative subcutaneous recombinant erythropoietin [46,47], preoperative autologous blood donation [34,48], preoperative placement of bilateral hypogastric artery balloon catheters [49,50], and intraoperative autologous blood donation with acute normovolemic hemodilution [51].

Erythrocyte salvage has increased in obstetrics during the past few years. The main concern in obstetrics is amniotic fluid embolism [52–55]. Complete elimination of fetal squamous cells from filtered erythrocyte salvage suspension using a leukocyte reduction filter has been suggested [56].

Rectal administration of misoprostol has been recommended (1000 μg) as a means to control excessive blood loss during the third stage of labor [57]. Recommended doses of 400 to 600 μg (oral or per rectum) have had some success compared with placebo [58]. Intravenous desmopressin reduces intraoperative hemorrhage by increasing platelet aggregation [51,59]; the dosage is 0.15 to 0.3 μg/kg over 30 minutes.

Intravenous recombinant factor VIIa was recently reported to control intractable hemorrhage [60,61]. Its use in obstetrics is limited. It activates factor Xa production and increases the rate and amount of thrombin generation. The dosage is 60 μg/kg. It has a short half-life and redosing may be necessary.

Hypertension

Maternal morbidity and mortality statistics

Hypertensive disease affects roughly 6% to 8% of all pregnancies and is the second leading cause of maternal morbidity and mortality in the United States, whereas in the United Kingdom it ranks fourth. It accounts for almost 15% of pregnancy-related maternal deaths and is a major risk factor for fetal morbidity and mortality [62,63].

In the United Kingdom, although the trend has been toward a decline in maternal deaths from hypertension, 46% of patients showed clear evidence

of substandard care in deaths that could have been avoidable. As the single largest cause of death, intracranial hemorrhage indicates failure of effective antihypertensive therapy. Late or failure to obtain consultation from an expert obstetrician was another factor in the mortality from hypertension.

Preeclampsia

Unique to humans, preeclampsia is a multiorgan disease of unknown origin. Symptoms present themselves in a normotensive woman after the 20th week of gestation. The risk for developing preeclampsia is greater in women who have preexisting conditions, such as chronic hypertension, diabetes, antiphospholipid syndrome, and collagen vascular disease.

Risk assessment

In risks associated with preeclampsia and eclampsia, the current CEMACH study (2000–2002) shows that among 14 accounted deaths from eclampsia and preeclampsia, 9 women died from intracranial hemorrhage, 1 from acute respiratory distress syndrome (ARDS), 2 from severe multiorgan failure, and 2 from disseminated intravascular coagulation [1]. Table 1 shows the causes of death compared with previous triennium reports. Risks from untreated hypertension, compounded by a low platelet count, place parturient women at increased risk for intracranial hemorrhage. Understanding the pathophysiology provides a basis for clinical and anesthetic management.

Table 1
Number of deaths by cause due to eclampsia and preeclampsia; United Kingdom 1988–2002

	Triennium				
Cause of death	1988–1990	1991–1993	1994–1996	1997–1999	2000–2002
Cerebral					
Intracranial hemorrhage	10	5	3	7	9
Subarachnoid	2	0	1	0	0
Infarct	2	0	0	0	0
Edema	0	0	3	0	0
Subtotal	14	5	7	7	9
Pulmonary					
ARDS	9	8	6	6	1
Edema	1	3	2	2	0
Subtotal	10	11	8	8	1
Hepatic					
Rupture	0	0	2	2	0
Failure/necrosis	1	0	1	1	0
Other	2	4	2	2	4
Subtotal	3	4	5	5	7
Total	27	20	20	20	14

From Neilson J. Pre-eclampsia and eclampsia. In: 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. p. 79–84; with permission.

Complications from severe preeclampsia include pulmonary edema and the development of ARDS, resulting in maternal mortality. Pulmonary edema, both cardiogenic and noncardiogenic, is a serious complication of severe preeclampsia with an incidence of approximately 3% [64]. Cardiogenic pulmonary edema is caused by impaired left ventricular systolic or diastolic function and is more prevalent in patients who have severe chronic hypertension, valvular heart disease, or cardiomyopathy. Noncardiogenic pulmonary edema results from increased capillary permeability, iatrogenic fluid overload, an imbalance between colloid osmotic pressure and hydrostatic pressure, or a combination of these factors [65].

Strategies to minimize risk and prevent morbidity and mortality

Hospitalization and bed rest are effective treatment for women who have mild preeclampsia. The primary goals of minimizing the risks and goals of management include (1) prevention of convulsions, (2) control of hypertension, and (3) stabilization of cardiovascular status and optimization of intravascular volume.

Prevention of convulsions

Because of several positive effects, magnesium sulfate (Mg^{++}) remains the preferred prophylactic treatment for seizure in the United States and is gaining popularity in the United Kingdom [66]:

1. It depresses both central and peripheral nervous systems; its mechanism of action involves generalized central nervous system depression, which is mediated by N-methyl-D-aspartate receptors
2. It reduces hyperreflexia
3. It acts at the neuromuscular junction through decreasing the amount of acetylcholine liberated from the presynaptic junction, the sensitivity of the motor end plate to acetylcholine, and the excitability of muscle membrane
4. It produces mild to moderate vasodilation
5. It depresses uterine hyperactivity to improve uterine blood flow
6. It suppresses cortical neuronal burst firing and electroencephalographic spike generation
7. It opposes Ca^{++}-dependant arterial constriction and relieves vasospasm

Because Mg^{++} impairs peripheral neuromuscular transmission at the neuromuscular junction, the intensity of the neuromuscular block after muscle relaxants are administered during general anesthesia correlates with elevated serum magnesium and decreased serum calcium levels [67].

Control of hypertension

Another important goal is to control blood pressure to prevent exacerbation with the propensity for intracranial hemorrhage. Traditionally, the

threshold for treating hypertension is a diastolic blood pressure of 105 to 110 mm Hg or a mean arterial pressure of 125 to 126 mm Hg. Diastolic blood pressure is a useful index of preeclampsia severity; current thinking is that the pressure during systole causes intracerebral hemorrhage. Recognition of this concept should be incorporated into guidelines to ensure effective reduction of systolic blood pressure. Therefore, the recommendation is for clinical protocols to identify a systolic blood pressure above which urgent and effective antihypertensive treatment is required [1].

Hydralazine. Hydralazine is no longer the preferred antihypertensive for acute blood pressure control during pregnancy. Recent studies have shown that it may decrease uterine blood flow by as much as 25% [68,69] and may be associated with neonatal thrombocytopenia [70]. Furthermore, the slow onset, delayed peak effect, and compensatory tachycardia make hydralazine a less than ideal agent for attenuating the hypertensive response to laryngoscopy and intubation during the administration of general anesthesia in women who have preeclampsia.

Labetalol. Labetalol a combined α- and β-adrenergic receptor antagonist (ratio 1:3 when given orally and 1:7 when given intravenously). Labetalol is found to be equally as, if not more, effective as hydralazine [71] in lowering the blood pressure. Labetalol decreases maternal systemic vascular resistance without increasing heart rate or decreasing cardiac index, uterine blood flow, or fetal heart rate [72]. The initial intravenous dose of labetalol is 10 to 20 mg, and this can be doubled every 10 minutes to a maximum dose of 300 mg. Labetalol crosses the placental barrier, but neonatal hypoglycemia and hypotension, initially believed to be a theoretic concern, are rarely seen [73]. Labetalol in doses of 1 mg/kg is effective in blunting the hypertensive response to tracheal intubation in patients who have preeclampsia undergoing general anesthesia [74].

Nicardipine, a calcium channel blocker, inhibits the influx of extracellular calcium into smooth muscle cells through the slow channels. The vascular effects predominate in arterial and arteriolar smooth muscle. Nicardipine has fewer negative inotropic effects and more selective action on peripheral vasculature than nifedipine, and effectively lowers blood pressure.

Optimization of intravascular volume

An important goal is to stabilize the patient's cardiovascular status and optimize the intravascular status, which includes (1) appropriate hemodynamic monitoring, (2) adequate volume resuscitation, and (3) adequate perfusion status. Evaluation of the patient's fluid balance must include a strict intake/output chart, placement of an indwelling urinary catheter, and (if possible) an assessment of the patient's current weight. The goal of fluid therapy is to provide an ideal intravascular volume, maintain a satisfactory urinary output, have immediate intravenous access for administration

of therapeutic agents, and compensate for any reduction in preload and afterload during administration of epidural anesthesia.

A paucity of data exists regarding the ideal volume and type of intravenous fluid (crystalloid or colloid) for patients who have preeclampsia. Preeclampsia is associated with a complex set of hemodynamic changes, and predicting how patients will respond to fluid loading is difficult [75]. Excessive administration of crystalloid or colloid may result in pulmonary or cerebral edema. Little evidence shows that colloid preloading before regional anesthesia is more beneficial than crystalloid solutions. However, the use of certain colloids is debatable. A study comparing albumin use for plasma volume expansion versus no albumin in nonpregnant patients showed that albumin increased risk for death [76]. Similarly, increased mortality was associated with the use of colloid for resuscitation compared with crystalloid [77].

Invasive monitoring may become necessary in patients who have low urinary output whose preeclampsia does not respond to multiple fluid challenges, patients who have pulmonary edema, and those who have intractable hypertension [65].

Anesthetic management

Preanesthetic evaluation

It is important for the anesthesiologist, as part of the interdisciplinary team, to be involved early to help control hypertension, stabilize the hemodynamic status, and optimize intravascular resuscitation. It is also prudent to have a well-planned, yet flexible, anesthetic strategy, because the situation may change suddenly.

Maternal monitoring

For patients who have mild preeclampsia, close routine monitoring with pulse oximeter and automated blood pressure cuff are often sufficient. For those who have severe preeclampsia, a radial arterial catheter is recommended for accurate monitoring of arterial blood pressure and blood sampling (arterial blood gases, complete blood count with platelets, coagulation panel, renal and liver function tests, and appropriate drug levels).

Analgesia for labor and delivery

The appropriate anesthetic intervention, particularly with the well-timed placement of an epidural block, can minimize or negate the unnecessary hazards and risks for general anesthesia, including exacerbation of hypertension during induction, intubation, and emergence, and the problem of encountering difficult or failed intubation.

Other benefits of epidural labor analgesia include complete pain relief during labor; attenuation of any exaggerated hypertensive response to

pain; reduction through sympathetic block of the circulating levels of catecholamines and stress-related hormones, which facilitates blood pressure control; vasodilation secondary to the sympathetic block, which improves intervillous blood flow; and stable cardiac output. Furthermore, the block can be extended to provide surgical anesthesia for instrumental or surgical delivery.

Maintenance of analgesia throughout labor can be accomplished with the use of a continuous infusion or patient-controlled epidural analgesia device. Currently, a mixture of a low concentration of bupivacaine (0.0625%) and a lipid-soluble narcotic such as fentanyl (2.0–2.5 μg/mL) has been shown to provide excellent sensory analgesia with minimal or no motor block [78]. Administration of 0.0625% or 0.125% bupivacaine with a lipid-soluble narcotic provides better analgesia than bupivacaine alone [79].

Anesthesia for cesarean section

General considerations

Patients who have preeclampsia scheduled for cesarean section have several important general considerations, including meticulous examination of the airway, administration of aspiration prophylaxis, availability of blood products, prevention of aortocaval compression, administration of increased FiO_2 (face mask), establishing a second peripheral intravenous line, immediate access to a difficult airway cart, application of standard American Society of Anesthesiologists (ASA) monitoring, invasive hemodynamic monitoring if required, and monitoring of the fetal heart rate pattern until the beginning of the surgery.

Regional anesthesia

Regional anesthesia (spinal or epidural) is the preferred method for cesarean deliveries because of the lower maternal morbidity compared to general anesthesia [80], it provides better hemodynamic control (to prevent exacerbation of blood pressure during induction and intubation), it blunts neuroendocrine stress response [81], patients are awake and able to interact with the infants, and it prevents transient neonatal depression associated with general anesthesia.

Epidural anesthesia is the regional anesthetic technique most commonly used for patients who have preeclampsia [82]. Investigators who have studied the systemic and pulmonary artery pressures in patients who have severe preeclampsia undergoing cesarean section have shown stable hemodynamic status with epidural anesthesia versus marked exacerbations in mean arterial pressure and pulmonary capillary wedge pressure during induction, intubation, and extubation with general anesthesia [82].

Ideally, in patients who have preeclampsia undergoing urgent cesarean section, a functioning epidural block should already be in place. The

preexisting block can be augmented with either 3% 2-chloroprocaine or pH-adjusted 2% lidocaine to provide rapid surgical anesthesia. The fetal heart rate should be monitored in the operating room until immediately before preparation for and initiation of surgery. Several studies have shown the usefulness and safety of spinal anesthesia in patients who have severe preeclampsia undergoing cesarean section [83]. Furthermore, the hemodynamic effects of spinal anesthesia are also found to be stable [84–86]. The combined spinal and epidural technique has also been shown to be safe and effective in women who have severe preeclampsia undergoing cesarean section [87].

General anesthesia

General anesthesia is required in cases of nonreassuring fetal heart rate requiring emergency cesarean section (no preexisting epidural catheter), coagulopathy that precludes the use of regional anesthesia, and patient refusal of regional anesthesia. The risks for general anesthesia in women who have preeclampsia include difficult tracheal intubation, the potential for aspiration of gastric contents, exacerbated hypertensive response to endotracheal intubation, impairment of intervillous blood flow, and drug interaction between magnesium and muscle relaxants.

Airway evaluation in patients who have preeclampsia undergoing general anesthesia is critical because endotracheal intubation may be difficult. Two of four maternal deaths in 442 cases reviewed [88] resulted from cerebral hypoxia secondary to failed intubation. Therefore, airway evaluation is crucial and a predicted difficult airway requires appropriate airway management preparation. The authors recently reported the successful use of intubating laryngeal mask (ILMA) after a failed tracheal intubation in a patient who had eclampsia undergoing emergency cesarean section. Rapid intervention with the ILMA averted maternal and fetal catastrophes and resulted in a positive outcome for mother and baby [89].

General anesthesia used in patients who have preeclampsia with hypertension causes exacerbation of blood pressure from stimulation during laryngoscopy, tracheal intubation, and surgical incision. Any acute increase in blood pressure, particularly in the face of coagulopathy, places the patient at risk for intracranial hemorrhage. Other reasons to attenuate the hypertensive response include a maternal risk for increased myocardial oxygen consumption leading to myocardial infarction, cardiac arrhythmias, and pulmonary edema. The fetus is also at risk from maternal hypertensive surges secondary to a significant reduction in uterine blood flow [90].

Postoperative care/critical care

After labor and delivery, all patients who have preeclampsia should be monitored in the recovery room or obstetric intensive care unit for the next 24 hours or until adequate diuresis is established. Management of

a subset of patients who have severe preeclampsia/eclampsia and HELLP syndrome who are critically ill requires involvement of a multidisciplinary team, including the obstetric, anesthetic, and critical care teams. Reassessment of this subgroup of critically ill patients in the immediate postpartum period is essential because of the high mortality rate. Patients who have multiorgan failure ARDS resulting from severe preeclampsia require postoperative mechanical ventilation [91] in the intensive care unit. Successful management of these patients who have high-risk severe preeclampsia/eclampsia requires good communication among the obstetric, anesthesia, neonatology, nursing, and critical care teams.

Anesthesia-related maternal mortality

Anesthesia-related death is the seventh leading direct cause of maternal mortality in the United States and United Kingdom, and accounts for 1.6% of all pregnancy-related deaths in the United States [3,29]. Since the 1980s, a significant reduction has been seen in anesthesia-related maternal deaths, with the success being attributed to increased use of regional anesthesia, the widely adopted policy of limiting oral intake during labor, and the effective measure of providing aspiration prophylaxis before operative delivery. In the current CEMACH study [1], six direct deaths that were associated with general anesthesia, suggesting a risk of 1 death per 20,000 maternal general anesthetics administered, similar to the statistics reported in the 1982 to 1984 triennium [1]. Most anesthesia-related deaths in United States and United Kingdom are associated with general anesthesia for operative delivery and are related to difficult/failed intubation. Difficult pulmonary ventilation, resulting in failure to oxygenate, or pulmonary aspiration remain the primary factors responsible for anesthesia-related maternal mortality [29,92]. A maternal death is devastating to all involved; however, in obstetric patients (parturients), mortality is 200% (mother and baby) with significant medicolegal implications.

There were no direct deaths attributed to regional anesthesia in this triennium. However, as the use of general anesthesia in obstetrics continues to decline, the anesthesia trainees' experience in basic airway management in obstetrics also continues to decline. Given the fact that 80% of anesthesia-related fatalities occur during emergency cesarean sections, the incidence of failed intubation is higher during emergencies that occur during nights and weekends. The CEMACH report highlighted the lack of supervision of trainees during emergencies as an area of concern.

Protocols to manage difficult or failed tracheal intubation in obstetric anesthesia are absolutely essential. Every anesthesia practitioner must have a preformulated strategy before induction to deal with a difficult or failed intubation. The decision to abandon repeated attempts at tracheal intubation must be made promptly. The importance of prompt and competent decision making in these critical situations, having appropriate equipment

immediately available in the labor and delivery suite operating room to deal with a difficult airway, and having advanced airway skills cannot be overemphasized.

Airway

Risk assessment

Pregnancy produces several anatomic and physiologic changes in the body, resulting in an increased risk for airway-related complications and pulmonary aspiration. Rocke and colleagues [93] suggested using the relative risk score to allow better prediction of difficult tracheal intubation in obstetric patients. This landmark study of obstetric patients showed that the relative risk for experiencing a difficult tracheal intubation compared with an uncomplicated class I airway assessment (risk ratio of 1.0) increased to 3.23 with class II; 7.58 with class III; 11.3 with class IV; 5.01 with a short neck indicating decreased mobility of neck; 8.0 with protruding maxillary incisors; and 9.71 with a receding mandible indicating decreased thyromental distance. Using the combination of risk factors showed that a combination of either Mallampati class III or IV airway plus protruding incisors, short neck, or receding mandible predicted a greater than 90% probability of encountering a difficult laryngoscopy. Other risk factors during pregnancy include, weight gain in pregnancy, increased breast size, and obesity. When these risk factors are encountered with a parturient who has a greater than 90% probability prediction of having a difficult airway, anesthesiologists should have a preformulated plan for dealing with a difficult airway or failed tracheal intubation.

In a recent review of maternal deaths in Michigan [94], anesthesia was the primary cause in eight of these deaths. However, no maternal deaths were associated with failed intubation during induction or with aspiration. Rather, all eight deaths occurred during emergence and recovery, which is becoming more frequent in surgical patients.

Strategies to minimize risk and prevent morbidity and mortality

Because most anesthesia-related deaths occur during operative delivery, strategies must be outlined to manage difficult or failed tracheal intubation. An emergency cesarean delivery is undertaken for either maternal or fetal indications, or both. Maternal hemorrhage or fetal distress dictates the need for an emergent or urgent delivery. Even in an emergency situation, a quick preanesthetic evaluation and determination of a difficult airway is possible, thus allowing for appropriate airway management [95].

The ASA Task force recently published a difficult airway algorithm (DAA) [96] for managing the difficult airway. The ASA Practice Guidelines for Obstetrical Anesthesia [97] recommend that labor and delivery units should have equipment (eg, basic airways, laryngeal mask airway [LMA], Combitube) and personnel readily available to manage airway emergencies,

including during regional anesthesia. These resources can provide life-saving oxygenation and pulmonary ventilation, enable access for securing the airway to prevent pulmonary aspiration, and reduce the incidence of maternal complications [98].

Fundamental steps outlined in the ASA/DAA can be applied to special situations, such as obstetric anesthesia. Prioritizing the airway management strategies after a failed initial attempt at tracheal intubation can influence the final outcome for mother and baby. The priorities and management goals after a failed initial attempt should be (1) maternal oxygenation, and thereby fetal oxygenation, (2) airway protection, (3) prevention of pulmonary aspiration, and (4) expeditious delivery. The flow chart for an unanticipated, difficult, or failed tracheal intubation is shown in Fig. 3.

After failed tracheal intubation, the DAA recommends calling for help, returning to spontaneous ventilation, or awakening the patient. In all but the most urgent situations (eg, maternal hemorrhage or severe fetal distress), the mother is awakened after the failed first attempt and the fetus is reassessed (see Fig. 3). However, in an acute emergency (eg, umbilical cord prolapse and severe fetal distress) awakening the patient may not be possible. Therefore, balancing the priorities of oxygenation, prevention of pulmonary aspiration, and delivery of the fetus become critical (see Fig. 3).

Oxygenation becomes extremely critical. If pulmonary ventilation using conventional facemask is successful, a quick assessment for failed tracheal intubation should be performed to enhance the success rate of the second attempt. Failure to visualize the cords may be related to incorrect positioning, inadequate laryngoscopy, inadequate muscle relaxation, or failure to apply adequate external laryngeal pressure.

The recommendation is that the second attempt at laryngoscopy be considered the best attempt at intubation. To increase the success rate, it should be performed by a reasonably experienced anesthesiologist, the optimal sniff position used, and external laryngeal manipulation applied. Additionally, the laryngoscope blade type and handle may need to be changed.

If the best attempt at tracheal intubation is unsuccessful, a third attempt is really not an option in this emergent scenario; nonsurgical airway management techniques that allow ventilation and oxygenation must be implemented immediately. These approaches include the use of LMA for the nonemergency pathway (cannot intubate/can ventilate), and the Combitube, rigid scopes, and transtracheal jet ventilation for the emergency pathway (cannot intubate /cannot ventilate). After failed conventional facemask ventilation and tracheal intubation, the ASA/DAA recommends the use of the LMA [95].

Laryngeal mask airway

Classic laryngeal mask airway. The classic LMA has been widely used for difficult obstetric airways [99–104] without any episodes of gastric regurgitation or pulmonary aspiration. In parturients undergoing elective cesarean

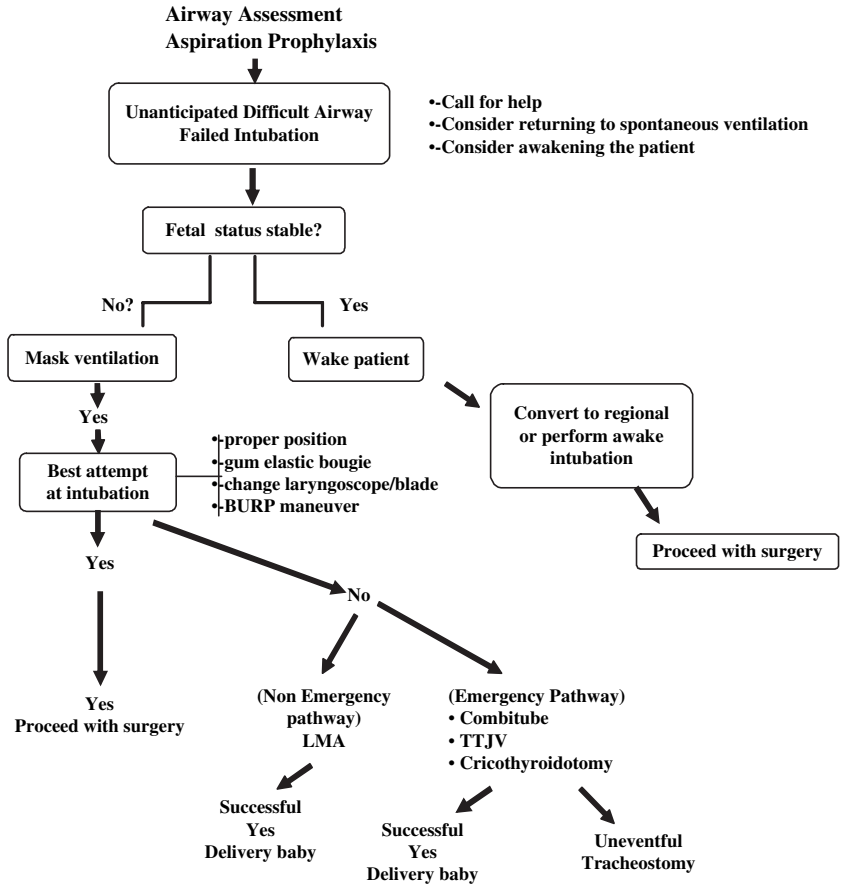

Fig. 3. Algorithm for an unanticipated difficult or failed intubation in a parturient undergoing an emergency cesarean section, with special emphasis on the goals of maintaining maternal oxygenation, airway protection, and delivery of the baby. BURP, backwards, upwards, rightwards pressure; LMA, laryngeal mask airway; TTJV, transtracheal jet ventilation.

delivery, Han and colleagues [105] reported the successful use of the classic LMA as a ventilatory device in 1060 of 1067 patients. No episodes of hypoxia, regurgitation, or aspiration occurred [105]. The successful use of the classic LMA after failed tracheal intubation in obstetrics has been reported in 17 instances.

Intubation through classic laryngeal mask airway. In patients requiring tracheal intubation, fiberoptic-guided tracheal intubation through the classic LMA is reliable [106]. However, a longer tracheal tube (eg, Endotrol, microlaryngeal, or nasal Ring-Adair-Elwyn) is needed. The Cook Aintree catheter

(Bloomington, Illinois) may also be used to facilitate intubation through the classic LMA.

Intubating laryngeal mask airway. The LMA Fastrach or Intubating LMA (ILMA) is designed to specifically overcome the problems associated with blind tracheal intubation through the classic LMA [107]. The ILMA is particularly useful during failed intubation in an emergency cesarean section because it provides oxygenation and a conduit for tracheal intubation, and prevents pulmonary aspiration. Several studies have shown the successful use of ILMA to help visually unassisted tracheal intubation in patients who have difficult airways [106–108]. The ILMA was used successfully after failed tracheal intubation during an emergency cesarean section in a patient who was morbidly obese and eclamptic [109]. The authors had a second case in which regional anesthesia had failed and was followed by general anesthesia, resulting in failed tracheal intubation. The ILMA again proved to be a life-saving device.

ProSeal laryngeal mask airway. The ProSeal LMA is a new, unique device that represents a substantial change in LMA design. The ProSeal LMA offers several advantages over the classic LMA for failed tracheal intubation in obstetrics: (1) the seal is 10 cm H_20 higher, giving it greater ventilatory capability [110]; (2) it enables correct positioning, isolating the glottis from the esophagus, and therefore may provide airway protection and protect against pulmonary aspiration [111,112]; (3) a gastric tube can be easily inserted to empty the stomach of fluid and air insufflated during difficult face mask ventilation. The ProSeal LMA has been used successfully in at least six case reports after failed intubation during emergency cesarean sections [111,113–117]. After failed tracheal intubation, and once the anesthesia practitioner is able to successfully achieve pulmonary ventilation and oxygenation, caution must be used in selecting a nonirritating inhalation anesthetic and an adequate depth of anesthesia. Recent studies show that sevoflurane provides rapid, smooth induction; adequate depth of anesthesia; and is the least irritating agent [118], and helps facilitate tracheal intubation through the LMA in patients who have a difficult airway [119].

Combitube. The Combitube should be considered in emergency airway situations, especially when patients are at risk for pulmonary aspiration and tracheal intubation has failed [120,121]. The DAA suggests using Combitube after failed pulmonary ventilation with conventional facemask and LMA [95]. The successful use of the Combitube has been described after failed tracheal intubation in an emergency cesarean section [120].

The DAA incorporated the use of Combitube in the emergency pathway (ie, the life-threatening "cannot ventilate, cannot intubate" situation. This situation is encountered in approximately 1 of 100,000 cases, and establishing ventilation and oxygenation is critical. Similarly, it may also be particularly

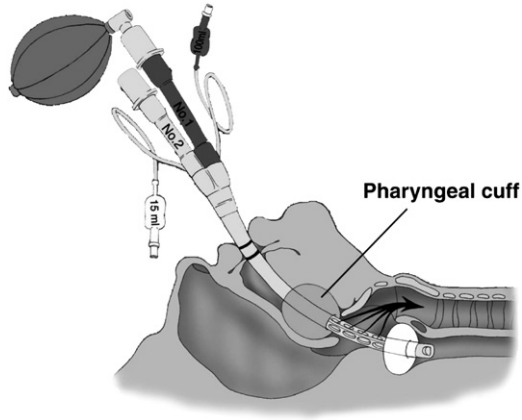

Fig. 4. Combitube in esophagus. The tube is advanced until the black rings are at the level of the teeth. The distal cuff is inflated with 10 mL of air to seal the esophagus, and the proximal cuff is inflated with 80 mL of air, securing the tube in position and occluding the nasal and oral passages. Ventilation is attempted through lumen.1. (*Courtesy of* Baylor College of Medicine, Houston, TX; with permission.)

useful for difficult or failed tracheal intubations in obstetric patients, who are especially at risk for gastric regurgitation and pulmonary aspiration. The Combitube may offer significant advantages over the LMA in the parturient. These advantages include isolation of the stomach from the glottic area and minimal preparation. Oxygenation and pulmonary ventilation can be achieved rapidly, especially because the parturient is prone to rapid arterial oxygen desaturation. The Combitube is shown to prevent pulmonary aspiration during cardiopulmonary resuscitation [122] and protect the airway from pulmonary aspiration of gastric contents during anesthesia [123]. Combitube

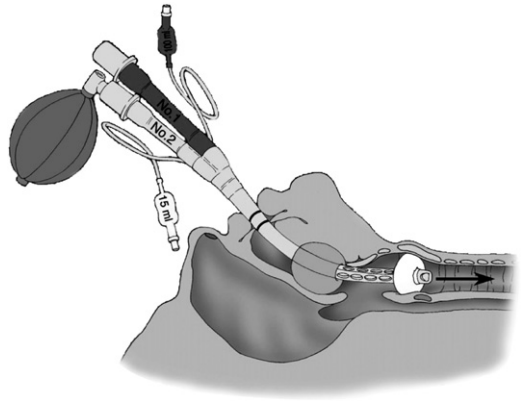

Fig. 5. Combitube placement in trachea. The Combitube is placed in trachea; the ventilation is shifted to lumen No. 2. (*Courtesy of* Baylor College of Medicine, Houston, TX; with permission.)

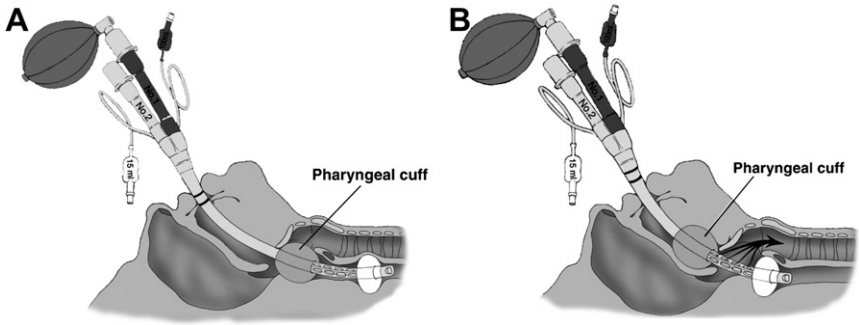

Fig. 6. (*A*) Improper placement of Combitube. Excessive insertion depth of the Combitube, causing obstruction of the glottic opening. Ventilation is not possible. (*B*) Correct position of the Combitube in the esophagus after readjustment. The Combitube is pulled back 2 cm (indicated by the two black rings) and ventilation from the side orifices into the trachea. (*Courtesy of* Baylor College of Medicine, Houston, TX; with permission.)

is a disposable double-lumen tube with two cuffs designed for blind insertion. Ventilation is initially attempted through lumen 1, which forces air into the trachea (Fig. 4).

Causes of failed ventilation with the Combitube include inadvertent tracheal placement (5%), deep insertion laryngospasm, and bronchospasm. The steps to troubleshoot are important to understand, especially when confronted with a critical airway. The Combitube is designed to enter the esophagus after blind insertion. If ventilation is difficult through the blue lumen 1, the Combitube could be in the trachea, which requires switching ventilation to the clear lumen 2, allowing air to enter the trachea directly through the open end (Fig. 5). Sometimes the Combitube is inserted too deep, in which case the pharyngeal balloon must be deflated, the Combitube pulled back 1 to 2 cm, and ventilation switched back to the blue lumen (Fig. 6A, B).

Other airway devices

Other airway devices include the LaryngealTube S (LTS) and the Airtraq. The LTS is a new supraglottic airway device. It has a second lumen for suctioning and gastric drainage. It may provide higher airway seal pressures than a classic LMA. In contrast to Combitube, it has only one adapter for ventilation. LTS may be useful in situations and patients who are at risk for aspiration. In a recent report, the LTS was used successfully to establish ventilation and oxygenation after failed intubation in an emergency cesarean section [124].

The Airtraq is a new disposable intubating laryngoscope. It is designed to provide a view of the glottis without alignment of oral, pharyngeal, and tracheal axes. Two cases of rapid tracheal intubation with Airtraq laryngoscope have been reported recently in morbidly obese parturients undergoing emergency cesarean delivery after failed tracheal intubation [125].

Cricothyroidotomy and transtracheal jet ventilation

When oxygenation and ventilation are not established with either the LMA or Combitube, this presents a grave life-threatening cannot ventilate, cannot intubate situation, and more invasive techniques such as cricothyroidotomy or transtracheal jet ventilation may become necessary [126].

Extubation

Having a strategy established for extubating the airway after difficult airway/failed tracheal intubation, including using an airway exchange catheter, is as crucial as the alternative plan for difficult tracheal intubation [95,127].

Local anesthetic toxicity

The problem of maternal deaths associated with local anesthetic toxicity has almost been eliminated in obstetrics.

Risk assessment

Almost 30 years ago, Albright [128] was the first to report several cases of fatal cardiac toxicity associated with use of the long-acting lipophilic local anesthetics, bupivacaine, and etidocaine in pregnant women. His editorial and a second one in 1984 [129], outlining scientific investigations and evidence of fatal cardiotoxicity, led to the eventual U.S. Food and Drug Administration withdrawal of 0.75% bupivacaine for epidural anesthesia in obstetrics. The common theme in those fatalities, who were otherwise healthy patients, was the apparent lack of response to standard resuscitative measures. Albright was prescient in his observation that both bupivacaine and etidocaine are lipophilic, a physical characteristic that has since been shown to correlate with particularly intransigent cardiac toxicity among local anesthetics [130].

Strategies to minimize risk and prevent morbidity and mortality

Albright's scientific observation led to implementation of practice standards that have reduced the incidence of cardiovascular catastrophes after local anesthetic use. These guidelines include implementing fractionated test doses, thus replacing the administration of concentrated local anesthetics as a bolus; an epinephrine-containing test dose to detect intravascular injection of local anesthetic through epidural catheter, and use of safe dose limits.

Clinical features of local anesthetic toxicity

The earliest clinical manifestations of local anesthetic toxicity include lightheadedness, altered mental status, agitation, slurred speech, and visual disturbance [131]. Early changes in vital signs include tachycardia and hypertension [131]. The clinical presentation of more severe toxicity is classically a combination of central nervous system excitation, cardiac arrhythmias, conduction blockade, and myocardial depression. Typically

severe bupivacaine cardiac toxicity presents as hypotension with bradycardia leading quickly to malignant ventricular arrhythmias and cardiovascular collapse that is highly resistant to standard resuscitation [128,132]. Most recommended drugs are supportive: sympathomimetics for blood pressure and inotropic support, and amiodarone for arrhythmias [130]. Use of β-adrenoceptor antagonists, calcium channel agonists, and local anesthetics for treating rhythm disturbances from local anesthesia toxicity are contraindicated.

Mechanism of toxicity

The standard model, established by Clarkson and Hondeghem [133], holds that the pronounced inhibition of cardiac voltage–gated sodium ion channels accounts for the differential toxicity of bupivacaine. Furthermore, it is well established that hypoxia exacerbates bupivacaine toxicity [134], suggesting that respiration is a clinically important target of bupivacaine.

Lipid reversal resuscitation

Animal studies have shown successful resuscitation from local anesthetic toxicity with the administration of lipid solutions. These observations suggest that lipid infusion might be useful in treating local anesthetic toxicity, and a recent editorial [135] described lipid rescue as a possible "silver bullet" for bupivacaine overdose.

Mechanisms of lipid rescue

Lipid-treated hearts showed a more rapid decline in myocardial bupivacaine content than controls; mean time constants (95% CIs) were 37 [32,43] and 83 seconds [66,107] for lipid-treated and control hearts, respectively ($n = 5$ for both groups, $P < .0002$). Stehr and colleagues [136] recently found in isolated rat hearts that lipid emulsion reverses bupivacaine-induced contractile depression at concentrations that are too low to provide a lipid sink effect, thus proposing a metabolic explanation for this beneficial effect.

Recommendations based on evidence of clinical efficacy for lipid rescue resuscitation

Given the highly reproducible benefit in reversing animal models of bupivacaine-induced toxicity, some have suggested that lipid emulsion should be stocked at sites where regional anesthesia is performed [137]. Currently, 0.5% bupivacaine is used for patients undergoing cesarean section with epidural anesthesia. Therefore, educating anesthesiologists is the first step to achieving this goal. Secondly, stocking lipid solutions in all labor and delivery suites is recommended. Experts have proposed that lipid be stocked and kept with a recommended dosing regimen in operating rooms and locations where regional anesthesia is performed.

Lipid infusion is reserved for use in cardiac arrest caused by local anesthetic toxicity that resists standard resuscitative measures. If asystole, malignant arrhythmias, or severe hypotension persist, standard advanced

cardiac life support (including ventilation with 100% oxygen and chest compressions) should be continued, and then 20% intralipid intravenously should be infused a bolus injection of 1.5 mL/kg, followed by continuous infusion 0.25 mL/kg per minute for 30 minutes. If no improvement is seen, the bolus should be repeated one to two times. For declining blood pressure, rate of infusion should be increased to 0.5 mL/kg per minute. Once sinus rhythm is restored, ventricular ectopy or other arrhythmias may persist, but additional bolus doses are probably not required. The infusion should continue for a full hour and may need to be restarted if blood pressure declines after it is stopped.

Propofol is formulated with 10% lipid and has been reported to improve bupivacaine toxicity, which has prompted some clinicians to consider, or perhaps confuse, using propofol as a lipid source during resuscitation from bupivacaine toxicity. However, propofol should not be used when there is any sign of cardiac compromise.

Infusing a lipid emulsion during resuscitation from local anesthetic toxicity reliably rescues animals from overwhelming and otherwise fatal bupivacaine overdose. Recent case reports of clinical efficacy of lipid rescue resuscitation [130] support the value of this technique in treating patients who have severe local anesthetic toxicity. To improve survival from this potentially catastrophic complication of regional anesthesia, general use of the regimen for treating local anesthetic cardiac toxicity should be incorporated into the standard approach for treating local anesthetic systemic toxicity.

Obesity

Maternal morbidity and mortality statistics

Obesity has increased worldwide, especially in developed countries, over the past decade. In the United States, one third of adult women were found to be obese based on the 1999–2002 National Health and Nutrition examination survey [138]. Additionally, women are delaying pregnancy until after 35 years of age, further compounding comorbidities [96,139]. Obese women also have a propensity to pregnancy-related complications, such as gestational diabetes, gestational hypertension, preeclampsia, fetal macrosomia, shoulder dystocia, failure to progress during labor [140–143], cesarean section [142], and spontaneous abortion after natural conception [144] or infertility treatment [145]. Intraoperative complications in obese parturients include prolonged operative time and excessive hemorrhage [146]. Postoperative complications include endometritis, obstructive sleep apnea [96,139], and wound infection [147]. Problems associated with regional and local anesthesia are increased in the obese parturient [148]. A maternal mortality review in Michigan from 1985 to 2003 showed eight anesthesia-related deaths; 75% of the patients were obese (body mass index >30) and 75% were African American [94], suggesting body weight and race are incriminating

factors. Obesity increases the risk for death during pregnancy [149] and obstetric anesthesia [150].

Risk assessment

Prepregnancy education is vital for obese women to have a successful and safe outcome for mother and baby. Obstetricians must discuss the importance of weight reduction, dietary planning, exercise regime, and behavior modification with their obese patients before they become pregnant [151]. Obese parturients should also be counseled about possible obstetric complications and worsening of preexisting medical comorbidities in the peripartum period. Screening for gestational diabetes should be provided at the earliest possible stage and followed up later. Obstetricians must educate obese parturients about intrapartum difficulties, such as estimating fetal weight, external fetal heart rate monitoring and tocodynamometry, and fetal access during emergency cesarean section [151].

Strategies to minimize risk and recommendations

Pregnancy-related complications, such as gestational diabetes, gestational hypertension, and preeclampsia, are greater in the pregnant patients who are obese. The incidence of fetal complications, such as prematurity, stillbirths, and neural tube defects, is higher in maternal obesity [140,152]. Fetal macrosomia is a common finding in obese parturients [153]. Cesarean section is recommended for fetal macrosomia (fetal weight >5000 g in the nondiabetic parturient [154] and fetal weight >4500 g in the diabetic parturient) [155]. Obstetricians must obtain multidisciplinary consultation from anesthesiology [156], neonatology, and cardiology [157] services. An anesthesiology consultation, early in labor, allows adequate time to develop an anesthetic plan [151]. Anesthetic challenges include difficult intravenous and arterial access, difficult sizing of noninvasive blood pressure cuff, positioning issues for regional and general anesthesia, transportation problems, and finding an adequately sized Herculean operating table. Obese parturients should receive aspiration and thromboembolism prophylaxis [151]. Antibiotic prophylaxis is recommended for elective and emergency cesarean section because of the higher incidence of wound dehiscence and infections in obese parturients [147]. Additional blood products may need to be available, because incidence of intraoperative hemorrhage is higher [146].

Neuraxial analgesia for labor and delivery may be provided through continuous epidural analgesia or continuous spinal analgesia. Combined spinal epidural technique is not desirable because of an untested epidural catheter in the first few hours after the initial spinal injection. A continuous neuraxial (epidural or spinal) catheter technique in labor allows for extension to continuous neuraxial anesthesia for cesarean section. Obese parturients are at risk for an unplanned cesarean section, difficult neuraxial block placement, and difficult airway management [148]. Hence, a well-functioning neuraxial

catheter is essential to proceed safely with an urgent or emergent cesarean section [158]. Neuraxial analgesia issues include difficulty identifying landmarks, which may require use of ultrasound guidance [159] with a 5.0 MHz curved array probe, and use of long neuraxial needles to identify the epidural space or obtain cerebrospinal fluid [148].

General anesthesia in the obese parturient has the compounded risk for pregnancy-related airway complications, obesity-related airway complications, and airway complications related to other comorbidities, such as preeclampsia and diabetes. Airway management strategies should include optimizing patient positioning, using a ramped pillow (straight line between sternal notch to external auditory meatus), an easily accessible difficult airway cart, and availability of experienced anesthesia personnel. The difficult airway cart should be stocked with 6- or 7-mm tracheal tubes, short-handle laryngoscope, Eschmann bougie, Levitan scope (fiberoptic stylet), videolaryngoscope, intubating laryngeal mask airway, Combitube, cricothyrotomy kit, and equipment for transtracheal jet ventilation [160,161].

Postoperatively, patients should be advised about the use of elastic compression stockings, adequate hydration, incentive spirometry, and early ambulation. Adequate analgesia must be provided, preferably neuraxially, to minimize postoperative pulmonary complications [162]. Dietetic education, exercise regimes, and weight reduction planning must be continued in the puerperium [151].

Summary

Maternal deaths in developed countries continue to decline and are rare. Maternal mortality statistics are essentially similar in the United States and United Kingdom. However, the situation is completely different in developing countries, where maternal mortality exceeds 0.5 million every year [163]. This article not only assesses morbidity risks in some of the leading causes of maternal death but also highlights strategies to minimize the risks and prevent maternal morbidity and mortality.

Venous thromboembolism is the leading cause of direct maternal mortality. Risk evaluation and reduction should begin before pregnancy and continue during and after pregnancy. LWMH is the preferred method for prophylaxis and treatment.

Obstetric hemorrhage can be life-threatening. Novel treatments, including nonsurgical and surgical options, should be attempted before cesarean hysterectomy, especially in young patients who desire future pregnancy. Excellent communication and teamwork among obstetric, anesthesia, nursing, and blood bank teams is critical for successful outcome.

Airway-related issues in obstetrics during induction and emergence continue to be problematic. Preoperative airway evaluation, risk assessment, having a preformulated strategy to deal with difficult or failed tracheal intubation, and acquisition of advanced airway skills are critical to avoid maternal morbidity and mortality from airway catastrophes.

Preeclampsia poses serious threats to mother and fetus. The dangers of high systolic blood pressure leading to intracranial hemorrhage require greater recognition and timely antihypertensive intervention. Magnesium sulfate is the preferred anticonvulsant. To avoid potentially serious consequences of fluid overload, careful monitoring of fluid input and output and invasive monitoring are essential. Regional anesthesia is the preferred technique, and offers several advantages over general anesthesia.

Finally, the virtual elimination of local anesthetic toxicity, particularly in obstetrics, is an anesthesia-related success story. Therefore, a heightened awareness of risks, proper communication among disciplines, and a multidisciplinary team approach toward the care of high-risk parturients helps physicians not only execute appropriate care and elevate standard care but also ensures safe outcomes for mothers and babies. Obesity, a growing epidemic, increases anesthesia-related maternal and fetal risks. Preoperative airway evaluation and functional neuraxial analgesia are critical in the safe management of mother and baby. Dietetic modification and behavioral therapy should be undertaken in the peripartum period.

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