Obstetric Mortality

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THERE has been a phenomenal reduction in obstetric death rates in the United States during the past quarter century (fig. 1). A number of factors have contributed to the decline during this period, namely, improved prenatal care, the use of antibiotics, the increasing use of blood banks, a higher incidence of hospital deliveries, and more ready availability of trained anesthetic personnel. The low prevailing maternal mortality rates have led to a philosophy in many quarters that an irreducible minimum has been reached and that the few remaining deaths must be inevitable. Nothing could be further from the truth. Lane recently reported that 77.8 per cent of the obstetric deaths in Chicago from 1956 through 1960 were preventable.[4] These might have been avoided through the application of current knowledge and facilities.

Further reduction in the number of obstetric deaths will follow recognition and attention to the most frequent existing causes. The relative role of the several factors contributing to maternal deaths varies from year to year and from practice to practice. As the number of deaths from one cause is reduced, another assumes a more prominent role. It is thus important that we continuously re-evaluate the factors involved in order to concentrate on the prevalent offending causes.

Sources of Information

Data on maternal mortality are available through the publication, *Vital Statistics of the United States*[1, 2, 3] and through reports in the medical literature. The former publication is based upon death certificates submitted to regional health departments. Although this reference is valuable in that it encompasses all the obstetric deaths in the nation, data available from this source are limited by the facts included on death certificates.

Reports on obstetric mortality in the medical literature emanate from community maternal mortality committees and from institutional surveys. Each of these offers more information about underlying factors contributing to obstetric deaths than reports on vital statistics, though, obviously, they cannot encompass the volume of cases included in the United States Public Health Service publication. Both community mortality committees and institutional surverys can offer the opportunity for careful review of the entire medical history of the case, though this occurs more frequently in the institutional studies.

Classification of Obstetric Deaths

Mortality data are classified in *Vital Statistics of the Unted States* according to the International Classification of Disease.[5] All deaths are classified according to a priority system for tabulating the cause from among those causes listed on the death certificate. The assignment of the cause of death is based upon the disease or condition which started the series of events leading to death. In this classification procedure, untoward effects of treatment, including the administration of drugs, operations, or anesthesia are not classified as a cause of death even though they may have been the immediate cause of the death.

Deaths due to "deliveries and complications of pregnancy, childbirth, and the puerperium" are sub-classified as resulting from complications of pregnancy, delivery with specified complications, complications of the puerperium, and abortion. Toxemia, sepsis or infection, hemorrhage, and "other causes" are listed in each of these categories as the most frequent underlying clinical entities causing death.

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FIG. 1. Obstetric deaths per 10,000 live births, United States—1938–1962.[1, 2, 3]

Since the sixth revision of the International Classification of Disease in 1949, deaths have been tabulated as obstetric only when complications of pregnancy have been indicated as the underlying cause of death. Deaths which are due to non-obstetric causes, such as rheumatic heart disease or hypertensive cardiovascular disease, and which are aggravated by the pregnant state are classified according to the primary disease entity; there is no indication in the vital statistics that the death was in any way related to pregnancy. Also, deaths due to anesthesia or other "therapeutic misadventures" are not indicated as such in any way in the national data. These deaths are classified according to the fundamental disease initiating the chain of events, and no cognizance is given to untoward responses to any form of therapy. The International Classification is reviewed every 10 years, and the next revision is scheduled for 1968. At that time a multiple cause tabulation will probably be established.* The first cause will indicate the disease initiating the chain of events, and the second cause will indicate the factor which was the immediate or direct cause of the death, in the event the latter is other than the original disease.

In figure 2 we note the relative contribution of hemorrhage, toxemia and infection to obstetric mortality in the United States during the past 25 years. Infection was by far the outstanding factor during the early part of this period; deaths from this cause were reduced at the time of the introduction of antibiotics, but they have risen slightly during recent years. The percentage of deaths from toxemia reached a peak during the middle part of this survey, and it has fallen markedly during recent years. The proportion of deaths from hemorrhage had an upward trend until just 10 years ago; this has fallen since, but very slightly.

Many maternal mortality committees report obstetric deaths according to "A Guide for Maternal Death Studies" published in 1957 by the American Medical Association.[6] This program includes a classification of obstetric deaths based upon the International Classification of Disease [5] and the Standard Nomenclature of Disease and Operations.[7] The death of any woman dying of any cause whatsoever while pregnant or within 90 days of the termination of pregnancy is classified as a maternal death. Hemorrhage, toxemia, infection, vascular accidents and anesthesia are listed as the principal causes of obstetric deaths. Thus, obstetric deaths that occur as the direct result of anesthesia given for an obstetric procedure are classified as due to anesthesia. In addition, there is a category of "indirect obstetric deaths," defined as "a death resulting from disease present before or developing during pregnancy (not a direct effect of pregnancy) which was obviously aggravated by the physiological effects of the pregnancy and caused the death." A death in an obstetric patient with rheumatic heart disease who died from pulmonary edema during labor is therefore classi-

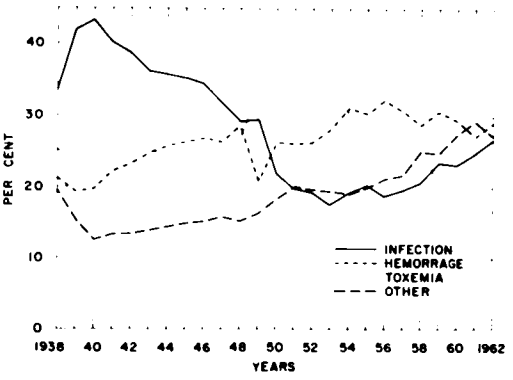

FIG. 2. Percentage of obstetric deaths by principal causes, United States—1938–1962.[1, 2, 3]

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TABLE 1. Obstetric Deaths—Direct Causes

	N. Carolina[8] (1946–1955) (%)	Illinois[8] (1948–1951) (%)	Mississippi[9] (1959) (%)	Baltimore[10] (1936–1958) (%)	Michigan[11] (1950–1952) (%)
Hemorrhage	28.8	34.7	54.3	29.7	38.7
Toxemia	33.7	26.7	31.4	16.3	16.5
Infection	8.0	13.3	5.7	34.3	24.1
Anesthesia	3.2	5.0	5.7	16.3	8.4
Other (including vascular accidents)	26.3	20.3	2.9	3.4	12.3
Total	100.0	100.0	100.0	100.0	100.0

fied as an indirect obstetric death and not a death from heart disease *per se*.

Data from regional maternal mortality studies are consistent with national vital statistics in that hemorrhage, toxemia and infection are the leading causes of obstetric mortality (table 1). In addition, however, anesthesia is included in most of these reports as one of the frequent contributing factors. Embolic deaths from non-septic thrombi and amniotic fluid emboli are also included as direct obstetric causes of death in many study reports. Heart disease is the most frequently reported cause of indirect obstetric death, and on occasion it is recorded as a more prominent factor than direct causes such as anesthesia and vascular accidents.[11]

Evaluation of the underlying factors responsible for obstetric mortality in each of the principal categories is based primarily upon community and institutional reviews. Means for further reduction of maternal mortality rates must be based upon reports of clinicians actively engaged in the field of obstetric practice. Utilization of classifications of obstetric deaths and of the terminology accepted in these classifications and established in the literature offer common denominators for comparison of experience and results.

Hemorrhage

In his text written in 1851, Ramsbotham reviewed the causes of 126 obstetric deaths.[12] Hemorrhage was the most frequent factor involved and accounted for 44 per cent of the total experience. More than 100 years later, hemorrhage is still the most frequent cause of obstetric death. Reports from a broad community survery[10] and from an active private hospital service[13] during comparable periods suggest that uterine trauma is by far the most frequent offender, with abruptio placentae, placenta previa and ectopic pregnancy contributing to a lesser extent (table 2). Grand multiparity, malpresentation, and intrauterine fetal death are also associated with an increasing incidence of obstetric hemorrhage.

Although placenta previa and abruptio placentae are among the major causes of obstetric hemorrhage, these clinical entities are responsible for relatively few obstetric deaths. The bleeding is either overt (placenta previa) or painful (abruptio placentae) and a potentially dangerous problem is readily recognized. The situation is dramatic, and all forces are mobilized in an atmosphere of vigilance, including anticipation of gross hemorrhage and surgical intervention. Similar alacrity applied to patients with suspected ectopic pregnancy and malpresentation would undoubtedly help avert many hemorrhagic deaths.

TABLE 2. Direct Obstetric Deaths
Hemorrhage

	Baltimore (1935–1958)		Magee-Womens Hospital (1937–1962)	
	Number	Per cent	Number	Per cent
Uterine trauma	67	41.36	24	70.59
Ectopic pregnancy	41	25.31	1	2.94
Placenta previa and premature separation	29	17.90	2	5.88
Other	25	15.43	7	20.59
Total	162	100.00	34	100.00

Seldom does an obstetric patient bleed faster than blood can be given, if a needle has been placed in a vein and if blood is available. Two common denominators thus account for the contribution of hemorrhage to obstetric mortality, namely, lack of recognition and lack of preparation. Matching of blood *before* bleeding occurs will make early transfusion possible. On admission of the patient to the delivery suite, a sample of blood should be sent to the laboratory to be readily available for cross-matching. In the presence of sudden, massive hemorrhage and hypotension, plasma volume expanders can be given while blood is being procured. Type-specific or even O-negative blood can be considered a lifesaving step. A cross-match for major blood group compatibility can be accomplished within several minutes, while complete processing will require well over an hour. The attending physician must decide whether to use a blood substitute or at which stage of the cross-matching blood should be utilized, balancing the risk of withholding whole blood against the risk of incomplete preparation. When it is anticipated that more than several units of blood will be given rapidly, the suggestion has been made that the blood should be warmed to 37° C.[14]

A postpartum patient who continues to bleed despite a firmly contracted uterus and in the absence of lacerations presents a serious problem for the attending physician. A blood coagulation defect should be suspected. The first and simplest diagnostic procedure is to place a sample of venous blood in a glass test tube for observation of clot formation. If a firm clot does not form within 10 minutes, the fibrinogen level is probably less than 100 mg. per cent. A rapid and accurate quantitative test for on-the-spot fibrinogen determinations is now available †, and serial determinations offer a valuable guide to the management of this disorder. The patient should be treated for hypofibrinogenemia unless other contributing factors are proven. Fibrinogen is very stable so that either fresh or stored whole blood and plasma provide a ready source; in the presence of profuse hemorrhage, fibrinogen can be given more quickly in concentrated form. Two grams of a solution of fibrinogen should be given intravenously,‡ and the clotting test should then be repeated. If clotting is inadequate, another two grams should be given. If clotting does not occur after four grams of fibrinogen, or if a formed clot subsequently liquifies, a fibrinolytic process should be suspected.

Recent studies suggest the sequence of events in fibrinolytic disorders.[15] Fibrinolysin (plasminogen) is present in the blood as a plasma protein and is converted to plasmin, the active factor in fibrinolysis. Plasmin normally aids in the dissolution of small intravascular clots. It may be present in excessive amounts to interfere with hemostasis in association with obstetric problems such as abruptio placentae and intra-uterine fetal death. If the fibrinolytic process is not arrested, continued infusion of fibrinogen would add additional plasminogen as a contaminant.

Several approaches have been developed in recent years to inhibit the fibrinolytic system. Epsilon amino-caproic acid (Amicar) inhibits the conversion of plasminogen to plasmin. If plasmin has already been produced in amounts sufficient to cause hemorrhage owing to excessive fibrinolysis, this drug will not be effective. Under these circumstances the polypeptide, Trasylol,§ has been found to inactivate the fibrinolytic agent, plasmin, that already exists. It has been suggested that a combination of Trasylol and epsilon amino-caproic acid may be the superior approach in the management of a fibrinolytic disorder.[15] Fibrinogen should be withheld as soon as evidence of clotting appears.

Toxemia

Toxemia of pregnancy is characterized by edema, hypertension, and weight gain, signs looked for in any program of antepartum care. Hospitalization and medical treatment alone

† The "Fi-Test" is available from Hyland Laboratories as a kit of reagents for a simplified bedside or operating room procedure to detect a state of hypofibrinogenemia.

‡ Fibrinogen (Human) Irradiated is supplied by Merck Sharp and Dohme in a vacuum type bottle containing 1 gram of fibrinogen and anti-hemophilic factor activity equivalent to 75–100 ml. of plasma not more than 18 hours old. This is restored for intravenous use by adding 100 ml. of sterile water.

§ Trasylol is a polypeptide with proteolytic enzyme inhibition activity. It is currently being investigated by FBA Division of Metachem, Inc.

often suffice for the satisfactory management of a toxemic patient, though it may be necessary on occasion to empty the uterus if the patient does not respond to usual therapy.

Those toxemic patients coming to delivery have several abnormalities worthy of note. Although they are edematous, the excess fluid is almost entirely interstitial, and a hypovolemia actually exists.[17, 18] A therapeutic regimen combining salt restriction with the use of thiazide diuretics may result in hyponatremia. This can usually be corrected with 500–1,000 ml. of a 5 per cent solution of sodium chloride.[19] Several facets of the picture thus combine to accentuate and yet obscure a hypovolemic state.

The sedative and vasodilator agents, hydralazine (Apresoline), veratrum viride (Unitensin), and magnesium sulfate are important in the management of severe toxemia. These drugs may, however, prevent the compensatory vasoconstriction following regional blockade with spinal and epidural anesthesia. Patients coming to delivery while medicated with these drugs, particularly if hypovolemic and hyponatremic, are likely to develop profound hypotension following spinal or epidural anesthesia, or after a moderate blood loss. Nevertheless, conduction anesthesia for labor and delivery can be used and may be the method of choice for the management of labor in a toxemic patient. When one of these techniques is anticipated for abdominal delivery, intramuscular magnesium sulfate should not be given within a half-hour of the administration of anesthesia, and intravenous infusion of vasodilator drugs should be discontinued at the time of the block.

More than half the obstetric patients who die from toxemia of pregnancy, on whom autopsies are performed, are found to have a cerebral hemorrhage, undoubtedly associated with an extreme hypertensive episode.[20] Suter suggests that all obstetric patients who convulse as a result of or in association with toxemia are likely to have some residual brain damage, even though this is not always discernible clinically.[21] Intracranial hemorrhage is certainly an anticipated finding among toxemic patients who die following a convulsion.

Immediate oxygenation is the first and most urgent treatment of the convulsing patient. This can be done with a reservoir bag-mask unit; a pharyngeal airway or an endotracheal tube may be necessary to overcome airway obstruction. The seizure may hamper attempts at re-oxygenation because of spasms of larynx, pharynx, and respiratory muscles, and salivation or vomiting. The convulsions can be controlled most quickly with the intravenous injection of succinylcholine. Relaxants should never be used, however, unless equipment and personnel are on hand to ventilate the lungs. The intravenous use of 300–600 mg. of a long-acting barbiturate such as sodium amobarbital has become established as the treatment of choice in obstetric circles.[22] The use of repeated injections of 50–100 mg. doses of a short-acting drug such as sodium thiopental (Pentothal) offers a more flexible approach to the management of seizures in the obstetric patient.[23] Barbiturates provide a theoretical advantage over succinylcholine in that they suppress cortical irritability and activity. As central nervous system depressants, however, they may accentuate central depression caused by pre-existing hypoxia.

Infection

Deaths from puerperal infection following either normal vaginal delivery or operative delivery have virtually disappeared from maternal mortality reports. Deaths from infection following spontaneous abortion, therapeutic abortion, or ectopic pregnancy are also decreasing in number. By contrast, we find that infection following self-induced and criminal abortion continues to be a major problem, and most deaths from infection are the result of non-professional efforts to interrupt pregnancy. The bacterial organism is usually a gram-negative bacillus, and the endotoxic shock that ensues carries with it a high mortality rate. A similar clinical picture will occasionally appear in association with amnionitis following prolonged rupture of the membranes.

In the initial phase of endotoxic shock, hypotension is related to a decrease in cardiac output secondary to a decreased venous return to the heart.[24] This is followed by an active vasospasm on both the arterial and venous sides. Vasospasm is not sustained, and peripheral resistance soon falls, suggesting a loss

of homeostatic mechanism. The clinical picture may be deceptive. Despite a systolic blood pressure as low as 50–70 mm. of mercury in the presence of vasodilation, a warm and pink skin is present, and the patient does not appear to be in shock. This is an example of failure of the circulatory reflexes to respond to hypovolemia and hypotension.[25, 26]

If endotoxic shock is suspected, blood cultures should be made, also cultures of any possible reservoir of infection, such as the uterine cavity. The treatment consists of the intravenous administration of large doses of corticosteroids (0.5 to 2 g. of hydrocortisone the first day), penicillin and streptomycin plus a broad spectrum antibiotic such as tetracycline or chloromycetin, intravenous fluids, and an increased oxygen content in the inhaled atmosphere. Shortly after the administration of antibiotics, hypotension may become more profound due to the release of endotoxin from bacteria. Antibiotics should be continued despite this adverse response, since the underlying source of the shock, the organism producing endotoxin, is being eliminated. Serial hemoglobin and hematocrit determinations and urinary output can serve as guides to the administration of electrolyte solutions, plasma elements or whole blood. Following a hypotensive state, metabolic acidosis is likely to result from inadequate tissue perfusion. It can be recognized by determinations of *p*H, P_{CO_2} and standard bicarbonate, and corrected if necessary with intravenous sodium bicarbonate. Vasopressors have been used and must be considered if the hypotension does not respond quickly to other measures and if there is danger of inadequate cerebral or coronary blood flow. Methamphetamine (Methedrine) and mephentermine (Wyamine) produce less peripheral vasoconstriction,[27] and therefore would be expected to impair tissue perfusion less than the potent vasoconstrictors, norepinephrine (Levophed) and methoxamine (Vasoxyl). Morris recently has compared the effects of angiotensin amide (Hypertensin), metaraminol (Aramine), and norepinephrine on dogs subjected to endotoxic shock; metaraminol proved the most effective in increasing arterial blood pressure, cardiac output, and renal and superior mesenteric artery blood flow.[24] Morris found that dilator drugs resulted in further deterioration of all circulatory parameters measured. The place of hypothermia has not yet been established, though cooling is indicated in the presence of extreme hyperpyrexia.

Anesthesia

Relief from the pain of childbirth has come to be the anticipated privilege of every parturient. Many agents and techniques are available so that a safe choice can be made for each patient. If a patient has been promised a specific type of anesthesia in advance of labor and delivery, there is always the possibility that this may not be the one of choice when the time arrives. The anesthetic, therefore, should be individually selected for each patient to answer the needs of the situation at hand.

Deaths have occurred with all agents and techniques used for obstetric anesthesia.[28] The leading causes in the past have been aspiration of vomitus and peripheral vascular collapse following major conduction techniques, such as spinal anesthesia. Based upon a review of two and one half million births, Merrill and Hingson estimated that there were about 100 maternal deaths each year from aspiration of vomitus alone[29]; about 66 per cent of these were associated with ether anesthesia. During recent years there has been an average of one death a year from aspiration of vomitus in communities with a population of about one million.[28] Edwards *et al.* in a review of 1,000 deaths associated with anesthesia reports that slightly more than 50 per cent of all obstetric anesthetic mishaps were due to aspiration of vomitus.[30]

La Salvia and Steffen observed that 40 per cent of a series of obstetrical patients vomited either during or immediately following inhalation anesthesia.[31] These investigators noted that pregnancy alone had little or no effect on the emptying time of the stomach. Sedation in the absence of labor decreased gastric motility; 10 to 11 per cent of a test meal was retained after 3 hours and 6 to 8 per cent after 5 hours. Sedation during active labor caused a 20 to 43 per cent retention at the end of 3 hours and 12 to 37 per cent at the end of 5 hours. In over half of all patients in labor with sedation, a fluid level was found in the

stomach 5 to 11 hours after the ingestion of the test meal.[31] No data are available to compare the amount of gastric retention at different time intervals between ingestion of the test meal and the onset of labor. A six hour interval between the last solid food or milk product and the onset of labor has been accepted by clinicians as a relatively safe interval for a patient who is to receive general anesthesia. Patients should be instructed to refrain from ingesting liquid or solid foods at the first sign of labor. As Bannister has pointed out, however, when labor and fasting are prolonged, acid gastric juices can collect in the stomach and these can be vomited or regurgitated during anesthesia for delivery.[32] Few patients in active labor can be trusted to have empty stomachs.

The use of regional anesthesia is the best way to avoid aspiration of vomitus in the presence of a full stomach. This is certainly the simplest and safest approach for normal vaginal delivery, though aspiration may still occur and continuous attention to the problem is imperative. Occasionally, general anesthesia is indicated because of the need for uterine relaxation or when spinal anesthesia might accentuate the hypotension resulting from hypovolemia. General anesthesia in the presence of a full stomach can be given safely only with a cuffed endotracheal tube in place, inserted either under a topical anesthesia prior to general anesthesia or following a rapid induction with a small dose of thiopental (100–250 mg.) and a paralytic dose of succinylcholine (40–60 mg.).

Hamilton [33] and Bannister [32] have outlined programs for managing patients who have aspirated vomitus. Facilities should be available for removing liquid and solid material from the tracheobronchial tree. These include a delivery table that can be put quickly into Trendelenburg position, effective suction apparatus, and endotracheal and bronchoscopic equipment. Many of the anesthetic deaths following general anesthesia are related to the aspiration of acid gastric juice. The chemical irritation causes a penumonitis that is resistant to any treatment if the mishap is not recognized and treated promptly.

Even if aspiration of vomitus is only suspected, an endotracheal tube should be inserted and the trachea suctioned. Solid materials in the trachea are an indication for immediate bronchoscopy. Oxygen should be given, and respirations should be assisted if not adequate. If oxygen is needed beyond the immediate emergency, it should be given with a warm aerosol mist. Bronchospasm may impede ventilation, and this can be helped by adding isoproterenol (Isuprel) to the inhaled mist and by giving aminophylline, 250–500 mg., intravenously. Large doses of hydrocortisone should be given intravenously, 100–200 mg. immediately, and at least 500 mg. during the day. These doses should be continued until the patient is symptom free. If the therapy is discontinued within five days, adrenocortical inactivity does not occur, and weaning is not necessary. If the aspirated material is of an acid nature, antibiotics will not help the inflammatory process, but will help to combat infection from secondary bacterial invasion. If the material suctioned is alkaline, this would indicate that intestinal fluids have been aspirated. Antibiotics should certainly then be given to combat the intestinal bacteria. Lewinski has shown recently that the instillation of hydrocortisone solution into the respiratory tree inhibits the inflammatory changes associated with the chemical pneumonitis.[34] Digitalization should be carried out if left-sided heart failure is superimposed upon the respiratory problem.

Death following spinal and epidural anesthesia is usually due to hypotension associated with sympathetic blockade. This is a common response to spinal anesthesia, and if recognized and treated should never result in a casualty. Kennedy noted that 17.7 per cent of 600 obstetric patients who received spinal anesthesia developed hypotension; this was corrected in 93.4 per cent of the patients by displacement of the uterus to the left to relieve uterine pressure on the vena cava.[35] Though respiratory depression can result from a high spinal block, this is an infrequent problem. Moya and Smith showed that the oxygen saturation was not altered significantly in 15 patients who received spinal anesthesia for cesarean section, even though the sensory level ranged from the sixth thoracic to the fifth cervical dermatome; in 14 of the patients the level was higher than the fourth thoracic.[36]

Egbert observed that respirations were depressed less by spinal anesthesia to the second to sixth thoracic levels than by the preanesthetic medication.[37] de Jong also has demonstrated that spinal anesthetic levels from the tenth thoracic to the fifth cervical dermatome had little effect on arterial oxygen and carbon dioxide tensions.[38]

Two gross transgressions of safe spinal anesthetic procedure have contributed in the past to many deaths with this technique. Excessive doses of spinal anesthetic drugs have been used, being limited, apparently, only by the amount of drug in the ampule.[39] In addition, many deaths have resulted because patients have been left unobserved, while delivered by the obstetrician who gave the spinal anesthetic.[40, 41] Every patient receiving a spinal anesthesia should have an intravenous infusion started and the vital signs should be monitored constantly. Vasopressor drugs should be readily available for correcting hypotension, as should facilities for administering oxygen by positive pressure in the event respiratory assistance is needed. Vasopressor drugs given prophylactically before conduction anesthesia for vaginal delivery cause a disturbingly high incidence of postpartum hypertension and should not be used routinely.[42] Those services using a prophylactic vasopressor prior to a spinal anesthesia for cesarean section, however, have fewer problems with hypotension during the operation.[36] Deaths from hypotension following spinal anesthesia have been virtually eliminated in recent years through attention to fundamental tenets of safe anesthetic management.[43]

Even the simplest anesthetic technique is not innocuous. Deaths have followed pudendal block or local infiltration.[28] In addition, anesthetic responsibility does not end when the patient has recovered from the anesthetic. Postpartum uterine bleeding is normally controlled by the firm contraction of the uterus. Ether and halothane are valuable drugs in obstetrics in that they can provide the uterine relaxation occasionally necessary for intrauterine manipulations. We must remember, however, that the relaxation associated with a surgical plane of anesthesia obtained with these drugs may continue into the postpartum period; the uterus remains less responsive to oxytocic drugs, and postpartum bleeding may be increased.[44]

Half the obstetric deaths at the Magee-Womens Hospital occurred 20 hours or more after anesthesia. This emphasizes the importance of close observation during the postpartum period.[13] Obstetric recovery rooms are available in very few hospitals in this country. Furthermore, only 44.6 per cent of the hospitals have recovery room facilities available for postanesthetic observation of patients who have had a cesarean section.[16] Obviously, patients cannot be observed closely during the postpartum period on a stretcher in the midst of an active delivery suite, or alone in a private room or on a general ward.

Vascular Accidents

Vascular accidents may be embolic, hemorrhagic or thrombotic. The largest number in most obstetric reports have been due to emboli, but the pattern of underlying factors in this category has changed during the past 25 years. Postpartum embolic deaths from nonseptic thrombi were most frequent during the early part of this period. These have almost disappeared, probably primarily due to early ambulation. Amniotic fluid embolism probably always has been a contributor to obstetric deaths. Prior to the description of this entity by Steiner and Lushbaugh,[45] these deaths were very likely listed as obstetric shock or heart failure. During recent years physicians have become more aware of this accident and have recorded the event in mortality reports with increasing frequency. Shnider and Moya[46] reported that more than half the deaths that occurred during the delivery period at the Sloane Hospital for Women in New York City were due to amniotic fluid embolism.

Amniotic fluid embolism nearly always follows trauma to the uterus, associated either with a difficult delivery or oxytocic stimulation. A traumatic or forceful delivery should serve to alert one to anticipate this development. Sudden respiratory distress and cyanosis at the time of delivery should suggest amniotic fluid embolism, if there is no other cardiorespiratory background or basis for the picture. The symptoms are due to an infusion of amniotic fluid, including cellular elements into the cir-

culation. There is a mechanical blockade of the pulmonary vessels and possibly a reflex pulmonary vasoconstriction, leading to increased pulmonary arterial pressure and hypoxia. Tachycardia and hypotension quickly ensue. The intravenous infusion of amniotic fluid, which is rich in thromboplastin, leads to a diffuse deposition of fibrin [47]; if the patient survives the initial stage, hemorrhage follows the depletion of fibrinogen.

Immediately upon recognition, oxygen by intermittent positive pressure is indicated, usually via an endotracheal tube. Isoproterenol, aminophylline, atropine and paraverine have been used to help overcome pulmonary vasoconstriction. An intravenous infusion should be started. Vasoconstrictor drugs are indicated only if hypotension threatens to impair cerebral or coronary arterial perfusion. Blood should be drawn for observation of clotting, and blood and fibrinogen should be available if bleeding follows hypofibrinogenemia.

Indirect Causes of Obstetric Mortality

Heart disease and pulmonary disease are responsible for the greatest number of indirect obstetric deaths. Heart disease is the most frequent contributor, and accounts for nearly half of all indirect obstetric deaths. Mitral stenosis and other vascular sequelae of rheumatic heart disease are the most frequent factors in obstetric deaths from heart disease; hypertensive cardiovascular disease and congenital cardiovascular defects, respectively, rank next in order of frequency.[48] It is estimated that some symptom of heart disease is present prior to labor in about one-half the obstetric deaths due to heart disease.[49] If heart disease is suspected, the assistance of a cardiologist should be sought. The patient with evidence of heart disease should be followed closely and hospitalized at any sign of failure. With careful medical preparation, few of these patients present serious obstetric or anesthetic problems.

Blood volume reaches its maximum at about 30 weeks gestation, and cardiac output reaches a peak increase of 37 per cent at 25 to 27 weeks. Cardiac output then falls at term to the non-pregnant level, rising again immediately after delivery.[50] Obstetric patients with heart disease should be observed most closely at those periods of maximum cardiac output.

Since uterine contractions and bearing down are essential for vaginal delivery, this component of the muscular work expended during labor cannot be eliminated other than by cesarean section. Pain relief can be offered, however, and the patient can be relieved of the work expended in reflex bearing-down and through extraneous activity by the use of a conduction technique, such as continuous caudal or lumbar epidural anesthesia. This approach has the additional advantage that there is no direct myocardial depression such as might be anticipated with the use of most general anesthetics.

Additional Factors Influencing Maternal Mortality

A number of other factors influence maternal mortality rates, even though they are not causes, *per se,* of obstetric death. Mortality rates in cesarean section based upon experience 25 to 35 years ago were reported to be as high as 9.7 per cent.[51] More recently several series of over 1,000 cesarean sections have been cited with no deaths [52, 36]; these suggest that the risk of delivery by cesarean section is hardly greater than the risk by vaginal route. The increasing safety of this procedure has increased the readiness with which a section is considered; thus the number of cesarean sections has increased in most institutions during the past 25 years.

Mortality rate has been cited as twice as great in the non-white as in the white race.[53] Moreover in the city of Baltimore the difference in obstetric mortality rates between the white and non-white populace has increased during recent years.[10] In the neighboring community of Washington, D. C., however, the difference in mortality rates between races has been reduced.[54] This suggests that the discrepancy is due, not to inherent racial characteristics, but to the influence of socio-economic factors on the quality of obstetric care.

Oxytocic drugs are among the more frequent iatrogenic factors contributing to obstetric mortality. Freeman reported 25 cases in which pitocin or pituitrin was considered a direct or contributing factor in death. These

accounted for 15 per cent of 164 maternal deaths reviewed [55]; 64 per cent of these were considered preventable. The oxytocic substances were considered causitive factors in 43 per cent of all deaths from uterine rupture, 35 per cent of deaths from amniotic fluid embolism, and 17 per cent of all deaths from hemorrhage.

The ergot alkaloids may cause marked hypertensive crises, and have resulted occasionally in convulsions and subarachnoid hemorrhage.[42, 56] The hypertensive response is more marked if the drug is given intravenously, and is particularly hazardous if the patient is toxemic. Hypertension may be accentuated if a vasopressor drug has also been given the patient.

Maternal age is an important factor in influencing both obstetric mortality and perinatal mortality.[22] The third decade is considered the best reproductive period: mortality rates rise progressively thereafter with increasing age. Parity is also important; this, obviously, is apt to be a factor with increasing age. During the period 1939–1961, patients at the Magee-Womens Hospital with a parity of 6 or more had a death rate of 30 per 10,000 live births, four times as great as the average parity of the entire series.[57] Similar observations led Solomons [58] to pronounce in 1934 that "it is altogether a mistake to suppose that in childbearing practice makes perfect." Israel and Blazar have reported on a more recent experience encompassing 134,000 deliveries.[59] They concluded that there was a significant increase in incidence of serious obstetric complications in patients of parity 7 or more, but that they are presently being cared for with no greater risk to life than other pregnant patients.

Summary

A phenomenal reduction in obstetric death rates has occurred during the past quarter century. An irreducible minimum has not been reached, however. More than three quarters of the remaining obstetric deaths are preventable; these might be avoided through the application of current knowledge and facilities. Further reduction in the number of obstetric deaths will follow recognition and attention to the most frequent existing causes.

Data on maternal mortality are available through the publication *Vital Statistics of the United States* and through reports in the medical literature emanating from community maternal mortality committees and from institutional surveys. Deaths in *Vital Statistics of the United States* are tabulated as obstetric only when complications of pregnancy have been indicated as the underlying cause of the death. Deaths due to nonobstetric causes such as rheumatic heart disease, aggravated by the pregnant state, are classified according to the primary disease entity; deaths due to anesthesia for an obstetric procedure are classified according to the fundamental entity (pregnancy) requiring anesthesia. Data reported in regional maternal mortality studies are classified according to a system designed by the Committee on Child Care of the American Medical Association. Anesthesia and vascular accidents are designated direct causes of obstetric death, and deaths due to medical complications aggravated by pregnancy are classified as indirect obstetric deaths.

Hemorrhage, infection, toxemia, anesthesia and vascular accidents are the most frequent direct causes of obstetric death. Heart disease is the most frequent indirect factor. Although not classified as etiological factors, age, multiparity, the use of oxytocics, and resort to cesarean section markedly influence maternal mortality rates.

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TRENDELENBURG POSITION The use of Trendelenburg position in gynecological operations is an additional source of stress of compensatory mechanisms and aggravates the course of anesthesia. Superficial endotracheal anesthesia with muscle relaxants prevents necessity for Trendelenburg position or allows limitation of the angle of inclination 10 to 20 degrees, which in fact improves the operative conditions. (*Gologorskii, V. A., and Tsirulnik, S. I.: Advantages of Modern Combined Anaesthesia With Use of Muscle Relaxants in Operative Gynaecology (Russian), Soviet Medicine 27: 60, 1964.*)

NEWBORN RESPIRATORY DISTRESS Causes of insufficient respiration of the neonate are amnionic fluid aspiration, upper respiratory obstruction, pneumothorax, and diaphragmatic hernia. Another cause is unilateral hydrothorax. The mother had a hydramnion in the fifth month of pregnancy. The newborn was pink at birth but respiration was insufficient with sternal retraction and respiratory standstill after 25 minutes. The anesthetist felt a high resistance to ventilation. Cardiac resuscitation was successful, but no spontaneous breathing occurred. A three-hour chest roentgenogram showed maximal inspiration, and homogenous opacity of the lungs, so-called white thorax. Liver, lung, heart and abdominal contents could not be separated on the roentgenogram. After six hours of artificial ventilation the child died. Postmortem lungs were collapsed by 40 ml. clear yellowish fluid. The syndrome must be differentiated from hyaline membrane disease, in which a white thorax is also found. Another case was observed by Perry, who achieved survival by pleural puncture and aspiration of 75 ml. fluid. The fluid prevents expansion of the lungs because the necessary negative pressure of 60 cm. water cannot be achieved. Probably this was a combination of hydramnion and hydrothorax. (*Peededeon, H.: Unilateral Hydrothorax as Cause of Severe Respiratory Distress in the Newborn (German), Fortschr. Roentgenstr. 102: 29 (Jan.) 1965.*)