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CHAPTER 15

BLEEDING PROBLEMS

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Joyful and natural though childbirth may be, the specter of death attends every birth. Women still bleed to death during childbirth, though modern medical care could save 75 to 90 percent of them. Once the leading cause of maternal mortality,[1] hemorrhage remains an important cause of death in developed countries despite elimination of maternal anemia and availability of blood transfusion.[2,3] Kaunitz and colleagues[4] found hemorrhage to be the third leading cause of reported maternal deaths in the United States between 1974 and 1978, though reports from individual states show wide variation in the rate of hemorrhagic death.[5-8] Hemorrhage remains among the leading causes of death in reports from the Nordic countries,[9] Sweden,[10] England, and Wales,[11] and Canada.[12]

Hogberg[13] estimates that worldwide reproductive mortality may approach a half million women per year, with maternal mortality rates for individual nations ranging from less than 10 to more than 2000 maternal deaths per 100,000 live births. Hemorrhage compounded by anemia represents the major cause of maternal mortality throughout the world.[14-21]

Women older than 35 suffer four times the obstetric mortality of women aged 20 to 34;[9,22] hemorrhage primarily causes death in this older group (14.3 deaths per 100,000 live births).[22] A religious group in the United States which rejects all medical care suffers 100 times the predicted maternal mortality, with 66 percent of the deaths due to hemorrhage.[23]

Pregnancy-related hemorrhage occurs not only with birth but also with ectopic pregnancy and abortion. Deaths from spontaneous abortion explain 5 percent of the United States maternal mortality; hemorrhage accounts for one in five.[24] Hemorrhage is the leading cause of death following legal abortion.[25,26]

Cesarean section introduces more risks, including hemorrhage, than vaginal delivery.[10,27,28] Petitti[29] identifies transfusion as the second most common source of significant morbidity after cesarean section.

Obstetric complications have become an uncommon cause of death in young women. Of North American women aged 15 to 24, 38 will die in motor vehicle accidents for each one who dies of any complication of pregnancy, and 40 more will die of murder, suicide, or nonvehicular accidents.[30] Obstetric bleeding problems

pose a public health problem not because of their numbers but because almost all women who bleed to death should not have died. A low death rate makes an individual death no less tragic, especially if prevention could have averted that death.

Physiology

This section briefly reviews placental function, maternal blood volume changes with pregnancy, and the features of normal hemostasis. For details, see Chapters 1 and 2.

FETAL ASPECTS

The parasitic human fetus draws life through the hemochorial placenta, a network of exteriorized fetal vessels implanted in maternal tissue. Maternal blood surrounds the fetal blood vessels of the placental villi, allowing metabolic exchange between fetal and maternal circulations. As much as a liter of maternal blood escapes the spiral arteries each minute to momentarily occupy extravascular areas within the placenta's intervillous space before re-entering maternal uterine veins through venous sinuses. Ironically, the very adaptation that enables fetal life imperils maternal life. Maternal blood may escape its accustomed vascular confinement to become irretrievably lost; amniotic fluid, air, infected debris, or thromboplastic material may invade the mother's once inviolable circulation (Fig. 15–1).

As the placenta separates from the uterine wall, maternal vessels supplying it shear off. Blood that would have perfused the placenta flows into the uterine cavity. The vascular system would empty in minutes if not for the simultaneous uterine contraction that normally compresses spiral arteries to stop blood loss. Not only can blood escape as the placenta separates, but material within the uterus or beneath the placenta can also enter the maternal circulation through venous sinuses. The hemochorial placenta which makes human reproduction possible thus introduces to childbirth the risks of bleeding and embolism.

MATERNAL ASPECTS

Pregnancy induces maternal physiologic adaptations to nourish a growing organism within the mother's body. Those changes essential to our consideration of bleeding problems include blood volume adaptations and alterations in the hemostatic mechanism.

Changes in Blood Volume

Pregnancy prepares patients well for the hemorrhage that inevitably accompanies delivery.

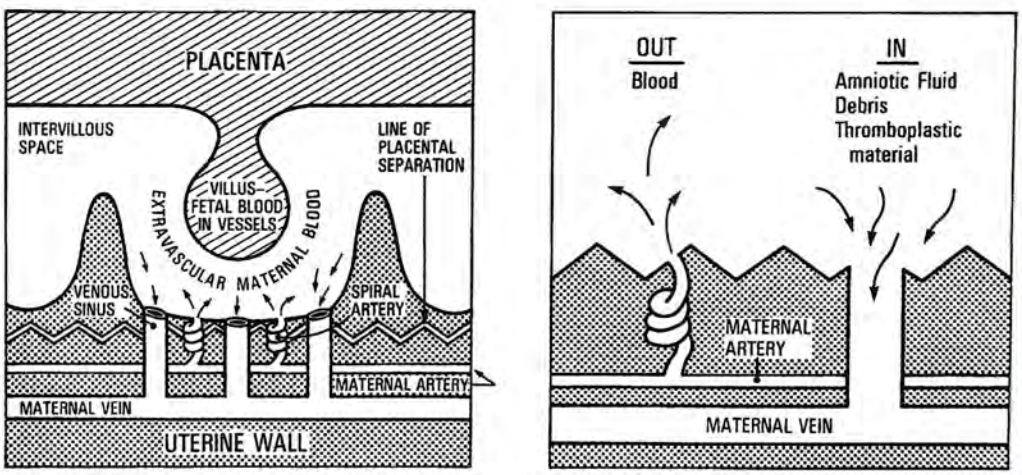

Figure 15–1. Maternal blood momentarily becomes extravascular as it perfuses the hemochorial human placenta *(left)*. After placental separation *(right)*, spiral arteries lose blood which would have perfused the placenta, and venous sinuses remain open to invasion by foreign material. Uterine contraction normally compresses these vessels, sealing the maternal circulation again.

Blood volume increases markedly with pregnancy; total blood volume near term exceeds nonpregnant volume by 35 to 50 percent, an increase of 1000 to 1500 ml.[31,32] Both plasma volume and red cell mass increase, but a greater increase in plasma volume produces the physiologic "anemia" of pregnancy by hemodilution. Pregnant patients withstand hemorrhage well because of their increased blood volume and because the blood-containing space becomes smaller immediately after delivery. The intervillous space disappears with the placenta, and uterine vasculature shrinks with uterine contraction to reduce the maternal vascular space by 300 to 500 ml.

Normal Hemostasis

Blood is a remarkable substance: free-flowing within the intact vascular system, it quickly blocks its own egress following vascular damage. Vascular injury initiates three groups of reactions that contribute to formation of a firm clot at the site of injury. First, a platelet plug forms at the injured site. Second, the coagulation mechanism evolves fibrin to stabilize the fragile platelet plug. Third, a group of limiting reactions restrict the clot to the area of injury.[33]

Platelet Plug. Vascular injury exposes subendothelial collagen, to which platelets adhere, releasing ADP and thromboxanes. Platelets aggregate in the presence of these substances, releasing more ADP and thromboxanes, causing further aggregation and making platelet phospholipids available for coagulation.

Formation of the primary hemostatic plug requires both functional platelets in adequate numbers and constriction of blood vessels in the injured area, since unimpeded blood flow would otherwise wash away any aggregating platelets. Because primary hemostasis is independent of coagulation, coagulation tests *do not* measure the ability to form this primary hemostatic plug; only bleeding time is a reliable test.

Coagulation. Secure hemostasis requires reinforcement of the temporary platelet plug by fibrin gel, a protein polymer composed of the fibrin monomers produced when thrombin digests fibrogen. The central reaction of the coagulation process involves generation of thrombin from prothrombin, with subsequent cleavage of fibrinogen to fibrin. The coagulation process (Fig. 15–2), a series of proteolytic reactions,[34] includes at each step conversion of an inactive precursor to an activated clotting factor, which, in turn, transforms the next factor into its active form.

Thrombin production proceeds through either of two pathways. Vascular injury activates the intrinsic pathway, so named because all of its components are present in circulating blood. The extrinsic pathway requires the presence of tissue factor, a generic term for certain proteins released by injured cells. Subsequent to activation of Factor X by either route (Fig. 15–2), the coagulation sequence follows the same common pathway, regardless of the initiating stimulus. The thrombin produced cleaves fibrinogen to the fibrin monomer. Fibrin monomers rapidly polymerize to form insoluble strands that wind around the platelet aggregates at the site of injury, providing a reinforced fibrin plug.

Limiting Reactions. Thrombin is a potent enzyme that not only cleaves fibrinogen to fibrin but also promotes its own formation, directly aggregates human platelets, activates Factor XIII, and converts plasminogen to plasmin.[35] Although unchecked generation of thrombin could rapidly clot all the blood in the body, a series of limiting reactions usually confine the clot to the injured area.

Several types of limiting reactions act simultaneously. Much of the thrombin generated becomes unavailable for further reaction after it is absorbed onto the surface of the developing fibrin gel. Simultaneously, blood flow to an injured area dilutes activated clotting factors and slows coagulation, while the force of the flow itself disrupts the clot and opposes further propagation. In addition, the liver clears blood of activated clotting factors. Conditions producing poor regional and hepatic blood flow, such as hemorrhagic shock, can unbalance the limiting mechanisms, an important effect in initiating disseminated intravascular coagulation (DIC).

Fibrinolysis is the most important limiting reaction. Plasmin, the major agonist in the fibrinolytic system, may serve as the most important defense against generalized clotting.[36] A potent proteolytic enzyme that digests both fibrin and fibrinogen, plasmin originates from plasminogen by thrombin, by activated Factor XIII, and by plasminogen activators from injured cells. Plasmin generation occurs as a normal part of the coagulation sequence.

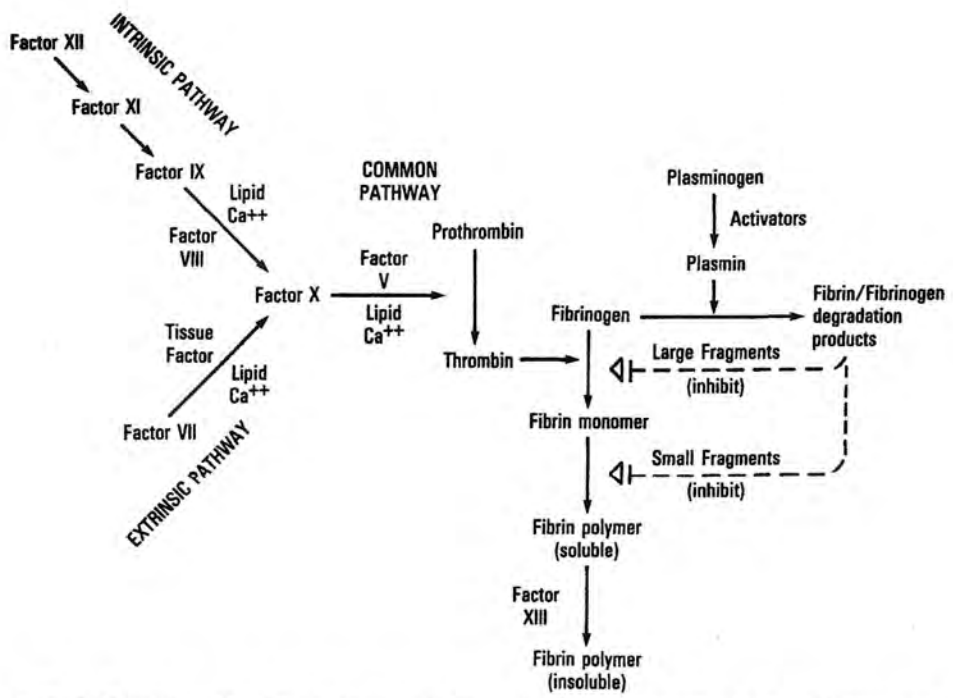

Figure 15–2. Thrombin evolves by one of two pathways, with a final common step to cleave fibrinogen to fibrin monomers, the structural units of the fibrin polymer, which stabilizes the platelet plug. In one of the limiting reactions, plasmin also cleaves fibrinogen to anticoagulant fragments which help confine coagulation to the site of vascular injury.

Plasmin digestion of fibrinogen removes a few small fragments first, leaving large fibrinogen remnants which can still react with thrombin but which clot very slowly (see Fig. 15–2). These large fragments act as potent anticoagulants, tying up thrombin with slowly reactive substrates. Further fibrinogenolysis produces smaller fragments, which no longer react with thrombin but, when incorporated into the developing fibrin gel, slow its rate of formation and weaken the resulting polymer. Therefore, the fibrin-fibrinogen degradation products produced when plasmin digests fibrinogen, interfere with production of further fibrin by thrombin. This point bears emphasis: Thrombin acts on fibrinogen to form fibrin; plasmin acts on fibrinogen to form anticoagulant degradation products. Both processes contribute to normal coagulation, and both normally take place only at the site of injury.

Changes With Pregnancy. Levels of Factors VII, VIII, IX, and X increase during pregnancy, though not significantly.[31] Plasma fibrinogen increases during pregnancy to levels of 400 to 600 mg per dl; severe pre-eclampsia may produce fibrinogen levels as high as 800 to 1000 mg per dl.[37] The platelet count does not change in normal pregnancy, remaining between 150,000 and 300,000 per mm^3. Pregnancy does not alter the results of coagulation tests.

The Anesthesiologist's Role in Obstetric Hemorrhage

An anesthesiologist caring for a hemorrhaging patient functions as resuscitator to prevent death and organ damage, and as anesthetist to facilitate diagnostic examination or operative repair of bleeding sites. In addition, an obstetric anesthesiologist may direct resuscitation of an infant born to a hemorrhaging patient.

Good care requires good communication. Obstetrician and anesthesiologist must clearly

define resuscitative needs and plans for therapy. An experienced obstetric anesthesiologist anticipates hemorrhage, understands the differential diagnosis, and participates in planning definitive therapy.

In the face of obstetric bleeding, one may need to summon additional help from within the obstetric suite or from elsewhere in the hospital. Don't hesitate—saving the life of a mother or her child takes priority over almost all other concerns within the hospital. A bleeding patient receives the best care from anesthesiologists who are fully involved in obstetric anesthesia care and who know the patient, the facility, and the members of the patient care team.

RESUSCITATION

Resuscitation during hemorrhage aims to ensure oxygen delivery to the tissues, maintaining good tissue perfusion by fluid with adequate oxygen content. Perfusion depends on both the fluid volume and the pump. Since hemorrhaging obstetric patients generally have sound hearts, the resuscitator can concentrate on fluid volume. Oxygen delivery depends on the oxygen content of the blood, a function of its hemoglobin content and of the quality of respiratory exchange within the lungs. Hemorrhaging obstetric patients usually start with healthy lungs, which allows the resuscitator to concentrate on hemoglobin content.

Effects of Hemorrhage

Volume Loss. Although pregnant patients may tolerate hemorrhage more readily, no study specifically delineates that tolerance. A healthy nonpregnant human can compensate for acute loss of up to 15 percent of the blood volume without change in blood pressure by constricting venous capacitance vessels. Loss of 15 to 30 percent of the blood volume leads to arterial vasoconstriction, tachycardia, and a slight drop in cardiac output. Acute volume loss of 30 to 50 percent produces decreased cardiac output with tachycardia, hypotension, and the α-adrenergic features of clinical shock, with preferential perfusion of heart and brain. Untreated loss of more than 50 percent of the blood volume leads to irreversible shock and death.[38]

Oxygen-Carrying Capacity. Little oxygen is dissolved in the fluid elements of blood compared with the amount carried by hemoglobin (0.30 ml dissolved oxygen per 100 ml of blood versus 20 ml per 100 ml of blood combined with hemoglobin at a PaO_2 of 100). Tissue under resting conditions normally extracts about 5 ml of oxygen from each 100 ml of blood. Dissolved oxygen represents less than 3 percent of that oxygen.[39] Even a patient breathing 100-percent oxygen with an arterial PO_2 over 600 will have only 2 ml of dissolved oxygen per 100 ml of blood—not even enough for half the resting tissue oxygen demand. Up to a point, the body can compensate for the loss of oxygen-carrying hemoglobin by increasing cardiac output and tissue perfusion. Beyond that point, tissue oxygenation will fail, leading to anaerobic metabolism, acidosis, and cellular death.

Hemostatic Ability. Hemorrhage per se has little effect on the blood's ability to remain fluid within blood vessels and to clot at the site of damage. However, a coagulopathy may develop upon depletion of clotting factors at the site of hemorrhage, and limiting reactions become unbalanced from the poor perfusion that accompanies hemorrhagic shock. Treatment of severe hemorrhage with massive fluid replacement often compounds a problem by diluting available clotting factors.

Extracellular Fluid. Hemorrhage initiates a shift of interstitial fluid into blood vessels to restore plasma volume and an apparent shift into cells to produce intracellular edema. The functional deficit of extracellular fluid thus induced may require up to 4 liters of clear fluid replacement beyond the correction of blood volume deficit.[40] Replacement of protein-containing intravascular fluid with noncolloidal fluid reduces colloid osmotic pressure, allowing fluid to shift to the interstitial space. Although colloid fluids should remain intravascular better than crystalloids, colloid resuscitation remains controversial owing to concerns about leakage into the interstitial space across shock-damaged capillaries.

Fluids Available to the Resuscitator

The resuscitator aims to restore intravascular volume and to maintain oxygen-carrying capac-

ity; correction of coagulation defects is initially of secondary importance but may later be essential for successful therapy. Table 15–1 lists major resuscitative fluids and clotting factor concentrates available in North America and indicates whether the fluid provides volume, oxygen-carrying capacity, colloid osmotic (oncotic) pressure, or hemostatic capacity. (Table 15–1 also identifies fluids acceptable to patients who refuse blood products.)

Crystalloid Solutions. Crystalloids, or balanced salt solutions, are mixtures of water and salts that approximate the composition of body fluids. Adding dextrose provides an energy source. Although they are inexpensive, easy to store, sterile, and virtually without risk of infection or immunoreactivity, crystalloids leave the vascular space readily and provide neither oxygen-carrying capacity nor hemostatic ability.

Ringer's lactate, the most ubiquitous crystalloid solution, is widely used in resuscitation. Some clinicians feel that the calcium in Ringer's lactate (3 mEq per liter) may reverse the anticoagulant effect of citrate in banked blood and discourage its use for diluting packed red blood cells (RBCs) prior to infusion. Others avoid Ringer's lactate in the presence of shock,[41] though no evidence links lactate in the solution (28 mEq per liter) to lactic acidosis. Normal saline is an acceptable crystalloid resuscitation solution and may be the fluid of choice for dilution of packed cells or for concurrent administration with blood.

Colloid Solutions. Large molecules in colloid solutions pass poorly through undamaged capillaries and, so, contribute to colloid osmotic pressure. Commercially prepared solutions, though sterile, easy to store, and free of infection risk, are more expensive than crystalloids, especially those protein products derived from blood. Untoward side effects are rare. Colloid solutions provide neither oxygen-carrying capacity nor hemostatic ability.

A 6-percent solution of hydroxyethyl starch (hetastarch, Hespan) expands plasma volume as effectively as albumin[42] but complicates blood typing and antibody screening if blood samples contain more than 30 percent starch.[43] Hetastarch has a lower cost and a longer shelf life than albumin.

Dextran 40 (Rheomacrodex) as "low–molecular weight dextran" (mw 40,000 dal-

Table 15–1. RESUSCITATIVE FLUIDS AND CLOTTING FACTORS

	Volume	Oncotic Pressure	Oxygen Carrying	Hemostasis
Crystalloids				
Ringer's lactate*	+			
Normal saline*	+			
Colloids				
Hetastarch*	+	+		
Dextran*	+	+		
Albumin, 5%	+	+		
Albumin, 25%		+		
Purified protein fraction	+	+		
Hemoglobin Substitutes				
Fluosol†			+	
Blood Products				
Fresh whole blood	+	+	+	+
Banked whole blood	+	+	+	
Packed red blood cells	+		+	
Fresh frozen plasma	+	+		+
Platelets				+
Fibrinogen				+
Cryoprecipitate				+
Factor VIII concentrate				+
Factor IX concentrate				+

*Products acceptable to a patient refusing all blood products. Such fluids (with the exception of Fluosol) possess neither hemostatic ability nor oxygen-carrying capacity.
†Experimental.

tons) reduces blood viscosity. Some have used it to improve microcirculation during low-flow states. Dextran 40 remains in the vascular space longer than crystalloids but not as long as dextran 70.

Dextran 70 (Macrodex), a glucose polysaccharide (mw 70,000 daltons), expands volume effectively but has little clinical use in the bleeding patient because it causes crossmatching problems, induces a bleeding diathesis due to reduced platelet adherence, and produces an occasional severe anaphylactic reaction.[44]

Albumin, 5-percent, in isotonic saline, a pooled plasma derivative, effectively expands volume, carries no risk of infection, and requires no ABO typing. However, it is expensive.

Albumin, 25-percent, in saline expands volume less effectively than the 5-percent solution, since it contains only one fifth the fluid volume of the more dilute solution. Water drawn from the extracellular fluid space by the concentrated solution may aggravate an extracellular volume deficit.

Plasma protein fraction (PPF), a heat-treated pooled plasma product, contains albumin plus alpha- and beta globulins. Though free of infection risk, PPF is expensive and has been associated with transient hypotension attributed to generation of bradykinin by an activator in the globulin fraction.

Hemoglobin Substitutes. Perfluorochemical emulsion (Fluosol-DA), the only resuscitative fluid besides blood that possesses any oxygen-carrying capacity, has limited usefulness, since it carries oxygen only at an arterial PO_2 above 300 mm Hg,[45] is available only to investigators, and has significant adverse effects.[46]

Blood Products. Human blood is the only available substance with both hemostatic properties and oxygen-carrying capacity. Though expensive, difficult to obtain and store, capable of transmitting all blood-borne diseases and of producing immune-mediated disease itself, blood and its components are irreplaceable in the treatment of hemorrhage. The availability of blood-banking services directly accounts for much of the recent decline in hemorrhagic maternal mortality. Fresh whole blood—if it is truly fresh—performs all the functions of a patient's own blood. However, clotting functions and, to a lesser extent, oxygen-carrying capacity decline with storage. Platelets lose their ability to function within 48 hours when stored at 4°C. After 1 week of storage, activity of the labile clotting factors (Factors V and VIII) falls to 50 percent of normal. Intracellular 2,3-DPG increases for the first 10 days of storage, then declines. Blood deficient in 2,3-DPG releases oxygen from hemoglobin less well, making less oxygen available to the tissues though the lower storage pH of blood partly counters this questionably significant clinical effect.[38] Despite the appeal of fresh whole blood, logistic difficulties usually make it unavailable as an emergency resuscitation fluid. Blood risks include not only infectious diseases such as hepatitis and acquired immune deficiency syndrome (AIDS), but also potentially fatal transfusion reactions which require crossmatching blood from donor and recipient before administration.

Platelets and labile clotting factors are deficient in stored whole blood in proportion to the time stored, while pH, 2,3-DPG levels, and perhaps temperature are lower than those of fresh blood. Risks and compatibility requirements parallel those for fresh blood. Stored blood should be warmed before administration and filtered through a standard 170-μ transfusion filter. Micropore filters are not needed. The need for acute replacement of both intravascular volume and oxygen-carrying capacity calls for administering whole blood. Difficulty in obtaining whole blood instead of components from some blood banks has led to its replacement by packed RBCs with supplemental colloid or crystalloid volume.

Removing most of the plasma from a unit of whole blood leaves packed RBCs—all the red cells of a unit of blood but a hematocrit near 70 instead of 40. Risks and compatibility requirements are those of whole blood. Packed cells can be diluted before administration with normal saline but not with Ringer's lactate (contains calcium) or 5-percent dextrose in water (forms aggregations of red cells).

Fresh frozen plasma, prepared from the plasma of a single unit of fresh blood, presents the same risks as a unit of blood, and should be ABO compatible. A unit of fresh frozen plasma contains most of the plasma proteins of a unit of fresh blood, including the labile Factors V and VIII. An NIH Consensus Development Conference in 1984 reported overuse of fresh frozen plasma, citing "little scientific evidence to sup-

port the increasing use of fresh frozen plasma in clinical medicine." Participants did agree on its value in replacement of documented isolated factor deficiencies and, rarely, after massive blood transfusion when Factors V and VII decrease to less than 25 percent of normal.[47]

Platelets, prepared from a single unit of fresh blood, present the same infectious risks as the unit of blood and should be ABO compatible. Each pack raises the platelet count by 7,000 to 10,000 platelets per mm^3. Platelet survival decreases significantly if platelets are refrigerated, but if stored at room temperature they remain viable for 5 days. To reduce platelet destruction, platelets should be administered through a filter no smaller than 170 microns, using a needle no smaller than 19-gauge. Platelets are used to treat thrombocytopenia (less than 75,000 platelets per mm^3) associated with clinical bleeding. Dilutional thrombocytopenia is the most common cause of a coagulation defect in a patient who has received multiple units of banked blood.[48]

Fibrinogen, prepared by Cohn fractionation of 500 to 1000 pooled plasma donations, carries a correspondingly high risk of hepatitis. If needed, one may obtain a better supply of fibrinogen from fresh frozen plasma (400 mg per 200 ml) or by cryoprecipitate (50 mg per 15 to 20 ml). Cryoprecipitate, possessing the same infectious risk as the single unit of blood from which it originates, commonly serves as treatment for hemophiliacs (Factor VIII deficiency).[49]

Freeze-dried concentrates of Factor VIII and Factor IX now available undergo heat treatment to reduce the risk of transmitting infection.

Emergency Availability of Blood

Each obstetric suite should have immediate access to a blood bank which can provide type-specific blood within minutes after saline crossmatch to patients who have previously undergone ABO and Rh typing and antibody screen. Such a system may not always be practical, especially in small hospitals or in cities with a single central blood bank, but alternatives can compromise patient safety.

A patient of unknown blood type who needs immediate transfusion can receive O-negative blood. However, this introduces the risk of a transfusion reaction in a patient with an undetected rare antibody to a non–ABO antigen. Further, the anti–A or anti–B antibodies which may be present in O negative plasma may be detrimental to recipients with type A, B, or AB blood (55 percent of the population). Giving only O negative packed cells reduces but does not eliminate this problem. A patient who has received 500 to 600 ml of type O plasma should be tested for anti–A and anti–B antibodies before she receives new units of the proper ABO type.[38]

Even in an emergency, obstetric patients should have type-specific blood available. Rarely is a patient's blood type unknown. In that situation, if the patient urgently needs blood but can be managed temporarily with other fluids; rapid ABO and Rh typing enable the patient to receive type-specific blood. Without antibody screen or crossmatch, an undetected rare antibody could still cause a reaction, but the risk with type-specific blood is less than with O-negative blood.

Performing blood type determination and antibody screening in advance enables the patient to receive type-specific uncrossmatched blood without delay and with minimal risk if the antibody screen was negative. In only a few more minutes, an immediate spin (saline) crossmatch provides a check against ABO incompatibilities and errors in typing. Shortening all incubation times to the minimum allows "stat" crossmatch in 30 to 45 minutes, while a complete crossmatch using optimal incubation times takes an hour.[50] Optimally, all blood administered will undergo full cross-matching. Given sufficient fluid, patients rarely die from lack of blood as they await crossmatch.

All patients at significant risk of hemorrhage (i.e., all patients in labor) should have blood drawn in advance for type and screen. Immediate access to blood banking allows patient typing and screening, which usually suffice for most obstetric situations, including most obstetric procedures. Patients at higher risk for transfusion (e.g., cesarean hysterectomy, cesarean section for low anterior placenta previa after previous cesarean section) should have fully crossmatched blood available. Predeposited autologous blood avoids the risks of homologous blood, but few obstetric and gynecologic patients have used this technique.[51]

Techniques of Infusion

Rapid intravenous fluid infusion initially restores tissue perfusion and oxygenation during hemorrhage. One must establish adequate perfusion before oxygen-carrying capacity becomes meaningful. Patients can tolerate severe hemorrhage if they receive *some* physiologic fluid infusion; obstetric patients die not because they receive the wrong fluid, but because they don't receive enough of any fluid to prevent irreversible shock and organ damage. Rapid fluid infusion initially takes priority over the choice of fluid or selection of hemodynamic monitoring.

Intravenous Equipment. Every parturient is a candidate for hemorrhage, and rapid fluid infusion may be life saving. Every parturient should have a large-bore intravenous cannula in place, inserted as painlessly as possible with local anesthesia. For ambulation a "heparin-locked" cannula can replace the connecting intravenous tubing. Optimistic individuals who resist dealing with the catastrophic potential of obstetrics find this recommendation controversial; it has not entirely gained favor even in the author's institution.

The importance of a large-bore cannula to fluid infusion cannot be overemphasized. A cannula smaller than 16-gauge should not routinely be used in obstetrics (Fig. 15–3). Routine use of plastic intravenous bags compressible by a pressure infuser set allows rapid fluid administration under pressure when necessary; an intravenous set with an in-line blood pump permits rapid pumping of fluid or medication boluses. When standard intravenous equipment seems inadequate, a special high-volume infusion system can infuse fluid through a central vein at rates up to 41 liters per hour.[52]

Fluid Management. Reported fluid management schemes for major hemorrhage and trauma resemble each other in concept but vary considerably in details. All investigators initially replace lost blood with clear fluid (crystalloid,

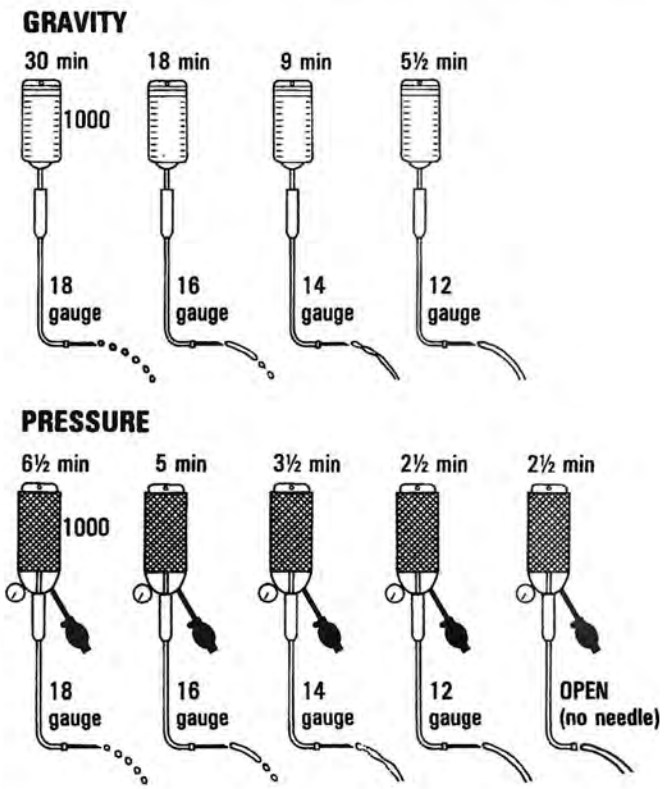

Figure 15–3. Intravenous cannula size affects rate of fluid administration. This figure shows approximate times to infuse 1 liter of fluid through various cannulas. Upper fluids infuse by gravity alone; lower fluids infuse under 300 mmHg constant pressure. Internal diameters of cannulas vary among manufacturers, even for identical gauge; the infusion rate rises with larger diameter and falls with longer length. Cannulas smaller than 16-gauge have no place in obstetrics.

colloid, or a combination) until losses approach 20 to 50 percent of the blood volume, then they administer erythrocytes as packed cells or whole blood.[53–56] For losses exceeding 100 percent of the estimated blood volume, most add platelets from either fresh whole blood[53] or platelet packs.[38,41,54,57] Recommended quantities of platelets range from one pack per six units of blood[41] to two or three packs per unit.[38,57] Most authors also recommend fresh frozen plasma when replacing more than the patient's original blood volume, 1 unit of FFP per 4 or 5 units of blood.[38,41] Prophylactic calcium replacement proves unnecessary,[38] though the physician may administer calcium for inotropic effect when blood replacement exceeds twice the original blood volume.[57]

Monitoring Volume Replacement and Oxygenation. The resuscitator faces the difficult decisions of when to start giving fluid and when to stop. In the nonpregnant patient, traditional signs of tachycardia, hypotension, and sympathetic hyperactivity may not surface until blood loss approaches 50 percent of blood volume. Since a pregnant patient in the lithotomy position can undergo substantial blood loss before manifesting unequivocal changes, vital sign surveillance alone does not adequately signal the onset of significant hemorrhage. An experienced obstetric attendant's observation of "excessive" blood loss probably constitutes the best early warning of hemorrhage.

Rapid infusion of fluids is the most important response to hemorrhage. Since the best approach to shock involves prevention, not treatment, recognition of excessive blood loss dictates infusion of fluids as rapidly as possible, even though vital signs remain unchanged. The infusion continues while the resuscitator assesses the patient, secures additional vascular access, seeks other appropriate fluids, and makes monitoring decisions. Initiating fluid resuscitation takes priority over all other steps.

Blood pressure, heart rate, and mental status guide the first moments of fluid replacement. An automated blood pressure device provides reliable data and frees hands for other tasks. Ideally, the clinical team should monitor shivering or significantly hypotensive patients with an indwelling arterial line after beginning vigorous fluid resuscitation.

Massive or prolonged blood loss justifies other monitoring. An indwelling urinary catheter measures urine output, a reliable indicator of renal perfusion in the absence of diuretics. Pritchard[31] recommends maintaining urine output at 60 ml per hr, 30 ml being the minimum acceptable output. A central venous pressure catheter measures right heart filling pressure, a good indicator of changes in intravascular volume in patients with a normal heart. If insertion of a jugular or subclavian line seems unwise in a patient with a coagulopathy, a long line from an antecubital vein serves the same function. A Swan-Ganz pulmonary artery catheter provides a more sensitive index of volume status in the presence of abnormal myocardial function or pulmonary edema.

Pulse oximetry aids assessment of oxygenation by giving beat-to-beat readings of hemoglobin saturation, though oximetry proves less helpful during shock, since it examines peripheral blood flow. In addition, oximetry may be a poor predictor of tissue oxygenation with very low hemoglobin levels. The best assessment of oxygenation remains periodic arterial blood gas sampling, to measure arterial PO_2, hemoglobin level, and acid-base balance. Metabolic acidosis may reflect inadequate tissue oxygenation with anaerobic metabolism.

Laboratory monitoring during volume replacement includes hemoglobin determination after clear fluid replacement, to evaluate the need for transfusion, as well as periodic assessment of platelet count and coagulation status during massive transfusion (blood replacement greater than original blood volume).

The Patient Who Refuses Blood

Many patients are reluctant to accept blood transfusion because of the medical risks, but few would prefer certain death to the risks of transfusion. Jehovah's Witnesses, however, refuse on religious grounds to accept blood and blood products, including packed cells, plasma, and platelets; most would accept death rather than submit to transfusion. Commercial products prepared from blood are apparently a matter of individual conscience, but serious Witnesses uniformly refuse transfusion.[58]

Jehovah's Witnesses now number more than 650,000 in the United States. Though the de-

nomination was founded in 1884, blood transfusions were not proscribed until July 1, 1945.[59] Biblical references supporting this proscription, such as *Genesis* 9:1–6, *Leviticus* 7:22–27 and 17:1–4, and *Acts* 17:22–29, prohibit eating blood and describe its sacrificial use.

The bleeding patient who refuses blood leaves few options for maintaining oxygen-carrying capacity. Prior withdrawal of blood for reinfusion at the time of need is useless in an emergency, and is usually unacceptable to Witnesses anyway.[60] Some patients refusing prior withdrawal may accept autotransfusion if the medical team can keep the blood in continuity with the circulation.[61] Acceptable systems for immediate preoperative blood withdrawal and contiguous storage[62,63] do not provide for emergency. Fluosol, though not widely available, has been used successfully in two patients whose hemoglobin values dropped to 3 mg per dl after postpartum hemorrhage.[64]

Patients can often survive hemorrhage without blood replacement, even when transfusion might normally have been given.[65,66] However, a hemorrhagic complication of pregnancy with coagulopathy means almost certain death if the patient refuses blood and blood products.[66,67] The obstetric anesthesiologist in particular often must contend with the painful dilemma created when a bleeding patient needs emergency care but refuses blood. An anesthesiologist on emergency call can neither refuse care nor engage in extensive discussion with the patient before providing care. Acceding to the patient's wishes may require the anesthesiologist to permit the preventable death of a young mother. Disregarding her wishes may expose the anesthesiologist to professional isolation and litigation, and the patient may suffer rejection by her own religious community.[68]

A physician who withholds blood in accord with a competent patient's wishes faces no legal jeopardy. A competent adult has the right to refuse a recommended therapy if fully informed of the consequences of refusal; the physician has no obligation to pursue legal efforts to force treatment. If requested, the court may order a mother to accept treatment intended to preserve the life and health of a fetus.[69] Courts are increasingly less likely to order a parent to accept medical treatment on behalf of minor children when other adult family members could care for the children if the parent dies.

A physician who gives blood despite a patient's refusal may face a law suit for battery and for any negligence in the medical treatment provided, though no one has yet successfully pursued such a suit. A decision to transfuse a Jehovah's Witness may commit an anesthesiologist to continuing care of a very ill patient with little support from physicians who disagree, to dealing with untoward consequences of transfusion in addition to the underlying illness, and to facing the displeasure of the patient and the patient's family.

ANESTHESIA

The bleeding patient's anesthesia needs depend upon the obstetrician's plans for diagnosis and definitive treatment, taking into account the individual patient's physical and emotional status. If the obstetrician can define plans and needs clearly, the anesthesiologist can provide the safest and most appropriate anesthetic.

Anesthesia for Examination

Examination of a patient with postpartum bleeding requires manual exploration of the uterus and inspection of the perineum, vagina, and cervix. A skillful examiner can accomplish this without anesthesia in a tolerant patient, but many patients need help. Examination requires no additional anesthesia in the patient with spinal, lumbar epidural, or caudal anesthesia for delivery. Analgesia without loss of consciousness or airway reflexes may permit brief examination of an unanesthetized patient. Nitrous oxide, 50- to 60-percent in oxygen, administered by an attentive anesthetist using mask or mouthpiece, effectively produces brief but profound analgesia without significant residual depression. Small doses of an intravenous narcotic can supplement inhalational analgesia, though the anesthetist must guard against loss of consciousness. Intravenous ketamine in 10- to 20-mg increments provides analgesia, amnesia, and immobility, and can likewise supplement inhalation analgesia. Though some vocal expression of discomfort may persist, patients tolerate examination and even definitive therapy well and seem to accept the technique readily.

Some patients recall having vivid dreams under low-dose ketamine, but if told in advance that dreams may occur, most do not find them unpleasant.

Conditions requiring general anesthesia call for endotracheal intubation to guard against regurgitation and aspiration of gastric contents. If the anesthetist cannot predict obstetric needs or patient response with confidence, general anesthesia with immediate intubation may be preferable to the risk of inducing anesthesia midway through a procedure when the patient is semiconscious and out of control.

Rapid induction of anesthesia with intravenous thiopental (3 to 5 mg per kg) or ketamine (0.8 to 1 mg per kg) and paralysis with succinylcholine (1 mg per kg) permits rapid tracheal intubation. Cricoid pressure applied by an experienced assistant until the airway is secure reduces the likelihood of regurgitation.[70] Pretreatment with a small dose of a nondepolarizing muscle relaxant (e.g., 3 mg d-tubocurarine) eliminates succinylcholine-induced fasciculations, though the dose of succinylcholine required for complete paralysis is then increased to 1.5 mg per kg.[71] A balanced technique maintains anesthesia: ventilate the lungs with nitrous oxide and oxygen, maintain muscle relaxation as necessary, and give small additional intravenous doses of thiopental, ketamine, or a narcotic. Volatile agents may be used in concentrations less than two-thirds MAC; uterine relaxation requires higher concentrations.

Perineal Anesthesia

Anesthesia of the perineum facilitates vaginal delivery, allows examination of the birth canal and repair of vaginal or perineal lacerations, and simplifies examination and manipulation of the uterus (since the examining hand must pass through the perineum). Local infiltration reduces perineal sensation, but bilateral pudendal block produces better and more widespread anesthesia. Major neuraxis blocks—spinal, lumbar epidural, or caudal—anesthetize the sacral roots of the pudendal nerve (S2–S4) but produce sympathectomy and loss of vasomotor tone in the affected areas, which may impair the normal vasoconstrictor response to hemorrhage. Thus major regional anesthesia is relatively contraindicated in an actively bleeding patient, and absolutely contraindicated in the presence of uncorrected hypovolemia. If regional anesthesia of the perineum is inadequate, contraindicated, or impossible to establish, analgesia for examination can be provided with inhaled nitrous oxide in oxygen or by small intravenous doses of a narcotic or ketamine. Balanced general anesthesia with tracheal intubation is an acceptable last resort.

Uterine Anesthesia

The uterus receives visceral innervation from fibers that enter the spinal cord at the T11 and T12 levels. Fibers from the fundus may pass to the tenth thoracic segment[72] (see Chap. 6). A patient may remain unaware of a punctate stimulus such as a suture needle in an area of visceral innervation, but stretching and distention of the cervix or uterus by cervical traction or intrauterine manipulation produces pain. Analgesia or anesthesia may be required, depending on the procedure, the skill of the operator, and the patient.

Patients with adequate spinal, lumbar epidural, or caudal anesthesia for delivery need no further uterine anesthesia. Initiation of major regional anesthesia after delivery can provide uterine anesthesia, though an actively bleeding or hypovolemic patient is a poor candidate for de novo block.

Inhaled nitrous oxide in oxygen, intravenous narcotics, or ketamine may provide adequate analgesia for brief intrauterine manipulation in selected patients. However, unless the obstetrician can ensure brief manipulation and the anesthetist feels confident that conscious analgesia seems appropriate for the patient, general endotracheal anesthesia from the outset may present the best choice. A volatile anesthetic added to the inhaled mixture can enhance uterine relaxation for rapid removal of retained placental fragments or reduction of an inverted uterus.

Uterine Relaxation

The bleeding patient seldom requires uterine relaxation. Indeed, relaxation (atony) is the commonest cause of postpartum hemorrhage. However, if the uterus contracts well but continues to bleed because remaining placental

fragments prevent complete contraction, then momentary uterine relaxation may allow the obstetrician to empty the uterus and facilitate complete contraction. Uterine relaxation may also facilitate reduction of an inverted uterus which has been allowed to contract in the inverted position. However, with prompt recognition and proper management of inversion, uterine relaxation not only proves unnecessary but probably contributes to the problem. Unless the uterus contracts promptly after reduction, reinversion may occur.

Halothane has become the standard anesthetic agent for uterine relaxation, replacing diethyl ether. Halothane concentrations above 2 MAC (1.6 percent) reversibly relax the uterus and block the response to oxytocin infusion,[73] though an equipotent dose of enflurane or isoflurane produces a similar degree of uterine relaxation in vitro[74] and is equally effective. Inhaled anesthetics produce rapid uterine relaxation which abates rapidly when the inhaled agent is withdrawn. The use of inhaled anesthetics as relaxants presents a major disadvantage: Effective concentrations also produce general anesthesia, with the attendant risks and side effects.

Despite the drawbacks of inhaled anesthetics as uterine relaxants, clinical experience with their emergency use is substantially greater than with nonanesthetic uterine relaxants such as tocolytic agents and amyl nitrite. Among tocolytics, selective β_2–adrenergic agonists such as ritodrine and terbutaline produce rapid but persistent uterine relaxation when given as an intravenous bolus; magnesium sulfate acts too slowly and persists too long for emergency use. Amyl nitrite produces rapid smooth muscle relaxation and vasodilation when inhaled from a crushed ampule and appears capable of producing rapid uterine relaxation. It may also produce hypotension in a hypovolemic patient.

Anesthesia for Laparotomy

The bleeding patient may require laparotomy for cesarean section, for ligation of the uterine blood supply, or for removal or repair of a ruptured or atonic uterus. Women who require laparotomy for control of obstetric hemorrhage are among the most desperately ill patients encountered in obstetrics. However, restoring circulatory volume and oxygen carrying capacity as completely as possible before anesthesia induction enables young, previously healthy patients to tolerate anesthesia and operation satisfactorily.

Major Conduction Anesthesia. Major conduction anesthesia (spinal, lumbar epidural, caudal) produces sympathetic block and is generally not appropriate for laparotomy in the actively bleeding patient. However, unexpected bleeding problems during cesarean section under regional anesthesia (e.g., placenta accreta) do not mandate conversion to general anesthesia unless this is required by the patient's emotional state or by the duration or anatomic extent of the procedure. If necessary, a vasopressor such as ephedrine, which affects both resistance and capacitance vessels, counters the pharmacologic sympathectomy of regional anesthesia. A patient facing possible major bleeding (e.g., repeat cesarean section with placenta previa) but currently neither actively bleeding nor hypovolemic should have the same anesthetic options offered to her as any other cesarean section patient. Quality of venous access carries much more significance to such a patient than choice of anesthesia.

Emergency cesarean section in the absence of a capable anesthetist may occasionally call for local infiltration anesthesia by the obstetrician, though obstetricians no longer receive adequate training in this approach and the absence of an anesthetist seems inexcusable except in the most isolated locations. This approach may sacrifice patient comfort but should not compromise patient safety. In the absence of an anesthetist, a physician member of the team should assume the role of resuscitator, monitoring and maintaining vital functions.

General Endotracheal Anesthesia. General endotracheal anesthesia is usually the best choice for laparotomy in a bleeding patient. An adequately resuscitated patient will tolerate rapid-sequence induction with either thiopental or ketamine, followed by succinylcholine-induced paralysis and rapid tracheal intubation with cricoid pressure. When rapid and uncontrollable blood loss prevents adequate fluid replacement prior to surgical control of the bleeding, no anesthetic technique appears ideal. If rapid-sequence induction seems inadvisable, the anesthetist can secure the airway of an uncon-

scious patient by inserting an orotracheal or nasotracheal tube without anesthesia, ventilate with oxygen, then administer a muscle relaxant to provide sufficient immobility to operate. For the conscious patient, small doses of ketamine (0.2 to 0.5 mg per kg) provide amnesia during airway manipulation. However, these approaches present real hazards. Unskilled attempts at intubation in unanesthetized patients can rapidly produce a bloody and compromised airway, perhaps causing hypoxia or provoking vomiting and pulmonary aspiration. Ketamine, which depends on catecholamine release to maintain blood pressure, may depress the cardiovascular system as much as any other anesthetic when given to the hypovolemic patient already experiencing maximal sympathetic activity.[75]

In short, there is no substitute for adequate resuscitation before anesthesia. General anesthesia cannot safely be provided to a hypovolemic patient hovering on the brink of profound shock. Though paralysis and ventilation with oxygen may suffice for surgical control of hemorrhage in a rapidly exsanguinating patient, the presence of an obstetric team prepared for resuscitation should preclude this desperate compromise.

Obstetric Hemorrhage

Obstetric hemorrhage arises either from the placental implantation site or from disrupted blood vessels in the uterus or birth canal. Uterine contraction normally arrests bleeding from the placental site by blood vessel compression, not by coagulation. In contrast, coagulation normally stops bleeding from disrupted blood vessels. Rapid bleeding or disruption of large vessels may require manual compression or surgical intervention to reduce blood flow so clotting can occur; if blood clots poorly, bleeding may persist despite effective control of large vessel bleeding.

This section examines the causes of obstetric hemorrhage. Obstetric considerations for each entity include the nature of the problem, its usual clinical presentation, and the treatment. Anesthesia considerations include brief discussion of both resuscitation and anesthetic management.

ANTEPARTUM HEMORRHAGE

Up to two thirds of antepartum bleeding episodes originate in the placental site, including nearly every instance of life-threatening hemorrhage.[76] Placenta previa and abruptio placentae account for nearly all serious antepartum bleeding; uterine rupture and fetal bleeding, though more grave, occur less frequently.

Placenta Previa

Obstetric Considerations. Placenta previa (literally, the placenta going ahead) implies abnormal implantation of the placenta on the lower uterine segment with some encroachment on the cervical os. A complete placenta previa covers the os entirely; a partial or marginal previa does so incompletely. Placenta previa occurs in 1 in 345 to 1 in 53 deliveries.[77–79] Singh, Rodriques, and Gupta[78] found twice the normal incidence (1 in 26) in patients with a previous cesarean section, peaking in the pregnancy immediately after the section. As noted later, the incidence of morbidly adherent placentas also increases with the number of previous cesarean sections.

Placenta previa causes significant morbidity, but rarely produces maternal death. Although nearly one third of the patients with placenta previa in one series[79] delivered without antepartum hemorrhage, others[80,81] have suggested that 90 percent of patients with placenta previa have at least one hemorrhage, and 10 to 25 percent develop hypovolemic shock. Patients with placenta previa and previous cesarean section run an increased risk for placenta accreta, massive hemorrhage, and hysterectomy to control bleeding.[82] Perinatal mortality, though it greatly exceeds maternal mortality, has improved. As high as 17 to 25 percent a decade ago,[76] perinatal mortality has more recently been reported to be 4.2 to 8.1 percent.[82,83]

Patients with placenta previa most commonly present with painless vaginal bleeding in the third trimester of pregnancy. The first bleeding episode usually ceases spontaneously, and fatalities rarely occur in the absence of rectal or vaginal manipulation. Subsequent episodes may occur at any time, and the bleeding may be severe. Ultrasound examination reliably establishes the diagnosis of placenta previa, largely eliminating the need for the risky double set-up

vaginal examination, which requires equipment and personnel in readiness for emergency cesarean section, should a vaginal examination provoke uncontrolled bleeding from the placenta.

Third-trimester bleeding is assumed to be a significant problem until proven otherwise. A bleeding patient should be admitted to the hospital, with immediate attention to vascular access and availability of resuscitative fluid during evaluation. A large-bore intravenous cannula allows immediate infusion of crystalloid fluid to restore intravascular volume; if bleeding has ceased and the obstetrician plans no immediate intervention, a type and screen suffices when the blood bank is located in the hospital. The blood bank director should be consulted with questions and kept aware of the clinical situation.

Management depends on the volume of blood lost, on the presence or absence of further bleeding, and on the gestational age. In all cases, resuscitation takes priority, and should be underway as clinical evaluation and diagnostic examination occur. Occasionally, bleeding from a disrupted placenta may be so heavy that complete fluid resuscitation becomes impossible without control of bleeding; resuscitation and definitive therapy then proceed simultaneously.

Indications for prompt delivery include continued bleeding that threatens the mother, fetal distress, or sufficient fetal maturity to render additional time in utero unnecessary (35 weeks or more). With firm diagnosis and satisfactory resuscitation, delivery proceeds at once, usually by cesarean section.

If mother and fetus remain stable, the obstetrician must weigh the maternal and fetal risks of keeping the fetus in utero against the fetal risks of premature delivery. For severe or repeated hemorrhage before 35 weeks in a stable patient the obstetrician will probably confirm fetal lung maturity by amniocentesis as soon as the fetus reaches 35 to 36 weeks and then proceed to cesarean section. If bleeding has been slight, expectant management may allow the fetus to mature beyond 35 weeks before delivery. Silver and colleagues[83] "aggressive expectant management" consists of multiple transfusions, volume expansion, tocolysis, and periodic determination of fetal lung maturity. Keeping the fetus in utero until 37 weeks' gestational age with mature lung function reduces the morbidity of prematurity and the cost of newborn care.[84] Patients managed expectantly usually remain in the hospital to minimize risks in the event of recurrent hemorrhage.

Cesarean section is almost always the preferred method of delivery with complete placenta previa to reduce maternal hemorrhage; rarely, vaginal delivery may be possible for a multiparous patient with a low-lying placenta. Even a dead fetus dictates delivery by cesarean section, for the mother is still at risk of hemorrhage.

Significant blood loss often accompanies cesarean section for placenta previa. Because the uterine incision may enter an anterior placenta, producing both maternal and fetal hemorrhage, the infant care team should recognize the possibility of fetal hemorrhage in all cases of placenta previa. The obstetrician may favor a vertical uterine incision to reduce placental injury and avoid extending the incision laterally through the poorly developed lower uterine segment into uterine vessels.

Placenta previa increases the risk of postpartum hemorrhage, since the lower uterine segment implantation site contracts less well than the usual fundal site and compresses spiral arteries poorly. Should this occur despite good uterine contraction, the obstetrician can oversew individual bleeding sites or compress the area with the overinflated balloon of a Foley catheter inserted through the cervix.[85,86] Ligation of the hypogastric (internal iliac) arteries may reduce pulsatile flow enough to achieve hemostasis. If not, the patient may require hysterectomy, which entails substantial additional blood loss (see the discussion of uterine atony in the section on postpartum hemorrhage).

Coagulation problems common with abruption rarely occur with placenta previa. Placenta accreta, more common with previous cesarean section, should lead to prompt hysterectomy without attempts to dislodge the placenta piecemeal. (See discussion of placental abnormalities in the section on postpartum hemorrhage.)

Anesthetic Considerations. Resuscitation of the patient bleeding from a placenta previa follows the general principles outlined in the section on resuscitation. The patient with placenta previa and a history of prior cesarean section especially needs additional massive fluid resuscitation. More than half of such patients in one

series[87] required hysterectomy for placenta accreta.

A patient with placenta previa requires anesthesia for cesarean section. Normovolemic patients can receive regional anesthesia, but those with uncorrected hypovolemia must not undergo this procedure. An unstable patient with continued bleeding and possible hypovolemia should receive general endotracheal anesthesia (see discussion of anesthesia techniques above).

Abruptio Placentae

Obstetric Considerations. Abruptio placentae (literally, breaking away of the placenta) involves separation of the normally implanted placenta before birth. Abruption occurs in 1 in 500 to 1 in 37 pregnancies.[76,77,88,89] More than half appear before the 36th week of gestation;[76] in a series of patients with premature rupture of the membranes, abruption occurred at two to eight times the normal rate.[90] Hypertension is unexpectedly common among patients with placental abruption;[91] one study found abruption in 23 percent of eclamptic patients, 10 percent of chronic hypertensives, and 2 to 3 percent of patients with pre-eclampsia, in contrast to 1.17 percent in the general population.[88] A history of abruptio placentae with a previous pregnancy is associated with a 10-fold increase in the rate of abruption in the current pregnancy.[89] The practice of medicine is itself associated with an increased risk of abruption; one report found evidence of abruption in 12 percent of pregnancies among female physicians.[92]

Though hemorrhage from abruptio placentae appears less dramatic than hemorrhage from placenta previa, abruption is the more lethal condition. Maternal mortality from abruption ranged in 1976 from 1.8 to 11 percent;[76] more recent reviews report maternal mortality from zero to 5 percent.[77,93] Perinatal mortality rates range from 19 to 67 percent with vaginal delivery and 8 to 22 percent with cesarean section.[77,94,95]

Abruption has been reported in association with up to 66 percent of abdominal trauma during pregnancy,[96] usually within the first 24 to 48 hours.[97] Though maternal death from trauma is the leading cause of traumatic fetal death,[98] when mothers survive their infants[99] abruption is the leading cause of fetal death. Individual reports describe abruption in association with snake bite,[100] cocaine use,[101] and congenital hypofibrinogenemia.[102]

Open venous sinuses beneath the abrupted placenta allow thromboplastic material to enter maternal veins and initiate DIC. Coagulation problems occur in up to 50 percent of patients with abruption,[77,95,103,104] becoming more severe with increasing placental separation. Abruption doubles the risk of postpartum hemorrhage; with DIC the incidence of postpartum hemorrhage quadruples again. The blood-suffused Couvelaire uterus of severe abruption responds normally to oxytocics and uterine massage, posing no additional threat of uterine atony.[105] Abruption severe enough to produce hypovolemic shock and DIC may initiate renal failure[103,105] or Sheehan's syndrome of anterior pituitary insufficiency secondary to posthemorrhagic necrosis.[106]

Although bleeding from placenta previa nearly always becomes visible, placental margins, adherent fetal membranes, or a fetal head that completely fills the lower uterine segment may conceal bleeding from abruptio placentae. Fewer than 80 percent of patients with abruption in one series experienced vaginal bleeding.[95]

A patient with severe abruptio placentae classically presents with vaginal bleeding, a painfully hypertonic uterus, hypovolemia, and absent or inaudible fetal heart tones. The extent and duration of separation determine the severity of presenting symptoms. Abruption presents a grave continuing threat to those fetuses who remain alive in utero. In one series, half of the fetuses alive at the time of hospital admission manifested fetal distress.[95]

A patient with suspected abruptio placentae should be evaluated in the hospital. Preventing hypovolemic shock avoids most maternal problems associated with abruption; fluid resuscitation should begin during evaluation of the patient. A large-bore intravenous needle allows restoration of circulating volume with crystalloid while awaiting blood; improved peripheral perfusion and hepatic blood flow reduce the risk of DIC. Blood bank personnel should be notified that the patient may need volume replacement for hemorrhage and component therapy for DIC.

Once the diagnosis of abruptio placentae has

been confirmed by ultrasound examination, obstetric management usually proceeds with vigorous maternal resuscitation and delivery of the infant. In the absence of fetal distress, the obstetrician may attempt vaginal delivery with careful fetal monitoring. Vaginal delivery is the preferred approach if the fetus is already dead, especially in the presence of coagulation problems, unless deteriorating maternal condition necessitates cesarean section. Pagano, Adey and Butterfield[107] recommend cesarean section for moderate to severe abruption when the fetus is viable, the uterus exhibits generalized tenderness and tenseness, and vaginal delivery is not imminent. For severe abruption with coagulopathy, Sher and Statland[108] recommend vigorous fluid resuscitation and expeditious vaginal delivery, avoiding cesarean section if the baby has died except to reduce worsening maternal complications. They also advocate the use of aprotonin (Trasylol), an antifibrinolytic, antithromboplastic, antikallikrein agent not available in the United States. Aprotonin reportedly produces rapid control of DIC, spontaneous resolution of uterine inertia, and rapid progress toward vaginal delivery. A later section discusses treatment of DIC.

Anesthetic Considerations. Since sudden exsanguinating hemorrhage occurs less frequently with abruption than with placenta previa, surgical intervention in the face of inadequate resuscitation seldom proves necessary. As with other hemorrhagic complications, fluid resuscitation should be the first course of action, not only to ensure oxygenation of maternal and fetal tissues but also to dilute activated clotting factors and restore hepatic blood flow as the first steps in preventing or treating DIC. Abruption severe enough to kill the fetus involves considerable maternal blood loss. Pritchard[31] found 1500 to 5000 ml of concealed hemorrhage in such cases, and Brame and associates[103] reported that patients with severe abruption required an average blood replacement of 2650 ml.

For the patient without hypovolemia or coagulopathy, the usual anesthetic options for labor, delivery, and cesarean section apply. Increased uterine irritability with abruption often produces rapid labor, which may reduce the duration if not the degree of pain relief needed. The compromised fetus requires careful electronic monitoring, since one cannot always predict the effects of an anesthetic on uterine blood flow and fetal oxygenation. As with any compromised fetus, supplemental oxygen administered to the mother may help maintain fetal oxygenation should placental perfusion fall.

Uterine Rupture

Obstetric Considerations. Rupture of the gravid uterus, a significant but often unrecognized cause of maternal and fetal mortality, classically occurs in a multigravid patient in tumultuous labor who experiences sudden severe abdominal pain. The abdominal contour changes, labor stops, and the patient becomes "shocky." Fetal heart tones diminish or disappear. However, this classic picture accurately describes only a portion of cases. Recent literature emphasizes a much wider variety of presentations and associations with uterine rupture.

Schrinsky and Benson's extensive review[109] notes the differences between incomplete rupture, usually due to dehiscence of a uterine scar, and the much more catastrophic true or complete rupture of an unscarred uterus. They describe traumatic rupture from instrumentation, violence, or obstetric causes (both direct application of force and indirect application by myometrial contraction) and spontaneous rupture, with or without a history of uterine surgery.

The incidence of uterine rupture of all types appears to fall between 1 in 1000 and 1 in 1500 deliveries in the United States,[109] with traumatic rupture in the majority. Spontaneous rupture represented only 25 percent of Schrinsky's own series, occurring before labor in 4.3 percent of cases. Uterine rupture happens much more commonly in developing countries. Reported African rates range from 1 in 943 in a South African hospital[100] to 1 in 93 at a rural Nigerian hospital.[111]

Uterine rupture contributes significantly to maternal mortality even in the United States, accounting for half the obstetric deaths from uterine hemorrhage and 5 percent of the total maternal mortality.[109] In Schrinsky and Benson's own series no maternal mortality followed rupture of a uterine scar, but maternal mortality rose to 13.6 percent with rupture of an intact

uterus. African reports indicate higher maternal mortality—from 3 percent at a South African hospital to 41 percent at a hospital in Nigeria, where half of the deaths occurred before the patients reached the operating room.[110,112–115] Two reports specifically mention that all maternal deaths were associated with rupture of the unscarred uterus;[113,115] one cites hemorrhage as the cause of eight out of nine maternal deaths.[115]

The fetus is even more likely to die than the mother when the uterus ruptures. Some larger series report fetal mortality over 80 percent after rupture of the intact uterus;[109] even Schrinsky's own series had fetal mortality of 28 percent after rupture of a scarred uterus, rising to 45.5 percent with rupture of the intact uterus.[109]

Uterine rupture most commonly originates from a scar at the site of prior cesarean section[116] or uterine surgery.[117] An unscarred uterus is more likely to rupture in the presence of thinning or increased fibrosis of the myometrium, previous curettage, infection, infarction, adenomyosis, and trophoblastic invasion.[118] The recent literature has firmly documented the relationship to trophoblastic invasion. A review of uterine rupture associated with placenta percreta (placental villi penetrating the myometrium) noted postpartum hemorrhage in 39 percent of patients, 90 percent of whom underwent postpartum hysterectomy.[119] All patients lacked decidua at the site of placental implantation; 30 percent had previously undergone cesarean section. Other reports document uterine rupture before labor due to placenta increta or percreta from 15 weeks to near term,[120–126] or during labor with a breech presentation at 34 weeks.[127] Though uterine rupture is frequent with placenta percreta (7 to 15 percent), the rarity of placenta percreta makes it an uncommon cause of uterine rupture.[121,126]

A number of reports describe uterine rupture when prostaglandins are used to induce labor. Prostaglandin $F_{2\alpha}$ has been associated with uterine rupture when used to induce abortion in the second trimester.[128,129] Uterine rupture has accompanied prostaglandin E_2 therapy for the induction of labor, usually after fetal demise.[122,130–134] In two of these instances a pre-eclamptic patient who suffered a seizure in the immediate postpartum period had a ruptured uterus at laparotomy.[133,134]

Several additional reports describe spontaneous rupture of the uterus in patients without evident predisposing factors at gestational ages ranging from 19 weeks to term,[135–138] emphasizing that uterine rupture may occur outside the intrapartum period.

Clinical presentations of uterine rupture vary; no individual sign is pathognomonic. Bleeding is the most common feature,[110,115] by one report present in two thirds of patients after rupture of the intact uterus and in 40 percent after rupture of a uterine scar.[115] Shock develops in one fourth of patients.[110] Abdominal pain or tenderness occurs more frequently when rupture has been unrecognized or untreated.[111] Fetal distress, uncommon with rupture of a scar, develops commonly with rupture of the intact uterus,[115] and delaying treatment increases its frequency.[111] Lenke[139] describes a single case in which rupture of a scar allowed the umbilical cord to prolapse into the abdomen, producing fetal distress.

Contractions may appear to cease if the uterus ruptures during labor, particularly if the uterine contents are extruded through the rupture into the abdominal cavity. In the presence of posterior rupture, a monitor may continue to show apparently normal contractions[140] or may show a hypertonic contraction pattern similar to that seen in abruption.[137] Significant rupture may remain entirely asymptomatic, only to be discovered as an incidental finding at the time of cesarean section[141,142] or postpartum tubal ligation.[143]

Effective treatment of uterine rupture cannot precede recognition. Even in a large center where uterine rupture occurred frequently, clinicians recognized only 35 percent of ruptures before delivery,[110] finding the remainder either at emergency cesarean section (20 percent) or after vaginal delivery (45 percent). Ultrasound examination can demonstrate scar rupture before it progresses to clinical catastrophe[116] and may help differentiate rupture from severe abruption. However, uterine rupture cannot be found unless it is sought. Gibbs[144] remarks on the tragedy of failing to consider uterine rupture because of its rarity, only to have it appear as a surprising autopsy finding.

Uterine rupture requires operation. Repair may be possible when the rupture has occurred in a scarred area; Fakhoury and coworkers even described fetal survival in utero after repair.[145] However, when an intact uterus ruptures or a scarred uterus tears beyond the scar, hysterectomy almost always becomes necessary. Since hemorrhage is the leading cause of maternal death with uterine rupture, this complication calls for an urgent laparotomy to control bleeding. Prompt maternal resuscitation and immediate operation may also save the baby. Ideally complete maternal resuscitation should take place before operation, but fetal distress or rapid blood loss may necessitate proceeding to operate as resuscitation continues.

Anesthetic Considerations. Prompt restoration of circulatory volume and replacement of blood loss are the most important measures in preparing to treat uterine rupture. Reports of catastrophic rupture describe 2000 to 3500 ml of obvious blood within the abdomen at the time of operation; eventual replacement volume sometimes exceeds 30 units of blood. Since control of bleeding requires surgical hemostasis, anesthesia may need to be induced while resuscitation continues to progress. The section on anesthesia for laparotomy describes anesthesia options.

The anesthesiologist should also consider whether analgesia provided for labor would interfere with recognition and prompt treatment of uterine rupture should it occur. Though some have questioned whether patients allowed to labor after previous cesarean section should receive epidural analgesia for labor,[146] no evidence suggests that epidural analgesia for labor impairs recognition of uterine rupture when it occurs.[147,148] Just as scar separation or significant rupture may be asymptomatic even without analgesia, uterine rupture can be painful even in the presence of epidural blockade.[149,150] Uppington[151] found no reason to withhold epidural block from patients undergoing trial of labor. Fetal distress remained the most common sign of scar separation independent of analgesia, while pain and tenderness were unreliable symptoms of rupture whether or not a patient had received epidural block.

Some have questioned the relationship between epidural block and rupture, noting that uterine rupture in one study occurred more frequently in patients given epidural anesthesia (1 in 596) than in unanesthetized patients (1 in 1362).[115] Since epidural anesthesia was freely available to patients in pain, this observation may simply emphasize the painful nature of yet-unrecognized uterine rupture. Others suggest a causal relationship between uterine rupture and extreme anteflexion in the sitting position for initiation of epidural anesthesia.[115,146] Although some authorities[148] dispute such a relationship, good sense dictates that anesthetists avoid the extremes of pressure that could result when an assistant forcefully flexes the patient by pulling down on the neck and shoulders.

Fetal Bleeding

Obstetric Considerations. Vaginal bleeding before birth almost always comes from the mother: fetal bleeding is unusual and difficult to identify but poses a much more immediate threat to the fetus. The great disparity between maternal and fetal blood volumes may render failure to identify a relatively small quantity of fetal bleeding fatal to the baby. The 80 ml of blood in a fully soaked perineal pad[152] is an insignificant portion of the mother's 5000-ml circulatory volume, but it could represent half the blood volume of a preterm fetus. As with uterine rupture, one must consider fetal bleeding in the differential diagnosis of every antepartum hemorrhage, especially in the presence of fetal distress. Diagnosis of fetal bleeding rests on identification of fetal hemoglobin by laboratory testing.

Iatrogenic fetal hemorrhage may follow disruption of fetal vessels in the placenta or, rarely, in the scalp. Incision through the placenta during cesarean section inevitably allows some fetal bleeding from incised placental villi and larger vessels, though such bleeding lasts only briefly. Fetal scalp blood sampling is rarely complicated by continued fetal bleeding,[153,154] but almost half of the reported cases of bleeding involve mortality.

Spontaneous fetal hemorrhage comes from disrupted placental or umbilical vessels. Acute fetomaternal hemorrhage from placental vessels directly into the intervillous space, invisible and

difficult to diagnose before birth, accounts for some cases of intrauterine death or hypovolemia at birth. Rupture of a vasa previa, a fetal umbilical vessel traversing the fetal membranes, produces visible hemorrhage with rapid fetal exsanguination.

Detection of fetomaternal hemorrhage, direct fetal bleeding into the maternal bloodstream, involves testing for fetal hemoglobin in maternal blood. The ratio of fetal cells to maternal cells allows calculation of the volume of fetal blood infused. Fetomaternal bleeding is not rare; for example, Gjode, Rasmussen, and Jorgensen[155] found evidence of some fetomaternal bleeding in 28 percent of mothers after attempted breech version. However, extensive bleeding is uncommon. Fay[156] estimates that macrotransfusion (fetomaternal bleeding in excess of 10 ml) occurs in 1 in 714 to 1 in 200 births; Hoag[157] reports massive transfusion (fetomaternal bleeding >150 ml) in 1 in 800 deliveries, accounting for 3 percent of perinatal mortality. In contrast to the high fetal mortality, fetomaternal bleeding carries no recognized threat of maternal mortality and little maternal morbidity unless maternal-fetal incompatibility exists. Unexplained fetal death or neonatal anemia should trigger a search for fetomaternal bleeding by testing for fetal hemoglobin in maternal blood.

Velamentous insertion of the umbilical cord occurs when the cord inserts into the fetal membranes rather than the surface of the placenta, allowing the ramifying fetal vessels to lie unsupported in the membranes. Vasa previa (literally, "vessel going ahead") exists when a vessel traverses the membranes in front of the presenting part. Velamentous insertion of the cord occurs in 1 in 417 to 1 in 56 deliveries,[158] nine times more frequently with twins, and most commonly with triplets.[159]

Vasa previa poses no threat to the mother, but the fetal risk is substantial. Perinatal mortality approaches 50 to 60 percent with intact membranes and increases to 75 to 100 percent when membranes rupture.[159] However, correct diagnosis apparently improves therapy; definitive antepartum or intrapartum diagnosis of vasa previa was associated with perinatal mortality in only 23 percent of cases in an extensive review.[158]

Signs of vasa previa include fetal bradycardia and hemorrhage. Fetal monitoring observations have included bradycardia with a "cord compression" pattern as the presenting part compressed an intact vasa previa,[160] and a sinusoidal pattern with vasa previa in twins.[159] Though vasa previa usually produces painless vaginal bleeding after rupture of the membranes, bleeding may occur in the presence of intact membranes or it may not begin for an hour or more after rupture.[161] Fetal distress appears as hemorrhage progresses; if diagnosis and delivery are not immediate, fetal death soon follows.

The obstetrician most readily diagnoses vasa previa before the vessel ruptures. He or she can sometimes palpate a pulsatile vessel on vaginal examination; amnioscopy allows direct examination of the membranes, and some physicians recommend it as a routine measure before artificial rupture of the membranes.[158,162] If hemorrhage has already begun, testing vaginal blood for fetal hemoglobin establishes the presence of fetal bleeding.[159] The hematocrit of a fetal scalp blood sample will resolve doubt about the significance of fetal bleeding, particularly if the bleeding has occurred slowly.[163]

Ruptured vasa previa represents a true obstetric emergency requiring immediate delivery, almost always by cesarean section. Fetal salvage requires a high index of suspicion, rapid action upon diagnosis, and skillful resuscitation of the baby at birth. Though the infant resuscitator must first establish adequate ventilation, an infant who has bled also needs immediate attention to volume replacement. Some of the baby's own blood drawn from vessels on the placental surface into a heparinized syringe can serve as an initial fluid bolus; however, adequate volume replacement usually requires balanced salt solution and perhaps colloid until type-specific or crossmatched blood arrives from the blood bank.

Anesthesia Considerations. As in many other situations, the anesthesiologist may have to function as the mother's guardian while all others concentrate on the distressed fetus. Upon diagnosis, immediate operation represents the appropriate response if the fetus remains alive. Anesthesia is risky, particularly for patients (e.g., those with airway abnormalities, severe asthma, cardiac defects, or malignant hyperthermia susceptibility). The anesthetist must not become so pressured by concern for the fetus that rescue imperils the mother.

In the absence of known maternal contraindications, general anesthesia may be the speediest anesthetic approach for immediate cesarean section. A skilled anesthesiologist can institute spinal anesthesia rapidly. Added maternal safety makes regional anesthesia the technique of choice for some patients.

POSTPARTUM HEMORRHAGE

Every mother bleeds at birth; few have bleeding problems. The contracting uterus normally arrests its own hemorrhage after expelling the placenta, while the mother's physiologic hypervolemia enables her to withstand normal blood loss even without intravenous fluid replacement. The wonder is not that postpartum hemorrhage occurs but that it so infrequently becomes a problem.

Postpartum hemorrhage has classically been defined as loss of more than 500 ml of blood in the postpartum period,[164] implying that substantially less blood loss usually occurs and that precise measurement of shed blood customarily takes place. Neither implication appears accurate. One recent study of measured blood loss in a small series found that 10 percent of patients lost more than 500 ml;[165] a larger study of weighed and measured loss found an average loss of 308 ml at vaginal delivery without episiotomy, 417 ml with episiotomy, and an association of greater blood loss with larger babies and episiotomy.[166] Older measurements of blood volume changes rather than measured blood loss suggest average blood loss of 600 ml with vaginal delivery, 1000 ml with cesarean section, and 1500 ml with cesarean hysterectomy.[167] Applying the above definition, "postpartum hemorrhage" might accompany more than half of all deliveries instead of the 5 percent now expected. In any event, the definition remains meaningless in practice. Estimates of obstetric blood loss are notoriously inaccurate, and clinicians seldom measure shed blood precisely. The implication of quantitative precision ignores reality.

For purposes of this discussion, postpartum hemorrhage includes any hemorrhage that occurs after the birth of the baby and threatens maternal health or survival. Most bleeding problems occur within the first 24 hours, primarily around the time of placental delivery. Donald's[168] dramatic statement, "The third stage is always a time of anxiety which no obstetrician ever wholly outlives," applies equally to the obstetric anesthetist. The anesthetist responsible for a patient's anesthetic care (e.g., a patient having epidural analgesia for labor) clearly must be available to the patient at birth and during the third stage of labor.

Excess postpartum bleeding usually comes from the placental implantation site, either because the uterus fails to contract (uterine atony) or because it cannot contract effectively (retained placental fragments, lower segment implantation, placenta accreta, or uterine inversion). Traumatically disrupted blood vessels in the uterus and birth canal also allow major bleeding. Only uterine atony surpasses lacerations as a source of postpartum hemorrhage. Uterine rupture can produce life-threatening hemorrhage that may not be recognized until after birth.

Uterine Atony

Obstetric Considerations. The normal uterus contracts as the placenta separates, compressing spiral arteries within the myometrium to stop the flow of blood in minutes. The ability of the uterus to arrest its own hemorrhage by contraction is largely a mechanical phenomenon, independent of blood coagulation. The myometrium fails to contract effectively in 2 to 5 percent of vaginal deliveries,[169] making uterine atony the commonest cause of pastpartum hemorrhage and the most frequent antecedent of maternal hemorrhagic death.[170]

Whenever conditions leading to ineffective myometrial contraction occur, the clinician should anticipate uterine atony. Since smooth muscle fibers do not contract effectively when stretched to their full fiber length, uterine atony may occur with the overdistension of polyhydramnios or multiple gestation. Even a macrosomic singleton fetus causes enough uterine distension to produce a sevenfold increase in postpartum hemorrhage when fetal weight exceeds 4500 grams (10 lb).[171] Metabolic interference with contraction may produce atony during hypoxia or ischemia; atony occurs more commonly with arrested labor or after pitocin has augmented labor.[87] Infection such as chorioamnionitis makes atony more probable and

increases the likelihood of hysterectomy for atony.[87]

Pharmacologic agents can impede or entirely abolish uterine contraction (see the section on uterine relaxation). Volatile anesthetics produce dose-related uterine relaxation that interferes with spontaneous uterine activity at about 1 MAC and blocks oxytocin-induced contractions entirely at 2 MAC. Tocolytic agents such as ritodrine, terbutaline, and isoxsuprine reduce uterine contractility and could produce atony if given within several hours of delivery, though Essed and co-workers[166] found no increase in peripartum blood loss in patients who received chronic β-sympathomimetic agents. Magnesium sulfate, a tocolytic agent also used to treat pre-eclampsia, reduces contraction in both smooth and skeletal muscle. Atony has followed use of intravenous dantrolene for malignant hyperthermia prophylaxis.[172] Atony occurs more commonly in the presence of DIC, leading to speculation that fibrin degradation products interfere with uterine contraction. Atony also occurs more often with increasing parity and in patients with a previous history of postpartum hemorrhage.

Uterine atony usually produces painless vaginal bleeding which continues longer than a few minutes after delivery of the placenta; yet bleeding due to atony is not always apparent, since the atonic uterus can hold a large volume of blood before vaginal bleeding begins. The uterine fundus should feel firmly contracted to the examining hand after placenta expulsion, and the fundal height should not change on subsequent examinations. A rising fundus without vaginal bleeding suggests intrauterine bleeding with the cervical os obstructed by blood clots.

Treatment of uterine atony consists of vigorous intravascular volume replacement while attempting to stimulate uterine contraction by uterine manipulation and appropriate pharmacologic maneuvers. Persistent bleeding may necessitate operative or embolic interruption of the uterine blood supply; uncontrollable bleeding may require hysterectomy.

Gentle kneading external massage of the uterus is the first step in stimulating uterine contraction; bimanual massage allows simultaneous exploration of the uterine cavity for placental fragments or disruption.[173] Although packing has not gained universal favor, some obstetricians may elect to pack an atonic uterus with gauze, removing the pack in 12 to 24 hours. Such a patient needs continued careful surveillance since bleeding behind the pack may not be evident.[173]

Pharmacologic agents that interfere with contraction should be withdrawn or discontinued, and pharmacologic stimulation of contraction should be initiated. Many obstetricians routinely administer oxytocin after delivery of the placenta, and this is essential if the uterus contracts poorly. Although intramuscular agents can be given, uterine atony should not be treated without a functioning intravascular line to permit fluid replacement. A patient who refuses an intravenous line for delivery should understand the risks and sign a waiver releasing the hospital and the medical staff from liability for harm caused by her refusal of recommended care.

Rapid intravenous infusion of synthetic oxytocin, 10 to 30 units per 1000 ml of balanced salt solution, can serve to supplement endogenous oxytocin and maintain uterine contraction. An intravenous bolus of more than 5 units rarely produces better contraction than a continuous infusion, and the bolus may be dangerous. Oxytocin causes significant but transient vasodilation;[174] rapid intravenous administration can produce profound hypotension in a hypovolemic patient, though Beeby[175] found no cardiovascular effects of a 10-unit bolus in healthy patients undergoing dilatation and curettage after spontaneous abortion. Oxytocin does have some antidiuretic activity, but brief postpartum administration in balanced salt solution has not been associated with water intoxication.

Calcium chloride is sometimes administered intravenously to treat uterine inertia, particularly that due to magnesium sulfate.

Ergot alkaloids produce rapid and sustained uterine contraction. Though usually administered intramuscularly in doses of 0.2 to 0.3 mg, either ergonovine or methylergonovine may be given cautiously in intravenous doses up to 0.2 mg if more rapid action is essential.[176] Ergot alkaloids can produce hypertension by a complex pharmacologic interaction which appears to include both agonist and antagonist activity

at α-adrenergic and tryptaminergic receptors; they should be used cautiously in patients receiving vasopressors, particularly intramuscular pressors. A hypertensive patient should not receive ergot preparations as arteriospasm with catastrophic consequences could result. Ergonovine has been implicated in coronary spasm causing myocardial infarction in a previously asymptomatic 22-year-old patient.[177]

Prostaglandins are local hormones that produce uterine contraction. Prostaglandin $F_{2\alpha}$ as the 15-methyl analog has been the most useful of the prostaglandins in treating postpartum uterine atony. Hayashi, Castillo, and Noah[178] reported that intramuscular or intramyometrial injection of 0.25 to 0.5 mg 15-methyl prostaglandin $F_{2\alpha}$ for intractable postpartum uterine atony successfully stopped hemorrhage in 86 percent of patients. Mild, infrequent side effects included nausea, vomiting or diarrhea, or slight temperature elevation. Among the patients in whom therapy was ineffective, more than half had chorioamnionitis. Buttino and Garite[179] reported similar results. They successfully arrested hemorrhage in 85 percent; half the failures had placenta accreta. The authors conclude that prostaglandins will successfully treat atony except in the presence of placenta accreta, lacerations, or retained products of conception.

Prostaglandin E_2 has served less frequently in the treatment of uterine atony. In one case vaginal prostaglandin E_2 effectively arrested late postpartum hemorrhage after uterine curettage and three doses of methylergonovine were ineffective.[180] In another, intravenous prostaglandin E_2 at 10 μg per min stopped a single case of primary postpartum hemorrhage within 20 min.[181]

When pharmacologic attempts to stimulate contraction fail, the obstetrician must interrupt the blood supply or remove the bleeding uterus. Interrupting the blood supply without surgery involves either direct infusion of a vasoconstrictor such as pitressin into the bleeding vessel or embolization of the bleeding vessel. Magrina and colleagues[182] successfully controlled hemorrhage with pitressin infusion after oxytocin, methylergonovine, prostaglandin $F_{2\alpha}$, subtotal hysterectomy, and hypogastric artery ligation had failed. Embolization of pelvic arteries with Gelfoam effectively controls postpartum hemorrhage,[183] though the 2-hour procedure more effectively controls pelvic bleeding after gynecologic surgery.[184] Complications such as nerve ischemia or tissue loss leading to vesicovaginal fistula occur more frequently in older patients who have received radiotherapy for malignancy.[185,186]

Ligation of the uterine blood supply can effectively reduce uterine hemorrhage. Blood comes to the uterus from uterine arteries and from anastomoses with ovarian arteries. The uterine artery branches from the hypogastric (internal iliac) artery, one of two branches of the common iliac artery. The posterior division of the hypogastric artery supplies the fascia, viscera, and muscles of the pelvis as well as the perineal, gluteal, and anorectal regions; the anterior division gives rise to internal pudendal, uterine, vaginal, and superior vesical arteries. When possible, Cruikshank and Stoelk[187] recommend ligation of the anterior division of the hypogastric arteries as well as the ovarian arteries.

Hypogastric artery ligation does not render the uterus avascular but reduces pulsatile flow and allows clotting to occur. Unilateral ligation reduces distal ipsilateral flow by only 48 percent but reduces pulse pressure by 77 percent (85 percent following ligation of both arteries).[188] Control of hemorrhage still requires an intact clotting mechanism. The rich collateral blood flow makes pregnancy possible even after uterine artery ligation. O'Leary[189] reported 12 pregnancies after uterine artery ligation; interestingly, 3 of the 12 women had a repeat postcesarean hemorrhage requiring a second hypogastric artery ligation.

Hypogastric artery ligation is drastic therapy requiring surgery in a seriously ill patient; it is neither uniformly effective nor innocuous. In one series of 19 patients treated with hypogastric artery ligation for bleeding, the procedure was effective in 42 percent; the remaining 58 percent went on to hysterectomy.[190] Another series of 18 patients achieved similar results,[191] though two serious complications occurred in this series. One patient developed not only ischemic lower motor neuron damage but also ischemic breakdown of the central pelvic area; the other developed obstruction of the right

common iliac artery and required femoral-femoral bypass grafting.

When all else fails, hysterectomy is the therapy of choice for uterine bleeding. Peripartum hysterectomy is a major operation. Maternal mortality approaches 1 percent in major centers,[146,192] a 10-fold increase over the mortality for cesarean section alone.[193] Reported blood loss before and during the procedure ranges from 500 to 10,000 ml,[194] with replacement of up to 35 units.[192,194] Indications for emergency peripartum hysterectomy include not only uterine atony unresponsive to other measures but also placenta accreta, uterine rupture, extension of a uterine incision laterally into vessels, abruption with DIC, chorioamnionitis, and leiomyomata. Unfortunately, only one-fourth of the patients who will require hysterectomy can be identified in advance.[87]

If hysterectomy fails to control bleeding, few options remain. Selective pitressin infusion and arterial embolization have been mentioned above. Jackson, Liebermann, and Smith[195] describe a single case in which deliberate hypotension induced by sodium nitroprusside slowed bleeding after hypogastric artery ligation in a patient who became hypertensive. Cassels, Greenberg, and Otterson[196] describe use of a mushroom pack for pelvic tamponade after hysterectomy for postpartum bleeding.

Anesthetic Considerations. Uterine atony may be treated simply by uterine massage, or it may require escalating therapy in a progressively more compromised patient, culminating in laparotomy and massive transfusion. The fundamental principal of resuscitative management with uterine atony is to maintain intravascular volume as attempts to treat atony proceed. Prompt and vigorous volume resuscitation should begin at once when the uterus fails to contract and should continue until hemostasis is established. Patients should not have to become hypotensive or exhibit signs of shock to command attention on an obstetric floor; any hint of excess bleeding should prompt vigorous volume replacement while diagnosis and therapy proceed.

Anesthesia depends on obstetric needs. Examination, uterine massage, fluid resuscitation, and oxytocics may require little anesthesia. Laparotomy for control of hemorrhage calls for balanced general endotracheal anesthesia in an adequately and continuously resuscitated patient. (See the section on anesthetic options above.)

Lacerations

Obstetric Considerations. Lacerations of the cervix, vagina, and perineum are the second most common cause of excessive maternal blood loss at delivery. Serious cervical or vaginal lacerations occur more frequently with operative delivery (e.g., use of forceps), particularly when the infant is high in the birth canal, the operator is relatively inexperienced, or the patient is poorly anesthetized and highly mobile. Vaginal rupture is a rare condition that may occur spontaneously or may follow traumatic delivery.[197]

Lacerations should be suspected when vaginal bleeding continues despite good uterine contractions. Diagnosis rests on direct visualization during examination. Vaginal rupture may allow the examiner to see intestinal loops with fatty epiploic appendages during vaginal exam, a presentation which could also be seen with ruptured uterus. Treatment of lacerations consists of maintaining intravascular volume, conducting a thorough examination of the uterus and birth canal, and repairing lacerations surgically. Vaginal rupture may require laparotomy;[197] other lacerations can be repaired vaginally. The obstetrician requires good lighting and assistance with retraction; the patient requires good anesthesia.

Anesthetic Considerations. Resuscitation should be as extensive as necessary for the amount of bleeding; lacerations do not usually produce major hemorrhage unless large vessels are involved. Anesthesia requirements depend on the affected areas. Perineal lacerations frequently involve only the area innervated by the pudendal nerve; when local infiltration does not suffice, careful bilateral pudendal nerve block with supplemental intravenous or inhalation analgesia often proves satisfactory even for extensive repairs. However, major conduction anesthesia (spinal, lumbar epidural, or caudal) produces more widespread and dependable block, allowing not only comfortable perineal repair but also uterine and cervical examination and repair. Repair of extensive lacerations may require general endotracheal anesthesia with

appropriate precautions to prevent aspiration of gastric contents. General anesthesia or major conduction anesthesia are equally appropriate for laparotomy in the well-resuscitated patient with vaginal rupture.

Retained Placenta

Obstetric Considerations. When all or part of the placenta stays within the uterine cavity after separation from the implantation site, the uterus cannot contract completely, and hemorrhage continues. Retention of the placenta or placental fragments is the third most frequent cause of postpartum hemorrhage. The diagnosis is suspected when bleeding continues from the cervical os in spite of apparently firm uterine contraction. Though therapeutic examination and curettage usually confirm the diagnosis, one group has reported detecting placental fragments and clots in the postpartum uterus on ultrasound examination, saving some patients the necessity of examination and curettage should the uterus prove empty.[198] Therapy for retained placenta may be as simple as brief manual examination with placental extraction or as complex as immediate hysterectomy for placenta accreta.

Anesthetic Considerations. Adequate resuscitation must take place before anesthetic intervention. The parturient seldom hemorrhages so rapidly from a retained placenta that resuscitation cannot be accomplished before induction.

Obstetric plans dictate anesthetic management. Good perineal and uterine anesthesia expedite manual removal of the placenta or uterine curettage. Analgesia using nitrous oxide, small doses of narcotics, or ketamine may permit expeditious removal if the placenta has already separated; if not, or if the obstetrician plans curettage or removal of small fragments, more adequate anesthesia is best instituted before beginning. Lumbar epidural, caudal, or spinal block suffice for all placental manipulations; if the patient was not anesthetized for delivery, the anesthesiologist can institute major regional anesthesia de novo for removal of placental fragments, particularly in the absence of hypovolemia and if the obstetrician anticipates extensive examination or curettage. The patient who needs more than brief analgesia but does not qualify for regional anesthesia can receive general endotracheal anesthesia. Inhalation of volatile anesthetics or amyl nitrite can provide uterine relaxation, when required.

Placenta Accreta, Increta, Percreta

Obstetric Considerations. Placenta accreta, an abnormality of placentation in which placental villi attach directly to the myometrium without intervening decidua basalis, makes separation of the intact placenta from the uterus virtually impossible. Placental villi invade or penetrate the myometrium in placenta increta and placenta percreta, respectively. Placenta increta and -percreta have been discussed earlier in relation to antepartum uterine rupture, with which they are strongly associated. Incidence of placenta accreta in recent reviews ranges from 1 in 2562 to 1 in 4348 pregnancies.[199,200] Read, Cotton, and Miller[199] estimate maternal mortality of 3.1 percent from placenta accreta in the period from 1970 to 1980, a decline from previous reports of 37 percent before 1934 and 10 percent between 1945 and 1969.

Placenta accreta shows a strong association with placenta previa and with previous uterine surgery. Not only does the incidence of placenta previa increase after cesarean section, but the incidence of placenta accreta among the previae increases markedly. Singh, Rodrigues, and Gupta[78] found placenta accreta in 15 percent of patients with placenta previa and prior cesarean section, but only 1 percent of patients with placenta previa and an unscarred uterus. Other studies confirm the relationship between placenta accreta, placenta previa, and previous cesarean section.[87,199,201] Clark, Koonings, and Phelan reported that the risk of placenta accreta with placenta previa and an unscarred uterus was 5 percent; with placenta previa and one prior cesarean section, the incidence rose to 26 percent and continued to rise until the risk of placenta accreta reached 67 percent with placenta previa and four or more previous sections.

Recognition of placenta accreta before the placenta is damaged by unsuccessful attempts at manual removal simplifies management, since a completely adherent placenta provokes no maternal bleeding. If untreated, a morbidly

adherent placenta undergoes slow postpartum involution over almost 2 months, with continued risk of infection or hemorrhage; methotrexate has successfully hastened involution,[203] with no ultrasound evidence of the placenta 13 days after delivery. However, this would seldom be the treatment of choice in a major North American medical center.

Placenta accreta usually calls for immediate peripartum hysterectomy.[87,119,199] Recognition of placenta accreta before placental disruption reduces blood loss, but morbid adherence may not be obvious until attempts at removal are unsuccessful. Partial or focal placenta accreta may be treated conservatively without hysterectomy,[199] though placenta accreta in these cases remains a clinical diagnosis without pathologic confirmation.

Anesthetic Considerations. Resuscitation is the first priority, but a partially destroyed placenta accreta may bleed so copiously that complete resuscitation is difficult without simultaneous surgical control of bleeding. In Clark and associates[87] series of emergency hysterectomies for hemorrhage, average blood loss with placenta accreta reached 3506 ml—less bloody than uterine atony (4013 ml) or extension of the uterine incision into the great vessels (4780 ml).

In most cases of placenta accreta, patients will need anesthesia for laparotomy. When placenta accreta becomes evident during cesarean section under regional anesthesia, continued regional anesthesia is appropriate if the patient tolerates additional time and surgical manipulation without difficulty. If enough supplemental sedation to cause loss of airway reflexes becomes necessary, establish general endotracheal anesthesia. Most anesthesiologists employ general endotracheal anesthesia when anesthesia is required de novo for hysterectomy in the presence of placenta accreta, particularly in the unstable or bleeding patient.

Lower Segment Placental Implantation

Postpartum hemorrhage often follows placental implantation in an area of the uterus that contracts poorly, since constriction of the placental bed may be ineffective. Postpartum hemorrhage may occur with placenta previa, with any implantation in the lower uterine segment, or with implantation at the site of an old uterine scar.

When massage and uterotonic agents cannot control the bleeding, surgical control may be necessary. In the patient already undergoing cesarean section, hemostatic suturing of discrete bleeding sites may control bleeding. If this proves ineffective, or control of bleeding requires laparotomy, ligation of uterine blood supply is usually the procedure of choice. Hysterectomy may be necessary as a last resort.

Uterine Rupture

The obstetric and anesthetic considerations with uterine rupture have been discussed in the section on antepartum hemorrhage. Uterine rupture deserves mention in a discussion of postpartum hemorrhage because the diagnosis may not become apparent until the postpartum period, even though rupture is usually an antepartum event. For example, 29 of 129 patients with uterine rupture in one series presented with postpartum bleeding; diagnosis took place in the postpartum period in 45 percent of all uterine ruptures.[110] We emphasize that differential diagnosis of postpartum hemorrhage must include uterine rupture to ensure proper diagnosis and appropriate treatment—in this case, hysterectomy or repair of the uterine defect.

Uterine Inversion

Obstetric Considerations. The inverted uterus actually everts, turning inside out so that the placental surface appears on the outside. Inversion may be partial, with the fundus inverted inside the lower uterine segment, or complete, with the inside-out uterus actually protruding from the vagina. Reported incidence ranges upward from 1 in 1700 deliveries,[204] though unreported institutional rates may be higher. Inversion can be fatal, yet mortality rates are not available from the literature.

Inversion produces profuse and continued postpartum bleeding. Hemorrhage occurred in 94 percent of such cases in one series;[204] 30 percent of these patients were in shock, but the shock related proportionally to blood loss. The uterus inverts more frequently when atonic or when the obstetrician has applied inappropriate

traction to the umbilical cord to hasten delivery of the placenta.[205,206]

Treatment of uterine inversion consists of immediate reduction of the inversion while restoring intravascular volume. Continued firm pressure against the fundus from inside the uterus is necessary for reduction, which can be performed with relative ease without uterine relaxation if undertaken at once. However, if inversion is not recognized or reduction is delayed until after a cervical contraction ring has formed, uterine relaxation may be required.[204,205] This is particularly likely in the hands of an inexperienced attendant; oxytocics and massage fail to stop the bleeding, but contract the inverted uterus so firmly on itself that reduction without relaxation is impossible. Even after reduction, a uterus which has been inverted may remain atonic, and recurrent inversion may occur. Heyl[206] describes successful management of such a patient with 15-methyl prostaglandin $F_{2\alpha}$ and uterine packing. Additional steps to control hemorrhage may be necessary, including laparotomy for control of uterine blood supply or for hysterectomy. (See the section on uterine atony for further discussion.)

Anesthetic Considerations. Resuscitation is the first priority. Though hypotension out of proportion to apparent blood loss was once considered characteristic of uterine inversion, this arose from our inability to estimate obstetric blood loss rather than from any special characteristic of obstetric shock. Postpartum hematocrits usually reflect blood losses much greater than initial estimates. A patient with uterine inversion needs vigorous replacement of current and continuing losses until the uterus is positioned and satisfactory uterine tone is restored.

Reduction of an inverted uterus that has begun to contract on itself can be painful. The process is easily accomplished in patients who have satisfactory regional anesthesia (spinal, caudal, or lumbar epidural) for delivery. Reduction can otherwise be accomplished under balanced general endotracheal anesthesia, usually without uterine relaxation unless the uterus has become tightly contracted on itself. Uterine relaxation, if needed, can be provided by adding a volatile agent to the inhaled anesthetic mixture. However, a patient who has been bleeding from an inverted uterus may be so hypovolemic despite vigorous resuscitation that high concentrations of a volatile agent are unwise. In our experience, uterine reduction has usually occurred by the time the patient is able to tolerate volatile agents.

Cesarean Section

Blood loss at cesarean section ranges from 500 to 1500 ml. Some of the loss comes from the placental site, just as in a vaginal delivery; the remainder comes from vessels disrupted by incisions in the abdominal wall and in the uterus. In spite of the relatively large blood losses, few patients require replacement of RBCs or even colloid. Crystalloid replacement of three to four times the estimated blood loss usually is satisfactory.

However, blood loss at routine cesarean section may approach 20 percent of a parturient's blood volume. Unanticipated bleeding—as from an atonic uterus, an adherent placenta, or a lateral extension of the uterine incision—could rapidly compromise intravascular volume and tissue oxygenation. For this reason, all patients undergoing cesarean section should have a large-bore intravenous cannula in place and should have blood available. When the blood bank is in the hospital, advance blood typing and antibody screen suffice. Except in cases where unusual blood loss is anticipated, patients having cesarean section do not need typed and cross-matched blood set up in advance.

COAGULATION DEFECTS

Congenital defects of the coagulation process, such as hereditary factor deficiencies, have been discussed in Chapter 14. Normal coagulation is reviewed in the physiology section at the beginning of this chapter. This section discusses acquired problems of hemostasis, which may be induced by drugs or by pathologic processes. The section on resuscitation covers dilution of coagulation factors during massive transfusion.

Drug-Induced Coagulation Defects

Heparin, oral anticoagulants such as warfarin, and drugs such as aspirin that alter platelet function can affect the coagulation process. Heparin acts through a plasma cofactor, an-

tithrombin III, to inactive thrombin and neutralize several activated clotting factors (XII, XI, X, IX, and VII).[207] Though heparin is used infrequently in pregnancy, patients with a history of deep venous thrombosis have received it in low doses. In the extremely rare event of bleeding due to anticoagulation with heparin, intravenous protamine (1 mg for every 100 units of heparin remaining in the patient) provides rapid reversal.

A pregnant patient rarely takes warfarin, except in the event of accidental ingestion. Warfarin inhibits production of vitamin K–dependent clotting factors (prothrombin, VII, IX, X) in the liver. Intravenous administration of up to 50 mg of vitamin K_1 serves as treatment for severe warfarin-induced hemorrhage;[207] continued intravascular volume support will be needed, however, as the drug requires several hours to produce effects. In a bleeding crisis the anesthesiologist may consider administration of the deficient clotting factors themselves.

Pregnant patients often have used aspirin because they regard it as a benign drug and because it may have some therapeutic role. For example, some have suggested that aspirin prevents maternal symptoms of pre-eclampsia,[208] although Goodlin[209] contends that only the mother benefits from such therapy. Aspirin interferes with platelet function by inhibiting release of ADP and inhibiting synthesis of the prostaglandins and thromboxane which stimulate aggregation. The effects on platelets are irreversible, lasting the life of the platelet. Prolonged bleeding times may persist for several days after aspirin therapy ceases.[207] Stuart and colleagues[210] found hematologic abnormalities in 60 percent of mothers and 90 percent of newborns when mothers took aspirin within 5 days of delivery.

Acquired Coagulation Defects

Blood's ability to clot helps stop bleeding; however, defective clotting does not by itself cause bleeding. Bleeding follows disruption of blood vessels; coagulation problems only make such bleeding more difficult to stop. The most common hemostatic defect is inadequate surgical hemostasis;[55] discovery of a coagulation problem should not divert attention from mechanical hemostasis. Surgical hemostasis must be even more meticulous when blood clots poorly.

Acquired coagulation defects with pregnancy include disseminated intravascular coagulation (DIC), coagulopathy with pre-eclampsia, and the dilutional changes with massive transfusion. The last has been discussed earlier in the chapter.

Disseminated Intravascular Coagulation. Generalized activation of the clotting system can occur when a disease causes widespread vascular damage or when thromboplastic materials enter the circulation.[33] Obstetric patients are particularly at risk for the latter because of the open venous sinuses beneath the placental site. Abruptio placentae is the commonest obstetric cause of DIC; significant coagulation abnormalities occur in 10 percent of abruptions overall but in up to 30 percent of severe abruptions.[211] Twenty percent of those with coagulopathy develop uterine inertia that is refractory to oxytocin.[212] The catastrophic cardiovascular collapse thought to accompany amniotic fluid embolism is accompanied by significant DIC so frequently that the presence of DIC helps establish the diagnosis. Additionally, DIC can also occur in the patient who carries a retained dead fetus in utero, as products of fetal or placental degeneration enter her circulation. The incidence of coagulation abnormalities exceeds 25 percent if the fetus has been dead for more than 4 weeks.[211]

Whatever the initiating event, DIC results from generalized activation of a usually local process. Thrombin and plasmin generation leads to the evolution not only of fibrin but also of fibrin-fibrinogen degradation products, which behave as anticoagulants. Though some patients remain asymptomatic, most patients will display signs of either excessive bleeding or of intravascular thrombosis, depending on the balance between the evolution of thrombin and the limiting reactions. Shock will worsen the problem by reducing peripheral blood flow and hepatic perfusion, thereby reducing dilution and clearance of activated clotting factors while producing further cell damage.

Diagnosis. No single laboratory test establishes the presence of disseminated intravascular coagulation, nor is any specific constellation of signs and symptoms unique. Clinical susp

cion prompts laboratory testing. Suspicion may be based on increased likelihood of DIC—the patient with abruption, amniotic fluid embolus, or retained dead fetus—or on a clinical presentation that unmistakably suggests a clotting problem, with bleeding from the gums, from venipuncture sites, from surgical wounds, and into the urine. The diagnosis rests on laboratory evidence that clotting factors are being consumed, with activation of both coagulation and fibrinolysis.

Laboratory findings consistent with DIC include decreased fibrinogen level, decreased platelet count, prolonged thrombin time, abnormal levels of fibrin-fibrinogen degradation products, and a peripheral smear showing red-cell fragmentation.

Plasma fibrinogen levels normally range from 400 to 600 mg per dl in pregnancy, with levels as high as 800 to 1000 mg per dl in severe pre-eclampsia. A "normal" level of 150 to 250 mg per dl may represent marked depletion in a pregnant patient. Comparison with a baseline value is helpful; serial tests establish a decline.

The platelet count remains normal in pregnancy. Laboratory levels that would otherwise be abnormally low are likewise low in pregnancy.

Fibrin-fibrinogen degradation products can be demonstrated by a variety of tests which fall into three groups: immunologic tests (Fi test, tanned red-cell agglutination), staphylococcal clumping, or paracoagulation (protamine sulfate precipitation, ethanol gelation). Though the effect of pregnancy on FDPs is not resolved, it appears that they should not normally be present, even in pregnancy.[37] Individual laboratories can best interpret the significance of the tests which they use.

The thrombin time, measured by adding dilute thrombin to plasma and recording the time necessary to form a clot, tests the reactivity of the fibrinogen in plasma. This reactivity remains normal in pregnancy but is reduced in DIC.

Treatment of DIC. Treating obstetric DIC consists primarily of maintaining circulatory volume while removing the stimulus that triggered DIC. Blood volume replacement and circulatory support are the resuscitator's first priorities. Replacement of specific clotting factors is usually unnecessary and may be dangerous. Treatment with heparin or with inhibitors of fibrinolysis appears to have little place in obstetrics.

Volume replacement follows the guidelines outlined earlier in the section on resuscitation. Restoring the circulation improves regional circulation and hepatic blood flow, both important as thrombin-limiting forces. If massive blood replacement is necessary, the deficiencies of stored blood (Factor V, Factor VIII, platelets) may become more of an issue. Fresh frozen plasma and platelets can be administered as needed in the presence of severe clotting factor deficiency. However, in the absence of frank bleeding from open vessels, replacement of clotting factors is usually unnecessary. Deficient factors will be repleted after the initiating stimulus has been corrected—in most obstetric cases, after the uterus has been emptied. If the stimulus persists, infusion of additional clotting factors may stimulate further consumption, perhaps tilting the balance in favor of clotting rather than lysis.

Fibrinogen replacement, once emphasized in treating obstetric DIC, now seems unsupportable. Bleeding in DIC results more from anticoagulant breakdown products of fibrinogen than from depletion of clotting factors; adding fibrinogen may worsen the process. The high risk of hepatitis with commercial pooled-plasma fibrinogen reduces its appeal even further.

Antifibrinolytic agents such as ϵ-aminocaproic acid have no role in treatment of obstetric DIC; such agents remove the important thrombin-limiting action of plasmin and may result in generalized thrombosis. Sher[93] has championed the use of aprotonin (Trasylol), an antifibrinolytic, antithromboplastic, antikallikrein agent currently unavailable in the United States, claiming improvement in consumption coagulopathy and in uterine activity with its use.

Although some suggest that antithrombin action of heparin should make it useful for treatment of DIC, nothing recommends its use. It has not been shown effective, may be frankly dangerous in the presence of open blood vessels, and is not necessary because most obstetric DIC clears by itself with adequate resuscitation and removal of the initiating stimulus.

Obstetric DIC has no specific treatment beyond applying sound principles of resuscitation to support the patient while removing the

initiating stimulus and recovery begins. Obstetric need, the patient's intravascular volume status, and the bleeding which follows all vascular injury dictate anesthetic considerations.

Coagulation Defects With Pre-eclampsia. Pre-eclamptic patients may exhibit coagulopathy manifested primarily by thrombocytopenia, with normal levels of fibrinogen, normal prothrombin time and partial thromboplastin time, and no evidence of fibrin degradation products.[211] Platelet counts may drop to 20,000,[213] often associated with a corresponding derangement of liver function.[214] Weinstein[215] has coined the acronym HELLP for the syndrome of *H*emolysis, *E*levated *L*iver enzymes, and *L*ow *P*latelets which appears as a variant of severe pre-eclampsia. Available evidence suggests that the thrombocytopenia of pre-eclampsia results not from a change in platelets themselves nor from increased thrombin activity but rather from an apparent increase in platelet turnover as platelets adhere to damaged vascular endothelium.[216–221] Weenink and associates[222] report that platelet counts and antithrombin III levels correlate inversely with the degree of placental infarction in pre-eclampsia and suggests that increased consumption in the maternal vasculature also depresses antithrombin III levels.

Treatment consists of supportive therapy, including platelet transfusions for counts under 20,000, while expediting delivery. If severe thrombocytopenia and significantly prolonged bleeding time exist, major neuraxis block (spinal, epidural, caudal) may present an unacceptable risk of bleeding into the subarachnoid or epidural space.

Other Acquired Problems. Two recent reports[223,224] describe patients with acquired Factor VIII inhibitors who experienced late postpartum bleeding (10 days after vaginal delivery or cesarean section). Patients were treated successfully with Autoplex, an anti-inhibitor coagulant complex, when other treatment had failed.

Nonobstetric Hemorrhage in the Obstetric Patient

In addition to the bleeding that follows trauma and accidents, obstetric patients face a greater risk of hepatic bleeding and intracranial hemorrhage. Pregnancy may complicate treatment of other nonobstetric hemorrhage.

HEPATIC BLEEDING

Intrahepatic bleeding, subcapsular hematoma, and rupture of the liver are rare complications of pregnancy associated with pre-eclampsia. The incidence is difficult to determine; maternal mortality ranges from 40 to 75 percent when the liver ruptures.[225,226] Liver enzyme elevation, coagulopathy, and right upper quadrant pain accompany hepatic bleeding. Ultrasonography or CAT scan can identify intraparenchymal or subcapsular bleeding before liver rupture.[227] Rupture may occur suddenly, usually in the right lobe, with rapid deterioration due to intra-abdominal bleeding.

Once the liver ruptures, prompt recognition of the problem facilitates resuscitation of the patient and surgical control of bleeding.[228] Control of hemorrhage almost always requires laparotomy, though Loevinger[225] reports a single case in which transcatheter embolotherapy using Gelfoam pledgets successfully treated bleeding from hepatic rupture.

Anesthesia considerations are those of other massive hemorrhage. Resuscitation is the first priority, but it may not be accomplished until bleeding is surgically controlled. Laparotomy calls for general endotracheal anesthesia. The patient, usually severely pre-eclamptic (see Chap. 21), will remain quite ill even if she survives the operation and merits attention to hemodynamic and ventilatory monitoring which should continue postoperatively.

INTRACRANIAL AND OTHER BLEEDING

Intracranial hemorrhage is a rare event, occurring in fewer than 1 : 10,000 pregnancies; however, the mortality exceeds 80 percent.[229] The risk of intracranial hemorrhage increases with pregnancy-induced hypertension. Treatment depends on the lesion and on the stage of pregnancy. Although a viable fetus might be removed by cesarean section at the time of craniotomy for bleeding, Reece and colleagues[229] report craniotomy for intracerebral hematoma at 24 weeks' gestation, after which the fetus was left in utero until vaginal delivery at 33 weeks.

Howard[230] reports recurrent epistaxis in a normotensive pregnant patient that was so severe that the patient underwent simultaneous cesarean section and external carotid artery ligation at 36 weeks' gestation. The patient recovered uneventfully after an episode of aspiration pneumonia secondary to inhalation of blood.

Summary

Birth is bloody, but bleeding at birth should seldom be fatal. Rapid and adequate fluid resuscitation is the most important response to obstetric hemorrhage, followed by immediate measures to stop the bleeding. The anesthetist must ensure resuscitation, assist in diagnosis, and facilitate repair.

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