Shock in the Obstetric Patient

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OBSTETRIC SHOCK is the most important and the most frequent emergency arising during pregnancy and the puerperium.[1] The causes may be divided into those which are truly obstetric and those of a more general nature, but in which the pregnant state is the exciting factor.[2] Obstetric shock, like shock in men and nonpregnant women, is attributable primarily to one of six factors:

(1) Hypovolemia (dehydration, hemorrhage)
(2) Impediment to Blood Flow (supine hypotension, amniotic fluid infusion, pulmonary embolism, air embolism)
(3) Cardiac Failure (rheumatic heart disease, toxemia of pregnancy)
(4) Neurogenic Dysfunction (spinal shock, inversion of uterus, postpartum vasomotor collapse)
(5) Bacteremia (endotoxins)
(6) Hypersensitivity Reactions (anaphylaxis, reaction to drugs)

Hemorrhage is the most common cause of obstetric shock.[3] In 1935, 13.6 per cent of maternal deaths in the United States were due to hemorrhage, whereas in 1952, that figure had increased relatively to 46.2 per cent. This does not imply that more parturients are dying from hemorrhage but that fewer women are dying from other causes.[4] In particular, the incidence of infection has declined markedly since the advent of antibiotic therapy.[5] On the other hand, the number of deaths from amniotic fluid infusion appears to have increased, although this increase may be more apparent owing to improved diagnostic methods.[6]

The treatment of shock in the obstetric patient consists of three phases: (1) prevention, (2) early recognition and accurate diagnosis, and (3) active therapy.[1] In prevention, good prenatal care is the most important single factor. Of 485 patients who died from obstetric hemorrhage in North Carolina in the 10-year period from 1946 to 1956, only 18 per cent had received "adequate" prenatal care, and 43 per cent had had no prenatal care at all.[1] Prompt diagnosis is aided by the astute and regular observation of the parturient's vital signs as well as cognizance of unusual pain, apprehension, or the cardinal signs of early shock.[1] Active therapy depends upon the etiology of the complication.

Recovery from obstetric shock may be followed by either of two sequelae: acute renal failure or anterior pituitary insufficiency.

Acute renal shut-down results in the main from reduced renal perfusion regardless of etiology. In a review of 94 obstetric deaths associated with renal failure, hemorrhage was the primary cause in 32 patients, toxemia in 37, infection in nine, and embolism in two.[7] Acute tubular necrosis has occurred particularly as a complication of severe abruptio placentae, possibly because the magnitude of blood loss is often underestimated[8] and circulating foreign substances are additive to the renal vasoconstriction. Anterior pituitary insufficiency, "Sheehan's syndrome," usually appears shortly after the puerperium but onset may be delayed for several years. In a survey of 18 women seen at the Mayo Clinic with the diagnosis of postpartum necrosis of the pituitary, sixteen presented a definite history of hemorrhage and obstetric shock.[9]

Shock from obstetric complications may occur almost from the time of conception onward; however, this discussion is limited to the shock states of late pregnancy and the puerperium.

Shock Due to Hypovolemia

Dehydration. Intravenous fluid therapy has resulted in a marked decline in the number of parturients presenting with "exhaustion" shock: shock due to dehydration, electrolyte

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imbalance and metabolic disorders. The incidence of these abnormalities was reported as 0.1 per cent in a study of 1,620 obstetric deaths [1] and as 1.7 per cent in a review of 234 maternal fatalities.[10] Labor is an energy-consuming process: oxygen consumption increases [11] and both aerobic and anaerobic carbohydrate metabolism rise steadily.[12] Depending on its intensity, labor can be compared with moderate to severe muscular exercise.[11] Water and salt are lost by sweating, often by vomiting. General symptoms include weakness and thirst. More specific signs include dry skin, dry tongue, and soft eyeballs. Hemoconcentration and metabolic acidosis are evident.

Prevention calls for the intravenous administration of fluids and dextrose in all cases of prolonged or complicated labor. Treatment consists of the immediate intravenous infusion of dextrose in saline or preferably Ringer's lactate solution. Anesthetic management must be designed to prevent further electrolyte and metabolic impairment. Since diethyl ether and methoxyflurane cause a trend toward metabolic acidosis, they should be avoided. Nitrous oxide and ethylene do not produce any significant metabolic change. Derangements in acid-base balance during cyclopropane, halothane, or fluroxene anesthesia are largely attributable to retention of carbon dioxide and can be prevented by adequate pulmonary ventilation. Nitrous oxide and cyclopropane appear to be the inhalation agents of choice for many procedures. Halothane may be used in minimal concentrations but any blood loss due to uterine relaxation may prove detrimental. Regional blocks do not cause metabolic alterations, but may lead to severe and possibly uncontrollable arterial hypotension in the hypovolemic mother and are, therefore, not as a rule recommended.

Hemorrhage. The early signs of blood loss, namely a decline in blood pressure coupled with a rise in pulse rate, may not develop in the normal obstetric patient until the blood volume has been reduced by 30–35 per cent, the equivalent of two liters of blood.[13] This is attributable, in part, to the young age and generally good health of the parturient and, in part, to the compensatory physiologic alterations of pregnancy. At term, total blood volume is increased by approximately 30 per cent, the red cell mass by 6 per cent, and hemoglobin by 15 per cent.[14, 15] The augmentation of blood volume, combined with hyperchromaticity of the erythrocytes, serves to hold blood in reserve in a dilated vascular system.[16] In addition, the lithotomy position, generally employed during parturition, helps the patient to maintain her blood pressure.[1] At least 500 ml. of blood are added to the effective circulation when the legs are placed in stirrups. The same amount is sequestered with lowering of the legs, and the clinical picture of shock may rapidly develop if the blood volume has been seriously reduced.

HEMORRHAGE FROM DISRUPTED BLOOD VESSELS: Bleeding may be externally evident or concealed. In the antepartum period, marginal sinus rupture, placenta previa, and abruptio placentae constitute the most frequent causes of external hemorrhage. Only abruptio placentae presents the additional hazard of concealed bleeding in the formation of a retroplacental hematoma.[1] Hemorrhage from a ruptured marginal sinus occurs whenever this relatively fragile vessel undergoes disruption of its mural structures.[2] The volume of blood temporarily trapped leads to mild discomfort from uterine contractions, and bladder urgency develops.[3] Bleeding from a low-lying, marginal, or partial placenta previa is the result of stretch and tension on the membranes during uterine contractions; this takes place through the previal portion itself in total placenta previa. The onset of hemorrhage is otherwise symptomless; bleeding is usually intermittent and painless because of the proximity of the cervix and an unobstructed escape.[2, 3] Development of placenta previa is favored by two factors: multiparity and atrophic changes in the endometrium.[17]

The hemorrhage of abruptio placentae is associated with extensive infarction of the placenta; necrosis of the chorionic epithelium in the infarcted areas liberates thromboplastin which, in turn, activates the clotting mechanism, producing large clots and placental separation. Excess fibrin formation in the general circulation may deplete blood fibrinogen sufficiently to result in hypofibrinogenemia.[18] Abruptio placentae is characterized by a sudden stormy onset, severe pain, and continuous

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hemorrhage.[2] Predisposing causes are hypertensive vascular disease, with or without toxemia, and trauma.[2, 18] In the 1964 California Maternal Mortality Survey,[19] among 58 cases in the "hemorrhage" group, placenta previa accounted for 8 fatalities; 6 patients died of hemorrhage, one of embolic blood clot, and one of sepsis. Abruptio placentae was the cause of 15 deaths; 10 from excessive blood loss, 3 from amniotic fluid infusion, and 2 of uremia.

Rupture of the uterus occurs most commonly near, or at term. The present-day etiologic classification includes the scarred and the intact uterus. Scarring as the result of previous cesarean section, myomectomy, or hysterotomy has emerged as the greatest single predisposing factor. Rupture of the intact uterus characteristically involves the distended and weakened lower segment. It is caused either by mechanical factors preventing the advance of the infant through the birth canal or by trauma attributable to difficult operative delivery, a blow, fall affecting the abdomen, or automobile accident.[1, 2, 17, 20] The use of oxytocics in obstructed labor and excessive fundal pressure are major contributing factors.[20] The resultant bleeding is both external and internal. In an analysis of 133 patients with uterine rupture, the estimated blood loss was greater than 1,000 ml. in approximately two-thirds of the group with an intact uterus; in the uterine scar group, only one fifth lost this amount.[20] Early signs of shock make diagnosis and surgical intervention possible before expulsion of the fetus into the abdominal cavity occurs.[21] Abdominal pain and uterine tenderness are less frequently observed.[20]

During parturition, hemorrhagic shock may result from traumatic operative delivery owing to difficult forceps application, breech extraction, version, or cesarean section, especially when performed in a patient whose condition is already jeopardized by prolonged labor, dehydration, anemia, or infection.[21]

Blood loss and shock in the postpartum period are, in general, attributable to one or more of five causes: uterine atony which accounts for approximately 80 per cent; bleeding from the placental site, 10 per cent; lacerations of the genital tract, 7 per cent; retained secundinae, 2 per cent; and defects in clotting mechanism, 1 per cent.[3, 4] Hemorrhage from an atonic uterus as a rule is preceded by prolonged labor, a history of multiparity, or multiple pregnancy. When sudden and massive, the typical hypovolemic shock picture is evident. When slow and insidious, appearing only as a heavier-than-usual lochia, most of the patient's potential reserve will have been lost by the time the symptoms of shock become apparent.[3] Bleeding from lacerations of the birth canal, particularly when uterus or both uterus and cervix are involved, may be largely arterial. If the hemorrhage is the result of a tear in the fundus, and blood is spilled into the peritoneal cavity, a neurogenic component of pain and spasm will potentiate the shock state.[3] Postpartum bleeding is not infrequently a slow continuous ooze; this may lead to underestimation of actual blood loss.[1, 21]

Rupture of the utero-ovarian or paravaginal veins may occur as result of the muscular exertion of labor, too vigorous manipulation during delivery, or as part of the third stage. The capacity of the veins of the broad ligament, and the ovarian veins principally, increases 60 times by the ninth month of gestation; hydrostatic pressure within these vessels is doubled. Rupture leads to profound hemorrhage which may be intra- or extraperitoneal, or both, and is associated with a mortality rate approaching 50 per cent. The clinical picture is that of any acute abdominal catastrophe.[22] With intraperitoneal bleeding, pain and signs of peritoneal irritation are generally present. When bleeding is retroperitoneal, dorsal or costovertebral angle pain may be noted.[23]

HEMORRHAGE OWING TO DEFECTS IN CLOTTING MECHANISM: Defects in clotting mechanism may result from hypofibrinogenemia, thrombocytopenia, aplastic anemia, anticoagulant therapy, and vitamin deficiency.[4] Only the first two entities are of importance statistically.

Hypofibrinogenemia: The genesis of hypofibrinogenemia is complex. There are at least three mechanisms by which hypocoagulability or incoagulability of the blood may develop [24]: (1) Plasma fibrinogen may be depleted. Continued bleeding and clotting within the uterine cavity at the site of placental separation, as in abruptio placentae, uses up large quantities

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of fibrinogen. In seven cases of abruptio placentae with hypofibrinogenemia, two-thirds of the fibrinogen calculated to be lost from the circulation was recovered as fibrin in clots evacuated from the uterus.[4] When tissues rich in thromboplastin become necrotic, such as occurs in abruptio placentae, macerated dead fetus, neglected missed abortion, or amniotic fluid infusion, massive amounts of thromboplastic substances are released into the circulation. This results in the conversion of fibrinogen to fibrin, the deposition of fibrin intravascularly, and exhaustion of the fibrinogen stores.[3] (2) Fibrin and fibrinogen may be destroyed by plasma fibrinolysins. Normally, a delicate balance exists between profibrinolysin, the inactive precursor of fibrinolysin, and its antagonist, antifibrinolysin. Transformation of profibrinolysin to fibrinolysin occurs when kinases present in endometrium or amniotic fluid enter the circulating bloodstream. Fibrinolysis appears to be increased by hypoxia, shock, and stress.[24, 25] (3) The conversion of prothrombin to thrombin may be blocked and thrombin may be inactivated by the release, systemically, of a heparin-like substance which is present in amniotic fliud.[24, 25] Decreased numbers of platelets or concentrations of accelerator factor or both have also been observed in abruptio placentae.[26]

The principal characteristic of a clotting defect is uterine hemorrhage with insufficient clot formation. In addition, there may be bleeding from the sites of venipuncture and from mucous membranes.

The blood fibrinogen level in the pregnant woman at term is normally about 450 mg./100 ml.[25] A level below 150 mg./100 ml. is considered potentially dangerous and a level below 100 mg./100 ml. is associated with inadequate clotting.[4] One of the most useful aids in the diagnosis of hypofibrinogenemia is the clot observation test; clotting is defective if a clot is not formed within 6 minutes, or if a soft clot forms and lyses within an hour. The presence of a circulating fibrinolysin is determined by mixing equal parts of unclotted maternal blood and normally clotted control blood; lysis of the normal clot represents a positive result. A circulating heparin-like factor is demonstrated by mixing equal parts of maternal blood and normal control blood in a test-tube; failure to clot establishes the diagnosis.[25]

Among the complications of pregnancy which are associated with the hazard of coagulation defects, abruptio placentae ranks first. Thirty-two of 48 cases of clotting abnormality observed over a seven-year period at the Los Angeles County Hospital were noted in cases of abruptio placentae. Postpartum hemorrhage contributed to hypofibrinogenemia in 4 women and prolonged intrauterine fetal death in 2. There was only one case of amniotic fluid infusion in this series.[25]

Idiopathic thrombocytopenic purpura: A review of 84 pregnancies in 62 mothers with idiopathic thrombocytopenic purpura disclosed that the maternal mortality rate was low; only one mother died (1.6 per cent); this rate does not exceed that expected from the disease alone. However, in the 84 pregnancies, there were 7 cases of excessive postpartum bleeding, an overall incidence of 8.3 per cent. These patients exhibited decreased platelet counts at the time of delivery, whereas postpartum hemorrhage was not recorded in patients with normal platelet counts.[27]

THERAPY: Hemorrhagic shock is treated by the prompt and adequate replacement of blood loss with compatible whole blood. Supportive measures include 15-degree elevation of the lower extremities and administration of high inspired oxygen concentrations. Coagulation defects should be suspected and defined. The specific therapy of clotting defects is based on the nature of the defect. Hypofibrinogenemia is corrected by giving fibrinogen in sufficient amounts to cause stable blood-clot formation. In order to elevate fibrinogen above the critical level of 100 mg./100 ml. in a patient with little or no circulating fibrinogen, a minimum of 4 g. of fibrinogen must be administered at a rapid rate. If clotting is not improved significantly within an hour, an additional 4 g. of fibrinogen should be given.[25, 26] Since there may be a decrease in the amounts of prothrombin, platelets, and/or accelerator factors in addition to fibrinogen, replacement of fibrinogen without administration of other blood constituents may not always correct the bleeding tendency.[26] When a circulating fibrinolysin is responsible for the clotting defect, the

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patient should receive epsilon-aminocaproic acid; an initial priming dose of 4 to 5 g., administered by intravenous infusion during the course of an hour, is followed by continuous infusion at the rate of 1 g. per hour for the next 8 hours, or until the bleeding has been controlled. When a heparin-like factor has been diagnosed, intravenous protamine sulfate is the drug of choice; 20 to 50 mg. should be given by slow intravenous injection.[25]

Anesthetic management must be designed to aid in the maintenance of circulatory homeostasis. Inhalation anesthesia with the highest possible oxygen concentration is the preferable technique. Cyclopropane and nitrous oxide appear to be the most suitable agents. The administration of halothane is associated with the danger of undue uterine relaxation, although the judicious use of low concentrations may be safe. Spinal and extradural blocks should not be employed in the presence of frank or potential hypovolemic shock because of the hazard of development of severe arterial hypotension.

Shock Due to Impediment to Blood Flow

Impediment to blood flow may occur on the venous or on the arterial side of the circulation.

Supine Hypotensive Syndrome. In late pregnancy, a picture has been observed which resembles shock in many respects. The cardinal features of hypotension, tachycardia, pallor, sweating, faintness, numbness, dyspnea, and nausea, develop gradually while the patient lies on her back, but disappear promptly when she turns on her side.[28] Described as "supine hypotensive" or "postural shock of late pregnancy," the syndrome is attributed to compression of the inferior vena cava by the enlarged noncontracted uterus. A rise in venous pressure caudad and a decrease in venous return to the heart lead to a reduction in cardiac output and a fall in arterial pressure. During contractions, the uterus is lifted off the great veins and does not exert any pressure posteriorly. Hence, the syndrome is more apt to occur in early labor and in elective cesarean section, and less often when uterine contractions are active.[28] An incidence of 11.2 per cent was observed in 160 consecutive obstetric patients who lay supine on admission for 3–7 minutes [29]; however, less than one per cent of 2,000 women approaching cesarean section near, or at term experienced this phenomenon.[30] Conduction anesthesia may enhance the development of the syndrome as suggested by two studies of vaginal delivery under spinal sacral or peridural block.[31, 32] In 100 consecutive deliveries under peridural anesthesia, eight patients developed hypotension to a level below 80 mm. of mercury which responded either to displacement of the uterus or change to a lateral position.[32] This suggested that sympathetic paralysis and pooling of blood in the splanchnic area may exaggerate the vena cava obstruction.[32]

Therapy is directed toward relieving the pressure on the inferior vena cava. This may be accomplished by manually lifting the uterus anteriorly and to the left or by turning the patient to a semilateral or lateral position. Cesarean section may be performed after elevation and support of the right hip to tilt the pelvis and uterus to the left side.[30]

Recognition of this syndrome precludes unnecessary treatment in the form of needless blood transfusion or emergency surgical intervention.[28]

Amniotic Fluid Infusion. Amniotic fluid infusion is a catastrophic situation encountered during labor, delivery, or immediately postpartum. Uterine trauma owing to precipitous or tumultuous labor or difficult delivery of a large infant is the common denominator in most cases.[33] The complication is caused by sudden entry of amniotic fluid into the maternal circulation via either the sinusoids at the uteroplacental site or the endocervical veins.[24] Following rupture of the membranes, amniotic fluid may dissect between the membranes and the uterine wall and enter the venous sinusoids [10]; or, the high intrauterine pressure associated with tetanic contractions may drive the fluid into the endocervical veins and those of the lower uterine segment.[31] In rupture of the uterus, cesarean section, placenta previa, or abruptio placentae, the amniotic fluid has direct access to the maternal blood stream.[10] The essential pathologic finding is pulmonary embolization associated with vasospasm distal to the occlusion; [24, 35] the circulation to kidneys, brain, and other vital organs may also be obstructed.[33] The embolic

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material consists of a mucin-like substance containing squamous cells, meconium, lanugo hairs, and polymorphonuclear leucocytes.[24, 35] The cardinal signs are respiratory distress, cyanosis, cardiovascular collapse, and coma. If the initial crisis is survived, hypofibrinogenemia with profuse uncontrollable hemorrhage may develop.

In one series of 234 obstetric deaths over a seven-year period, amniotic fluid infusion ranked fifth as the cause of fatality, an incidence of 6.4 per cent.[10] In other surveys, the incidence was reported as high as four in 21 deaths (19.5 per cent)[25] or as low as 12 in 959 deaths (1.3 per cent).[1]

The following therapeutic measures have been recommended: oxygen administration under positive pressure, preferably via an endotracheal tube: blood replacement, adequate but not excessive: fibrinogen, when hypofibrinogenemia is present: if vasopressors are necessary, those without pulmonary vasoconstricting properties such as mephentermine or ephedrine should be used: bronchodilators, antihistaminics, steroids, and digitalis may also be helpful.[21]

The prognosis is generally poor, yet not hopeless. Survivals have been reported,[33, 34] including one parturient who recovered from amniotic fluid infusion associated with hypofibrinogenemia and acute renal failure.[37] After-effects may then develop. Obliteration of the small vessels in the lungs may cause difficulty in the circulation through the pulmonary vessels and lead to pulmonary arterial hypertension and right ventricular hypertrophy. Such cases may masquerade as primary pulmonary hypertension.[35]

Pulmonary Embolism. Pulmonary embolism usually occurs later in the puerperium and only rarely shortly after labor. The complication carries an immediate 65–70 per cent mortality [38, 39] and has been observed to be three times as frequent after operative than normal delivery.[40] Many fatalities appear to be not so much due to the mechanical occlusion of pulmonary arteries by thrombi detached from uterine, pelvic, or leg veins, but to an associated reflex sympathetic vasospasm. Leriche and his co-workers found, in 30 per cent of fatal cases, that there was insufficient pathologic change to explain the death of the patient. They postulated that a stellate ganglion block could have been life-saving.[41]

The clinical picture is marked by sudden and intense dyspnea, cyanosis, and cough productive of bloody sputum. Initial substernal pain is followed by pain confined to one side of the chest, aggravated by respiration. Ventilatory excursion may be diminished. Shock may ensue rapidly with a large embolus, or more slowly with repeated small emboli. The usual finding on roentgenographic examination is a poorly defined, homogenous shadow indistinguishable from the consolidation of pneumonia; the characteristic wedge-shaped outline is rarely found. The electrocardiographic changes are those resulting from strain and dilation of the chambers of the right side of the heart.[42]

Prophylaxis plays an important role since circulatory stasis favors the production of thrombosis. Early ambulation and conservative obstetrics with prevention of infection have led to a decrease in the incidence of pulmonary embolism.[40] A course of anticoagulant therapy is indicated when venous thrombosis is recognized.[42]

Treatment consists of the immediate administration of high concentrations of oxygen, general supportive measures, the injection of morphine for pain relief, and the performance of a stellate ganglion block for vasodilation.[41] Where facilities are available, pulmonary embolectomy should be attempted in the moribund patient.

Air Embolism. Air embolism is a rare cause of shock and sudden death during labor or delivery. The complication has occurred during obstetric operations, especially for placenta previa,[2] or as result of transfusion of blood under pressure.[43]

The signs and symptoms of venous air embolism are caused by mechanical obstruction to the outflow tract of the right ventricle. A loud churning "mill-wheel" murmur is heard almost immediately after the air has entered the venous circulation. The venous pressure rises to a marked degree, retrograde circulatory failure appears, and evidence of forward circulatory failure follows.[44] The patient presents with chest pain, dyspnea, cough, cyanosis, venous distention, and tachycardia. Shock develops rapidly and cardiac arrest ensues

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within seconds if large amounts of air have entered, and if treatment is delayed.[43]

The patient must immediately be placed in the left lateral position with the head lowered so as to release the air block and to prevent air from entering the cerebral circulation. If these measures fail, cardiac compression must be instituted while ventilation is maintained.[44] Following cardiac massage a definitive diagnosis of air embolism may not be possible even if, on autopsy, the heart is opened beneath a water seal. It must be recognized that the cardiac chambers can be experimentally emptied of air by massage, and that the absence of crepitation during cardiac massage is not a reliable means of disproving the presence of air in the cardiac chambers.[48]

Cardiac Failure

Severe cardiac failure in pregnancy and the puerperium is manifested commonly by acute, fulminating pulmonary edema which is frequently of an intractable nature.[46] This complication has been observed in two distinct groups of patients: in rheumatic heart disease and in toxemia of pregnancy. The physiologic alterations of pregnancy, as manifested by the retention of sodium and water, the hypoproteinemia, and the increases in blood volume, heart rate and cardiac output, tend to favor the development of heart failure and pulmonary edema.[47] In rheumatic heart disease, pulmonary edema is preceded by a sudden intensification of pulmonary congestion and a rise in pulmonary capillary pressure.[46] In toxemia of pregnancy, the left heart is strained by the arterial hypertension,[48] the increase in extracellular fluid volume is further accentuated as compared to normal pregnancy, and there is also evidence of increased capillary permeability.[47]

The ability of a patient with heart disease to survive pregnancy, labor, and the puerperium depends upon: (1) the capacity of the heart for work and (2) the extent of the total demand for work placed upon the heart during pregnancy. The load includes the cardiac work necessary for normal metabolic processes, and the extra burden imposed by activity, emotional stress, intercurrent illness, lack of sleep and other factors. The first principle in managing the combination of heart disease and pregnancy or labor is to remove enough of the ordinary load to compensate for the increased burden of pregnancy and labor.[49]

At term, cardiac output has been found to be about 11 per cent higher than at the beginning of pregnancy.[50] During each contraction of effective labor, an additional rise of approximately 20 per cent occurred and was associated with significant increases in pulse rate, blood pressure, and left ventricular work. Stroke volume, circulation time, and total peripheral resistance were not altered appreciably. However, following spinal sacral block or peridural anesthesia, such changes in the circulation did not occur. This suggests that the parturient's response to pain and anxiety and voluntary muscle activity play a more important role in raising the cardiac output than the addition to the systemic circulation of approximately 500 ml. of blood from the uterine sinuses.[51] It follows that continuous conduction anesthesia via the lumbar or caudal route is the method of choice for the parturient with potential cardiac failure, for comfort and analgesia and to prevent bearing down efforts. Immediately following delivery, cardiac output was observed to rise about 20 per cent and to remain elevated during the first one or two weeks postpartum. This increase, in contrast to that accompanying uterine contractions during labor, was accomplished by an increase in stroke volume and a decline in heart rate: thus, it appeared to be caused primarily by an augmentation of venous return.[51] Perhaps for this reason, conduction anesthesia should not be discontinued immediately following parturition but should be tapered off gradually over a day or two. Continuous monitoring of venous pressure during labor, delivery, and the first postpartum days may be helpful in detecting the onset of congestive heart failure.

Acute pulmonary edema is probably the most important cardiac cause of death in pregnancy; hence, its prevention is essential. Respiratory tract infections should be treated promptly. Digitalization should be started as soon as practical. Antihypertensive drugs should be used if indicated. If anemia is present, transfusion of packed cells will increase the red cell mass without increasing total circulating blood volume. Intravenous infusions should be kept to a minimum, prefer-

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ably without excess salt. The use of pitocin should, in general, be limited to the third stage of labor.

The treatment of pulmonary edema is the same as that in the nonpregnant patient. Positive pressure oxygen therapy should be instituted immediately; bubbling oxygen through 95 per cent ethyl alcohol has proved most effective as an antifoaming agent. The patient should be placed in a semi-sitting position. If continuous conduction anesthesia has not been instituted, narcotics and rotating tourniquets are indicated. If regional block is in effect, bloodless phlebotomy can be accomplished by raising the level of sympathetic blockade, judiciously. Digitalis, aminophyllin, and diuretics should be given as needed.

Shock Due to Neurogenic Dysfunction

Neurogenic factors constitute the basis for three vastly different syndromes of circulatory shock occasionally encountered during or after parturition.

Spinal Shock. "Spinal shock" or severe arterial hypotension leading to ultimate circulatory and respiratory failure, is the result of high sympathetic and motor blockade following spinal or peridural anesthesia. Arterial hypotension in the pregnant woman under conduction anesthesia is the result of the same factors which cause the decline in blood pressure in the nonpregnant patient. However, several additional alterations accentuate the cardiovascular response to sympathetic denervation. The neurogenic vasoconstrictor tone which supports arteriolar and venous systems is increased at the end of gestation.[52] The large amount of blood present in the gravid uterus will cause a proportionally greater quantity of blood to be trapped in the peripheral circulation following sympathetic blockade. If the parturient is in early labor and is kept in the dorsal decubitus after anesthetization, onset of the supine hypotensive syndrome may reduce venous return from the lower extremities. If, on the other hand, parturition is imminent and the patient actively bears down with contractions, the increased intra-abdominal pressure may decrease the venous return to the heart.[53]

Spinal shock is further abetted by one or more of the following factors: the use of conduction anesthesia in patients suffering from severe hypovolemia or hemorrhage, the administration of excessive doses of local anesthetic drugs, or the subarachnoid injection of the local anesthetic during a uterine contraction. It has been recognized that, during the last trimester of pregnancy, the dose requirement for both spinal and peridural anesthesia is reduced about one-third below that commonly needed in the nonpregnant patient.[53, 54] Uterine contractions *per se* do not influence cerebrospinal fluid pressure. However, marked fluctuations in cerebrospinal fluid pressure have been observed during myometrial contractions as result of voluntary muscle contractions in response to pain. These fluctuations appear to promote the spread of the local anesthetic solution.[55]

Spinal shock can be treated successfully by supporting the patient's circulation and ventilation until the level of sympathetic blockade has receded. Arterial pressure is raised by the intravenous administration of vasopressors; ephedrine and phenylephrine appear most beneficial. Ephedrine possesses both cardiac stimulatory and peripheral vasoconstricting properties, the latter including venoconstriction. Phenylephrine infusion is advantageous because of controllable titration. The administration of 100 per cent oxygen via a tight-fitting mask, or preferably an endotracheal tube, is mandatory. In the event that endotracheal intubation is not performed, gastric dilatation must be watched for and treated by insertion of a nasogastric tube.

In a study of the factors responsible for the decline of maternal mortality in Bronx County, New York,[5] the greatest improvement in regard to anesthesia was noted in the management of spinal anesthesia. Spinal shock accounted for 9 maternal deaths in 10,000 live births in the period 1946–1951, but no death from this cause occurred after 1952. This was attributed to the increased availability of trained anesthetists for obstetric analgesia and the greater interest of the obstetrician in obstetric analgesia.

Inversion of Uterus. Inversion of the uterus is a rare but important cause of postpartum shock. Three factors are necessary for its occurrence: (1) attenuation of the uterine wall, (2) pressure from above or traction on cord or

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placenta, and (3) a patulous cervical canal. The complication may develop spontaneously as the result of intra-abdominal pressure or from the mere weight of the intestines, but in most cases it is attributable to force resulting from the vigorous employment of Credé's maneuver or to traction on the cord.[17]

Inversion of the uterus is followed by vasomotor collapse, the patient presenting evidence of shock out of proportion to blood loss. Severe uterine pain may occur and induce the patient to bear down, which, in turn, increases the degree of inversion.[2] Unexplained shock following delivery should suggest the possibiliyt of inversion and makes imperative immediate vaginal examination, by which means the diagnosis is readily established.[17]

Treatment consists of immediate reposition of the uterus. If the patient has hemorrhaged, manipulation should be postponed until transfusion has been started. Since the procedure is painful, anesthesia should be employed.[17] Cyclopropane appears to be the agent of choice for rapid induction unless relaxation of the lower uterine segment is desired, in which case halothane should be used.

Postpartum Vasomotor Collapse. Patients with toxemia of pregnancy occasionally develop shock within the first 24 hours postpartum. This seems to be related to two factors. The sudden dilation of the splanchnic veins resulting from the abdominal decompression incident to delivery may lead to pooling of blood and insufficient venous return to the heart; this may be accentuated by the lability of blood pressure in toxemic women. In addition, toxemia is associated with a low plasma concentration of sodium and a high concentration of potassium. It has been postulated that the two alterations may act synergistically to lower the blood pressure.[17]

Bacteremic Shock

Shock secondary to overwhelming infection results from the release into the bloodstream of massive doses of bacteria or a bacterial endotoxin which has been identified as a lipoprotein-carbohydrate complex.[3, 56]

The organisms responsible, in two-thirds of the cases, belong to the Gram-negative group.[3] The elimination by antibiotic treatment of the competitive Gram-positive bacteria appears to favor infection and invasion by the relatively nonpathogenic strains, in particular *Escherichia coli, Proteus vulgaris* or *Pseudomonas aeruginosa*.[56] The most common source of infection is the uterus as a consequence of contaminated delivery, prolonged or traumatic labor, or premature rupture of the membranes with chorioamnionitis and placentitis.[3, 56, 57] Urinary tract infection, a not infrequent complication of pregnancy, also may provide the portal for bacterial invasion.[56] Infection is usually accompanied by anemia, hemorrhage, and tissue destruction predisposing to the development of bacteremic shock.[3]

The main target organs of bacterial endotoxin are the coagulation system and the vascular bed.[56] The pathophysiology is not fully understood. Studies in animals have shown that a massive dose of endotoxin results in immediate death. Smaller doses cause disseminated intravascular coagulation and formation of renal thrombi. In man, thrombi have also been observed in the lungs, liver, spleen, adrenals, and intestines.[58] The intravascular clotting process leads to a gradual decrease of circulating fibrinogen, progressive reduction of factors V and VII, and the rapid disappearance of platelets.[59] Coagulation time, *in vivo,* is decreased by approximately 50 per cent.[57] In nonpregnant animals, the Shwartzman reaction is induced by injection of two doses of endotoxin one day apart, the first being a sensitizing dose. In pregnant animals, however, a single dose of toxin produces a similar reaction.[56, 58] This has been attributed to an increase in the circulating level of corticosteroids during gestation.[56, 58] The most prominent vascular change is a generalized vasodilation leading to arterial hypotension and subsequent reduction in cardiac output. Venous pressure is highest in the portal vein.[55] Intermittent local vasoconstriction also occurs and is thought to be caused by potentiation by endotoxin of the action of endogenous catecholamines.[58]

The clinical picture begins with a chill or spiking fever, usually 12 to 48 hours after delivery.[3, 57] Tachycardia frequently develops. The initial symptoms may subside only to be followed by episodes of chills and fever, and eventually by circulatory collapse which leads rapidly to metabolic and renal deterioration, and finally coma. Additional findings include

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leucocytosis, an increase in hematocrit secondary to hemoconcentration, azotemia, and lowered values for sodium, potassium, and chlorides.[3]

The death rate is high. Survival is dependent upon the inherent reserves of the patient and the speed and intensity of treatment.[3] Since bacteremia precedes the elaboration of toxin, antibiotic therapy in the form of chloramphenicol, kanamycin, and broad spectrum antibiotics is warranted.[56, 60] Blood pressure is maintained at levels adequate for the perfusion of vital organs. Blood loss is replaced and, if a coagulation defect exists, the regimen outlined in the section on hemorrhage is followed. Corticosteroids in doses larger than 300 mg. daily are beneficial, not because adrenocortical insufficiency exists, but because the systemic reaction to endotoxin is suppressed. In addition, corticosteroids may increase cardiac output.[60] Hypothermia has provided favorable results by lowering the oxygen requirements of tissues, thus placing the cells in a better metabolic environment.[61] If improvement is not noted within 6–10 hours, curettage or hysterectomy may be required.[58]

In anesthetic management, cardiovascular collapse, renal failure, and fever are the major considerations. Attempts should be made to lower the body temperature by external means before the administration of anesthesia. Cyclopropane is the agent of choice. Nitrous oxide supplemented by low concentrations of halothane or by minimal amounts of succinylcholine, the only muscle relaxant which is hydrolyzed almost completely. It must, however, be recognized that diminished urinary excretion may lead to the accumulation in the blood of the first breakdown product, succinylmonocholine, which, in turn, tends to slow down the hydrolysis of the parent compound.

Shock Due to Hypersensitivity Reactions

Anaphylactic shock is a reaction that occurs in a previously sensitized individual during or shortly after the injection of a foreign serum or a drug. The patient complains of anxiety and impending death, exhibits restlessness, apprehension, coughing or "asthmatic" breathing, and sinks rapidly into profound peripheral cardiovascular collapse. In acute anaphylactic death in man, changes are noted predominantly in the lungs; extensive focal emphysema and exudation of protein-rich serum in the alveoli are characteristic. Other pathologic findings include dilatation of the right ventricle, and multiple petechial hemorrhages in the heart, lungs, kidneys and adrenals.[62]

Data concerning the incidence of hypersensitivity reactions in pregnancy are few. However, the drugs most frequently implicated in anaphylactic reactions, such as penicillin or procaine, are commonly used in pregnant women. The possibility of this type of shock must, therefore, be kept in mind.

The majority of patients affected by hypersensitivity reactions die so rapidly as to preclude treatment. In the remainder, oxygen therapy, vasopressors, and corticosteroids are most useful. If bronchospasm is present, epinephrine by injection or inhalation is indicated.

Conclusions

This review has indicated the type of assistance which the anesthesiologist is able to provide in cases of obstetric shock. Aside from administering the optimal anesthetic for a specific problem, he is able to recognize and treat emergencies as they arise. This is of importance in the immediate survival of the parturient. It is hoped that close cooperation between obstetrician and anesthesiologist will lead to a further reduction in the incidence and greater improvement in the prognosis of obstetric shock.

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The woman, in an early maternity dress (sixteenth century), eases her burden of multiple embryos with a wooden hoop.

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