Acute Myocardial Infarction Associated with Pregnancy

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Purpose: To review available information on the epidemiology, cause, diagnosis, prognosis, and treatment of acute myocardial infarction during pregnancy or in the early postpartum period and to develop guidelines for the management of this condition.

Data Sources: MEDLINE and Index Medicus searches and a manual search of bibliographies from reviewed articles.

Study Selection: Published reports of well-documented acute myocardial infarction during pregnancy or the early postpartum period or potentially relevant information.

Data Extraction: 125 well-documented cases of myocardial infarction were identified.

Data Synthesis: The highest incidence seems to occur in the third trimester and in multigravidas older than 33 years of age. Acute myocardial infarction during pregnancy is most commonly located in the anterior wall. The maternal death rate was 21%; death occurred most often at the time of acute myocardial infarction or within 2 weeks of the infarction and was usually related to labor and delivery. Most fetal deaths were associated with maternal deaths. Coronary artery morphology was studied in 54% of described patients. Coronary atherosclerosis with or without intracoronary thrombus was found in 43% of patients, coronary thrombus without atherosclerotic disease in 21%, coronary dissection in 16%, and normal coronary arteries in 29%.

Conclusions: Acute myocardial infarction during pregnancy or the early postpartum period is rare but may be associated with high risk. Although atherosclerosis can be documented in many cases, coronary dissection and arteries that are normal on angiography are common, especially in acute myocardial infarction occurring in the peripartum or postpartum period. Early diagnosis is often hindered by the normal changes of pregnancy and low level of suspicion. Management should follow the usual principles of care for acute myocardial infarction. However, selection of diagnostic and therapeutic approaches may be greatly influenced by fetal safety.

Acute myocardial infarction rarely occurs in women of childbearing age and has been estimated to occur in only 1 in 10 000 women during pregnancy [1, 2]. Since the first report in 1922 [3], many additional cases have been published in the literature [4-109], indicating the unique features of acute myocardial infarction that can significantly affect maternal and fetal outcomes. With the current trend of childbearing at an older age and the ongoing effects of cigarette smoking, stress, and cocaine use, the occurrence of acute myocardial infarction during pregnancy can be expected to increase. We review the clinical aspects of acute myocardial infarction in the gestational and early postpartum period in 125 well-documented cases and attempt to establish recommendations for the management of this condition.

Methods

A literature search for acute myocardial infarction during pregnancy was done using MEDLINE and Index Medicus. All original articles were obtained from the University of Southern California library, interlibrary communications, or the authors of the articles. Translators were used to translate all original articles written in foreign languages. Only cases of acute myocardial infarction that were documented by chest pain, standard electrocardiographic criteria, and enzymatic changes (or histologic changes in patients who died) were selected for review. Six cases that were described as acute myocardial infarction but did not fulfill the aforementioned criteria were excluded from the analysis.

Epidemiologic data were used to compare selected patients who had acute myocardial infarction in the antepartum (as many as 24 hours before labor), peripartum (within 24 hours before or after delivery), and postpartum (from 24 hours to 3 months after delivery) periods. We make recommendations on the basis of available information, with the understanding that the 125 cases...
identified in our review may not represent all patients who have acute myocardial infarction associated with pregnancy and that reporting may be biased in favor of unusual cause, presentation course, and outcome.

Epidemiology

Although acute myocardial infarction has occurred in pregnant women at all stages of pregnancy and at ages between 16 and 45 years, this event usually occurs in the third trimester in women older than 33 years (Table 1). Patients who had acute myocardial infarction in the antepartum (mean age ±SD, 33 ± 6 years) or peripartum (mean age, 34 ± 5 years) period. In addition, acute myocardial infarction associated with pregnancy has been noted to occur most commonly in multigravidas and is most commonly located in the anterior wall. Most maternal deaths occurred either at the time of infarction (usually resulting in an undelivered child) or within 2 weeks of infarction [1] (usually in association with labor and delivery). No association could be found, however, between maternal death and method of delivery (Table 2). The cumulative reported maternal mortality rate was 21%; rates in the peripartum or postpartum period were higher than those in the antepartum period. Most fetal deaths (10 of 16 [62%]) were associated with maternal deaths, and maternal survival was usually accompanied by normal fetal outcome. The remaining fetal deaths resulted from spontaneous abortion or unexplained stillbirth. Overall, fetal mortality (13%) was lower than maternal mortality.

When coronary artery morphology was studied (through use of angiography or at autopsy) during pregnancy or shortly thereafter (54% of reported patients), coronary atherosclerosis with or without intracoronary thrombus was found in 43% of patients (29 of 68) and definite or probable coronary thrombus without evidence of atherosclerotic disease was present in 21% of patients (14 of 68). Atherosclerotic disease was found more commonly in women with acute myocardial infarction in the antepartum period (58%) than in those with acute myocardial infarction in the peripartum (12%) or postpartum (29%) period. Coronary dissection was found in 16% of patients (11 of 68) and was the primary cause of infarction in the postpartum period (33%). Normal coronary arteries were reported in 29% (20 of 68) of all cases and 75% of those with acute myocardial infarction in the peripartum period.

Causes

Risk factors for myocardial infarction in young women generally include a family history of coronary artery disease; familial hyperlipoproteinemia; low levels of high-density lipoprotein cholesterol, high levels of low-density lipoprotein cholesterol, or both; diabetes mellitus; cigarette smoking; and previous use of oral contraceptives [110]. Although it seems to be the primary cause of acute myocardial infarction during pregnancy, atherosclerotic disease was found in less than half of patients in whom coronary anatomy was investigated (Table 1). Other potential causes include thrombosis, coronary artery spasm (either spontaneous [37] or induced by bromocriptine mesylate [7, 17]), coronary artery dissection [15, 31, 71], collagen vascular disease [16, 41], Kawasaki disease [18], cocaine use [24], aortic valvular stenosis, aortic prosthetic valve thrombosis [23], sickle cell chronic lung disease [65], pheochromocytoma [93], and fibrosis of a coronary ostium secondary to repeated trauma by a papillary fibroelastoma with a long stalk that was strategically located in front of the ostium [100]. Despite the many potential causes, their rarity during childbearing age may explain the low rate of acute myocardial infarction during pregnancy.

Twenty-nine percent of patients in whom coronary artery anatomy was defined were found to have normal coronary arteries (Table 1). Because a thrombus was found without atherosclerotic disease in 21% of the patients, a transient coronary spasm resulting in acute coronary thrombosis as a result of the hypercoagulable state of pregnancy is a possible explanation. Failure to identify similar cases of thrombosis in other patients may be related to doing angiography in late stages of pregnancy. Because acute myocardial infarction has been related to pregnancy-induced hypertension and preeclampsia, enhanced vascular reactivity to angiotensin II [111] and norepinephrine [112] and endothelial dysfunction [113] (which have been reported in these conditions)
may also promote coronary constriction. Other suggested causes of coronary spasm are 1) decreased uterine perfusion in the supine position, leading to renin release and angiotensin production [32] and 2) ergot derivatives that are used to control postpartum [33] or postabortion [21] hemorrhage or to suppress lactation [7, 13, 14].

The profound alterations in the coagulation and fibrinolytic system that occur during pregnancy increase the risk for thrombosis. These alterations include decreased releasable tissue plasminogen activator (t-PA) [114-116], increased fast-acting t-PA inhibitor [116, 117], change in the level of coagulation factors [115, 118], and reduction in functional protein S levels [26, 119, 120]. Cigarette smoking during pregnancy further increases risk for thrombosis because of enhanced platelet aggregability [121]. Hypercoagulation is further augmented at the time of separation of the placenta, which is a major source of t-PA inhibitor [119].

Coronary arterial dissections related to pregnancy have been reported to occur in the antepartum and postpartum periods, but most cases seem to occur in the immediate postpartum period Table 1 [122, 123]. It has been suggested [15] that angiographically normal coronary arteries seen in many patients who had acute myocardial infarction in the peripartum period may represent healed or spontaneously repaired coronary dissections. The cause for dissection may be hormonally mediated biochemical and histologic changes that have been reported to occur in arterial walls during gestation, such as loss of normal corrugation in elastic fibers, fragmentation of reticular fibers, and decrease in the content of acid mucopolysaccharide [124, 125].

The marked increases in blood volume, stroke volume, and heart rate that are usually seen during pregnancy [126] can increase myocardial oxygen demand. At the same time, the physiologic anemia and decreased diastolic blood pressure that occur during gestation may reduce myocardial oxygen supply and contribute to the development of myocardial ischemia when coronary blood supply is compromised. Anxiety, pain, and uterine contraction may further augment these ischemic events during labor and delivery and may be associated with as much as a threefold increase in oxygen consumption. In the puerperium, increased hemodynamic load may be further increased by enhanced return of venous blood to the heart with relief of caval compression and shift of blood from the contracting emptied uterus into the systemic circulation. The hemodynamic changes that normally occur during late pregnancy and during labor, delivery, and the puerperium [126] may contribute to poor outcome in women who have an acute myocardial infarction in the peripartum period.

### Diagnosis and Clinical Considerations

Early diagnosis of acute myocardial infarction during pregnancy is often hindered by mistaking its signs and symptoms for normal manifestations of pregnancy [127] and because of a low level of suspicion. Diagnosis in pregnant women is confirmed as it is in nonpregnant patients, primarily by electrocardiographic and changes in enzyme levels. Therefore, it is important to note that electrocardiographic changes that mimic myocardial ischemia have been reported in as many as 37% of parturients having elective cesarean section [128, 129]. Echocardiography can be done safely during pregnancy to confirm the presence of ischemia by showing wall motion abnormalities that correspond to electrocardiographic changes. Although myocardial ischemia is often associated with cardiac arrhythmias [8], this association is less helpful to diagnosis in pregnant women when various arrhythmias (especially those characterized by multiple atrial and ventricular premature beats but also supraventricular and ventricular tachycardia) occur in women with normal hearts [130, 131].

The use of radiation during pregnancy should generally be kept to a minimum. The amount of fetal exposure to radiation during chest radiography is extremely small and should probably be considered safe and used when appropriate [132]. Radionuclide ventriculography using technetium 99m and myocardial perfusion scanning using thallium-201 or technetium 99m sestamibi is expected to yield less than 0.01 Gy to the conceptus but should be used during pregnancy only when absolutely necessary. Cardiac catheterization and interventional procedures may also result in fetal exposure to less than 0.01 Gy. However, difficult procedures requiring longer fluoroscopy time and several cine views could easily yield a fetal radiation exposure of 0.05 to 0.10 Gy. Termination of pregnancy is not generally recommended for fetal doses of radiation less than 0.05 Gy but may be considered when the dose exceeds that amount.

### Treatment

Although the management of acute myocardial infarction and its complications should follow the usual principles of care, fetal considerations may affect the choice of therapy. Close consultation among the attending obstetrician, internist or cardiologist, and anesthesiologist is essential to optimize maternal and fetal well-being. Ideally, the patient should be treated in an intensive care unit that is capable of providing maternal and fetal monitoring along with a comprehensive obstetric service. A plan for prompt rescue of a potentially viable fetus in the case of sudden maternal deterioration should be established.
Morphine Sulfate

This drug has not caused congenital defects, but because it crosses the placenta, it may cause neonatal respiratory depression when given shortly before delivery [133]. Morphine enters breast milk only in trace amounts and is considered compatible with breast-feeding [134].

Thrombolytic Therapy

Thrombolytic therapy is a first-line treatment of acute myocardial infarction but has had limited use in pregnancy [135-148]. In fact, pregnancy currently constitutes a contraindication for thrombolytic treatment [86, 94], although the basis for such prohibition is strictly theoretical and has probably been adopted from research protocols in which pregnancy was an exclusion criterion. Streptokinase and t-PA do not cross the placenta in animals. However, no information is available on the passage of streptokinase in the human placenta during early pregnancy; streptokinase has not been shown to cross the placenta in late pregnancy and has only minimal placental passage during labor [135, 139]. On the other hand, streptokinase antibodies were detected in neonatal spinal cord blood after women received streptokinase several weeks before delivery [137]. Urokinase was not found to be teratogenic in rats or mice [149]. Clinical experience with use of thrombolytic therapy during pregnancy has been mostly with streptokinase, and this experience has been reported primarily in patients with massive and hemodynamically significant pulmonary embolism [136, 142, 145, 146], deep venous thrombosis [137], or prosthetic valve thrombosis [138, 143, 144, 148, 150]. Because thrombolytic therapy has played a pivotal role in modern treatment of acute myocardial infarction, the reported experience with this therapy during pregnancy is shown in Table 3.

Available reports describe the use of therapeutic doses of streptokinase [137, 141-144], t-PA [145-147], and urokinase [42, 136, 138, 145] between the 9th and 38th week of gestation [147]. Delivery occurred during thrombolytic therapy or 1 to 2 days after therapy was stopped in several cases [141, 142, 145-147]. Although maternal and fetal outcomes were normal in some reports, documented complications include maternal hemorrhage, preterm delivery, and fetal loss [135, 137, 141, 142]. Hemorrhagic complications included spontaneous abortion, minor vaginal bleeding that did not require intervention [135, 138], spontaneous hematoma in the inguinal and axillary region that required blood transfusion [135], fatal abruptio placentae with fetal death [137], uterine bleeding that required emergency cesarean section [137, 148], and postpartum hemorrhage that required transfusion [141, 142]. The risk for hemorrhagic complications seems to increase when thrombolytic therapy is given at the time of delivery. Fetal hemorrhage or teratogenic effects have not been reported, and fetal outcome has been favorable in most reported cases.

Thrombolytic therapy was used during the first trimester in seven cases [135], and fetal loss occurred in one case. Most reported cases of fetal loss in patients receiving thrombolytic therapy do not seem to be related to treatment. In one case, however, fetal death was caused by abruptio placentae during therapy [137]. In another case, fetal heartbeat stopped during thrombolytic therapy, and a treatment-related cause could not be ruled out [150]. In addition, intracranial hemorrhages were reported in a newborn infant whose mother had received t-PA during pregnancy; the infant died of the acute distress syndrome 14 days after delivery [146].

In summary, the few available reports on the use of thrombolytic therapy during pregnancy do not support a teratogenic effect. Although most reported cases were associated with favorable maternal and fetal outcomes, therapy is associated with a risk for maternal hemorrhage, especially when thrombolytic agents are given at the time of delivery. The occasional fetal loss that was reported did not seem to be related to therapy in most instances, although such a relation could not always be ruled out. More information is needed before fetal safety during thrombolytic therapy can be established.

Anticoagulation Therapy

Because of its large molecular size, heparin does not cross the placenta, is not teratogenic, and is therefore the anticoagulant of choice during pregnancy [151]. Because its effect may persist for as long as 28 hours, discontinuation of therapy 24 hours before elective induction of labor is desirable [151]. Heparin therapy should be discontinued with the onset of spontaneous labor, and judicious use of protamine sulfate may be needed to reduce the risk for bleeding and to allow safe pudendal and epidural anesthesia [151, 152]. Hemostatic stitches should be used to prevent bleeding caused by episiotomy; uterine contraction should be stimulated by massage after delivery; and oxytocin or ergot derivatives should be used to stop bleeding. Heparin therapy can be resumed after delivery as soon as hemostasis seems adequate. Heparin is not secreted in breast milk [134].

Aspirin

The safety of high doses of aspirin during pregnancy is debatable, especially during the third trimester. At this time, high doses may lead to increased maternal and fetal hemorrhage, congenital abnormalities, and premature closure of the ductus arteriosus.
On the other hand, the safety of low dosages of aspirin (150 mg/d), which effectively inhibit thromboxane synthesis, has been shown during the second and third trimester in many hypertensive pregnant women. The safety of aspirin during the first trimester is still unclear. Although aspirin is secreted in breast milk in low concentrations, cautious use of aspirin by lactating women has been recommended and adverse effects have not been reported.

**Organic Nitrates**

Intravenous or oral nitrates have been used during pregnancy to treat hypertension, myocardial ischemia, and infarction and to arrest preterm labor. No known adverse effects of nitrate therapy during pregnancy have been seen; however, careful titration is recommended to avoid maternal hypotension, which can lead to fetal distress. No data are available on breast-feeding in women treated with these drugs.

**ß-Adrenergic Blocking Agents**

Substantial experience with the use of propranolol, atenolol, labetalol, and metoprolol in pregnancy suggests that they are safe for use during gestation, although such side effects as fetal growth retardation, bradycardia, hypoglycemia, hyperbilirubinemia, and apnea at birth have been anecdotally reported. Because nonselective ß-blockers may facilitate an increase in uterine activity, use of ß₁-selective agents may be preferable. All ß-blockers are weak bases and accumulate in greater concentrations in breast milk than in plasma. Nursing infants should therefore be monitored for adverse effects.

**Calcium Antagonists**

Increasing experience with use of nifedipine during gestation for treatment of hypertension, preeclampsia, myocardial ischemia, and tocolysis has shown the safety of this drug. Information about the use of verapamil and diltiazem during pregnancy is more limited. A recent surveillance study suggested that diltiazem may have teratogenic effects. Nifedipine, verapamil, and diltiazem are all considered to be compatible with breast-feeding.

**Magnesium Sulfate**

Although it is still debated whether magnesium sulfate should be used routinely in pregnant patients who have had acute myocardial infarction, early administration of the drug to high-risk patients (especially those who do not receive thrombolytic therapy) has been shown to be useful. Magnesium sulfate is commonly used in pregnancy as a tocolytic agent and as an anticonvulsant agent for toxemia. Wide clinical experience with this drug during pregnancy has not shown teratogenic effects or other forms of toxicity. It should be noted, however, that magnesium sulfate has rarely been used during the first trimester. Newborn infants of mothers treated with magnesium sulfate close to delivery should be observed for signs of respiratory depression, muscle weakness, and loss of reflexes, all of which were reported in a hypertensive woman treated with 11 g of magnesium sulfate within 3.5 hours of delivery. Maternal hypothermia with maternal and fetal bradycardia caused by intravenous magnesium sulfate use has been described. In addition, hypotension and pronounced muscle weakness have been reported when the drug was used in combination with nifedipine. Magnesium sulfate is considered to be compatible with breast-feeding.

**Complications**

**Congestive Heart Failure**

Diuretics should be used cautiously to prevent diuresis of too great an extent, hypovolemia, and subsequent reduction in uteroplacental blood flow. Nitrates are effective, but careful dose titration is recommended to avoid reducing blood pressure. Sodium nitroprusside has been used in some patients during pregnancy, but its safety is unknown. A large dose of sodium nitroprusside resulted in fetal death in animals, showing a potential for fetotoxicity. Dopamine has had only limited use in pregnant humans. Studies in animals have shown both an increase and a decrease in uterine blood flow. The drug has been used to increase renal blood flow in oliguric, eclamptic patients and to treat spinal anesthesia-related hypotension during cesarean section without apparent adverse effects to the fetus or newborn infant. Information about the use of dobutamine during pregnancy is similarly limited. Short-term use of this drug in one patient with myocardial infarction at 18 weeks gestation and in a patient with pulmonary hypertension before delivery was not associated with adverse effects. Angiotensin-converting enzyme inhibitors are contraindicated during pregnancy because of increased incidence of fetal illness and death. Reported complications include oligohydramnios, intrauterine growth retardation, premature labor, fetal and neonatal renal failure, bone malformations, limb contractures, persistent patent ductus arteriosus, pulmonary hypoplasia, the respiratory distress syndrome, prolonged hypotension, and neonatal death. If heart failure persists or the clinical condition of the patient deteriorates, more
aggressive approaches should be pursued. In one case of severe hemodynamic instability, treatment with intra-aortic balloon counterpulsation was successful and allowed postponement of delivery until hemodynamic stability could be achieved [20]. Cardiogenic shock accompanying acute myocardial infarction was reported to improve dramatically after cesarean section [11].

Arrhythmias

Drug Therapy

On the basis of substantial clinical experience, use of digoxin and quinidine during gestation is considered to be safe for the fetus [162]. Procainamide and disopyramide have been successfully used to treat maternal and fetal arrhythmias, but experience with these drugs is limited. Lidocaine has been used safely during pregnancy mainly for epidural or local anesthesia; it can also be used as an antiarrhythmic agent as long as blood levels are closely monitored. Elevated levels can cause apnea, hypotonia, dilated pupils, seizures, and bradycardia in infants [162]. Relatively few pregnant women have been treated with newer antiarrhythmic agents, such as mexiletine [173], flecainide [174], propafenone, and sotalol [175]. Because information is limited, the safety of these drugs is unknown. Use of amiodarone during pregnancy has been associated with significant side effects, including hypothyroidism and congenital malformations in the newborn infant [176]. The drug should therefore be used only in refractory cases of maternal tachyarrhythmias. A recent survey identified 34 women who received intravenous adenosine during pregnancy to treat maternal supraventricular arrhythmia [177] and showed the efficacy and safety of adenosine use during pregnancy.

Cardiac Pacing

Indications for cardiac pacing in pregnant patients who have had acute myocardial infarction are similar to those used for nonpregnant patients. On the basis of reported experience in many pregnant patients, no problems are anticipated with the use of temporary pacemakers [178]. These devices, however, should be inserted without fluoroscopy when possible. The recent introduction of external transcutaneous pacing [179] provides clinicians with an ideal alternative for cardiac pacing during pregnancy.

Direct-Current Cardioversion

Cardioversion is recommended for treatment of maternal tachyarrhythmias associated with systemic hypotension or heart failure. Several investigators [1, 4-68, 34] have documented the safety and efficacy of cardioversion and defibrillation in the setting of myocardial infarction during pregnancy. If possible, monitoring of fetal heart rate is advisable whenever maternal cardioversion is done.

Postinfarction Angina

If postinfarction angina pectoris is poorly controlled with maximal medical therapy in the early stages of pregnancy (20 weeks), consideration should be given to termination of the pregnancy. The approach to this condition after the 20th week should be similar to that in nonpregnant patients. Cardiac catheterization and angiography, if indicated, should be done with fetal shielding using the brachial or radial approach to reduce scattered radiation to the fetus. Although experience with percutaneous transluminal coronary angioplasty or coronary artery bypass grafting is limited, these procedures have been done successfully during pregnancy or the early postpartum period [12, 17, 25, 180]. However, these forms of therapy should be avoided in pregnancy if possible, especially during the first trimester, because of the potential deleterious effects on the fetus of both radiation and cardiopulmonary bypass [181].

Cardiopulmonary Resuscitation

Before the onset of fetal viability (at about the 24th week of gestation), the objectives of cardiopulmonary resuscitation can be guided almost exclusively by maternal considerations; later, consideration should also be given to fetal safety. Success of cardiopulmonary resuscitation in pregnancy may be hampered by several factors. The thorax is less compressible to external pressure because of cephalad displacement of abdominal contents. Chest compressions in the supine position may fail to produce sufficient cardiac output because of reduced venous return and increased obstruction to arterial forward flow. Because of the elevated diaphragm, resistance to airflow during artificial respiration and to thoracic compressions is increased, making resuscitation efforts even more difficult [182].

Because of increased oxygen consumption and increased carbon dioxide and hydrogen ion production by fetoplacental metabolism, any delay in establishing effective ventilation magnifies circulatory compromise of the uteroplacental unit and may inhibit resuscitation efforts despite adequate chest compression [183]. To minimize the effects of the gravid uterus on venous return and cardiac output, a wedge (such as a pillow) should be placed under the flank of the right abdomen and hip to displace the uterus to the left side. If having the mother in the left lateral decubitus position makes resuscitative efforts clumsy and ineffective, continuous manual displacement to the left or positioning the back of the resuscitated patient on the thighs of a person kneeling on the floor and sitting back on their heels (the so-called human wedge maneuver) [184] may be used as alternative methods. To date, no studies comparing closed- and open-chest cardiac massage on the hemodynamics of the human uterus and
placenta have been done. The uteroplacental circulation, however, offers minimal vascular resistance as long as oxygenation and acid-base balance are not profoundly distorted. Favorable outcomes reported in some cases [185] suggest that the pressure gradients generated even by standard cardiopulmonary resuscitation may be adequate to sustain fetal life and should be attempted. At the same time, early evacuation of the uterus by bedside cesarean section resulted in recovery of blood pressure in a patient who did not generate adequate blood pressure despite vigorous resuscitative measures. Lee and colleagues [186] recommended thoracotomy and open-chest cardiac massage between the 24th and 32nd week of gestation. Cesarean section after the 32nd week should be considered if standard cardiopulmonary resuscitation is ineffective.

Survival of the infant has been directly proportional to the time interval between the death of the mother and delivery [187]. Delivery taking place more than 15 minutes after maternal death rarely produces a viable infant, and almost all surviving infants had some neurologic sequelae. On the other hand, all surviving infants delivered within 5 minutes after maternal death were healthy. Successful rescue and long-term maintenance of brain-dead and comatose mothers have been accomplished, allowing for delivery at a time that was more beneficial to the fetus [187, 188]. To maximize the chances of survival for both the mother and infant [183-190], rapid cesarean section (within 4 to 5 minutes of cardiac arrest) has been recommended.

Several issues about the use of drugs that are commonly administered during resuscitation should be addressed. Routine administration of bicarbonate has been discouraged in nonpregnant patients during resuscitation because combined respiratory and metabolic alkalosis caused by vigorous ventilation and bicarbonate administration may be deleterious. During resuscitation of a pregnant woman, however, the rate of acid metabolite production increases. Because acidosis increases the -adrenergic reactivity of the uteroplacental vasculature, use of bicarbonate during resuscitation of a pregnant patient seems logical. Although ß-agonists or combined ß- and -agonists do not cause any hemodynamic derangement in the uteroplacental blood flow during normal pregnancy, hypoxemia or hypotension enhances sensitivity to the vasoconstrictor action of epinephrine and norepinephrine [191] and thus may further impair uteroplacental flow. For this reason, reduced uteroplacental blood flow due to hypotension may not improve with vasopressors.

**Labor**

To allow adequate healing of the infarction, delivery should be postponed, if possible, for 2 to 3 weeks after acute myocardial infarction. Because of the increased hemodynamic requirements during labor and uterine contractions [126], myocardial ischemia and cardiac decompensation may occur. In addition, reduced oncotic pressure may predispose patients to the development of pulmonary edema.

The mode of delivery in a pregnant patient with gestational myocardial infarction should be determined by obstetric reasons and the clinical status of the mother. Both vaginal and cesarean deliveries have advantages and disadvantages. Review of the outcomes for all 125 cases in this paper supports the conclusion reached by Cohen and colleagues [4], who found no convincing support for one method of delivery over the other, and suggests that an individual approach be used. Advantages of elective cesarean section include control of the time of delivery and avoidance of long or stressful labor. Vaginal delivery, on the other hand, eliminates the risks associated with anesthesia and a major surgical procedure and avoids potential postoperative morbid conditions (including hemodynamic fluctuations, blood loss, pain, infection, and respiratory complications) [2, 30]. With measures aimed to reduce cardiac workload and oxygen demands, vaginal delivery can be accomplished relatively safely [5, 6, 10, 40, 192]. Instrumental delivery is recommended to avoid excessive maternal efforts, and the patient should receive supplemental oxygen. For optimization of cardiac output, a left lateral position is preferred. Hemodynamic values, as well as oxygen saturation, electrocardiogram, and fetal heart rate, should be monitored continuously. During labor and delivery, the patient's pain, fear, and apprehension, all of which can increase myocardial oxygen demand, must be minimized and controlled. Tachycardia and hypertension should be prevented or, if they occur, promptly corrected. In view of the possibility of coronary spasm, it may be wise to avoid oxytocin infusion during labor and ergonovine in the postpartum period. Ischemia that develops during labor and delivery can be treated by intravenous nitroglycerin, ß-blockers, and calcium antagonists. Nitroglycerin, as well as such calcium antagonists as diltiazem and verapamil, have shown some tocolytic effect and may prolong labor [159, 167].

In summary, selection of the method of delivery for patients after myocardial infarction should be made on an individual basis. Most patients with coronary artery disease can tolerate vaginal delivery and should therefore have this type of delivery. Cesarean section is only indicated for obstetric purposes and in patients with unstable ischemic or hemodynamic conditions.

**Hospital Discharge**
The timing of hospital discharge after acute myocardial infarction needs to be determined on an individual basis. If the pregnancy has advanced into the third trimester, then continued hospitalization with restricted exertional activities until elective delivery may be appropriate.

Low-risk patients and patients in earlier stages of pregnancy may be discharged if their clinical condition is stable. Left ventricular function and presence of residual myocardial ischemia can be assessed using stress echocardiographic study. A recent report described such an evaluation done 9 days after infarction at 27 weeks of gestation. Because of the potential for fetal distress during exercise, a submaximal exercise protocol and fetal monitoring during exercise are recommended. In cases in which high-quality echocardiographic evaluation is not possible for technical reasons, thallium exercise testing should be considered.

Because myocardial oxygen consumption will progressively increase with the progression of pregnancy, close follow-up of the pregnant patient who has had acute myocardial infarction is recommended after hospital discharge. Assessment of fetal maturity and reassessment of cardiac status after the 32nd week of gestation are important for determination of management plan and feasibility of early delivery, if indicated.

**Subsequent Pregnancies**

Data on maternal and fetal outcome of pregnancy in patients with a history of myocardial infarction are extremely limited. Reported cases of pregnancy after myocardial infarction have not been associated with death but with an increased incidence of short- and long-term illness. These data must be interpreted with caution because the number of patients was small, complete obstetric and cardiovascular details were not always presented, and the period between infarction and pregnancy varied substantially (range, 6 months to 7 years). The risks associated with subsequent pregnancy probably depend on many factors, including the cumulative amount of myocardial damage, residual left ventricular function, underlying coronary anatomy, and ongoing myocardial ischemia.

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