the risk of cardiac failure with these conditions. As a rule, mitral valve prolapse does not present problems for the pregnant patient, and aortic stenosis, unless very severe, is well tolerated. In the most severe cases of aortic stenosis, limitation of activity or balloon valvuloplasty may be indicated.

CONGENITAL HEART DISEASE

(See also Chap. 282) Reparative surgery has markedly increased the number of women with surgically repaired congenital heart disease. Maternal morbidity and mortality are greater among these women than among those without surgical repairs. When pregnant, these patients should be jointly managed by a cardiologist and an obstetrician familiar with these problems. The presence of a congenital cardiac lesion in the mother increases the risk of congenital cardiac disease in the newborn. Prenatal screening of the fetus for congenital cardiac disease with ultrasound is recommended. Atrial or ventricular septal defect is usually well tolerated during pregnancy in the absence of pulmonary hypertension, provided that the woman's prepregnancy cardiac status is favorable. Use of air filters on IV sets during labor and delivery in patients with intracardiac shunts is recommended.

OTHER CARDIAC DISORDERS

Supraventricular tachycardia (Chap. 276) is a common cardiac complication of pregnancy. Treatment is the same as in the nonpregnant patient, and fetal tolerance of medications such as adenosine and calcium channel blockers is acceptable. When necessary, pharmacologic or electric cardioversion may be performed to improve cardiac performance and reduce symptoms. This intervention is generally well tolerated by mother and fetus.

Peripartum cardiomyopathy (Chap. 287) is an uncommon disorder of pregnancy associated with myocarditis, and its etiology remains unknown. Treatment is directed toward symptomatic relief and improvement of cardiac function. Many patients recover completely; others are left with progressive dilated cardiomyopathy. Recurrence in a subsequent pregnancy has been reported, and women who do not have normal baseline left-ventricular function after an episode of peripartum cardiomyopathy should be counseled to avoid pregnancy.

SPECIFIC HIGH-RISK CARDIAC LESIONS

Marfan Syndrome (See also Chap. 427) This autosomal dominant disease is associated with a high risk of maternal morbidity. Approximately 15% of pregnant women with Marfan syndrome develop a major cardiovascular manifestation during pregnancy, with almost all women surviving. An aortic root diameter <40 mm is associated with a favorable outcome of pregnancy. Prophylactic therapy with beta blockers has been advocated, although large-scale clinical trials in pregnancy have not been performed. *Ehlers-Danlos syndrome* (EDS) may be associated with premature labor, and in type IV EDS there is increased risk of organ or vascular rupture that may cause death.

Pulmonary Hypertension (See also Chap. 304) Maternal mortality in the setting of severe pulmonary hypertension is high, and primary pulmonary hypertension is a contraindication to pregnancy. Termination of pregnancy may be advisable in these circumstances to preserve the life of the mother. In the *Eisenmenger syndrome*, i.e., the combination of pulmonary hypertension with right-to-left shunting due to congenital abnormalities (Chap. 282), maternal and fetal deaths occur frequently. Systemic hypotension may occur after blood loss, prolonged Valsalva maneuver, or regional anesthesia; sudden death secondary to hypotension is a dreaded complication. Management of these patients is challenging, and invasive hemodynamic monitoring during labor and delivery is recommended in severe cases.

In patients with pulmonary hypertension, vaginal delivery is less stressful hemodynamically than cesarean section, which should be reserved for accepted obstetric indications.

DEEP VENOUS THROMBOSIS AND PULMONARY EMBOLISM

(See also Chap. 300) A hypercoagulable state is characteristic of pregnancy, and deep venous thrombosis (DVT) occurs in about 1 in 500 pregnancies. In pregnant women, most unilateral DVTs occur in the left leg because the left iliac vein is compressed by the right iliac artery and the uterus compresses the inferior vena cava. Pregnancy is associated with an increase in procoagulants such as factors V and VII and a decrease in anticoagulant activity, including proteins C and S. Pulmonary embolism is one of the most common causes of maternal death in the United States. Activated protein C resistance caused by the factor V Leiden mutation increases the risk for DVT and pulmonary embolism during pregnancy. Approximately 25% of women with DVT during pregnancy carry the factor V Leiden allele. Additional genetic mutations associated with DVT during pregnancy include the prothrombin G20210A mutation (heterozygotes and homozygotes) and the methylenetetrahydrofolate reductase C677T mutation (homozygotes).

TREATMENT DEEP VENOUS THROMBOSIS

Aggressive diagnosis and management of DVT and suspected pulmonary embolism optimize the outcome for mother and fetus. In general, all diagnostic and therapeutic modalities afforded the nonpregnant patient should be utilized in pregnancy except for D-dimer measurement, in which values are elevated in normal pregnancy. Anticoagulant therapy with low-molecular-weight heparin (LMWH) or unfractionated heparin is indicated in pregnant women with DVT. LMWH may be associated with an increased risk of epidural hematoma in women receiving an epidural anesthetic in labor. Four weeks prior to anticipated delivery, LMWH should be switched to unfractionated heparin. Warfarin therapy is contraindicated in the first trimester due to its association with fetal chondrodysplasia punctata. In the second and third trimesters, warfarin may cause fetal optic atrophy and mental retardation. When DVT occurs in the postpartum period, LMWH therapy for 7–10 days may be followed by warfarin therapy for 3–6 months. Warfarin is not contraindicated in breast-feeding women. For women at moderate or high risk of DVT who have a cesarean delivery, mechanical and/or pharmacologic prophylaxis is warranted.

ENDOCRINE DISORDERS

DIABETES MELLITUS

(See also Chaps. 417–419) In pregnancy, the fetoplacental unit induces major metabolic changes, the purpose of which is to shunt glucose and amino acids to the fetus while the mother uses ketones and triglycerides to fuel her metabolic needs. These metabolic changes are accompanied by maternal insulin resistance caused in part by placental production of steroids, a growth hormone variant, and placental lactogen. Although pregnancy has been referred to as a state of "accelerated starvation," it is better characterized as "accelerated ketosis." In pregnancy, after an overnight fast, plasma glucose is lower by 0.8–1.1 mmol/L (15–20 mg/dL) than in the nonpregnant state. This difference is due to the use of glucose by the fetus. In early pregnancy, fasting may result in circulating glucose concentrations in the range of 2.2 mmol/L (40 mg/dL) and may be associated with symptoms of hypoglycemia. In contrast to the decrease in maternal glucose concentration, plasma hydroxybutyrate and acetoacetate levels rise to two to four times normal after a fast.

TREATMENT DIABETES MELLITUS IN PREGNANCY

Pregnancy complicated by diabetes mellitus is associated with higher maternal and perinatal morbidity and mortality rates. Preconception counseling and treatment are important for the diabetic patient contemplating pregnancy and can reduce the risk of congenital malformations and improve pregnancy outcome. Folate supplementation reduces the incidence of fetal neural tube defects, which occur with greater frequency in fetuses of diabetic mothers. In addition, optimizing glucose control during