Changes in thyroid hormone concentrations that are characteristic of hyperthyroidism must be distinguished from physiological changes in thyroid hormone economy that occur in pregnancy, especially in the first trimester. Approximately one to two cases of gestational hyperthyroidism occur per 1000 pregnancies. Identification of hyperthyroidism in a pregnant woman is important because adverse outcomes can occur in both the mother and the offspring. Graves disease, which is autoimmune in nature, is the usual cause; but hyperthyroidism in pregnancy can be caused by any type of hyperthyroidism—eg, toxic multinodular goitre or solitary autonomously functioning nodule. Gestational transient thyrotoxicosis is typically reported in women with hyperemesis gravidarum, and is mediated by high circulating concentrations of human chorionic gonadotropin. Post-partum thyroiditis occurs in 5-10% of women, and many of those affected ultimately develop permanent hypothyroidism. Antithyroid drug treatment of hyperthyroidism in pregnant women is controversial because the usual drugs—methimazole or carbimazole—are occasionally teratogenic; and the alternative—propylthiouracil—can be hepatotoxic. Fetal hyperthyroidism can be life threatening, and needs to be recognized as soon as possible so that treatment of the fetus with antithyroid drugs via the mother can be initiated. In this Review, we discuss physiological and pathophysiological changes in thyroid hormone economy in pregnancy, the diagnosis and management of hyperthyroidism during pregnancy, severe life-threatening thyrotoxicosis in pregnancy, neonatal thyrotoxicosis, and post-partum hyperthyroidism.