Thyrotoxicosis is associated with increased cardiovascular morbidity and mortality, primarily due to heart failure and thromboembolism. Palpitations, caused by sinus tachycardia and occasionally by atrial fibrillation, are the most frequent cardiovascular symptom. As atrial fibrillation may be the only manifestation of thyrotoxicosis, thyroid hormone excess should routinely be excluded in patients with this rhythm disturbance. Heart failure occurs mostly in the presence of underlying heart disease or tachycardia-induced cardiomyopathy in patients with long-standing atrial fibrillation. On occasion, long-standing hyperthyroidism may lead to heart failure even in the absence of concomitant cardiac conditions. Beta-blockers offer symptomatic relief and at the same time slow the ventricular response in patients with atrial fibrillation. Amiodarone, and occasionally iodinated contrast agents, may cause iodine-induced thyrotoxicosis. Clinical suspicion is essential in the diagnosis of amiodarone-induced thyrotoxicosis (AIT), because the antiadrenergic effect of the drug may conceal symptoms. AIT should be considered in any patient on amiodarone in the presence of new-onset or recurrent atrial arrhythmias or unexplained weight loss. Beyond discontinuation of amiodarone, treatment options include propylthiouracil or methimazole, potassium perchlorate, steroids, lithium and, if pharmacological treatment fails, surgery. Amiodarone may potentially be used less frequently in the future since recent studies have shown that this drug is inferior to implantable cardioverter defibrillators in prevention of sudden cardiac death in patients with severe heart failure. In addition, non-iodinated amiodarone analogues are currently in advanced phase of clinical testing.