Graves' disease was introduced 70 years ago, and it is estimated that more than one million individuals have been treated with ^{131}I for hyperthyroidism (Chapman, 1983). The use of radioactive iodine has been detailed for more than 1200 children (Rivkees et al., 1998). Patients as young as 1 yr of age have been treated with ^{131}I with excellent outcomes (Rivkees et al.1998). Some studies have reported remission rates that exceed 95%, with very rare complications (Levy et al., 1988, Read et al., 2004, Rivkees et al.1998). Properly administered radioactive iodine remains an ideal form of treatment for Graves' disease in the pediatric population and it is an effective cure for Graves' disease which is associated with few acute side effects (Rivkees & Dinauer 2007). When radioiodine is used at appropriate doses, there is a very high cure rate without increased risks of thyroid cancer or genetic damage (Rivkees, 2001). Also data of Moll & Patel (Moll & Patel, 1997) from nearly 8 years experience with ^{131}I therapy support consideration of RAIT within 3 to 6 months of diagnosis of pediatric Graves´ disease as an effective, efficient, and probably safe alternative approach to the traditional antithyroid therapy of Graves´ disease in children.

For 70 years radioidine has been used to treat most cases of Graves´ disease and thyroid autonomies. The thyroid gland utilizes iodine for the synthesis of thyroid hormones. The cells do not differentiate between stable and radioactive iodine. If radioactive iodine is administered, it is trapped and then organified by thyroid follicular cells like nonradioactive iodine. The therapeutic effects of ^{131}I sodium iodide are due to the emission of ionizing radiation from the decaying radionuclide. The therapy is based on short-range beta radiation from radioactive ^{131}I. The beta particles, due to their high mean energy (190 keV, with typical beta-decay spectra present) have a tissue penetration of 0.6 to 2 mm (Skugor, 2006). Radioiodine administration should be preceded by pharmacological normalization of fT4 and fT3 levels, beacause post-radiation thyrocyte destruction and thyroid hormone release can lead to hyperthyroidism exacerbation (Gurgul & Sowinski, 2011). In some cases it is possible to withdraw antithyroid drugs 10-14 days before radioiodine administration to restore appropiate iodine uptake. It is known that one of the cause of reduced ^{131}I uptake and shortened effective half life is pre-treatment with antithyroid drugs (ATD), which may have an additional radio-protective effect. It can influence the outcome of ^{131}I therapy (Sabri et al., 2001, Walter et al., 2002) with possibility a risk of treatment failure for lower delivered radioiodine doses to the target tissue (Dunkelmann et al., 2005). Tuttle et al. describe that antithyroid drug therapy has been associated with relative radioresistance and with a decrease in radioiodine therapy effectiveness (Tuttle et al., 1995). A similar effect as longer discontinuation of ATD (10-14 days) can be obtained when ATD is discontinued 24 hours before ^{131}I administration („bounce effect") (Gurgul & Sowinski, 2011). But in severe hyperthyreoidism it is not possible to withdraw ATD for a long time, that is why discontinuation of ATD starting two or three days before ^{131}I is now widely accepted (Walter et al., 2002). 2-day ATD withdrawal prior to radioiodine administration sufficiently increases the radioiodine uptake and does not exacerbate hyperthyroidism compared to 7-day withdrawal in Graves´ disease patients. There is not significant diference in RAIT outcomes between 2-day and 7-day groups. In order to prevent serum thyroid hormone increase after ATD withdrawal and RAIT, a 2-day ATD withdrawal period before RAIT may be useful for high-risk patients such as the elderly and patients with cardiac complications (Kubota et al., 2006). Pretreatment with propylthiouracil but not methimazole, discontinued even 15 days before radioiodine administration, reduces the rate of success of ^{131}I therapy. Propylthiouracil pretreatment and large goiters are related to failure of radioiodine therapy.