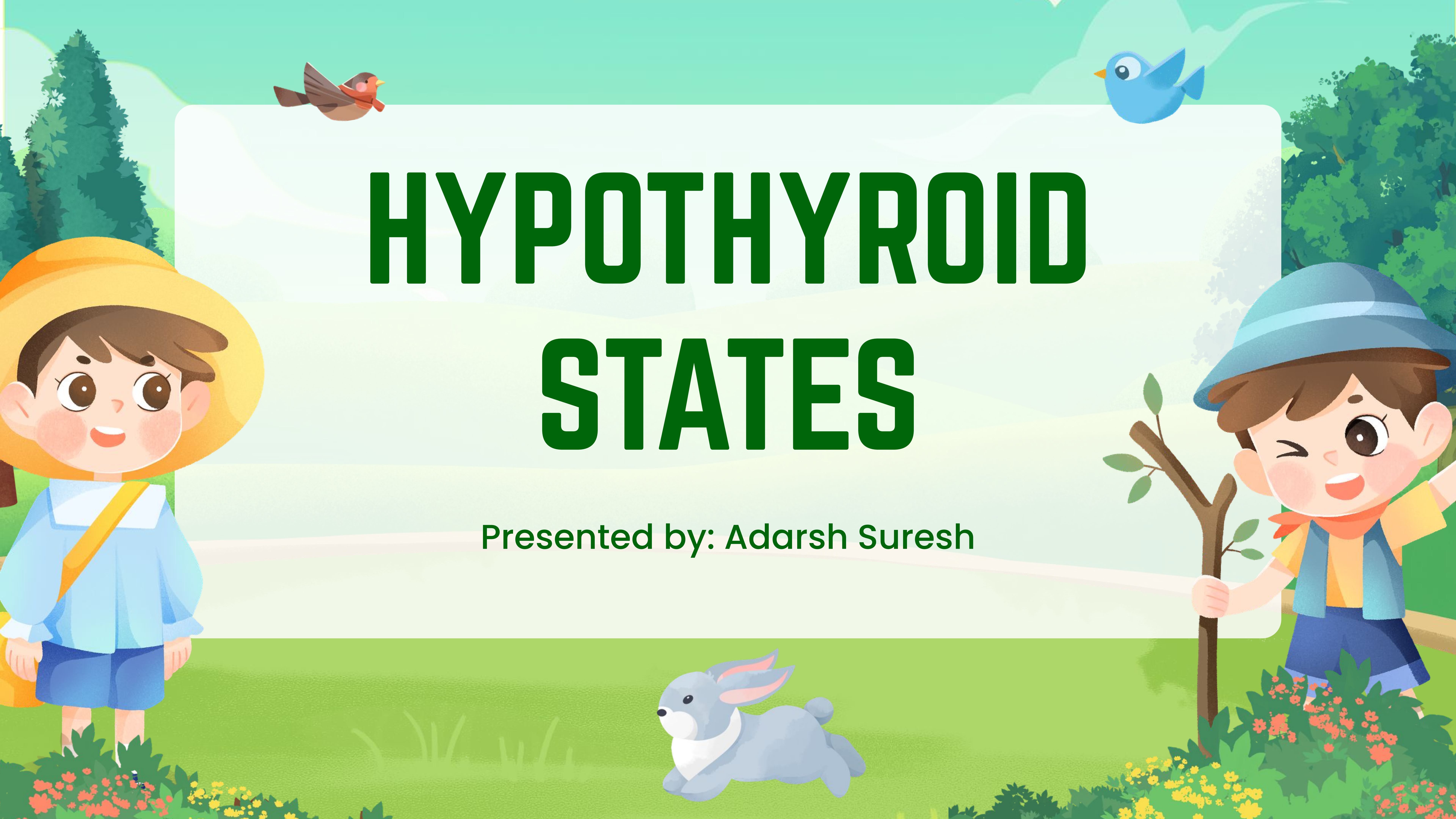
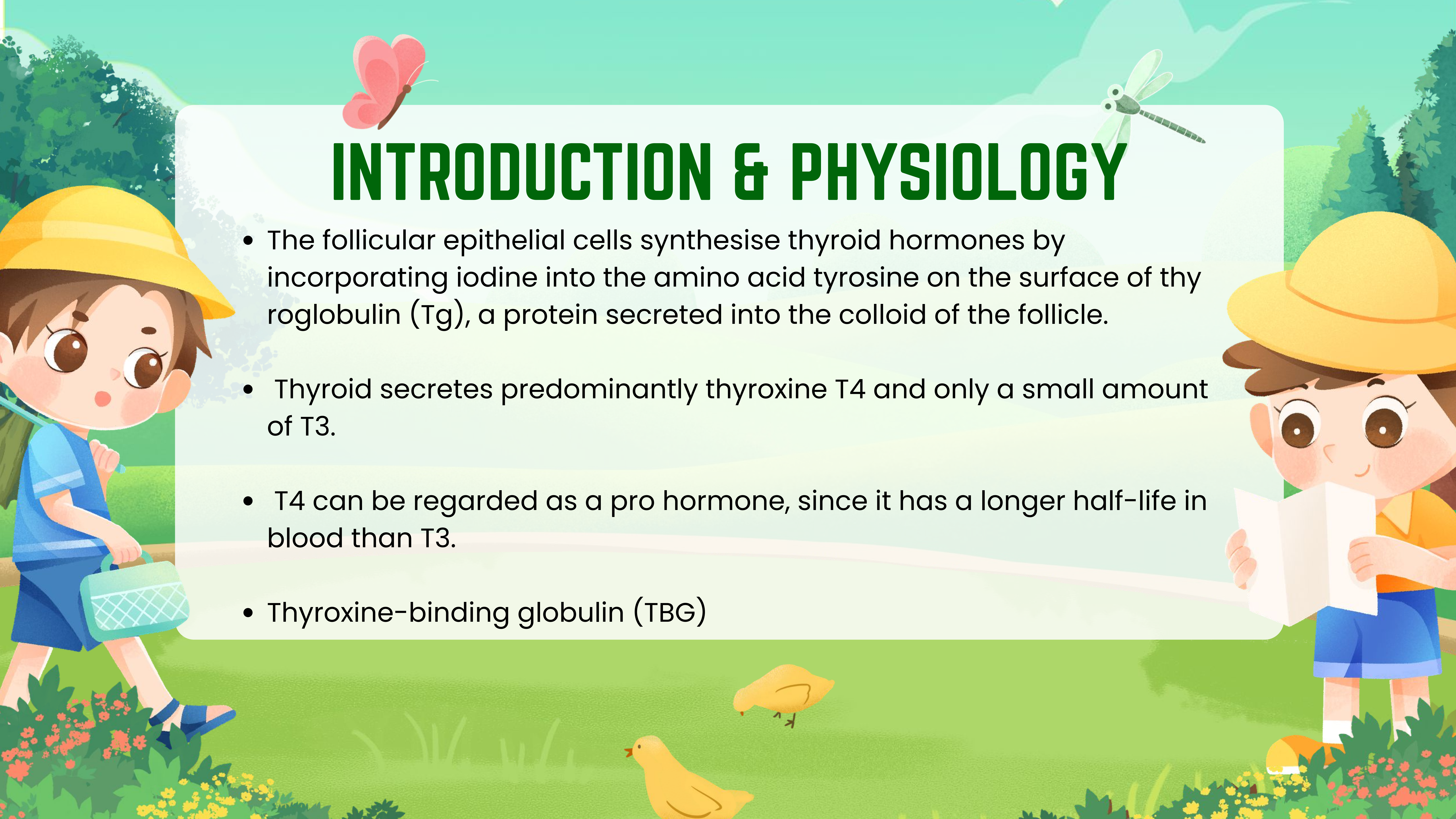

HYPOTHYROID STATES
Presented by: Adarsh Suresh


INTRODUCTION & PHYSIOLOGY
• The follicular epithelial cells synthesise thyroid hormones by incorporating iodine into the amino acid tyrosine on the surface of thy roglobulin (Tg), a protein secreted into the colloid of the follicle.
• Thyroid secretes predominantly thyroxine T4 and only a small amount of T3.
• T4 can be regarded as a pro hormone, since it has a longer half-life in blood than T3.
• Thyroxine-binding globulin (TBG)

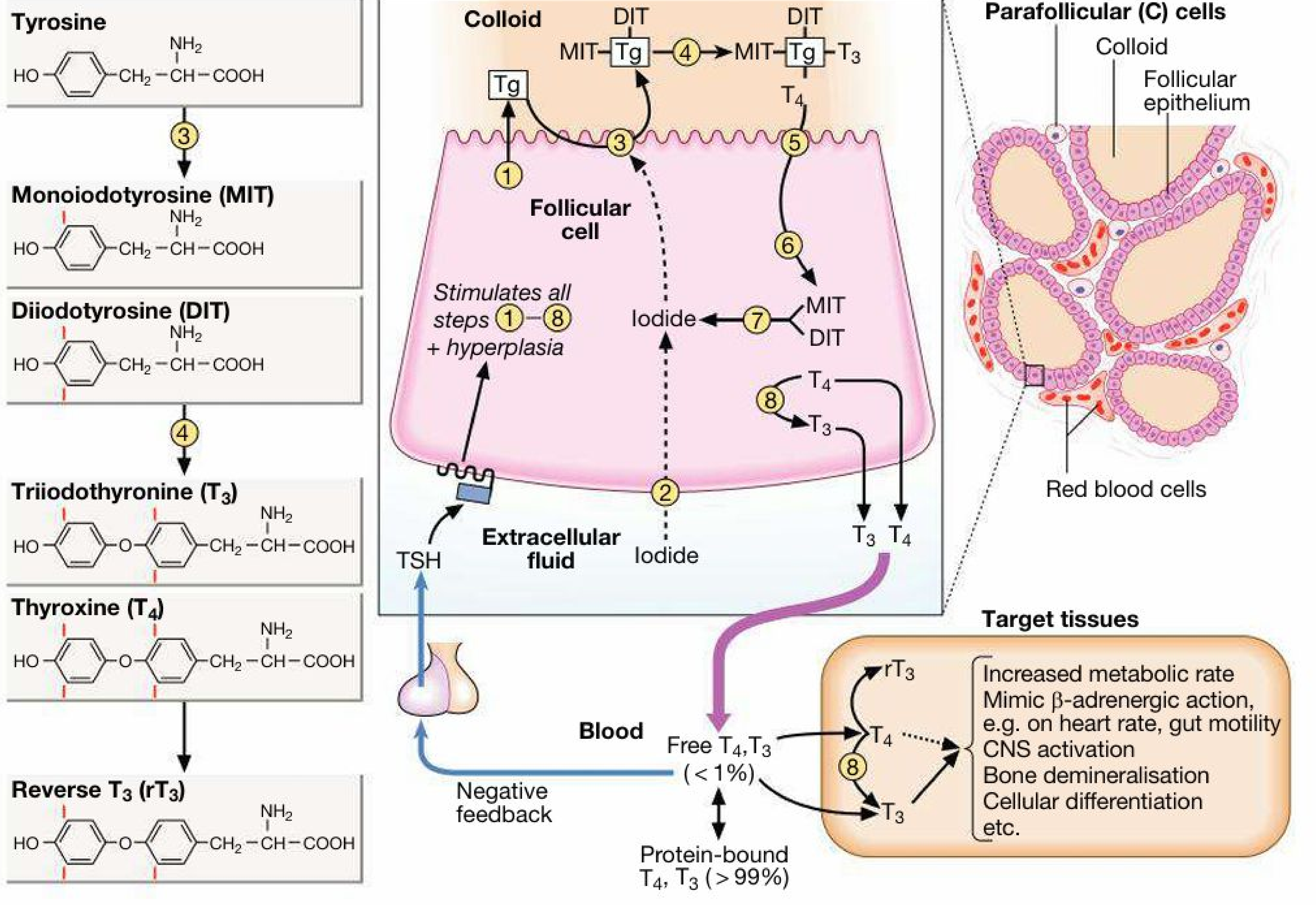
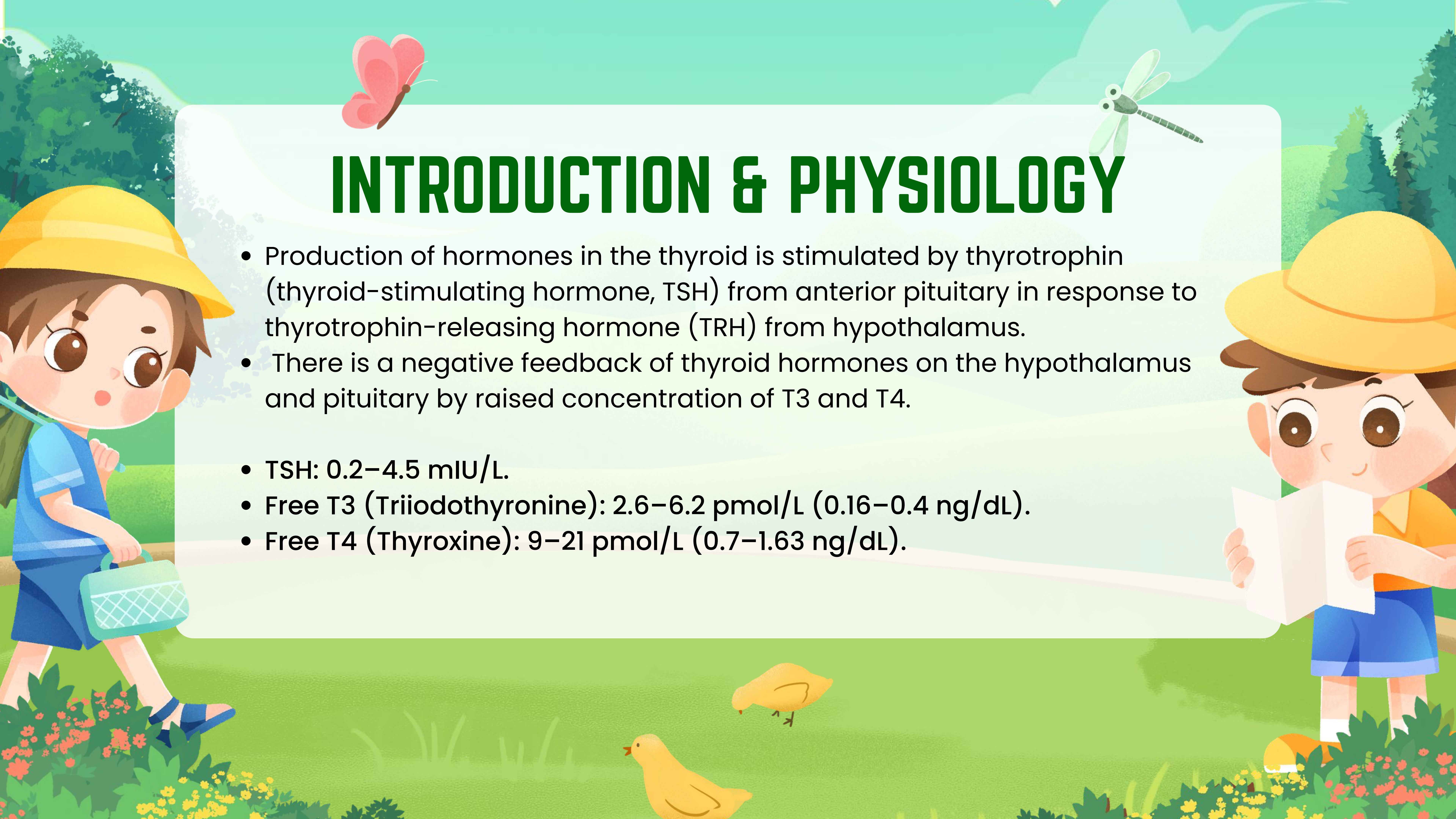


Fig. 20.3 Structure and function of the thyroid gland. (1) Thyroglobulin (Tg) is synthesised and secreted into the colloid of the follicle. (2) Inorganic iodide (I^-) is actively transported into the follicular cell ('trapping'). (3) Iodide is transported on to the colloidal surface by a transporter (pendrin, defective in Pendred syndrome, p. 667) and 'organified' by the thyroid peroxidase enzyme, which incorporates it into the amino acid tyrosine on the surface of Tg to form monoiodotyrosine (MIT) and diiodotyrosine (DIT). (4) Iodinated tyrosines couple to form triiodothyronine (T_3) and thyroxine (T_4). (5) Tg is endocytosed. (6) Tg is cleaved by proteolysis to free the iodinated tyrosine and thyroid hormones. (7) Iodinated tyrosine is dehalogenated to recycle the iodide. (8) T_4 is converted to T_3 by 5′-monodeiodinase. (CNS = central nervous system; TSH = thyroid-stimulating hormone)

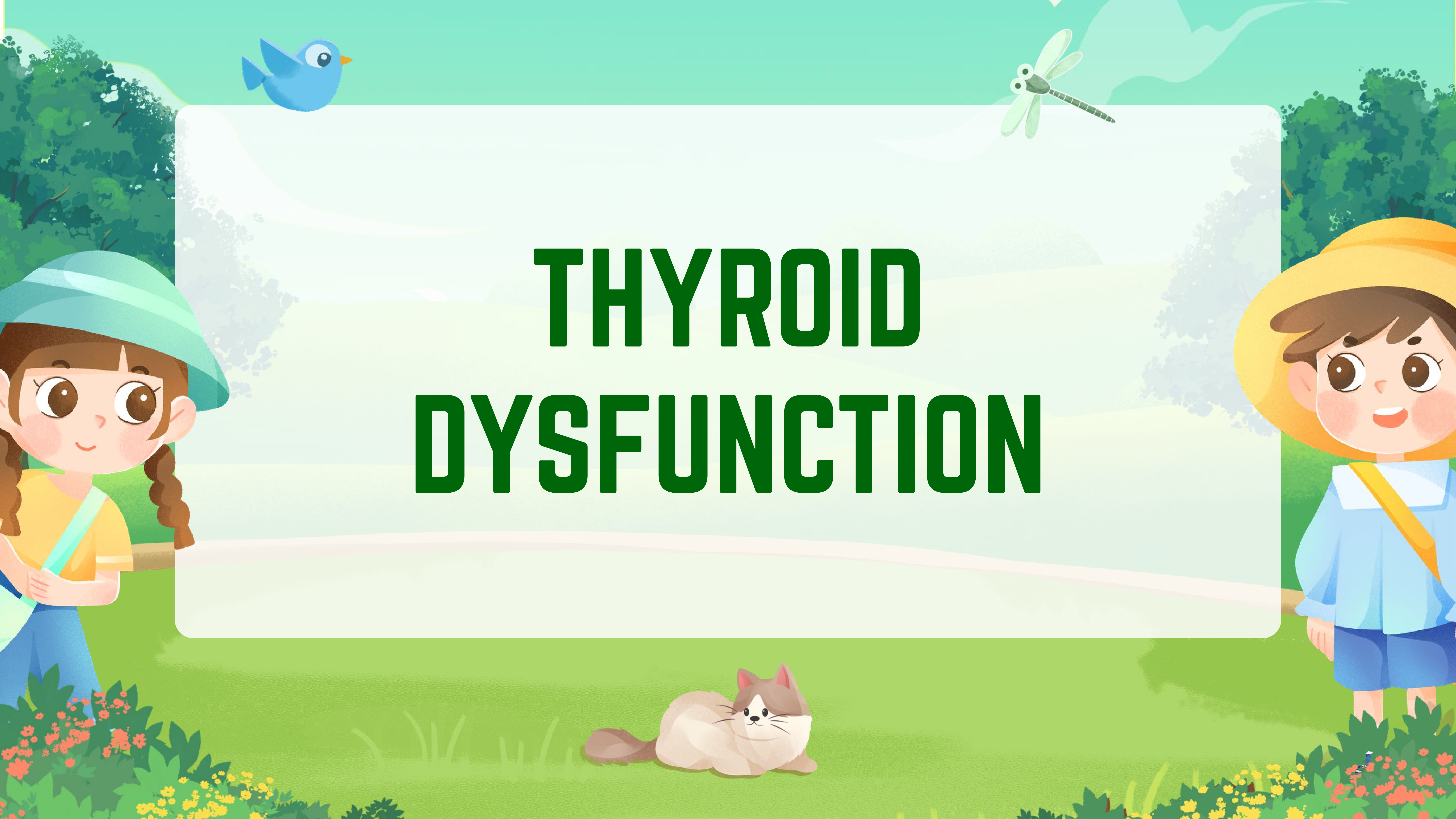

INTRODUCTION & PHYSIOLOGY
• Production of hormones in the thyroid is stimulated by thyrotrophin (thyroid-stimulating hormone, TSH) from anterior pituitary in response to thyrotrophin-releasing hormone (TRH) from hypothalamus.
• There is a negative feedback of thyroid hormones on the hypothalamus and pituitary by raised concentration of T3 and T4.
• TSH: 0.2–4.5 mIU/L.
• Free T3 (Triiodothyronine): 2.6–6.2 pmol/L (0.16–0.4 ng/dL).
• Free T4 (Thyroxine): 9–21 pmol/L (0.7–1.63 ng/dL).

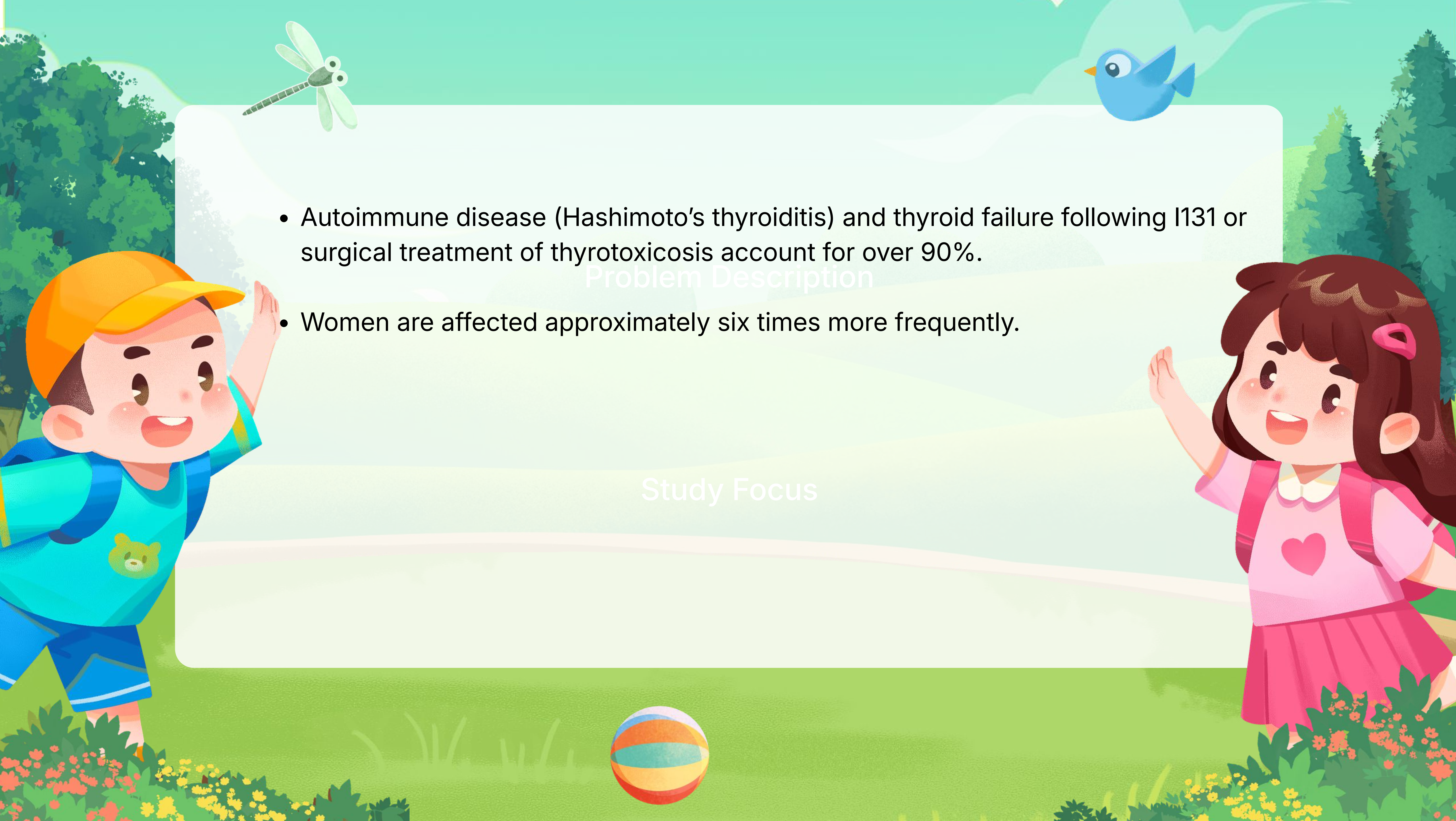
20.5 How to interpret thyroid function test results

TSH	T_4	T_3	Most likely interpretation(s)
Undetectable	Raised	Raised	Primary thyrotoxicosis
Undetectable or low	Raised	Normal	Over-treatment of hypothyroidism with levothyroxine Factitious thyrotoxicosis
Undetectable	Normal[1]	Raised	Primary T_3 toxicosis
Undetectable	Normal[1]	Normal[1]	Subclinical thyrotoxicosis
Undetectable or low	Raised	Low or normal	Non-thyroidal illness Amiodarone therapy
Undetectable or low	Low	Raised	Over-treatment of hypothyroidism with liothyronine (T_3)
Undetectable	Low	Low	Secondary hypothyroidism[4] Transient thyroiditis in evolution
Normal	Low	Low[2]	Secondary hypothyroidism[4]
Mildly elevated 5–20 mIU/L	Low	Low[2]	Primary hypothyroidism Secondary hypothyroidism[4]
Elevated >20 mIU/L	Low	Low[2]	Primary hypothyroidism
Mildly elevated 5–20 mIU/L	Normal[3]	Normal[2]	Subclinical hypothyroidism
Elevated 20–500 mIU/L	Normal	Normal	Artefact Heterophilic antibodies (host antibodies with affinity to the animal antibodies used in TSH assays)
Elevated	Raised	Raised	Non-adherence to levothyroxine replacement – recent 'loading' dose Secondary thyrotoxicosis[4] Thyroid hormone resistance

[1]Usually upper part of reference range. [2]T_3 is not a sensitive indicator of hypothyroidism and should not be requested. [3]Usually lower part of reference range. [4]i.e. Secondary to pituitary or hypothalamic disease. Note that TSH assays may report detectable TSH.

(TSH = thyroid-stimulating hormone)


THYROID
DYSFUNCTION


• Autoimmune disease (Hashimoto's thyroiditis) and thyroid failure following I131 or surgical treatment of thyrotoxicosis account for over 90%.
Problem Description
• Women are affected approximately six times more frequently.
Study Focus

20.10 Causes of hypothyroidism

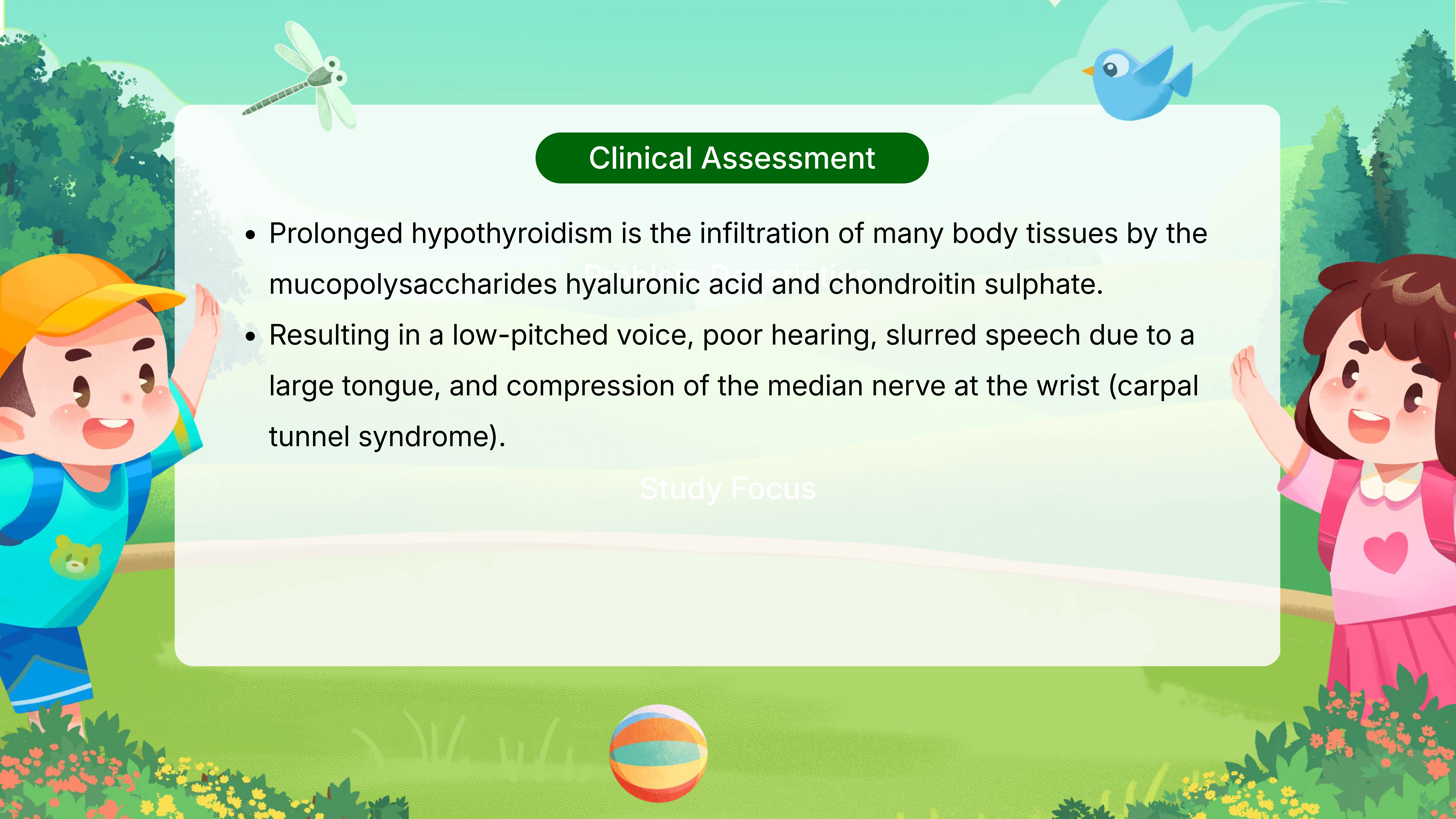
Causes	Anti-TPO antibodies[1]	Goitre[2]
Autoimmune		
Hashimoto's thyroiditis	++	±
Spontaneous atrophic hypothyroidism	–	–
Graves' disease with TSH receptor-blocking antibodies	+	±
Iatrogenic		
Radioactive iodine ablation	+	±
Thyroidectomy	+	–
Drugs		
Carbimazole, methimazole, propylthiouracil	+	±
Amiodarone	+	±
Lithium	–	±
Transient thyroiditis		
Subacute (de Quervain's) thyroiditis	+	±
Post-partum thyroiditis	+	±
Iodine deficiency		
e.g. In mountainous regions	–	++
Congenital		
Dyshormonogenesis	–	++
Thyroid aplasia	–	–
Infiltrative		
Amyloidosis, Riedel's thyroiditis, sarcoidosis etc.	+	++
Secondary hypothyroidism		
TSH deficiency	–	–

[1]As shown in Box 20.8, thyroid autoantibodies are common in the healthy population, so might be present in anyone. ++ high titre; + more likely to be detected than in the healthy population; – not especially likely. [2]Goitre: – absent; ± may be present; ++ characteristic.
(TPO = thyroid peroxidase; TSH = thyroid-stimulating hormone)

TABLE 395-1 Causes of Hypothyroidism

Primary
Autoimmune hypothyroidism: Hashimoto's thyroiditis, atrophic thyroiditis
Iatrogenic: ^{131}I treatment, subtotal or total thyroidectomy, external irradiation of neck for lymphoma or cancer
Drugs: iodine excess (including iodine-containing contrast media), amiodarone, lithium, antithyroid drugs, *p*-aminosalicylic acid, interferon α and other cytokines, aminoglutethimide, tyrosine kinase inhibitors (e.g., sunitinib), immune checkpoint inhibitors (e.g., ipilimumab, nivolumab, pembrolizumab)
Congenital hypothyroidism: absent or ectopic thyroid gland, dyshormonogenesis, TSH-R mutation
Iodine deficiency
Infiltrative disorders: amyloidosis, sarcoidosis, hemochromatosis, scleroderma, cystinosis, Riedel's thyroiditis
Overexpression of type 3 deiodinase in infantile hemangioma and other tumors
Transient
Silent thyroiditis, including postpartum thyroiditis
Subacute thyroiditis
Withdrawal of supraphysiologic thyroxine treatment in individuals with an intact thyroid
After ^{131}I treatment or subtotal thyroidectomy for Graves' disease
Secondary
Hypopituitarism: tumors, pituitary surgery or irradiation, infiltrative disorders, Sheehan's syndrome, trauma, genetic forms of combined pituitary hormone deficiencies
Isolated TSH deficiency or inactivity
Drugs: bexarotene, mitotane
Hypothalamic disease: tumors, trauma, infiltrative disorders, Prader-Willi syndrome

Abbreviations: TSH, thyroid-stimulating hormone; TSH-R, TSH receptor.


Clinical Assessment
Prolonged hypothyroidism is the infiltration of many body tissues by the mucopolysaccharides hyaluronic acid and chondroitin sulphate.
Resulting in a low-pitched voice, poor hearing, slurred speech due to a large tongue, and compression of the median nerve at the wrist (carpal tunnel syndrome).

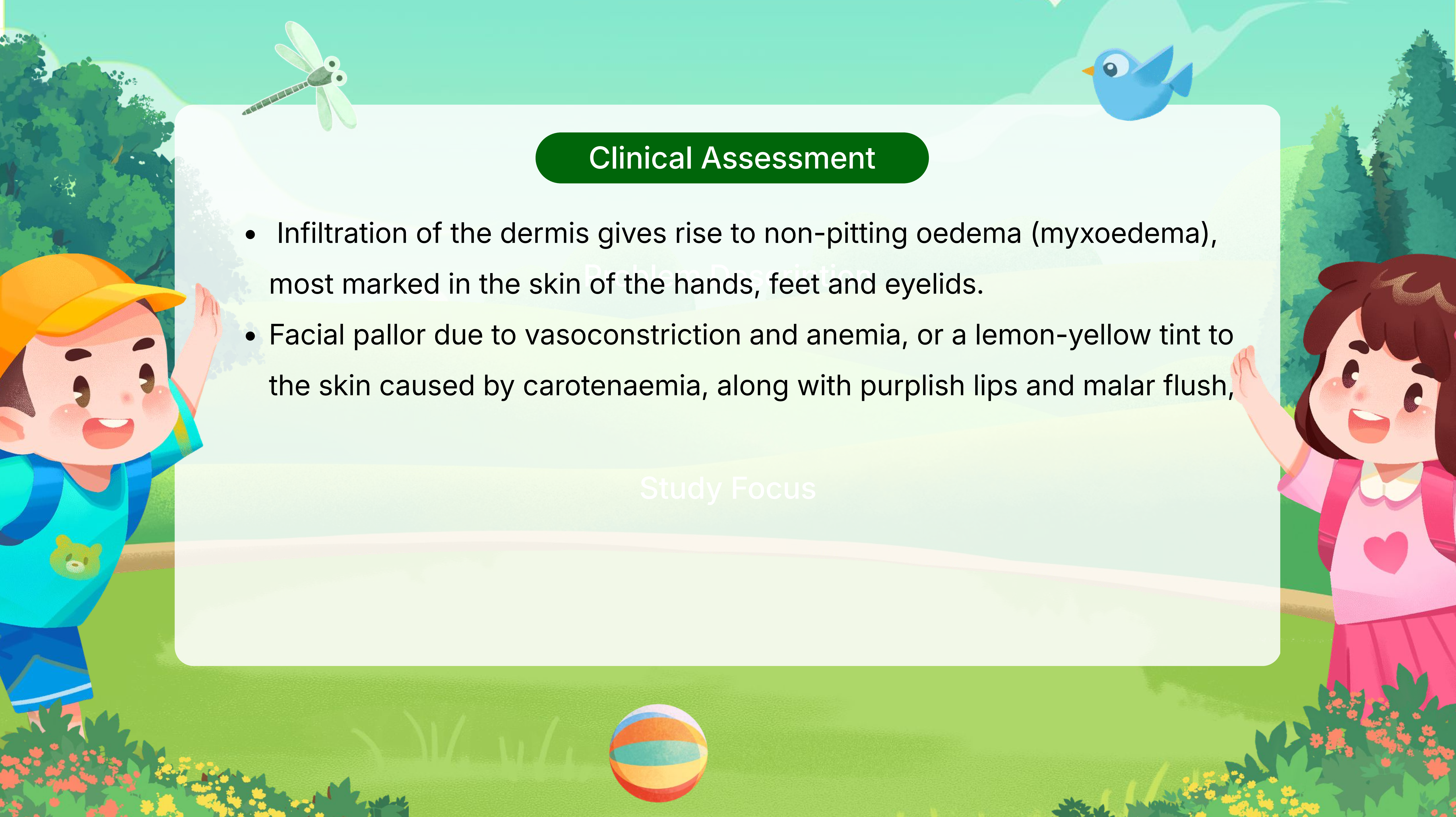

Clinical Assessment
• Infiltration of the dermis gives rise to non-pitting oedema (myxoedema), most marked in the skin of the hands, feet and eyelids.
• Facial pallor due to vasoconstriction and anemia, or a lemon-yellow tint to the skin caused by carotenaemia, along with purplish lips and malar flush,
Study Focus

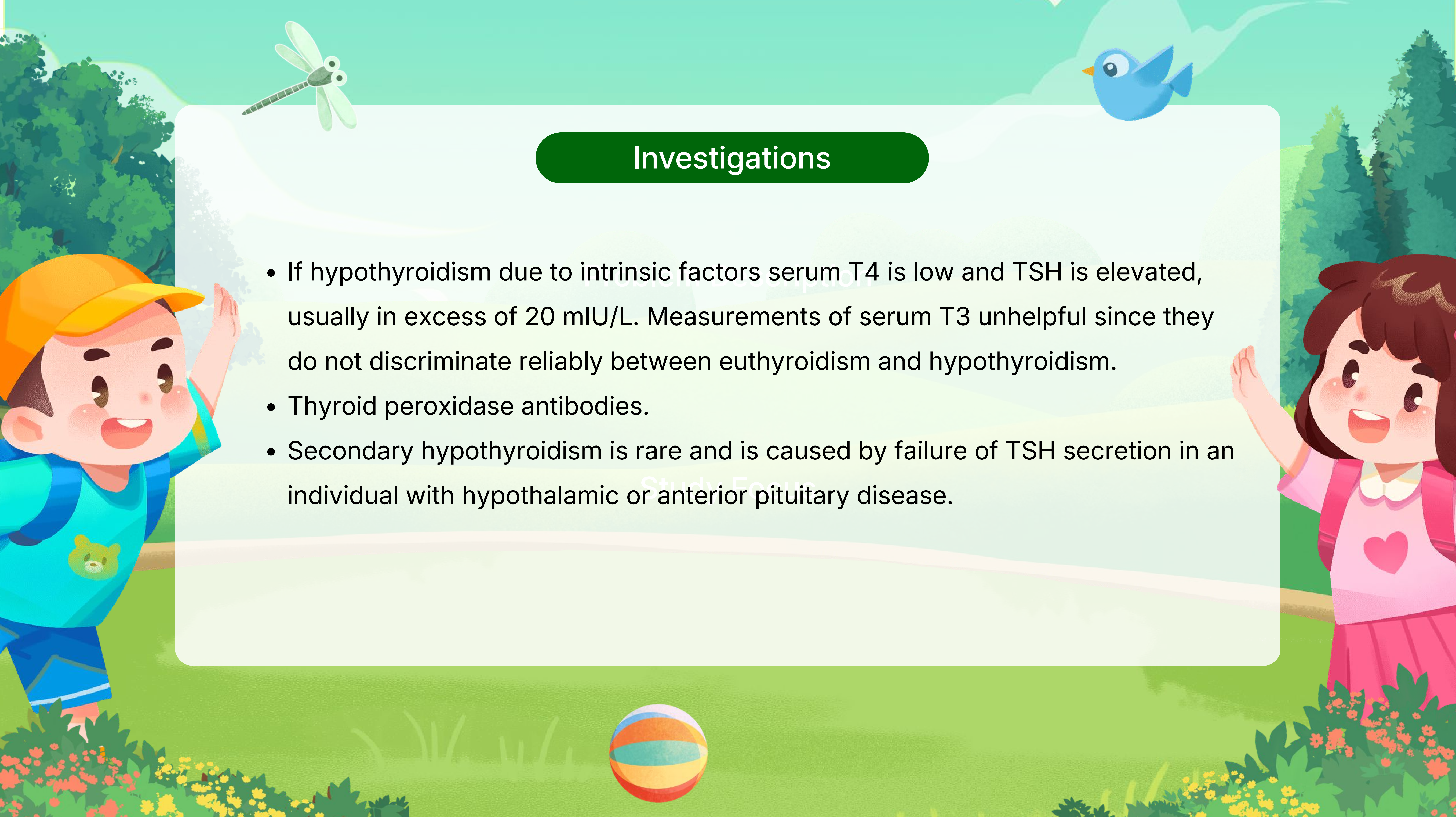
TABLE 395-3 Signs and Symptoms of Hypothyroidism (Descending Order of Frequency)

SYMPTOMS	SIGNS
Tiredness, weakness	Dry coarse skin; cool peripheral extremities
Dry skin	Puffy face, hands, and feet (myxedema)
Feeling cold	Diffuse alopecia
Hair loss	Bradycardia
Difficulty concentrating and poor memory	Peripheral edema
Constipation	Delayed tendon reflex relaxation
Weight gain with poor appetite	Carpal tunnel syndrome
Dyspnea	Serous cavity effusions
Hoarse voice	
Menorrhagia (later oligomenorrhea or amenorrhea)	
Paresthesia	
Impaired hearing	

20.7 Clinical features of thyroid dysfunction

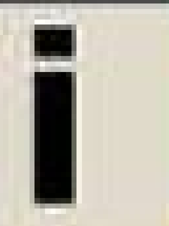
Thyrotoxicosis		Hypothyroidism	
Symptoms	**Signs**	**Symptoms**	**Signs**
Common			
Weight loss despite normal or increased appetite	Weight loss	Weight gain	Weight gain
Heat intolerance, sweating	Tremor	Cold intolerance	
Palpitations, tremor	Palmar erythema	Fatigue, somnolence	
Dyspnoea, fatigue	Sinus tachycardia	Dry skin	
Irritability, emotional lability	Lid retraction, lid lag	Dry hair	
		Menorrhagia	
Less common			
Osteoporosis (fracture, loss of height)	Goitre with bruit[1]	Constipation	Hoarse voice
Diarrhoea, steatorrhoea	Atrial fibrillation[2]	Hoarseness	Facial features:
Angina	Systolic hypertension/increased pulse pressure	Carpal tunnel syndrome	Purplish lips
Ankle swelling	Cardiac failure[2]	Alopecia	Malar flush
Anxiety, psychosis	Hyper-reflexia	Aches and pains	Periorbital oedema
Muscle weakness	Ill-sustained clonus	Muscle stiffness	Loss of lateral eyebrows
Periodic paralysis (predominantly in Chinese and other Asian groups)	Proximal myopathy	Deafness	Anaemia
Pruritus, alopecia	Bulbar myopathy[2]	Depression	Carotenaemia
Amenorrhoea/oligomenorrhoea		Infertility	Erythema ab igne
Infertility, spontaneous abortion			Bradycardia hypertension
Loss of libido, impotence			Delayed relaxation of reflexes
Excessive lacrimation			Dermal myxoedema
Rare			
Vomiting	Gynaecomastia	Psychosis (myxoedema madness)	Ileus, ascites
Apathy	Spider naevi	Galactorrhoea	Pericardial and pleural effusions
Anorexia	Onycholysis	Impotence	Cerebellar ataxia
Exacerbation of asthma	Pigmentation		Myotonia

[1]In Graves' disease only. [2]Features found particularly in older patients.



Investigations

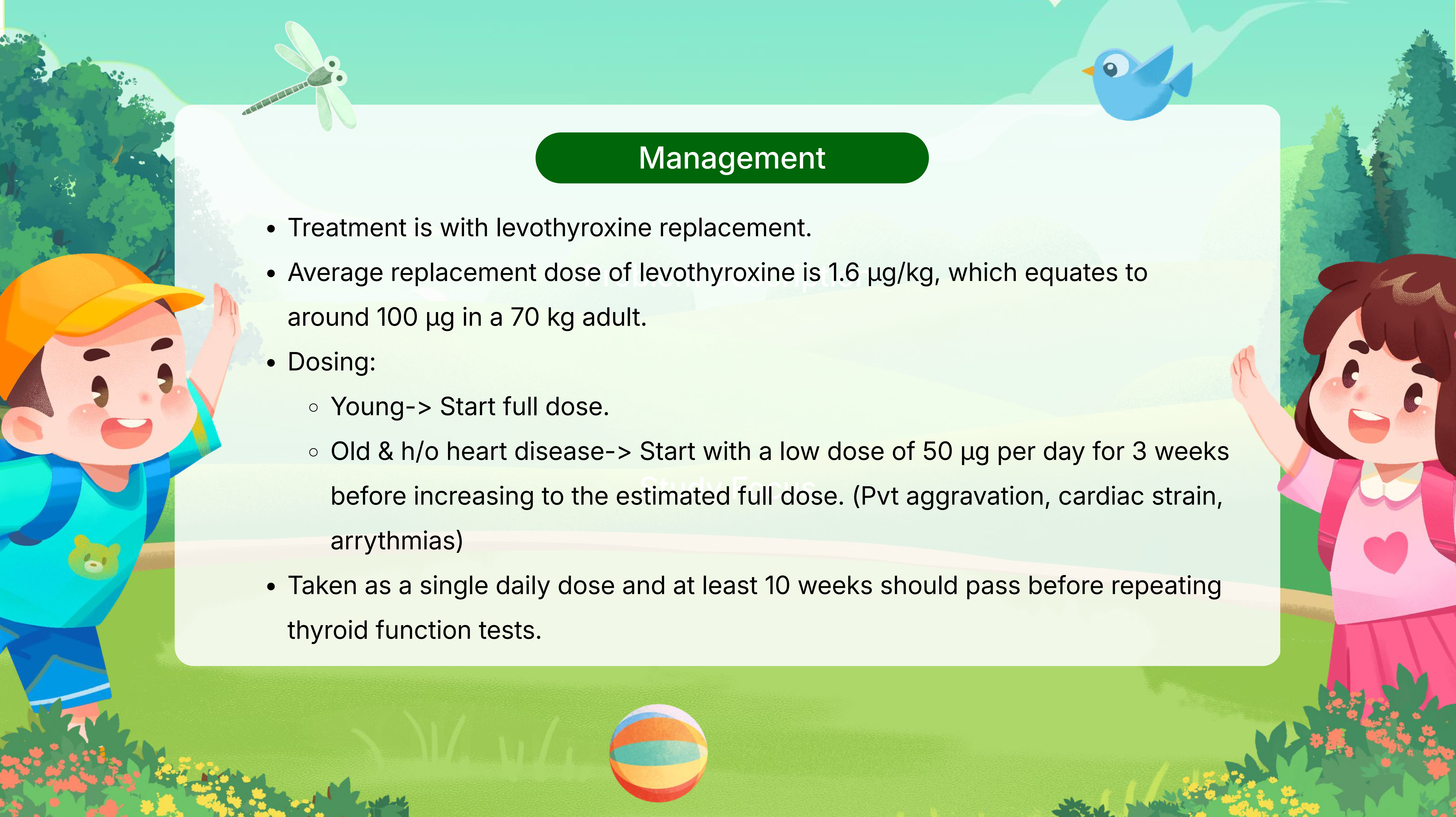
- If hypothyroidism due to intrinsic factors serum T4 is low and TSH is elevated, usually in excess of 20 mIU/L. Measurements of serum T3 unhelpful since they do not discriminate reliably between euthyroidism and hypothyroidism.
- Thyroid peroxidase antibodies.
- Secondary hypothyroidism is rare and is caused by failure of TSH secretion in an individual with hypothalamic or anterior pituitary disease.

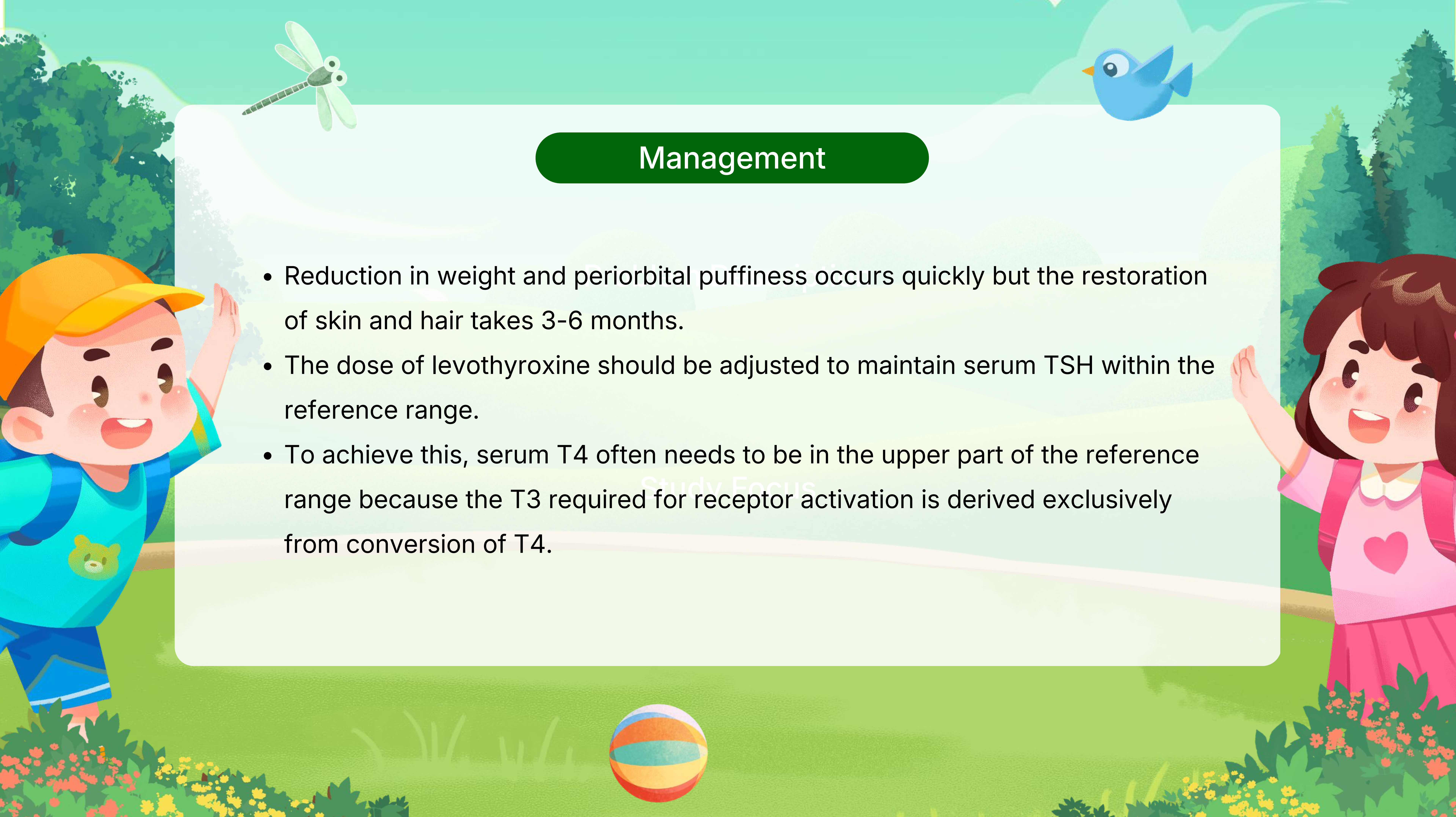


20.9 Non-specific laboratory abnormalities in thyroid dysfunction*

Thyrotoxicosis
• Serum enzymes: raised alanine aminotransferase, γ-glutamyl transferase (GGT), and alkaline phosphatase from liver and bone • Raised bilirubin • Mild hypercalcaemia • Glycosuria: associated diabetes mellitus, 'lag storage' glycosuria
Hypothyroidism
• Serum enzymes: raised creatine kinase, aspartate aminotransferase, lactate dehydrogenase (LDH) • Hypercholesterolaemia • Anaemia: normochromic normocytic or macrocytic • Hyponatraemia

*These abnormalities are not useful in differential diagnosis, so the tests should be avoided and any further investigation undertaken only if abnormalities persist when the patient is euthyroid.


Management
Treatment is with levothyroxine replacement.
Average replacement dose of levothyroxine is 1.6 µg/kg, which equates to around 100 µg in a 70 kg adult.
Dosing:
Young-> Start full dose.
Old & h/o heart disease-> Start with a low dose of 50 µg per day for 3 weeks before increasing to the estimated full dose. (Pvt aggravation, cardiac strain, arrythmias)
Taken as a single daily dose and at least 10 weeks should pass before repeating thyroid function tests.


Management
• Reduction in weight and periorbital puffiness occurs quickly but the restoration of skin and hair takes 3-6 months.
• The dose of levothyroxine should be adjusted to maintain serum TSH within the reference range.
• To achieve this, serum T4 often needs to be in the upper part of the reference range because the T3 required for receptor activation is derived exclusively from conversion of T4.

Management
Problem Description
Some patients remain symptomatic despite normalization of TSH and wish to take extra levothyroxine, which suppresses TSH. However, suppressed TSH is a risk factor for osteoporosis and atrial fibrillation.
Measure thyroid function every 1–2 years once the dose of levothyroxine is stabilized.
Study Focus

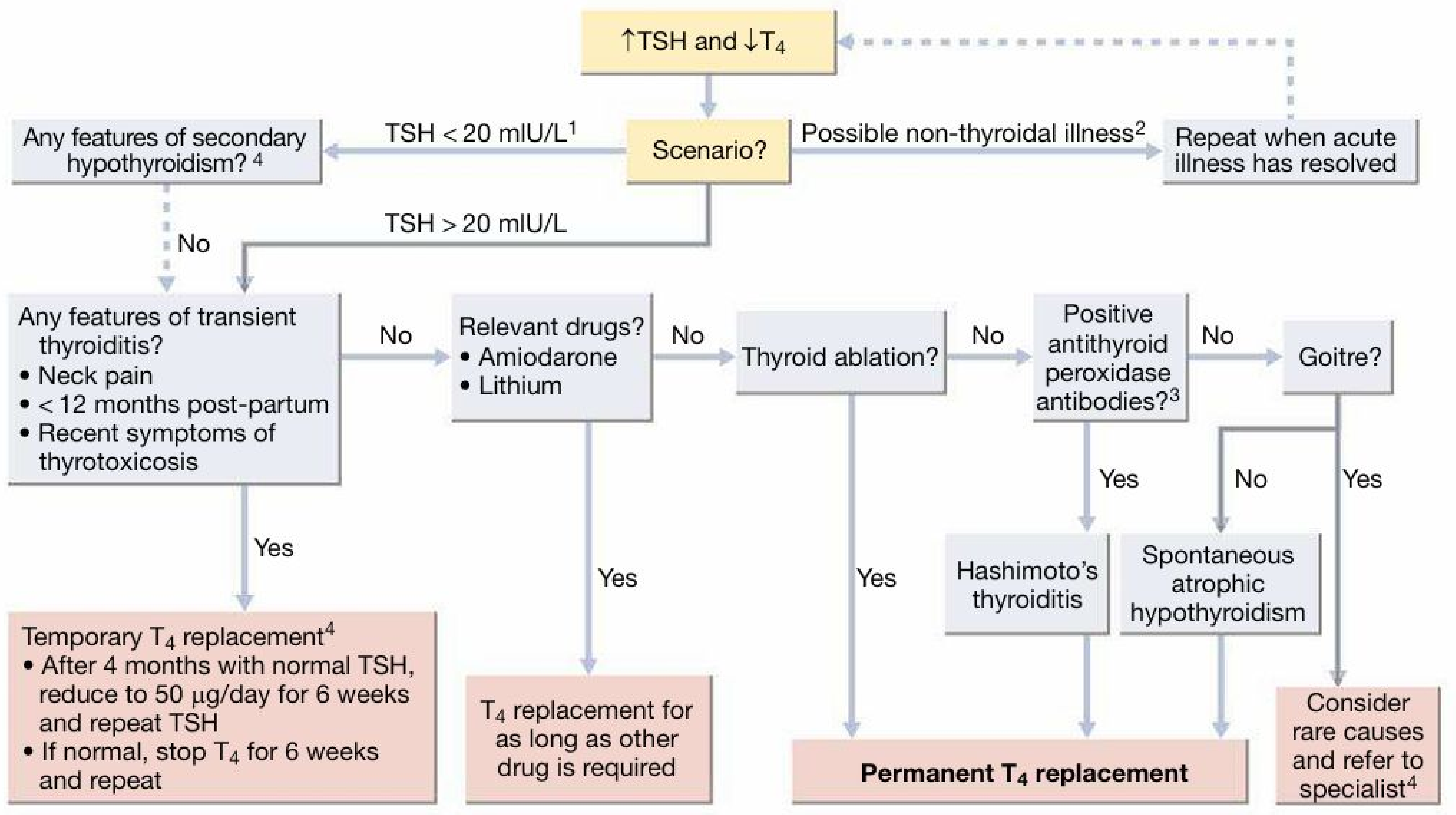
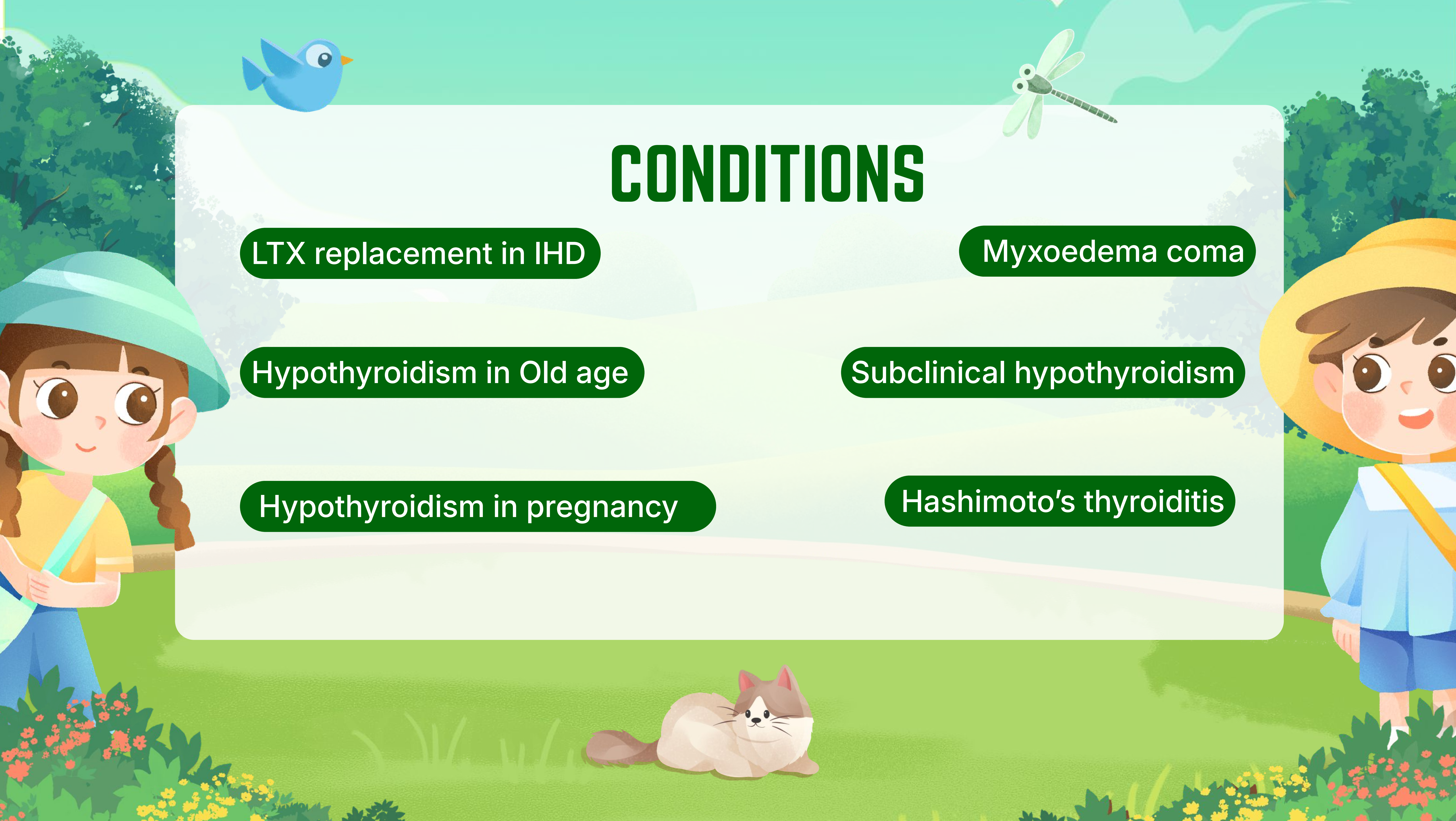
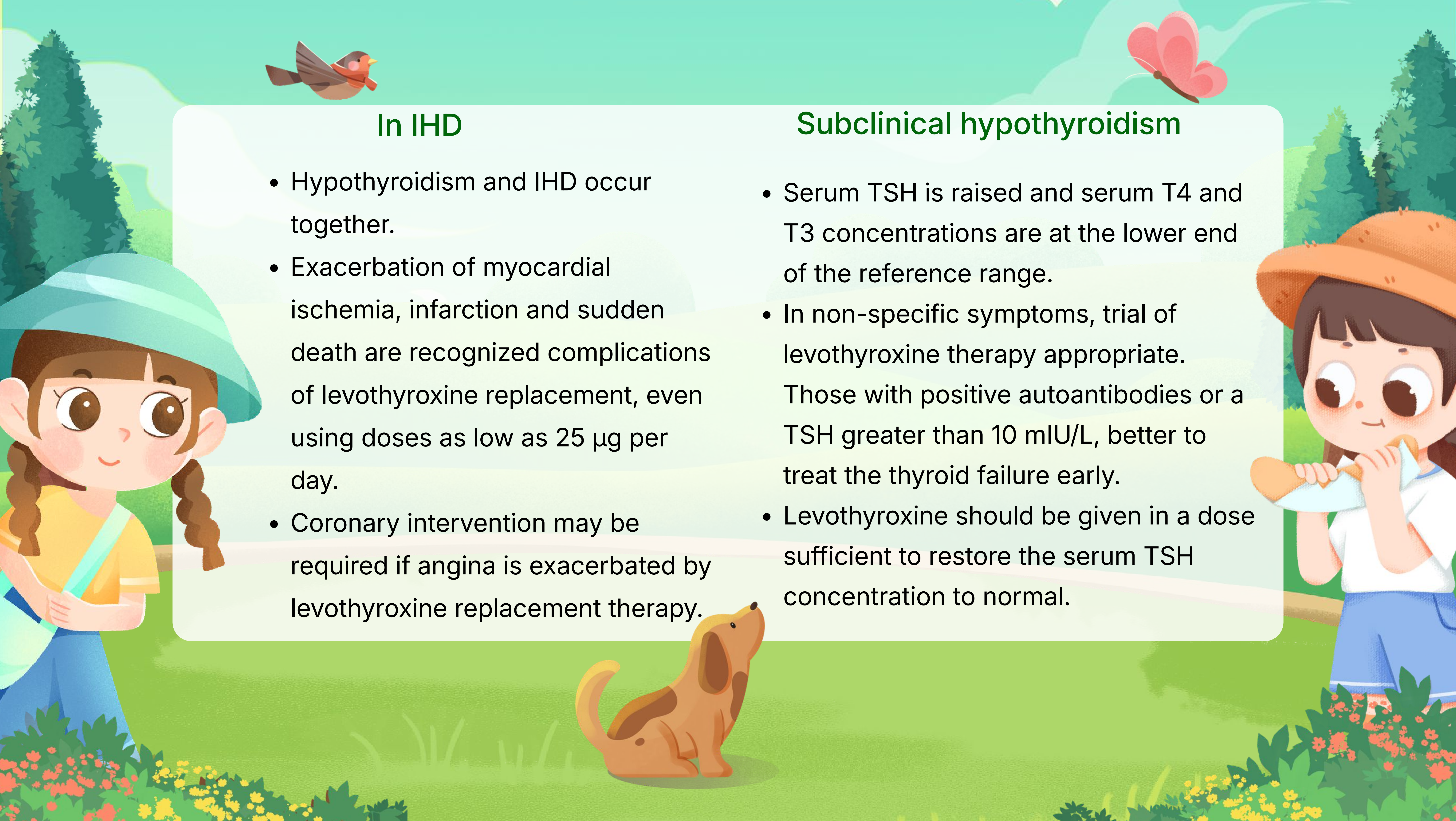


Fig. 20.6 An approach to adults with suspected primary hypothyroidism. This scheme ignores congenital causes of hypothyroidism (see Box 20.10), such as thyroid aplasia and dyshormonogenesis (associated with nerve deafness in Pendred syndrome, p. 667), which are usually diagnosed in childhood. [1]Immunoreactive thyroid-stimulating hormone (TSH) may be detected at normal or even modestly elevated levels in patients with pituitary failure; unless T_4 is only marginally low, TSH should be >20 mIU/L to confirm the diagnosis of primary hypothyroidism. [2]The usual abnormality in sick euthyroidism is a low TSH but any pattern can occur. [3]Thyroid peroxidase (TPO) antibodies are highly sensitive but not very specific for autoimmune thyroid disease (see Boxes 20.8 and 20.10). [4]Specialist advice is most appropriate where indicated. Secondary hypothyroidism is rare, but is suggested by deficiency of pituitary hormones or by clinical features of pituitary tumour such as headache or visual field defect (p. 695). Rare causes of hypothyroidism with goitre include dyshormonogenesis and infiltration of the thyroid (see Box 20.10).


CONDITIONS
LTX replacement in IHD
Myxoedema coma
Hypothyroidism in Old age
Subclinical hypothyroidism
Hypothyroidism in pregnancy
Hashimoto's thyroiditis


In IHD
Hypothyroidism and IHD occur together.
Exacerbation of myocardial ischemia, infarction and sudden death are recognized complications of levothyroxine replacement, even using doses as low as 25 µg per day.
Coronary intervention may be required if angina is exacerbated by levothyroxine replacement therapy.
Subclinical hypothyroidism
Serum TSH is raised and serum T4 and T3 concentrations are at the lower end of the reference range.
In non-specific symptoms, trial of levothyroxine therapy appropriate.
Those with positive autoantibodies or a TSH greater than 10 mIU/L, better to treat the thyroid failure early.
Levothyroxine should be given in a dose sufficient to restore the serum TSH concentration to normal.

20.12 The thyroid gland in old age

Thyrotoxicosis

- **Causes**: commonly due to multinodular goitre.
- **Clinical features**: apathy, anorexia, proximal myopathy, atrial fibrillation and cardiac failure predominate.
- **Non-thyroidal illness**: thyroid function tests are performed more frequently in older patients but interpretation may be altered by intercurrent illness.

Hypothyroidism

- **Clinical features**: non-specific features, such as physical and mental slowing, are often attributed to increasing age and the diagnosis is delayed.
- **Myxoedema coma**: more likely in older people.
- **Levothyroxine dose**: to avoid exacerbating latent or established heart disease, the starting dose should be 25–50 μg daily. Levothyroxine requirements fall with increasing age and few patients need more than 100 μg daily.
- **Other medication** (see Box 20.10): may interfere with absorption or metabolism of levothyroxine, necessitating an increase in dose.

20.13 Thyroid disease in pregnancy

Normal pregnancy

- **Trimester-specific reference ranges**: should be used to interpret thyroid function test results in pregnancy.

Iodine deficiency

- **Iodine requirements**: increased in pregnancy. The World Health Organization (WHO) recommends a minimum intake of 250 µg/day.
- **Iodine deficiency**: the major cause of preventable impaired cognitive development in children worldwide.

Hypothyroidism

- **Impaired cognitive development in the offspring**: may be associated with hypothyroidism that is not adequately treated.
- **Levothyroxine replacement therapy dose requirements**: increase by 30%–50% from early in pregnancy. Monitoring to maintain TSH results within the trimester-specific reference range is recommended in early pregnancy and at least once in each trimester.

Thyrotoxicosis

- **Gestational thyrotoxicosis**: associated with multiple pregnancies and hyperemesis gravidarum. Transient and usually does not require antithyroid drug treatment.
- **Graves' disease**: the most common cause of sustained thyrotoxicosis in pregnancy.
- **Antithyroid drugs**: propylthiouracil should be used in the first trimester, with carbimazole substituted in the second and third trimesters.

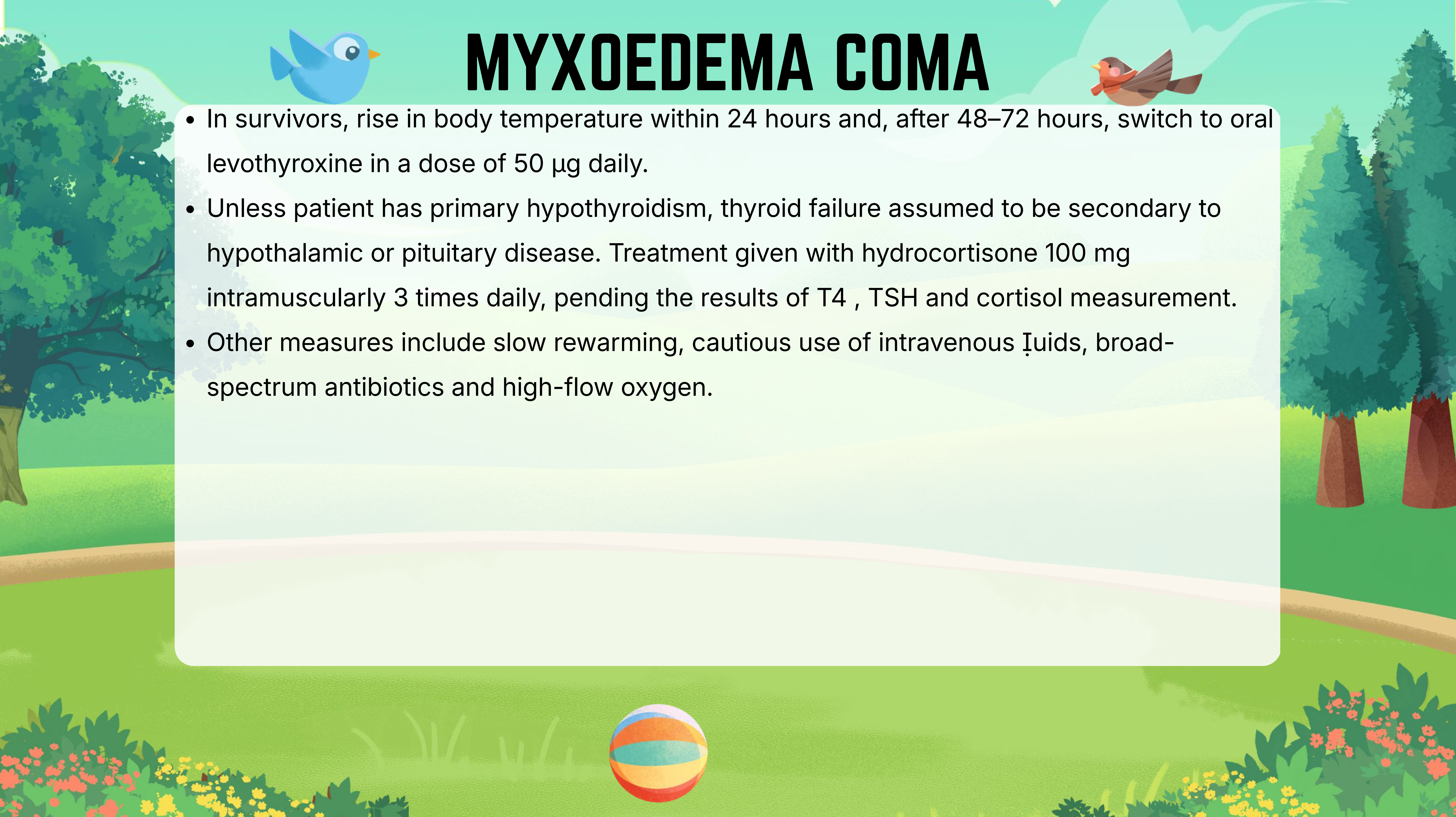
Post-partum thyroiditis

- **Screening**: not recommended for every woman, but thyroid function should be tested 4–6 weeks post partum in those with a personal history of thyroid disease, goitre or other autoimmune disease including type 1 diabetes, in those known to have positive antithyroid peroxidase antibodies, or when there is clinical suspicion of thyroid dysfunction.

	TSH range
First trimester	>0.1 mIU/L and <2.5 mIU/L
Second trimester	>0.2 mIU/L and <3.0 mIU/L
Third trimester	>0.3 mIU/L and <3.0 mIU/L

MYXOEDEMA COMA

- Depressed level of consciousness, usually in an older patient who appears myxoedematous.
- Body temperature as low as 25°C, convulsions are not uncommon, and cerebrospinal fluid (CSF) pressure and protein content are raised.
- Altered consciousness level, cardiac failure, pneumonia, dilutional hyponatremia and respiratory failure.
- Suspected cases should be treated with an intravenous injection of 20 µg liothyronine, followed by further injections of 20 µg 3 times daily until there is sustained clinical improvement.


MYXOEDEMA COMA
In survivors, rise in body temperature within 24 hours and, after 48–72 hours, switch to oral levothyroxine in a dose of 50 μg daily.
Unless patient has primary hypothyroidism, thyroid failure assumed to be secondary to hypothalamic or pituitary disease. Treatment given with hydrocortisone 100 mg intramuscularly 3 times daily, pending the results of T4 , TSH and cortisol measurement.
Other measures include slow rewarming, cautious use of intravenous fluids, broad-spectrum antibiotics and high-flow oxygen.

MYXEDEMA COMA

Triggers

- Infection
- Cold exposure
- Drugs
- Trauma

Labs

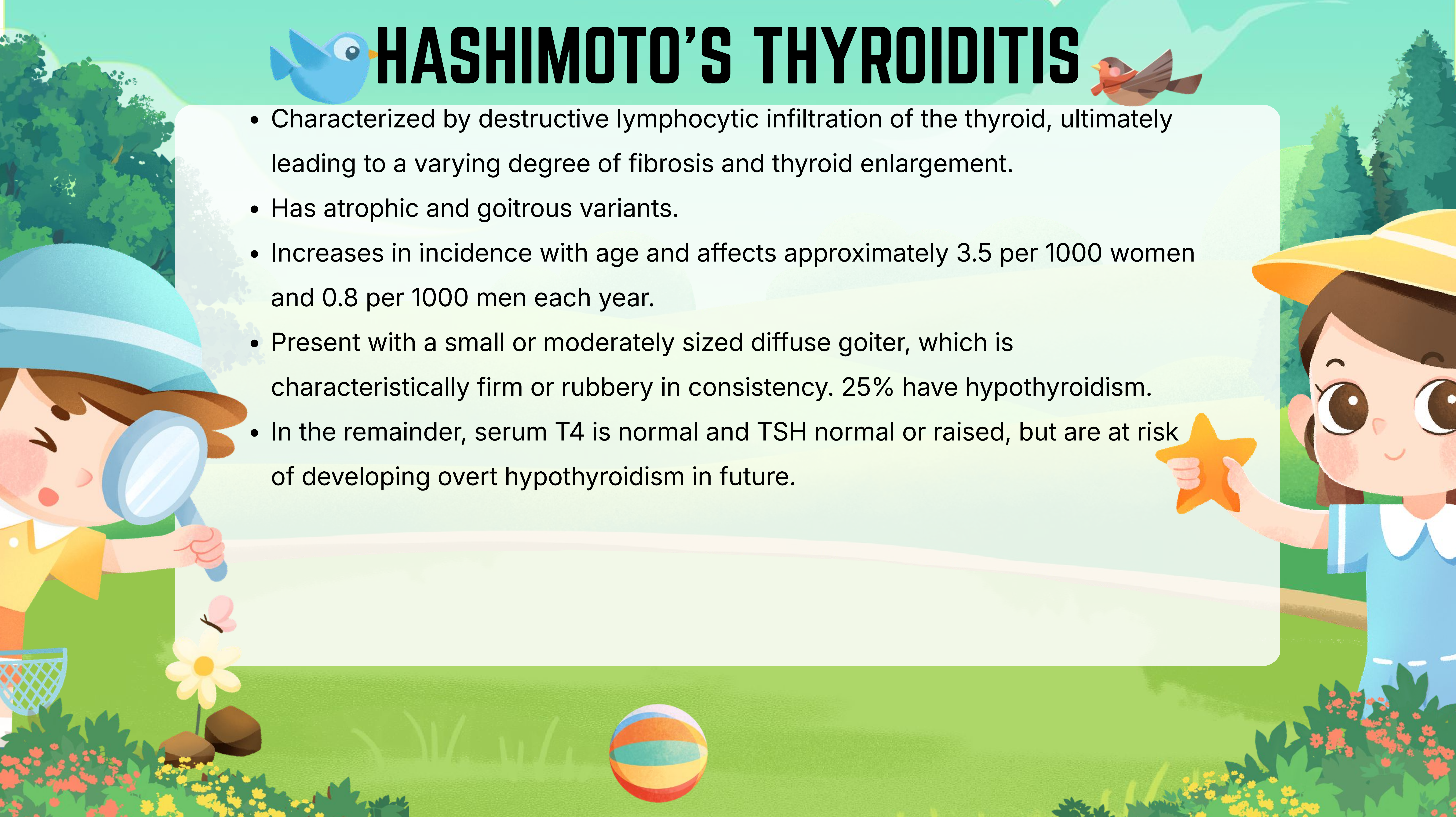
↑ TSH
↓ T3, T4
↓ Sodium
↓ Glucose

Signs and Symptoms

- Alterd mental
- Hypothermia
- Bradycardia
- Hypotension

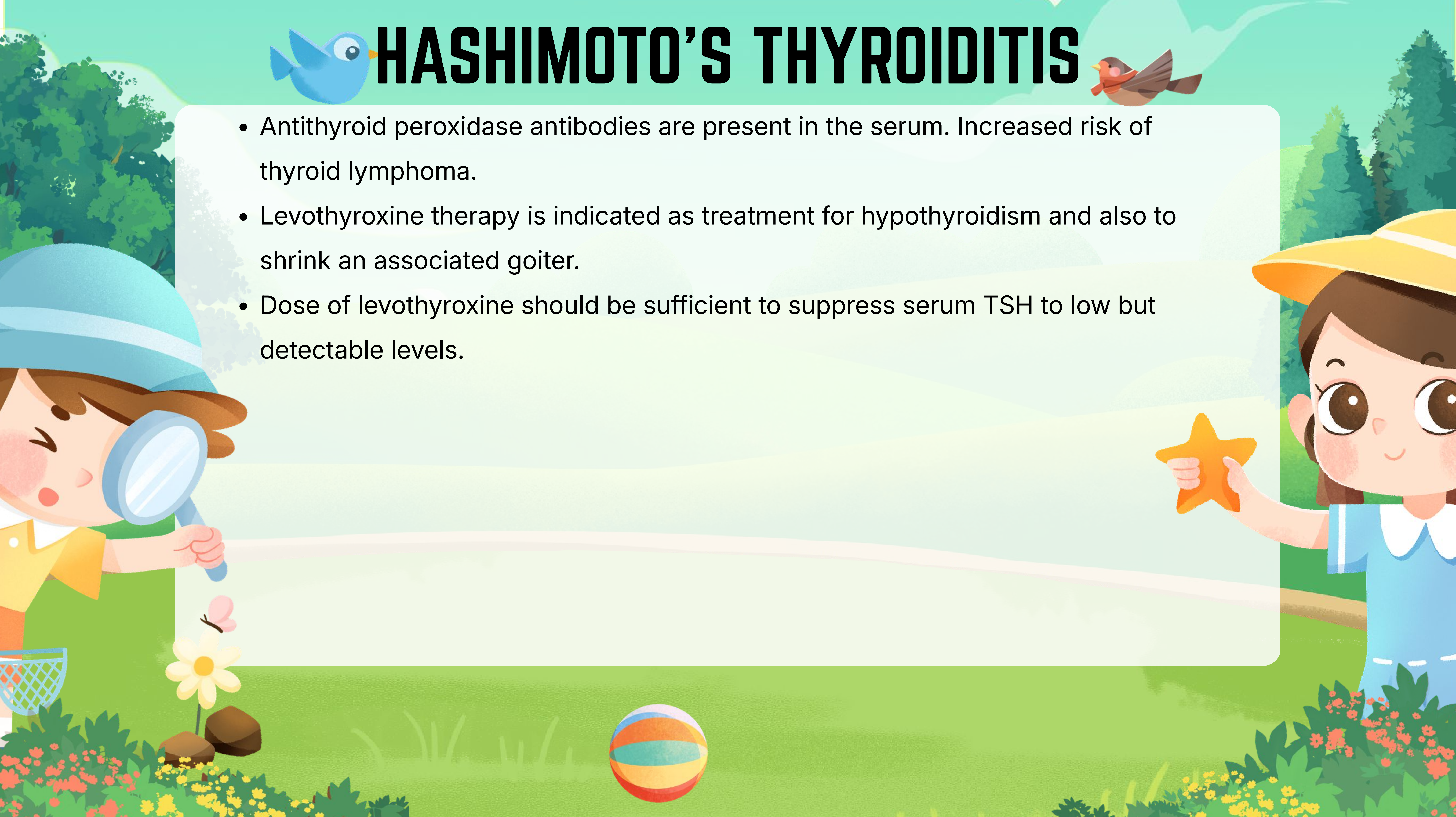
Treatment

- IV throid hormone
- IV hydrocortisone
- Passive rewarming
- Treat underlying cause
- Supportive care



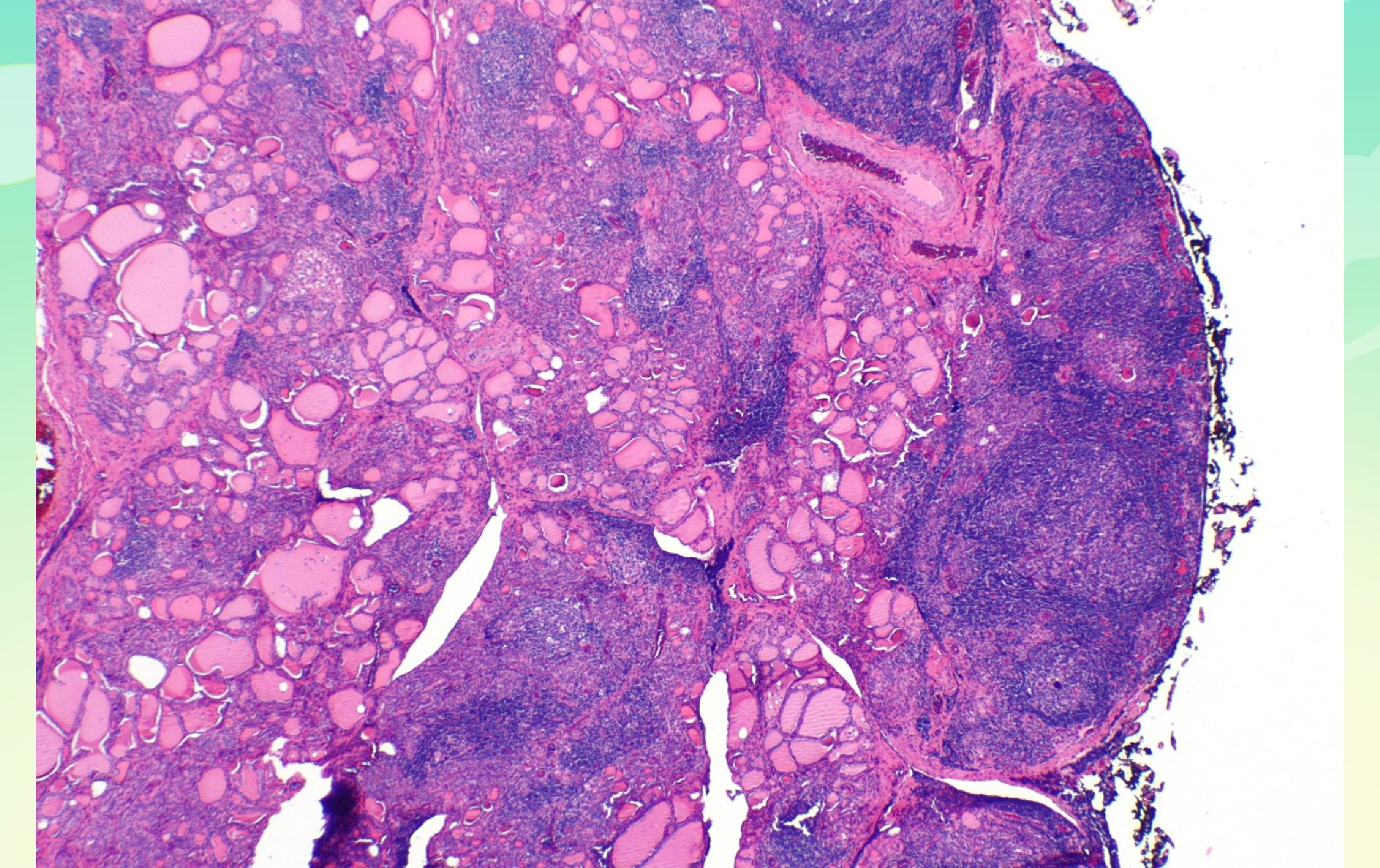
HASHIMOTO'S THYROIDITIS

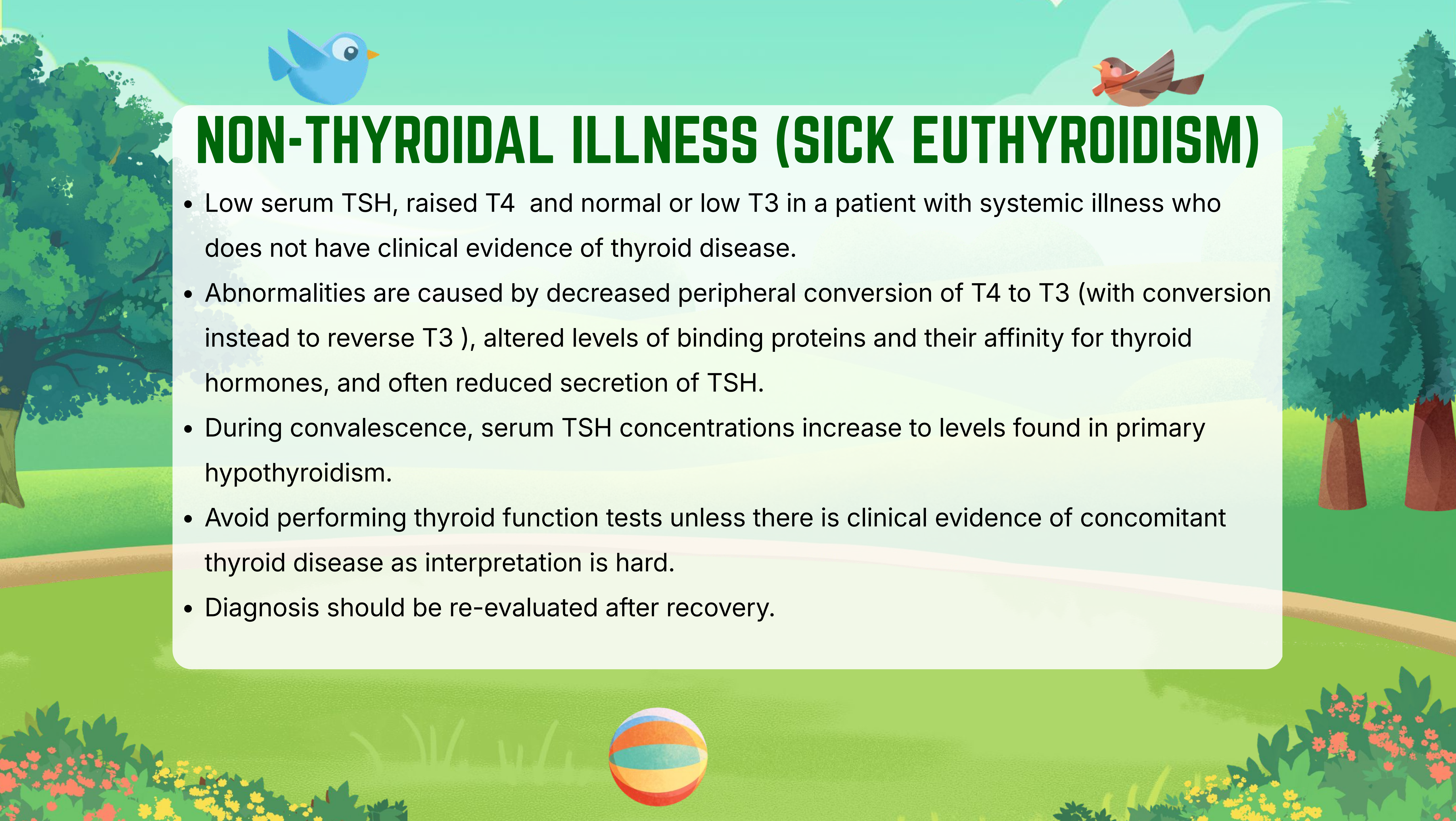
- Characterized by destructive lymphocytic infiltration of the thyroid, ultimately leading to a varying degree of fibrosis and thyroid enlargement.
- Has atrophic and goitrous variants.
- Increases in incidence with age and affects approximately 3.5 per 1000 women and 0.8 per 1000 men each year.
- Present with a small or moderately sized diffuse goiter, which is characteristically firm or rubbery in consistency. 25% have hypothyroidism.
- In the remainder, serum T4 is normal and TSH normal or raised, but are at risk of developing overt hypothyroidism in future.

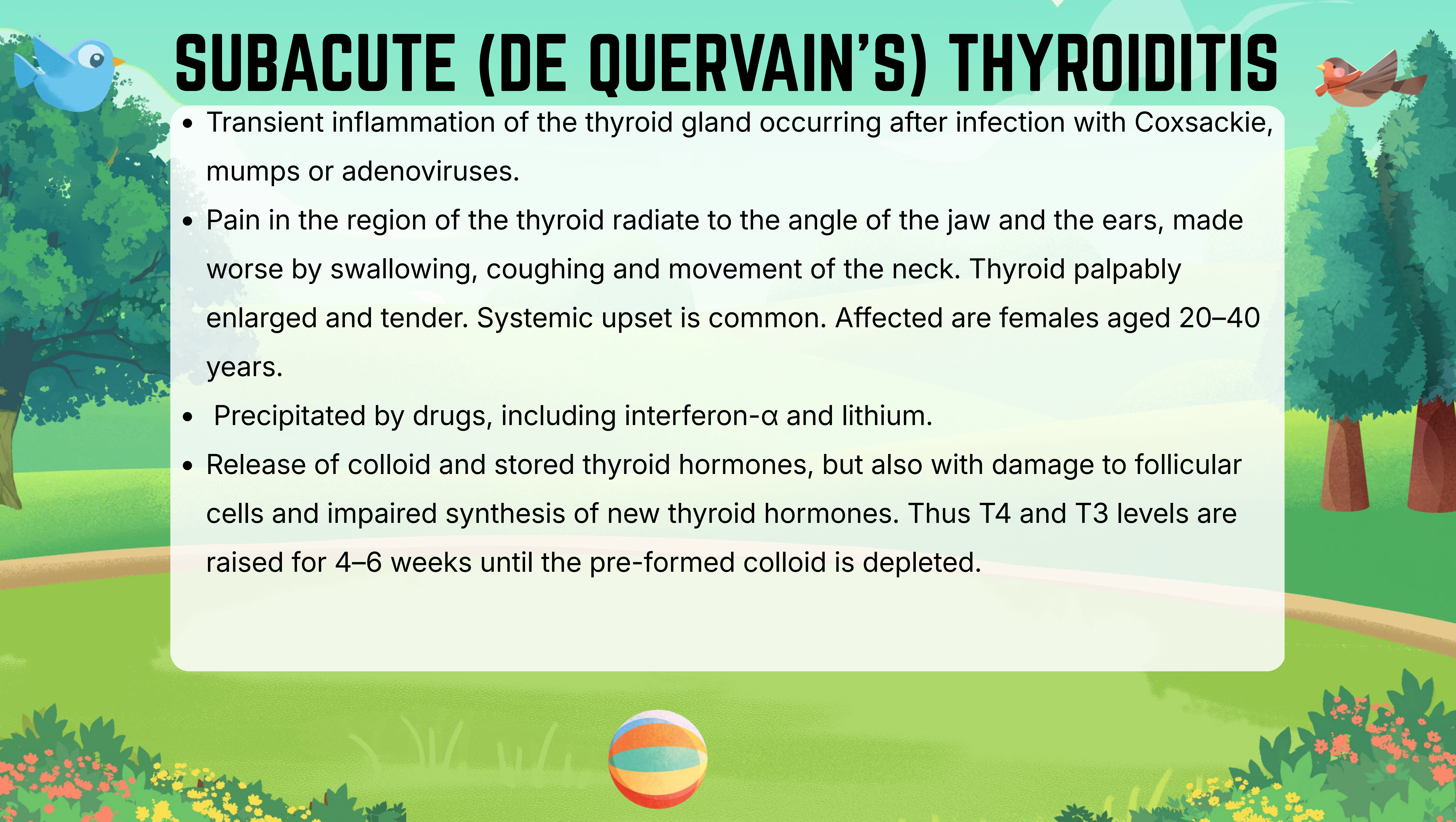


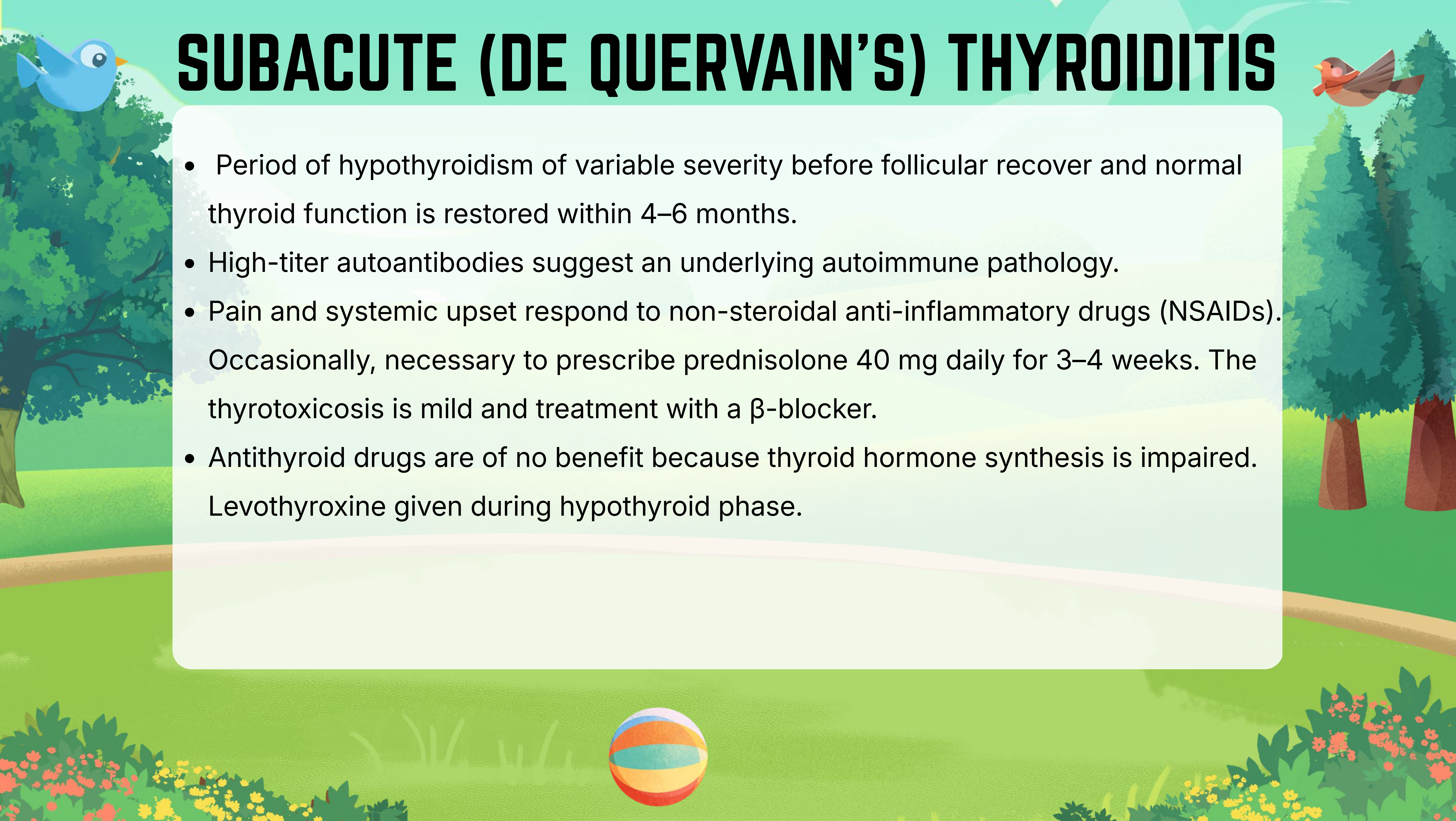
HASHIMOTO'S THYROIDITIS

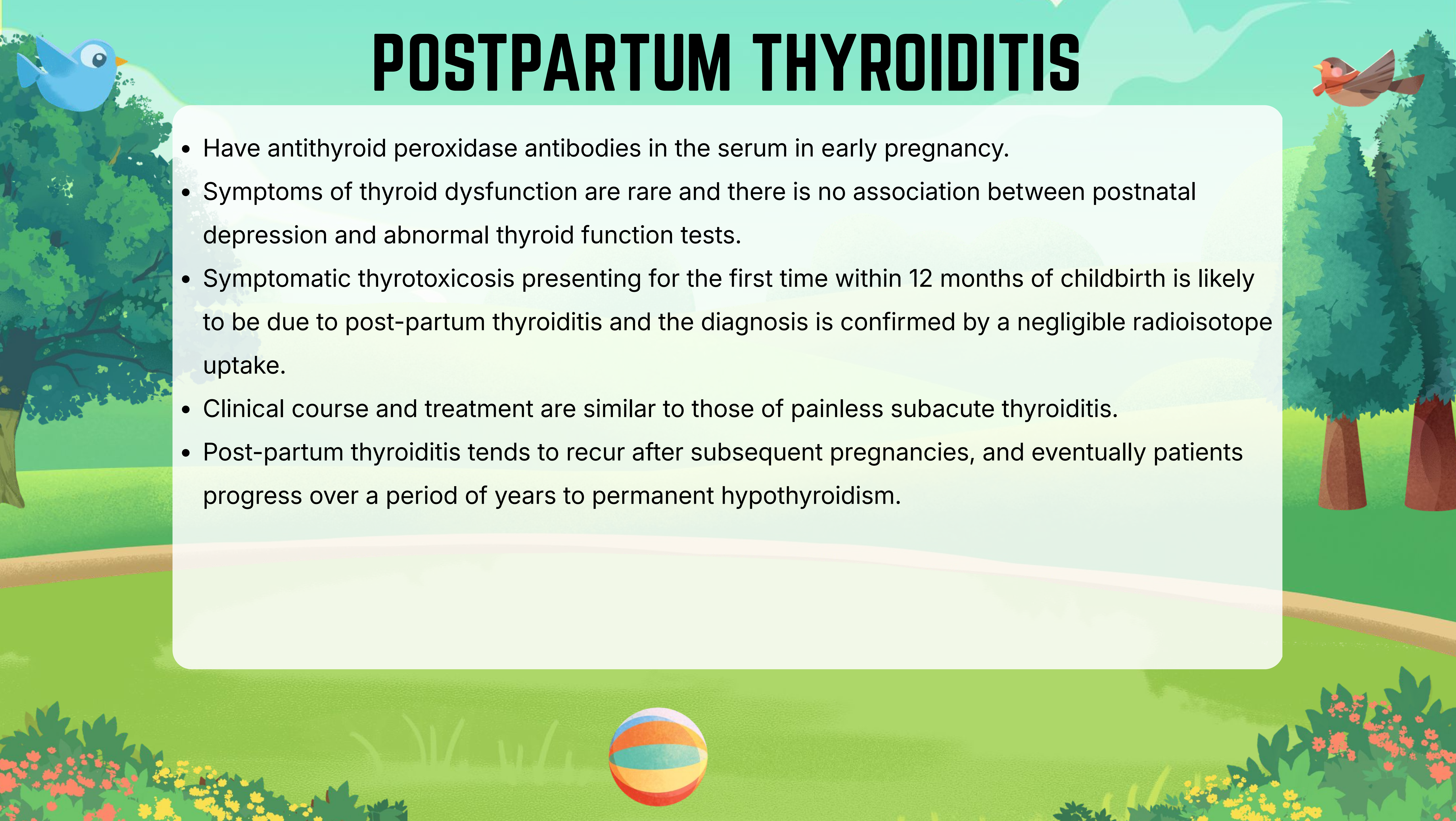
- Antithyroid peroxidase antibodies are present in the serum. Increased risk of thyroid lymphoma.
- Levothyroxine therapy is indicated as treatment for hypothyroidism and also to shrink an associated goiter.
- Dose of levothyroxine should be sufficient to suppress serum TSH to low but detectable levels.




NON-THYROIDAL ILLNESS (SICK EUTHYROIDISM)
Low serum TSH, raised T4 and normal or low T3 in a patient with systemic illness who does not have clinical evidence of thyroid disease.
Abnormalities are caused by decreased peripheral conversion of T4 to T3 (with conversion instead to reverse T3), altered levels of binding proteins and their affinity for thyroid hormones, and often reduced secretion of TSH.
During convalescence, serum TSH concentrations increase to levels found in primary hypothyroidism.
Avoid performing thyroid function tests unless there is clinical evidence of concomitant thyroid disease as interpretation is hard.
Diagnosis should be re-evaluated after recovery.


SUBACUTE (DE QUERVAIN'S) THYROIDITIS
Transient inflammation of the thyroid gland occurring after infection with Coxsackie, mumps or adenoviruses.
Pain in the region of the thyroid radiate to the angle of the jaw and the ears, made worse by swallowing, coughing and movement of the neck. Thyroid palpably enlarged and tender. Systemic upset is common. Affected are females aged 20–40 years.
Precipitated by drugs, including interferon-α and lithium.
Release of colloid and stored thyroid hormones, but also with damage to follicular cells and impaired synthesis of new thyroid hormones. Thus T4 and T3 levels are raised for 4–6 weeks until the pre-formed colloid is depleted.


SUBACUTE (DE QUERVAIN'S) THYROIDITIS
Period of hypothyroidism of variable severity before follicular recover and normal thyroid function is restored within 4–6 months.
High-titer autoantibodies suggest an underlying autoimmune pathology.
Pain and systemic upset respond to non-steroidal anti-inflammatory drugs (NSAIDs). Occasionally, necessary to prescribe prednisolone 40 mg daily for 3–4 weeks. The thyrotoxicosis is mild and treatment with a β-blocker.
Antithyroid drugs are of no benefit because thyroid hormone synthesis is impaired. Levothyroxine given during hypothyroid phase.


POSTPARTUM THYROIDITIS
Have antithyroid peroxidase antibodies in the serum in early pregnancy.
Symptoms of thyroid dysfunction are rare and there is no association between postnatal depression and abnormal thyroid function tests.
Symptomatic thyrotoxicosis presenting for the first time within 12 months of childbirth is likely to be due to post-partum thyroiditis and the diagnosis is confirmed by a negligible radioisotope uptake.
Clinical course and treatment are similar to those of painless subacute thyroiditis.
Post-partum thyroiditis tends to recur after subsequent pregnancies, and eventually patients progress over a period of years to permanent hypothyroidism.

GOITRE

Diffuse

1. Simple/ non-toxic
    a. Physiological- Puberty, pregnancy
    b. Iodine deficiency
2. Toxic goitre: Graves disease
3. Autoimmune/ inflammatory:
    a. Hashimoto's thyroiditis.
    b. Ridel’s thyroditis
4. Dyshormonogenesis

Nodular

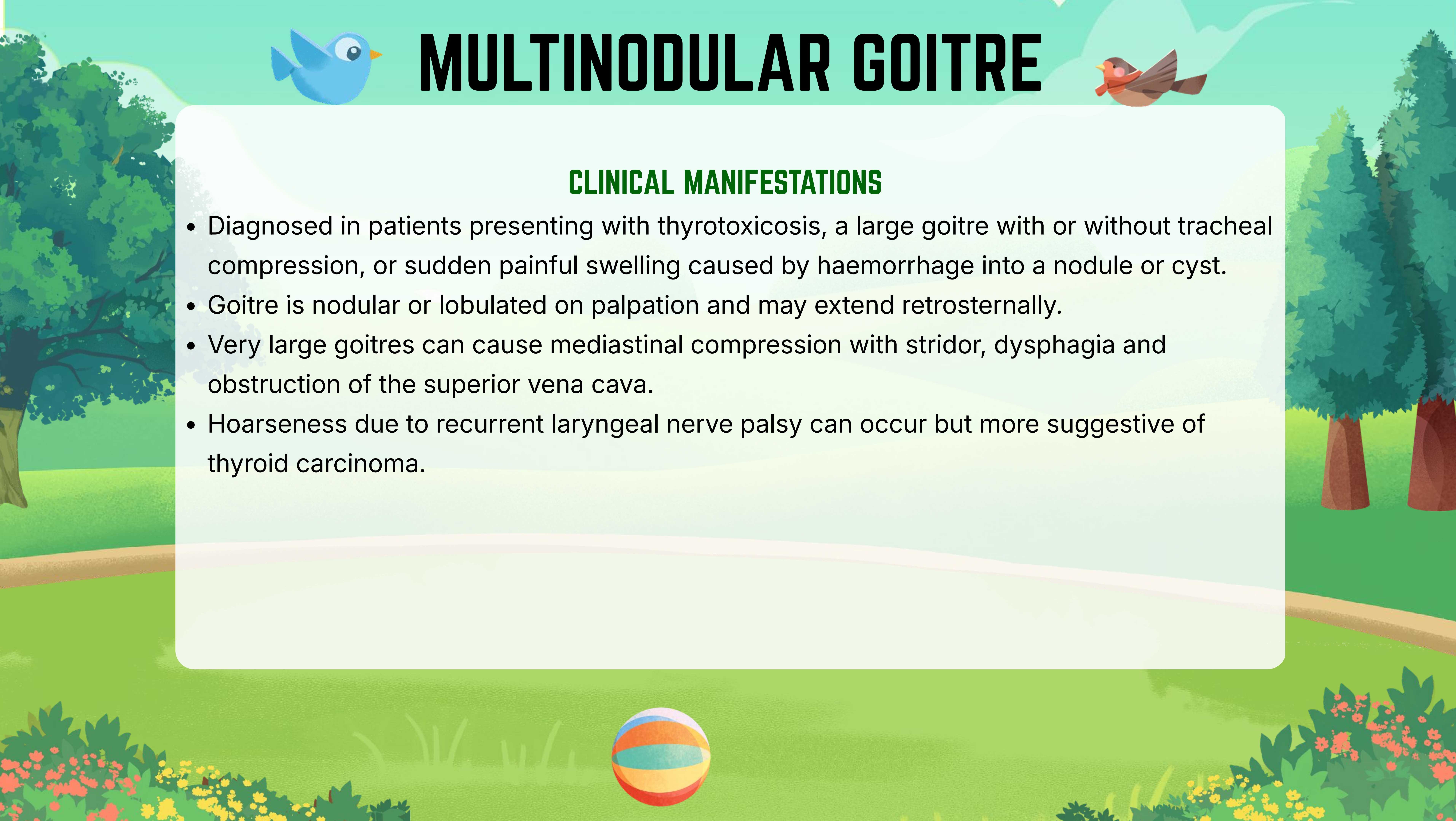
1. Multinodular Goitre, toxic multinodular goitre.
2. Solitary nodular goitre.
3. Cysts
4. Tumors:
    a. Benign: Adenomas
    b. Malignant: Carcinoma and lymphomas
5. Miscellaneous: Sarcoidosis and tuberculosis

SIMPLE DIFFUSE GOITRE
Presents between the ages of 15 and 25 years, often during pregnancy.
Tends to be noticed by friends and relatives rather than the patient.
Occasionally, tight sensation in the neck, particularly when swallowing.
Goitre is soft and symmetrical, and the thyroid enlarged to two or three times normal.
No tenderness, lymphadenopathy or overlying bruit.

SIMPLE DIFFUSE GOITRE
Concentrations of T3 , T4 and TSH are normal.
No thyroid autoantibodies are detected in the serum.
No treatment is necessary and the goitre usually regresses.
In some unknown stimulus to thyroid enlargement persists and recurrent episodes of hyperplasia and involution during the following 10–20 years, the gland becomes multinodular with areas of autonomous function.

Age (in years)	15–25	26–55	> 55
Goitre	Diffuse	Nodular	Nodular
Tracheal compression/ deviation	No	Minimal	Yes
T_3, T_4	Normal	Normal	Raised
TSH	Normal	Normal or undetectable	Undetectable

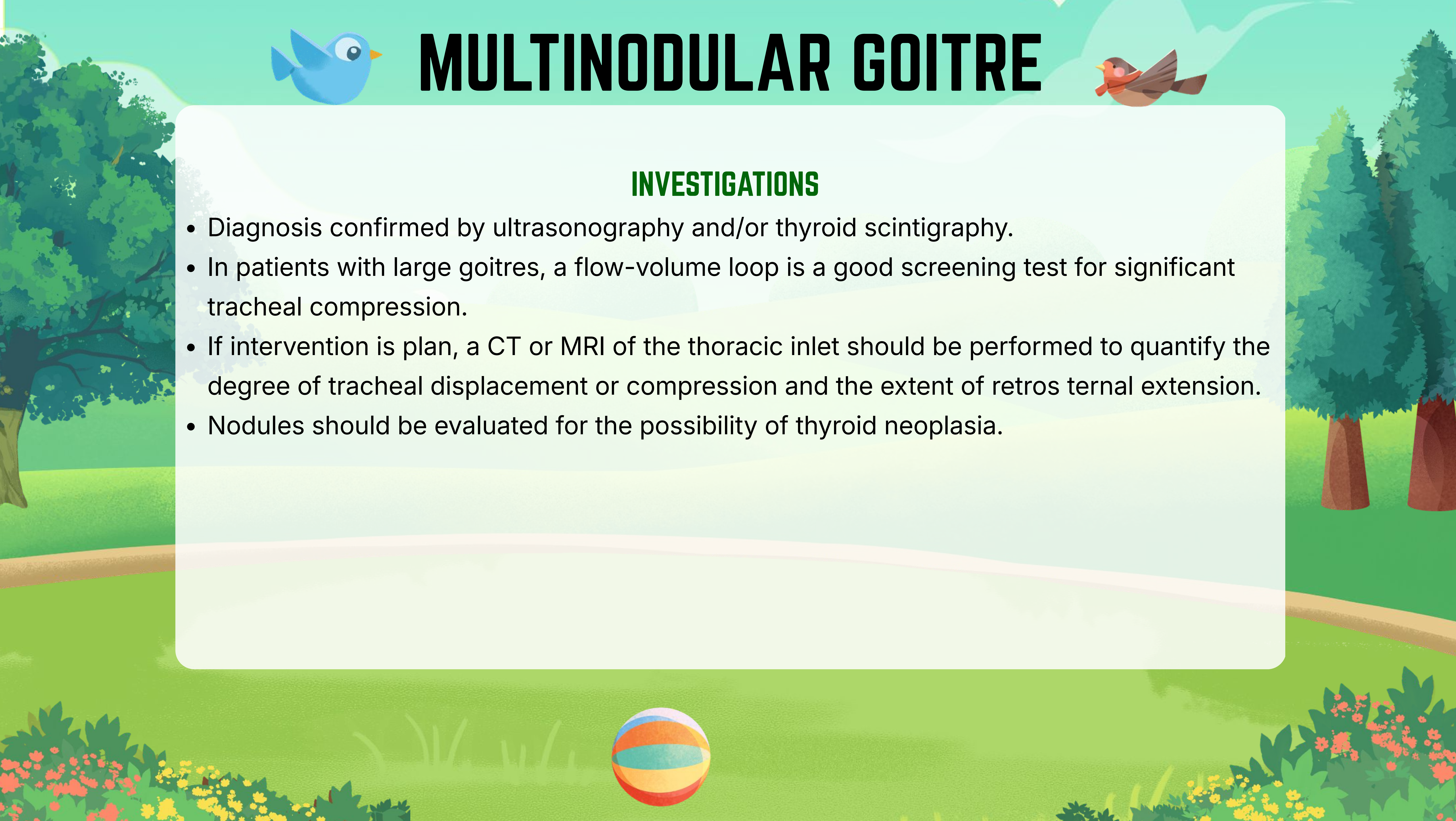
g. 20.11 **Natural history of simple goitre.** (T_3 = triiodothyronine; T_4 = thyroxine; H = thyroid-stimulating hormone)



MULTINODULAR GOITRE

CLINICAL MANIFESTATIONS

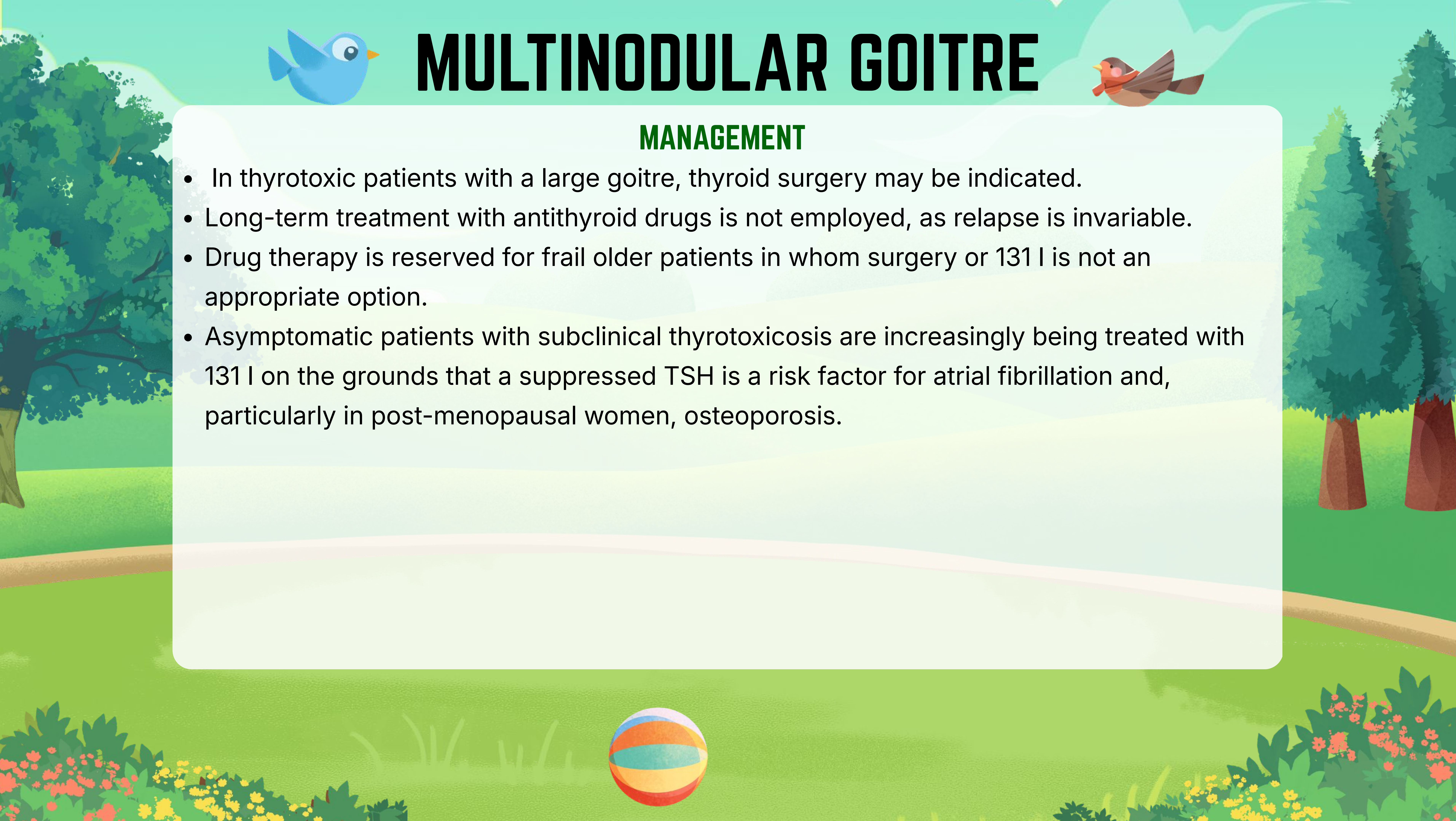
- Diagnosed in patients presenting with thyrotoxicosis, a large goitre with or without tracheal compression, or sudden painful swelling caused by haemorrhage into a nodule or cyst.
- Goitre is nodular or lobulated on palpation and may extend retrosternally.
- Very large goitres can cause mediastinal compression with stridor, dysphagia and obstruction of the superior vena cava.
- Hoarseness due to recurrent laryngeal nerve palsy can occur but more suggestive of thyroid carcinoma.


MULTINODULAR GOITRE
INVESTIGATIONS
Diagnosis confirmed by ultrasonography and/or thyroid scintigraphy.
In patients with large goitres, a flow-volume loop is a good screening test for significant tracheal compression.
If intervention is plan, a CT or MRI of the thoracic inlet should be performed to quantify the degree of tracheal displacement or compression and the extent of retros ternal extension.
Nodules should be evaluated for the possibility of thyroid neoplasia.

MULTINODULAR GOITRE

MANAGEMENT

- Goitre is small, no treatment is necessary but annual thyroid function testing done as the natural history is progression to a toxic multinodular goitre.
- Thyroid surgery is indicated for large goitres that cause mediastinal compression or that are cosmetically unattractive. I131 can result in a significant reduction in thyroid size and value in older patients.
- Levothyroxine therapy is of no benefit in shrinking multinodular goitres in iodine-sufficient countries.
- In toxic multinodular goitre, treatment is usually with 131 I. Iodine uptake is lower than in Graves' disease, so a higher dose may be administered (up to 800 Mbq (approximately 20 mCi)) and hypothyroidism is less common.



MULTINODULAR GOITRE

MANAGEMENT

- In thyrotoxic patients with a large goitre, thyroid surgery may be indicated.
- Long-term treatment with antithyroid drugs is not employed, as relapse is invariable.
- Drug therapy is reserved for frail older patients in whom surgery or 131 I is not an appropriate option.
- Asymptomatic patients with subclinical thyrotoxicosis are increasingly being treated with 131 I on the grounds that a suppressed TSH is a risk factor for atrial fibrillation and, particularly in post-menopausal women, osteoporosis.

THANK YOU
FOR YOUR ATTENTION