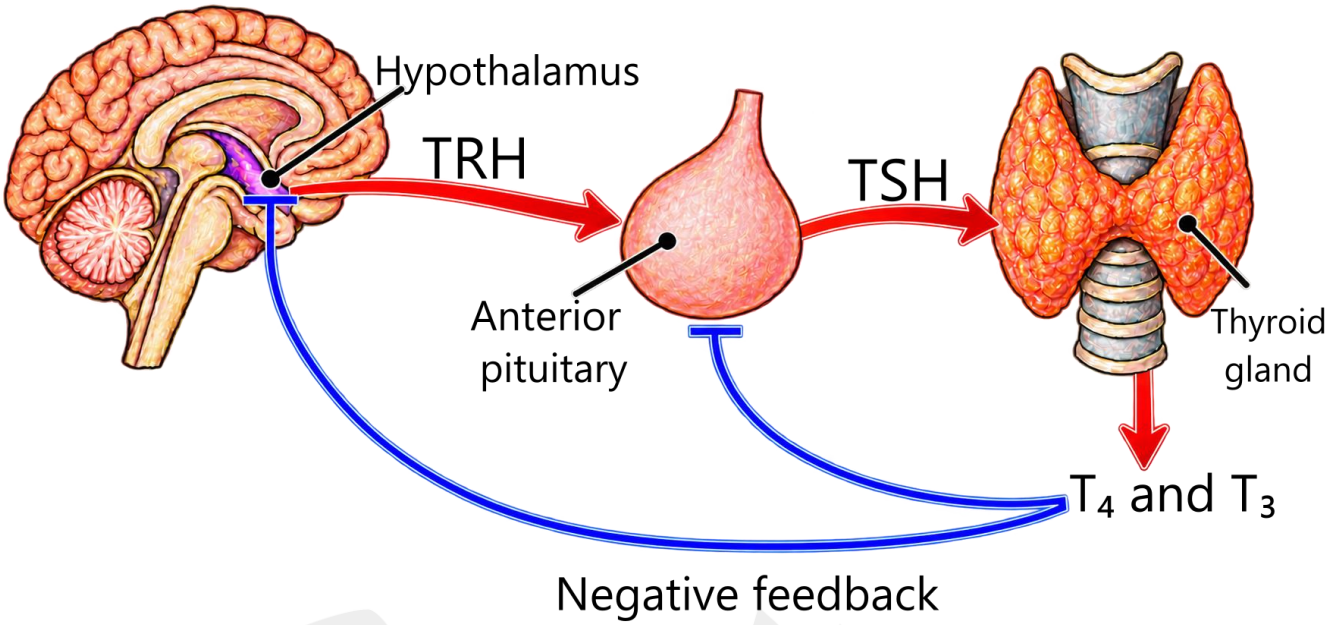
Pediatrics - Topic 30 - Endocrinology

Hypothyroidism in Childhood

Hormone Function and Causes



Thyroid hormone production depends on hypothalamic and pituitary signals and is regulated by negative feedback

- Thyroid hormone supports brain development, linear growth and normal metabolic activity.
- Primary hypothyroidism begins in the thyroid gland, while central hypothyroidism results from inadequate hypothalamic or pituitary stimulation.

Causes by Time of Onset

A. Congenital Causes

- The thyroid may be absent, underdeveloped or located in an abnormal position.
- A normally positioned gland may have an inherited defect in hormone synthesis.
- Iodine deficiency, excessive iodine exposure, maternal medicines or maternal antibodies may cause transient dysfunction.
- Central disease may occur with other pituitary hormone deficiencies and can be missed by a screening programme that measures only TSH.

B. Acquired Causes

- Autoimmune thyroiditis is an important cause in older children and may produce a goitre.
- Other causes include iodine imbalance, thyroid surgery, irradiation and medicines that interfere with thyroid function.
- Pituitary disease should be considered when poor growth accompanies other hormone deficiencies or neurological symptoms.

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Age-Related Clinical Features

1. Newborn and Infant Findings

- An affected newborn may initially look well because maternal thyroid hormone has provided some protection before birth.
- Prolonged jaundice, poor feeding, constipation, a large tongue, a hoarse cry or an umbilical hernia may develop.
- Hypotonia, a large fontanelle and delayed development are additional warning signs.
- Waiting for a typical appearance can delay treatment during a critical period of brain development.

2. Older Children

- Slowing of height gain with preserved or increasing weight is a useful clue.
- Fatigue, cold intolerance, dry skin, constipation, bradycardia and reduced school performance may occur.
- Bone age and puberty may be delayed, although severe longstanding disease can occasionally cause unusual early sexual changes.
- Obesity alone with normal growth does not establish hypothyroidism.

Steps in Screening Interpretation

1. Check the Sample and Timing

- Confirm the infant's identity, gestational age, sample date, test method and the laboratory's recall threshold.
- The normal TSH surge soon after birth can affect an early sample, while prematurity or illness may delay a TSH rise.
- Follow the programme's repeat-sampling plan for premature, low-birth-weight, sick or otherwise high-risk infants.
- Screening thresholds differ between blood-spot and serum assays and should not be treated as interchangeable numbers.

2. Confirm an Abnormal Result Promptly

- Arrange venous free T4 and TSH promptly after an abnormal screen and contact the paediatric endocrine team.
- A clearly low free T4 with raised TSH indicates primary hypothyroidism and needs urgent treatment.
- Low free T4 with an inappropriately normal or low TSH suggests central hypothyroidism, although illness and assay factors also need consideration.
- A borderline result requires an explicit repeat-testing plan rather than being labelled normal without follow-up.

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- Symptoms suggesting hypothyroidism still require evaluation even when the newborn screen was reported as normal.

Components of Thyroid Replacement

1. Starting Levothyroxine

- Start oral levothyroxine promptly after confirmation, ideally within the first two weeks of life for congenital disease.
- Severe congenital hypothyroidism commonly requires an initial dose of 10–15 micrograms/kg once daily under specialist supervision.
- Milder biochemical disease may need a lower starting dose, so the dose must reflect the free T4 result and clinical context.
- Imaging can identify thyroid anatomy but must not delay treatment.
- If central disease is suspected, assess adrenal function and treat associated cortisol deficiency before thyroid replacement when necessary.

2. Safe Administration and Monitoring

Consistent Dosing and Absorption

- Give the prescribed formulation consistently and avoid mixing the entire dose into a full bottle that the infant may not finish.
- Iron, calcium and some foods can reduce absorption, so discuss practical dose separation with the team.

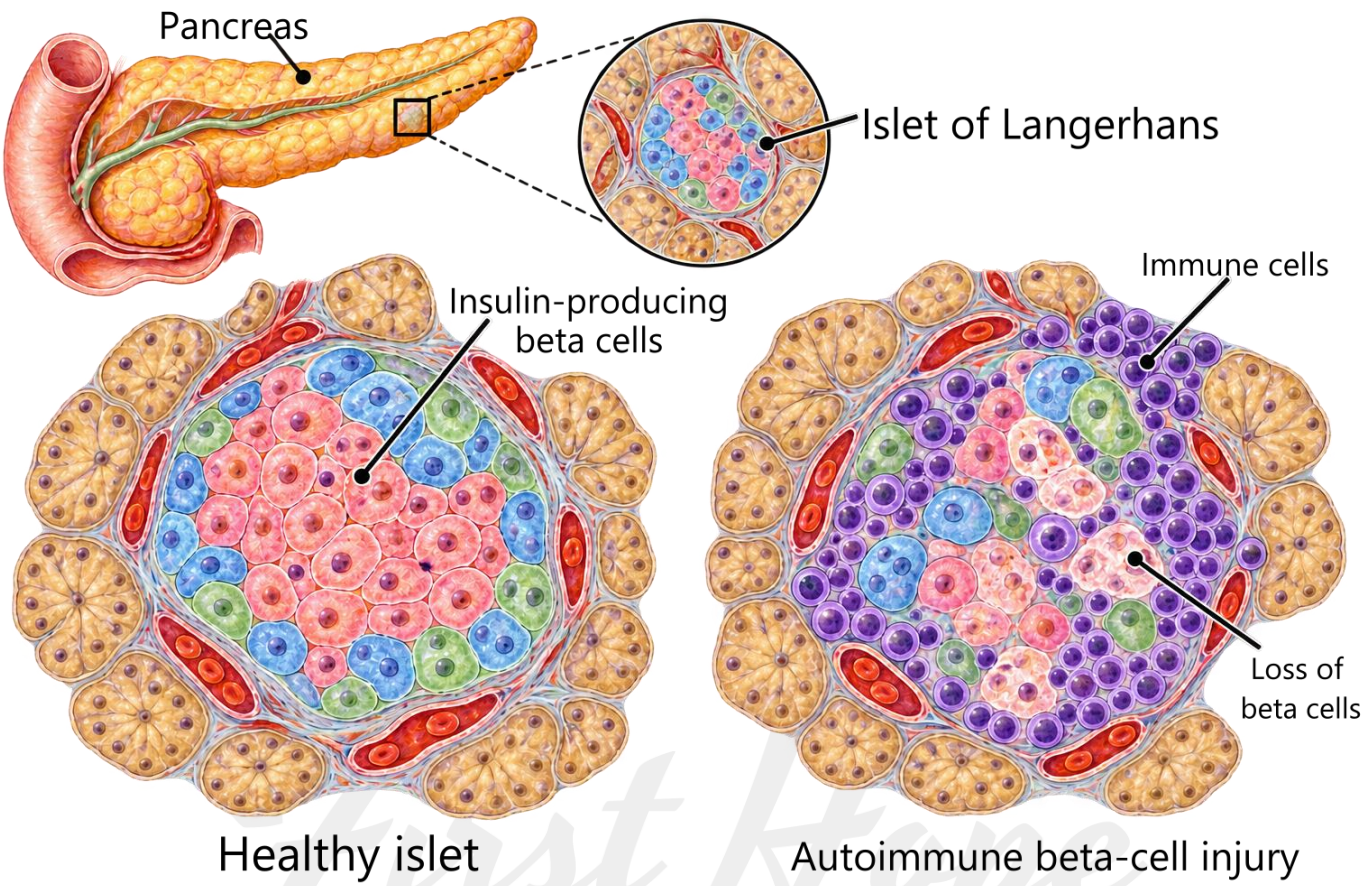
Monitoring and Supervised Reassessment

- Check free T4 and TSH about one to two weeks after treatment begins and repeat frequently until biochemical control is established.
- Continue age-specific monitoring of growth, development, adherence and hormone levels during childhood.
- Older children need individualized replacement, particularly when hypothyroidism has been severe and longstanding.
- A supervised reassessment may establish whether congenital disease is transient when the original diagnosis did not prove permanent disease.
- Do not stop treatment simply because the child appears well while taking levothyroxine.

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Diabetes Mellitus in Children

Clinical Types and Mechanisms



Autoimmune injury reduces insulin-producing beta cells in type 1 diabetes

1. Type 1 Diabetes

- Immune-mediated destruction of pancreatic beta cells leads to progressive insulin deficiency.
- Insulin deficiency causes high blood glucose and, when severe, breakdown of fat with ketone production.
- The condition can occur at any childhood body size and is not caused by eating sweets alone.

2. Type 2 and Other Diabetes

- Type 2 diabetes combines insulin resistance with impaired insulin secretion and increasingly affects adolescents.
- Obesity, acanthosis nigricans, family history and associated metabolic disease support suspicion but do not prove the type.
- Monogenic diabetes should be considered with diabetes in early infancy, a strong multigenerational pattern or atypical findings.
- Pancreatic disorders, cystic fibrosis, endocrinopathies and medicines such as glucocorticoids can also cause diabetes.

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- Diabetes diagnosed before six months usually warrants genetic evaluation rather than an automatic label of type 1 disease.

Symptoms and Diagnostic Criteria

Symptoms and Ketoacidosis Warning Signs

- Polyuria, polydipsia and weight loss are the main presenting symptoms.
- A previously dry child may develop bedwetting, and an infant may have unusually heavy wet nappies.
- Fatigue, blurred vision, recurrent candidiasis or poor growth may accompany hyperglycaemia.
- Vomiting, abdominal pain, deep breathing or drowsiness suggest ketoacidosis and need emergency assessment.

Test	Diagnostic threshold
Fasting plasma glucose	At least 126 mg/dL after at least eight hours without calories.
Two-hour oral glucose tolerance test	At least 200 mg/dL after 1.75 g/kg glucose, up to 75 g.
Random plasma glucose	At least 200 mg/dL with classic symptoms or a hyperglycaemic crisis.
Laboratory HbA1c	At least 6.5% using a validated standardized assay.

Confirmation and Classification

- Without unequivocal symptomatic hyperglycaemia, confirm an abnormal diagnostic result with repeat or additional laboratory testing.
- Use a laboratory plasma glucose result for diagnosis and do not delay emergency care while waiting for confirmation in a sick child.
- HbA1c can be misleading with altered red-cell survival, some haemoglobin variants or rapidly evolving diabetes.
- Islet autoantibodies and selected C-peptide or genetic tests help classify diabetes when the clinical type is uncertain.

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