Notes for the Practical lesson in Internal Medicine

Iodine deficiency disorder (IDD) of the thyroid gland (1) & Thyrotoxicosis (2)

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http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3063534/
Plan of the Notes

• Iodine deficiency disorder (IDD) of the thyroid gland (1)
• Thyrotoxicosis (2)
• Clinical Endocrinology’ Tests
• Recommended literature
1) IDD of the thyroid gland

How much iodide is taken up by thyroid glands for synthesis of the thyroid hormones?
1) IDD of the thyroid gland: Iodine

- Iodine is a micronutrient of crucial importance for the health and well-being of all individuals
- Iodine is a trace element, just 5 gm of which are sufficient to meet the life-time needs of an individual with a life-span of 70 years
- Iodine is mostly concentrated in thyroid gland
- A healthy adult body contains 15-20 mg of iodine, 70-80% of which is stored in the thyroid gland
- Daily intake of iodine by an individual amounts to 500 micrograms; daily physiological requirement during adult life is 150 micrograms; during pregnancy and lactation period is 200 micrograms; and during neonatal period is 40 micrograms
- Normally about 120 micrograms of iodide are taken up by the thyroid gland for the synthesis of thyroid hormones

http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3063534/
1) IDD of the thyroid gland

What are the sources of iodine?
What is the cause of endemic spread of IDD?
1) IDD of the thyroid gland: the cause of endemic distribution

- Oceans are the world's main repositories of iodine and very little of earth's iodine is actually found in the soil
- The deposition of iodine in the soil occurs due to volatilization from ocean water, a process aided by ultraviolet radiation
- The coastal regions of the world are much richer in iodine content than the soils further inland; here the problem gets more compounded by continuous leeching of iodine from the soil
- The crops grown in such soil remain iodine deficient; even ground water in these areas is deficient in iodine
- This explains the endemic distribution of Iodine Deficiency Disorder (IDD) in the world
- In 1998, one-third of the world's population lived in iodine deficient areas
- The two major factors responsible for IDD are inadequate iodine intake and inadequate iodine utilization

http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3063534/
1) IDD of the thyroid gland: the cause of endemic distribution

IDD occurs primarily in mountainous regions such as the Himalayas, the European Alps, and the Andes, where iodine has been washed away by glaciation and flooding

1) IDD of the thyroid gland

Which thyroid hormones do you know?
1) IDD of the thyroid gland: iodine metabolism

- Iodine is mostly obtained from food sources particularly vegetables grown on iodine-rich soil; the remaining requirement is met from drinking water
- Thyroid gland plays a central role in the metabolism of iodine
- Iodine trapping is the first step in the metabolism of iodine
- Synthesis and secretion of thyroglobulin is the second step
- The third step is the oxidation of iodide with iodination of tyrosine and formation thyroxine (T4) hormone and triiodothyronine (T3) hormone
- In the blood stream, T4 and T3 may circulate in the bound or free form; whereas 99 percent of T4 and T3 circulate in the bound form, less than 1 percent circulates in an unbound (biologically active) form
- About 80 percent of circulating T3, the most active thyroid hormone is derived from peripheral deiodination of T4 hormone

http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3063534/
1) IDD of the thyroid gland

How thyroid hormones secretion is regulated?
1) IDD of the thyroid gland: feed-back in thyroid secretion regulation by pituitary gland through TSH

- Thyroid secretion is regulated by pituitary gland through TSH which operates on a feed-back mechanism tuned to T4 level in blood
- A fall in T4 level stimulates the pituitary to increase its TSH secretion which in turn stimulates the thyroid gland to release T4 in circulation to maintain normal level of the hormone in blood
- Thyroid gland secretes 80 micrograms of iodine in the form of T3 and T4 hormones per day; 40 micrograms of iodine secreted appear in extracellular fluid (ECF) per day
- T3 and T4 are metabolized in liver which releases about 60 mcg of iodine into ECF and 20 mcg of iodine into the bile to be excreted in stools
- On an average, 480 mcg of iodine get excreted in urine and 20 micrograms in stools per day

http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3063534/
1) IDD of the thyroid gland: feed-back in thyroid secretion regulation by pituitary gland through TSH
1) IDD of the thyroid gland

What are the main symptoms of IDD?
What are the complications of severe iodine deficiency during pregnancy?
Why mild iodine deficiency is complicated with learning disabilities in children?
1) IDD of the thyroid gland: consequences

- The WHO defines iodine deficiency as a median urinary iodine concentration less than 50 μg/L in a population.
- Iodine deficiency is associated with goiter and hypothyroidism.
- When severe iodine deficiency occurs during pregnancy, it is associated with cretinism and increased neonatal and infant mortality.
- In addition, mild iodine deficiency is associated with thyroid enlargement and learning disabilities in children.
- Adult population inhabiting the iodine deficient areas is characterized by a high degree of apathy, reduced mental functioning, lack of physical energy and reduced work output, all contributing to poor quality of life.
- Iodine deficiency has emerged as a socio-medical problem of vast dimensions associated with physical and mental retardation, neurological disorders, feeble mindedness, low educability, poor performance, social handicaps, dependability and disfigurement.

http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3063534/
1) IDD of the thyroid gland

Can you list spectrum of IDD?
1) IDD of the thyroid gland: spectrum

<table>
<thead>
<tr>
<th>Stage</th>
<th>Conditions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fetus</td>
<td>Miscarriage, Stillbirths, Congenital anomalies, Increased perinatal morbidity, and mortality, Endemic cretinism</td>
</tr>
<tr>
<td>Neonate</td>
<td>Neonatal goiter, Neonatal hypothyroidism, Endemic mental retardation</td>
</tr>
<tr>
<td></td>
<td>Increased susceptibility of the thyroid gland to nuclear radiation</td>
</tr>
<tr>
<td>Child and adolescent</td>
<td>Goiter(Subclinical) hypothyroidism, Impaired mental function, Retarded physical development, Increased susceptibility of the thyroid gland to nuclear radiation</td>
</tr>
<tr>
<td>Adult</td>
<td>Goiter with its complications, Hypothyroidism, Impaired mental function, Spontaneous hyperthyroidism in the elderly, Iodine-induced hyperthyroidism, Increased susceptibility of the thyroid gland to nuclear radiation</td>
</tr>
</tbody>
</table>

[www.thyroidmanager.org/...iodine-deficiency-disorders](http://www.thyroidmanager.org/...iodine-deficiency-disorders)
1) IDD of the thyroid gland

What types of goitre do you know?
1) IDD of the thyroid gland: goiter

- Goiter is the most obvious manifestation of iodine deficiency.
- Low iodine intake leads to reduced thyroxine (T4) and triiodothyronine (T3) production, which results in increased thyrotropin (TSH) secretion in an attempt to restore T4 and T3 production to normal.
- The goiter is initially diffuse but eventually becomes nodular because the cells in some thyroid follicles proliferate more than others.
- In regions of iodine deficiency, children and adolescents generally have diffuse goiters, while adults who lived in conditions of longstanding iodine deficiency have nodular goiter.
- For many individuals, iodine-deficiency goiter is only a cosmetic problem, however, particularly in older adults, it may be large enough to cause compression of the trachea or esophagus.
1) IDD of the thyroid gland: goiter

WHO classification of goiter’ grade

- 0 - no palpable or visible
- 1 - palpable but not visible when the neck is in the normal position, thyroid nodules in a thyroid which is otherwise not enlarged fall into this category
- 2 - clearly visible when the neck is in a normal position and is consistent with an enlarged thyroid when the neck is palpated
1) IDD of the thyroid gland: goiter

A young girl with a soft diffuse goitre

http://www.thyroidmanager.org/chapter/the-iodine-deficiency-disorders/
1) IDD of the thyroid gland: goiter

An elderly woman with a huge, longstanding multinodular goiter
1) IDD of the thyroid gland

Can you list symptoms of cretinism?
1) IDD of the thyroid gland: cretinism

- Cretinism is a condition associated with iodine deficiency and goiter, commonly characterised by mental deficiency, deafness, squint, disorders of stance and gait, stunted growth and hypothyroidism
- As a result of restricted diet, isolation, intermarriage, etc., as well as low iodine content in their food, children often had peculiar stunted bodies and retarded mental faculties, a condition later known to be associated with thyroid deficiency
- According to World Health Organization, in 2007, nearly 2 billion individuals had insufficient iodine intake, a third being of school age

https://en.wikipedia.org/wiki/Iodine_deficiency
1) IDD of the thyroid gland: cretinism

The appearance of Cretinism sufferers
1) IDD of the thyroid gland

How do you diagnose iodine deficiency?
1) IDD of the thyroid gland: diagnosis

- The diagnostic workup of a suspected iodine deficiency includes signs and symptoms as well as possible risk factors (thyroid size is a sensitive marker for iodine deficiency)

- A 24-hour urine iodine collection is a useful medical test, as approximately 90% of ingested iodine is excreted in the urine
  - For the standardized 24-hour test, a 50 mg iodine load is given first, and 90% of this load is expected to be recovered in the urine of the following 24 hours.
  - Recovery of less than 90% is taken to mean high retention, that is, iodine deficiency
  - The recovery may however be well less than 90% during pregnancy, and an intake of goitrogens can alter the test results

- In regions of iodine deficiency, the frequency of supranormal TSH concentrations (>5mU/L) in blood spots collected as part of neonatal screening programs is higher than in iodine sufficient areas and roughly correlates with the severity of iodine deficiency

1) IDD of the thyroid gland

How is iodine deficiency prevented and treated? Are there any problems with taking too much iodine?
1) IDD of the thyroid gland: prevention and treatment

• Iodine supplementation in areas deprived of iodine rich food is viewed as the most cost effective solution to address the problem of IDD

• Iodine deficiency can be corrected by adding iodine to dietary media like salt, oil, water, sauces etc.

• The methods of proven value for mass use are iodized salt and iodized oil

• To this end, fortification of salt with iodine has been identified and considered to be the most suitable method of fortification

• Being not only technically feasible, this food item is consumed worldwide in standard amounts by all sections of the population
1) IDD of the thyroid gland: prevention and treatment

- The World Health Organization (WHO) recommends 90 mcg of iodine daily for infants and children up to five years, 120 mcg for children 6 to 12 years, 150 mcg daily for children ≥12 years and adults, and 250 mcg daily during pregnancy and lactation.

- Too high iodine intake (over dosage of iodine supplements) can have toxic side effects:
  - It can lead to hyperthyroidism and consequently high blood levels of thyroid hormones (hyperthyroxinemia).
  - In case of extremely high single-dose iodine intake, typically a short-term suppression of thyroid function (Wolff–Chaikoff effect) occurs.
2) Thyrotoxicosis Clinical Case
(from the American Thyroid Clinic Website)

- The patient is a 22 year old female college student from Waco, referred to the center by an Austin endocrinologist.
- She was diagnosed with Graves’ Disease in January of this year and started on Tapazole (Methimazole) and Toprol (Metoprolol succinate).
- Mild to moderate exophthalmos was present.
- She and her family were already well informed about hyperthyroidism, and she was well versed in the various treatment options available to her.
- As a young female of child bearing age, she rejected the idea of radiation exposure and she also did not want to take long term anti thyroid medication.
- Total Thyroidectomy was the treatment plan that she and her family chose.
2) Thyrotoxicosis Clinical Case
(from the American Thyroid Clinic Website)
2) **Thyrotoxicosis Clinical Case**  
*(from the American Thyroid Clinic Website)*

**Methimazole**

- Methimazole is used to treat hyperthyroidism
- Methimazole inhibits the thyroperoxidase, which normally acts in thyroid hormone synthesis, facilitating iodine's addition to tyrosine residues on the hormone precursor thyroglobulin, a necessary step in the synthesis of triiodothyronine (T3) and thyroxine (T4)
- Dosage: 5 mg ; 10 mg ; 15 mg ; 20 mg
- Methimazole usually is taken three times a day, approximately every 8 hours, with food
- Side effects: skin rash, itching, abnormal hair loss, upset stomach, vomiting, loss of taste, abnormal sensations (tingling, prickling, burning, tightness, and pulling), swelling, joint and muscle pain, drowsiness, dizziness, decreased white blood cells, decreased platelets
2) Thyrotoxicosis Clinical Case
(from the American Thyroid Clinic Website)

Metoprolol succinate

• A beta₁-selective (cardioselective) adrenoceptor blocking agent, for oral administration, available as extended-release tablets

• Metoprolol succinate has been formulated to provide a controlled and predictable release of metoprolol for once-daily administration.

• The tablets comprise a multiple unit system containing metoprolol succinate in a multitude of controlled release pellets

• Each pellet acts as a separate drug delivery unit and is designed to deliver metoprolol continuously over the dosage interval

• The tablets contain 23.75, 47.5, 95 and 190 mg of metoprolol succinate equivalent to 25, 50, 100 and 200 mg of metoprolol tartrate, USP, respectively
2) **Thyrotoxicosis** Clinical Case  
(from the American Thyroid Clinic Website)

**Lab/Imaging:**
- Jan/03
  - T4 25.2 (n=4.4–12.5)
  - T3 Uptake 52.5 (n=20-38.5)
  - TSH <.01
- Feb/03
  - Thyroid scan & Uptake – 24 hour uptake 85%, diffusely enlarged gland
- Jun/03
  - Free T4 1.8 (n=0.8-1.8)
  - Free T3 690 (n=230-420)
  - TSH <.01
2) Thyrotoxicosis Clinical Case
(from the American Thyroid Clinic Website)

Clinical Course

• With return of her thyroid function tests to normal and near normal, the patient was started on SSKI (a saturated solution of potassium iodide) 10 days prior to surgery.
• Total thyroidectomy was performed as an outpatient in early July.
• Her post operative calcium levels were normal and she was started on a low dose of Synthroid (levothyroxine) and referred back to her endocrinologist for further care and thyroid hormone maintenance.
Levothyroxine

- A synthetic thyroid hormone that is chemically identical to thyroxine (T₄)
- Levothyroxine is used to treat hypothyroidism
- Levothyroxine for systemic administration is available as an oral tablet, an intramuscular injection, and as a solution for intravenous infusion
- Dosages vary according to the age groups and the individual condition of the patient, body weight and compliance to the medication and diet
2) Thyrotoxicosis Clinical Case
(from the American Thyroid Clinic Website)

Final Pathology

- Total thyroidectomy: changes consistent with Graves’ Disease; left sided parathyroid tissue was identified with the specimen. An adjacent lymph node was benign.
2) Thyrotoxicosis Clinical Case
(from the American Thyroid Clinic Website)

Comments

• Total thyroidectomy is well proven as a quick, safe, and efficient treatment plan for patients with Graves’ Disease.
• Of all the therapeutic options, total thyroidectomy probably offers the best opportunity for improvement and perhaps even resolution of the oculopathy that sometimes accompanies this disorder.
• Radioactive iodine therapy and long term anti thyroid medication are also acceptable treatment options.
• As with most therapies, the treatment plan should be tailored to the patient’s best interests.
2) Thyrotoxicosis

What causes of thyrotoxicosis do you know?
2) **Thyrotoxicosis: etiology**

- The second most prevalent endocrine disorder
- Effects women eight times more frequently than men
- May appear after an emotional shock, stress, or an infection
- Graves’ disease: excessive output of thyroid hormones
- Other common causes of hyperthyroidism include thyroiditis and excessive ingestion of thyroid hormone (toxic adenoma, Plummer's disease (toxic multinodular goiter))
2) Thyrotoxicosis

Please list the symptoms of hyperthyroidism
2) Thyrotoxicosis: clinical symptoms

**Emotional** (nervousness, irritability)

**Exophthalmos**

**Goitre** (diffuse enlargement of thyroid, bruit)

**Thyroid dermopathy** (pretibial myxedema & TSH-R Ab↑)

**Heat intolerance**

**Cardiovascular** (palpitation, atrial fibrillation, CHF, dyspnea, angina)

**Gastrointestinal** (weight, appetite, diarrhea)

**Reproductive** (amenorrhea, oligo- menorrhea, infertility, gynecomastia)

**Bone** (Osteoporosis, Thyroid acropachy)

**Neuromuscular** (nervousness, tremor, emotional labiality, proximal myopathy, myasthenia gravis, hyperreflexia, clonus, periodic hypokalemic paralysis)

**Skin** (pruritus, onycholysis, vitiligo, hair thinning, palmar erythema, spider nevi)
2) **Thyrotoxicosis**: clinical symptoms

- Bulging eyes
- Goiter
- Thick or red skin on the shins
2) Thyrotoxicosis

When does thyroid storm occur?
2) **Thyrotoxicosis:** thyroid storm (crisis)

- Occurs in a severely hyperthyroid patient caused by a precipitating event such as:
  - Infection
  - Surgical stress
  - Stopping antithyroid medication in Graves’ disease
2) **Thyrotoxicosis**

What clinical signs of thyroid storm do you know?
2) **Thyrotoxicosis:** thyroid storm (crisis)

- Clinical clues
  - fever $\rightarrow$ hyperthermia
  - marked anxiety or agitation $\rightarrow$ coma
  - anorexia
  - tachycardia $\rightarrow$ tachyarrhythmias
  - pulmonary edema/cardiac failure
  - hypotension $\rightarrow$ shock
  - confusion
2) **Thyrotoxicosis**: diagnosis

Positive thyroid nodule on PET CT imaging
2) **Thyrotoxicosis: diagnosis**

The hyperplastic thyroid nodule, also termed a colloid or adenomatous nodule

https://www.med-ed.virginia.edu/courses/rad/Thyroid_Ultrasound/images/Adenomatous%20nodule.jpg
2) **Thyrotoxicosis: diagnosis**

The thyroid nodule ultrasound-guided needle biopsy

http://www.jaypeejournals.com/eJournals/_eJournals%5C192%5C2011%5CSeptember-December%5Cimages/4_img_2.jpg
http://www.jaypeejournals.com/eJournals/_eJournals%5C192%5C2011%5CSeptember-December%5Cimages/4_img_3.jpg
2) Thyrotoxicosis

Please list Blood’ Thyroid Panel components
2) Thyrotoxicosis: diagnosis

Blood’ Thyroid Panel

- Thyroid-Stimulating Hormone (TSH) evaluates overall thyroid function
- Total Thyroxine (T4) evaluates the total amount of T4 produced by the thyroid gland
- Free Thyroxine (T4) evaluates the amount of T4 available to the cells and tissues
- Free Tri-iodothyronine (T3) measures the amount of T3 (the active form of the hormone) available to the cells and tissues
2) Thyrotoxicosis

What changes in thyroid hormones indicate hyperthyroidism?
## 2) Thyrotoxicosis: diagnosis

<table>
<thead>
<tr>
<th>TSH</th>
<th>FREE T4 (FT4)</th>
<th>FREE OR TOTAL T3</th>
<th>PROBABLE INTERPRETATION</th>
</tr>
</thead>
<tbody>
<tr>
<td>High</td>
<td>Normal</td>
<td>Normal</td>
<td>Mild (subclinical) hypothyroidism</td>
</tr>
<tr>
<td>High</td>
<td>Low</td>
<td>Low or normal</td>
<td>Hypothyroidism</td>
</tr>
<tr>
<td>Low</td>
<td>Normal</td>
<td>Normal</td>
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<td><strong>Low</strong></td>
<td><strong>High or normal</strong></td>
<td><strong>High or normal</strong></td>
<td><strong>Hyperthyroidism</strong></td>
</tr>
<tr>
<td>Low</td>
<td>Low or normal</td>
<td>Low or normal</td>
<td>Non-thyroidal illness; rare pituitary (secondary) hypothyroidism</td>
</tr>
<tr>
<td>Normal</td>
<td>High</td>
<td>High</td>
<td>Thyroid hormone resistance syndrome (a mutation in the thyroid hormone receptor decreases thyroid hormone function)</td>
</tr>
</tbody>
</table>

2) Thyrotoxicosis: diagnosis

- TSH↓, High FT4↑ and/or FT3↑
  - If eye signs are present, the diagnosis of Graves’ disease can be made without further tests
  - If eye signs are absent and the patient is hyperthyroid with or without a goitre, a radioiodine uptake test should be done
  - Radioiodine uptake and scan (diffuse increased uptake)
  - TSH-R Ab is specific for Graves’ disease, may be useful in the “apathetic” hyperthyroid patient or who presents with unilateral exophthalmos without obvious signs or laboratory manifestations of Graves’ disease
2) Thyrotoxicosis

Please list components of antithyroid pharmacotherapy
2) **Thyrotoxicosis: treatment**

- Treatment includes symptom relief, as well as antithyroid pharmacotherapy, beta blockers, radioactive iodine-131 ($^{131}$I) therapy (the preferred treatment of hyperthyroidism among US thyroid specialists), or thyroidectomy.

- Antithyroid medications are not effective in thyrotoxicosis in which scintigraphy shows low uptake of iodine-123 ($^{123}$I), as in patients with subacute thyroiditis, because these cases result from release of preformed thyroid hormone.

- Adverse effects: agranulocytosis or hepatitis from the antithyroid medications.

- Discussing adverse effects before starting therapy, patients should be given written or documented verbal instruction to the effect that if they develop high fever (> 39.5°C) or a severe sore throat, they should stop the medication and seek medical attention.

2) **Thyrotoxicosis**

Please list antithyroid medications
2) **Thyrotoxicosis**: antithyroid medications

- **Examples**

<table>
<thead>
<tr>
<th>Generic Name</th>
<th>Brand Name</th>
</tr>
</thead>
<tbody>
<tr>
<td>methimazole</td>
<td>Tapazole</td>
</tr>
<tr>
<td>propylthiouracil</td>
<td>Propyl-Thyracil or PTU</td>
</tr>
</tbody>
</table>

- **How It Works**

  - Antithyroid medicines cause thyroid gland to make less thyroid hormone
  - Antithyroid medicine works more quickly than radioactive iodine therapy
  - It also does not permanently damage thyroid gland

2) Thyrotoxicosis

Please list medications for thyroid storm treatment
2) **Thyroid storm: treatment**

- Antiadrenergic drugs
- Thionamides
- Iodine preparations
- Glucocorticoids
- Bile acid sequestrants
- Treatment of the underlying condition
- Rarely plasmapheresis
2) **Thyrotoxicosis**: Clinical Case 2 (1)

- A 72 year old woman was referred because of typical complaints of hyperthyroidism, including weight loss of about 5 kg during the previous 6 months in spite of a normal appetite, excess sweating, fatigue and several soft stools a day.

- There was no history of thyroid disease in her family or her past and no evidence of iodine deficiency in her area.

- During the previous year she had been taking 4-6 tablets of sea-kelp (Vitalia, Norway), each tablet containing 0.7mg of iodine, a total of 2.8-4.2 mg iodine per day.

[http://pmj.bmj.com/content/62/729/661.full.pdf/](http://pmj.bmj.com/content/62/729/661.full.pdf/)
2) **Thyrotoxicosis**: Clinical Case 2 (2)

- Physical examination revealed diffuse enlargement of the thyroid without a bruit
- Thyroid function tests (Table I) were compatible with a diagnosis of hyperthyroidism
- The radioactive uptake (131I uptake) value was very low after 2 and 24 hours and a thyroid scan with technetium 99m was normal
- There was complete suppression of thyrotropin stimulating hormone (TSH) after injection of 200 gg thyrotropin releasing hormone (TRH)
- Thyroid antibodies were undetectable
- Erythrocyte sedimentation rate and routine haematology and biochemistry were normal
2) **Thyrotoxicosis**: Clinical Case 2 (3)

- The patient was advised to stop the sea-kelp tablets after which her complaints disappeared, she gained weight and her thyroid gland became no longer palpable.
- Six months after stopping the sea-kelp tablets plasma levels of thyroid hormones had returned to normal and a normal TSH.
Clinical Endocrinology’ Tests
A 35-year-old female patient has gained 20 kg weight within a year with the normal diet. She complains of chill, sleepiness, shortness of breath. The patient’s mother and sister are corpulent. Objectively: height - 160 cm, weight - 92 kg, BMI - 35,9. Obesity is uniform, there are no striae. The face is amimic. The skin is dry. The tongue is thickened. Heart sounds are muffled. HR- 56/min, AP- 140/100 mm Hg. The patient has constipations, amenorrhea for 5 months. TSH-28 mkME/l (normal rate - 0,32-5). Craniogram shows no pathology. What is the etiology of obesity?

A. Hypothyroid
B. Hypo-ovarian
C. Hypothalamic-pituitary
D. Alimentary and constitutive
E. Hypercorticoid
A 19-year-old patient complains of fever up to 37,4°C during the last 2 months after recovering from ARVI. Objectively: malnutrition, diffuse grade II enlargement of the thyroid gland feeling dense on palpation, exophthalmos, tachycardia. What kind of pathological syndrome is it?

A. Thyrotoxicosis
B. Hypothyroidism
C. Hypoparathyroidism
D. Hyperparathyroidism
A 47-year-old woman underwent a thyroid gland resection on account of nodular euthyroid goiter. What preparations are most likely to prevent the disease recurrence?

A. Thyroid hormones
B. Mercazolil
C. Thyrotropin
D. Antistruminum (potassium iodide)
E. Radioactive iodine
A 39-year-old patient complains of a tumour on the anterior surface of her neck. The tumour has been observed for 2 years. It is nonmobile and has enlarged recently. The patient has a changed tone of voice, a sense of pressure. Objectively: in the left lobe of the thyroid gland a 3 cm node is palpable; it is very dense, tuberous, painless. Cervical lymph nodes are enlarged. Functional status of the thyroid gland is unchanged. What is the most likely diagnosis?

A. Thyroid gland cancer
B. Nodular euthyroid goiter
C. Nodular hyperthyroid goiter
D. Chronic lymphomatous Hashimoto’s thyroiditis
E. Chronic fibrous Riedel’s thyroiditis
A female patient consulted a doctor about gain in weight, chill, edemata, dry skin, sleepiness, problems with concentration. Objectively: the patient’s height is 165 cm, weight is 90 kg, gynoid body proportions, to- 35,8oC, ESR-58/min, AP- 105/60mm Hg. Heart sounds are weakened, bradycardia is present. Other internal organs have no changes. Thyroid gland is not palpable. Mammary glands ooze milk droplets. Hormonal study revealed rise of TSH and prolactin concentration, reduction of T4. What factor caused obesity?
A. Primary hypothyroidism
B. Secondary hypothyroidism
C. Prolactinoma
D. Hypopituitarism
E. Adiposogenital dystrophy
A 26 y.o. Male patient with postoperative hypothyroidism take thyroxine 100 mcg 2 times a day. He has developed tachycardia, sweating, irritability, sleep disorder. Determine further treatment tactics.

A. To decrease thyroxine dosage
B. To increase thyroxine dosage
C. To administer betablockers
D. To add mercasolil to the treatment
E. To administer sedatives
A patient of 32 y.o. complains of severe weakness, tremor of extremities. Objective examination: body weight loss, wet and warm skin. The thyroid gland is enlarged up to the 3-rd degree, painless, elastic. Ps-108/min. BP-160/55 mm Hg. There are no other abnormalities. The diagnosis is:

A. Diffuse toxic goiter of the 3-rd degree, thyrotoxicosis of the average degree
B. Diffuse euthyroid goiter of the 3-rd degree
C. Chronic autoimmune thyroiditis, hypertrophic type
D. Chronic fibrous thyroiditis
E. Toxiferous adenoma of the thyroid gland
A 39-year-old patient complains of a tumour on the anterior surface of her neck. The tumour has been observed for 2 years. It is nonmobile and has enlarged recently. The patient has a changed tone of voice, a sense of pressure. Objectively: in the left lobe of the thyroid gland a 3 cm node is palpable; it is very dense, tuberous, painless. Cervical lymph nodes are enlarged. Functional status of the thyroid gland is unchanged. What is the most likely diagnosis?

A. Thyroid gland cancer
B. Nodular euthyroid goiter
C. Nodular hyperthyroid goiter
D. Chronic lymphomatous Hashimoto’s thyroiditis
E. Chronic fibrous Riedel’s thyroiditis
Test 9

A 40-year-old female patient complains of having a bulge on the anterior surface of neck for 5 years. Objectively: Ps- 72 bpm, arterial pressure - 110/70 mm Hg, in the right lobe of thyroid gland palpation reveals a mobile 4x2 cm node, the left lobe is not palpable, the basal metabolic rate is 6%. What is the most likely diagnosis?

A. Nodular euthyroid goiter
B. Nodular hyperthyroid goiter
C. Riedel’s thyroiditis
D. Mixed euthyroid goiter
E. The median cervical cyst
On the first day after a surgery for diffuse toxic goiter a patient developed difficulty breathing, cold sweats, weakness. Objectively: pale skin, body temperature - 38,5oC, RR - 25/min, Ps- 110/min, AP-90/60 mm Hg. What early postoperative complication occurred in the patient?

A. Thyrotoxic crisis
B. Hypothyroid crisis
C. Postoperative tetany
D. Acute thyroiditis
E. Compression of the trachea by the hematoma
A 39-year-old female patient complains of rapid fatigability, drowsiness, dry skin, hair loss, swelling of the face. A month ago, she underwent a surgery for thyrotoxicosis. The patient has the following gland dysfunction:
A. Thyroid (hypothyroidism), due to inadequate operative technique
B. Pituitary, due to a tumor
C. Adrenal
D. Parathyroid, due to the gland removal during surgery
E. Ovarian, due to a tumor
Recommended literature