



DIABETES MELLITUS. DIFFUSE TOXIC GOITER

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Diabetes mellitus is

- a chronic endocrine - metabolic disease, basically the occurrence of which is insulin deficiency (diabetes - 1),
- or tissue insensitivity to it (diabetes - 2),
- accompanied by a violation of all types of metabolism, but mainly - carbohydrate,
- resulting in increased blood glucose levels (hyperglycemia), urinary glucose excretion (glucosuria),
- which subsequently leads to dysfunction of vital organs and systems.

Etiological classification of diabetes mellitus

There are 4 types of diabetes:

1. Type 1 diabetes mellitus (diabetes mellitus -1)

2. Type 2 diabetes mellitus (diabetes mellitus -2)

3. Other or specific types
(endocrinopathies, diseases of the pancreas, genetic defects of β -cells, infections, drug or chemically-induced diabetes)

4. Gestational diabetes (gestational diabetes, GDM)

- The clinical picture of diabetes is a consequence of acute or chronic insulin deficiency,
- which in turn can be absolute or relative

Acute insulin deficiency causes the rapid development of metabolic decompensation, above all carbohydrate

which is manifested by a vivid clinical picture in the form:

hyperglycemia	glucosuria	polydipsia	polyuria	weight loss on the background of increased	appetite (polyphagia)	a significant decrease in efficiency
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Classification of diabetes according to clinical features

Severity of diabetes

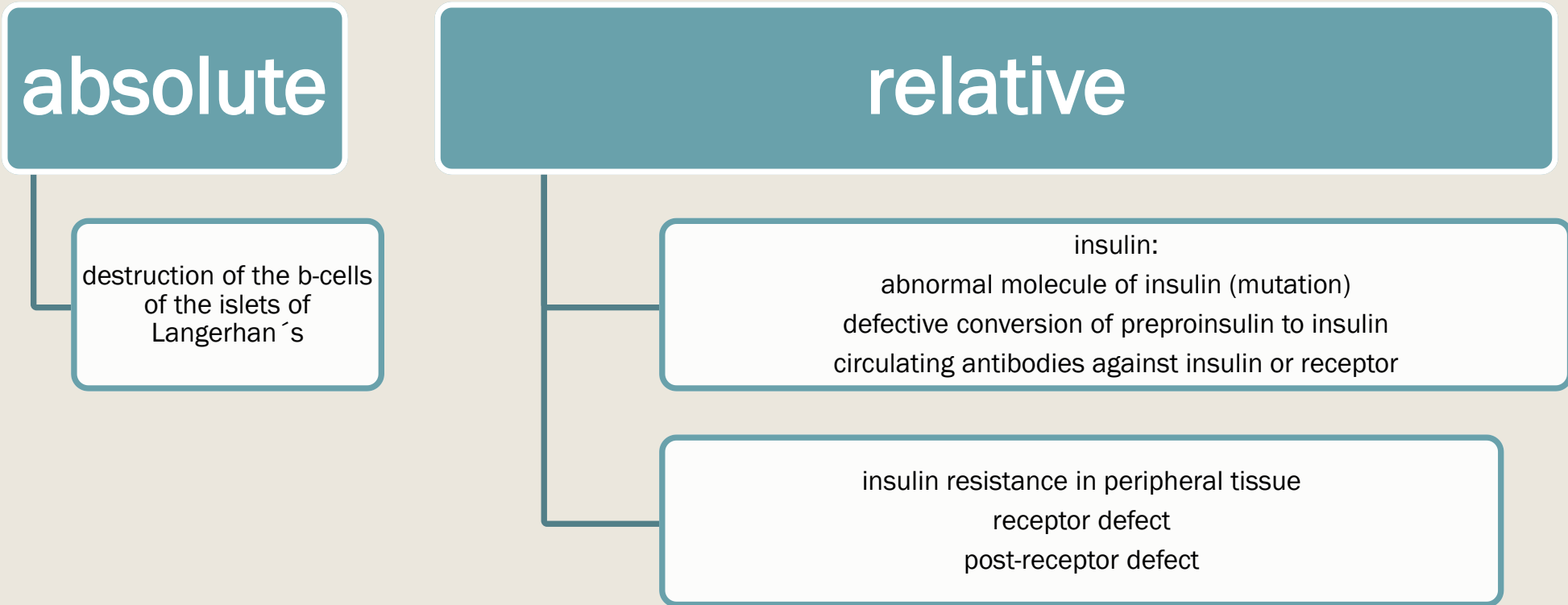
Compensation state

Complications

Type 1 diabetes mellitus

- Characterised by absolute insulin deficiency.
- Most cases result from autoimmune pancreatic beta-cell destruction in genetically susceptible individuals.
- Usually presents with acute symptoms or ketoacidosis in childhood or adolescence.
- Lifelong insulin therapy is required.

Causes of insulin deficiency



absolute

destruction of the b-cells
of the islets of
Langerhan's

relative

insulin:

abnormal molecule of insulin (mutation)
defective conversion of preproinsulin to insulin
circulating antibodies against insulin or receptor

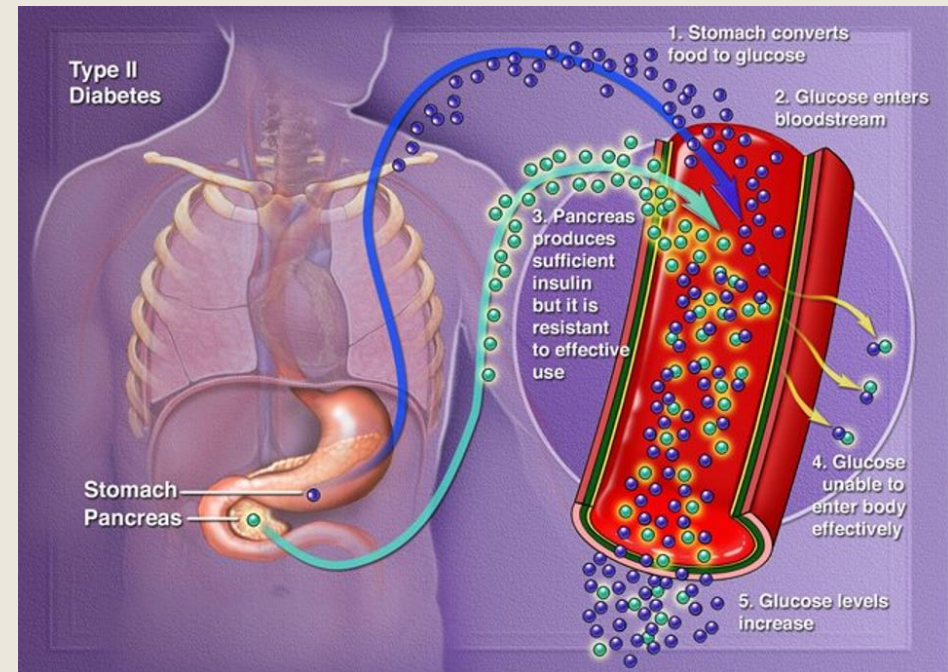
insulin resistance in peripheral tissue
receptor defect
post-receptor defect

Type 2 diabetes mellitus in adult

- Common disorder characterised by insulin resistance and relative insulin deficiency

Type 2 diabetes mellitus in adult

- Most patients are asymptomatic
- and are diagnosed through screening
- (abnormal fasting plasma glucose, haemoglobin A1c, and/or oral glucose tolerance test).



Strong risk factors of the Type 2 diabetes mellitus in adult include:

- older age
- overweight/obesity
- physical inactivity
- prior gestational diabetes
- pre-diabetes
- non-white ancestry
- family history of diabetes, or polycystic ovary syndrome

- Modification of cardiovascular risk factors (e.g., hypertension and dyslipidaemia) are important treatment considerations, along with glycaemic control to prevent microvascular complications.

Main symptoms of Diabetes

blue = more common
in Type 1

- Central**
- Polydipsia
 - Polyphagia
 - Lethargy
 - Stupor

- Eyes**
- Blurred vision

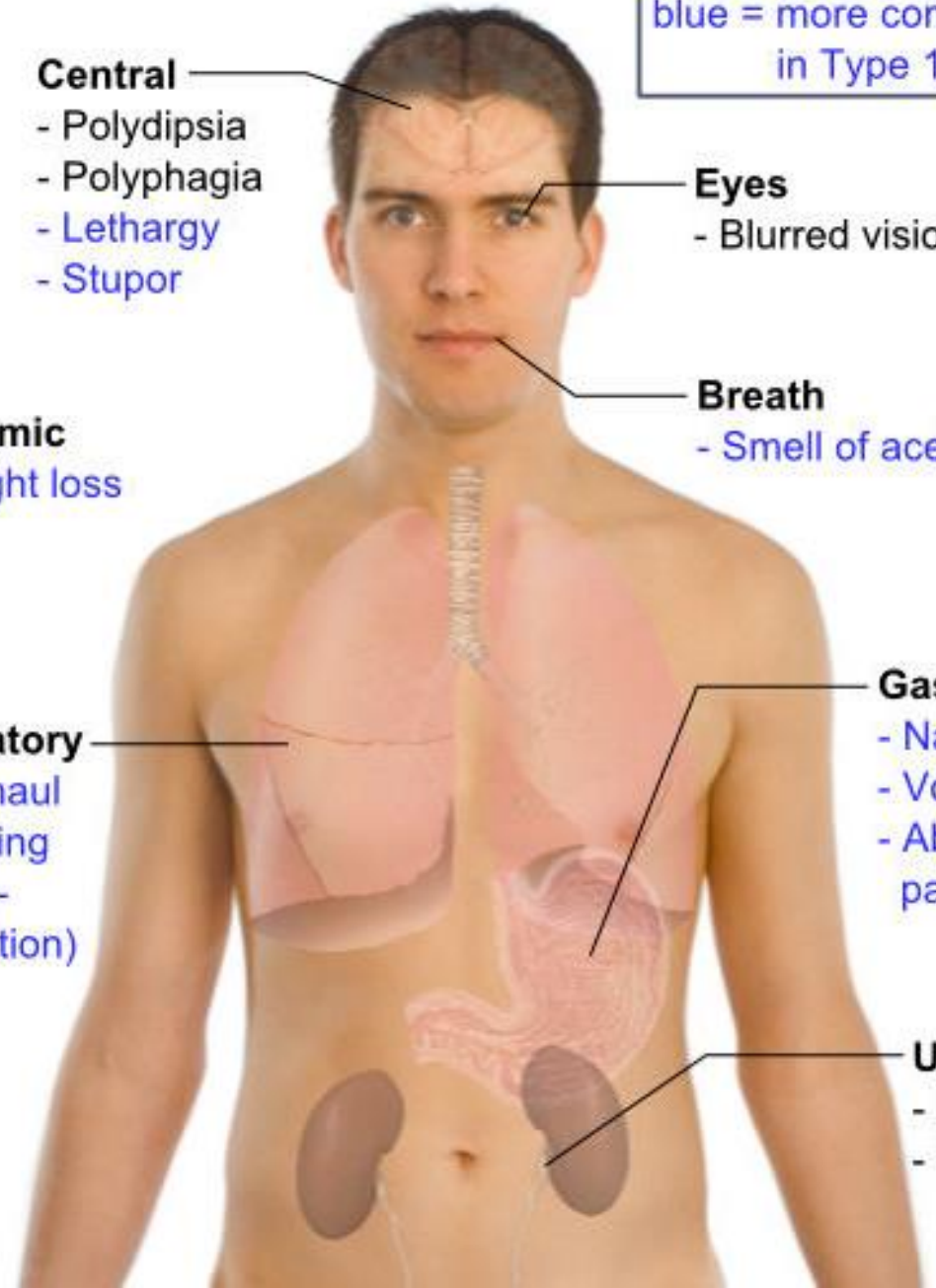
- Systemic**
- Weight loss

- Breath**
- Smell of acetone

- Respiratory**
- Kussmaul breathing (hyper-ventilation)

- Gastric**
- Nausea
 - Vomiting
 - Abdominal pain

- Urinary**
- Polyuria
 - Glycosuria



Main characteristics of T1DM and T2DM

■	T1DM	T2DM	
clinical manifestation		often acute	mild or none
onset		childhood	adults
genetic disposition		yes (oligogenic)	yes (polygenic)
autoimmunity		yes	No
insulin resistance		no	Yes
dependence on insulin		yes	No
obesity		no	yes

Type 2 diabetes mellitus in children

- Obesity, leading to insulin resistance, is the primary cause of type 2 diabetes in children.
- The majority of cases are diagnosed after the age of 10 years.
- Commonly accompanied by acanthosis nigricans (90% to 95% of patients).

Acanthosis nigricans



Gestational Diabetes



High blood glucose levels in mother



Brings extra glucose to baby



Causes baby to put on extra weight



(1) Mother's blood brings extra glucose to fetus

(2) Fetus makes more insulin to handle the extra glucose

(3) Extra glucose gets stored as fat and fetus becomes larger than normal

Gestational diabetes

- Defined as any degree of glucose intolerance with onset or first recognition occurring during pregnancy. It is usually recognised at 24 to 28 weeks of gestation on the basis of abnormal glucose tolerance testing.

Gestational diabetes

- Strong risk factors include:
- advanced maternal age (>40 years)
- obesity
- personal history of gestational diabetes or macrosomia of previous child
- polycystic ovary syndrome,
- non-white ancestry
- family history of diabetes mellitus.

Gestational diabetes

- It is uncommon for patients to present with symptoms (e.g., urinary tract infections or vulvovaginal candidiasis).
- Occasionally it may be difficult to distinguish GD from undiagnosed pre-existing type 2 diabetes.
- Infrequently, type 1 diabetes may present during pregnancy.
- Medical nutrition therapy is central to control of GD and most women are adequately treated with diet alone.

Interpretation of glycemia

- **FPG:**
 - **<6.1 mmol/l = normal glycemia**
 - **6.1-7.0 mmol/l = IGT (impaired glucose tolerance)**
 - **7.0 mmol/l = diabetes**
- **oGTT – 2h PG:**
 - **<7.8 mmol/l = normal glucose tolerance**
 - **7.8 – 11.1 mmol/l = IGT**
 - **11.1 mmol/l = diabetes**

Type 1 DM (formerly IDDM)

selective destruction of b - cells of LO in genetically predisposed individuals

- chrom. 6 – HLA (DR3-DQ2 a DR4-DQ8), chrom. 11 – insulin gene
- initiation by infection (viruses: mumps, coxsackie B₄), toxic chemical agents or destructive cytotoxins and antibodies released from sensitized immunocytes
- autoimmunity mediated by T-lymphocytes, antibodies against b - cells (ICA, GAD) though, manifestation typically in childhood

absolute dependence on exogenous supplementation by insulin

Type 2 DM (formerly NIDDM)

imbalance between secretion and affect of insulin

genetic predisposition – polygenic

- insulin resistance
- impairment of secretion

clinically manifested T2DM has concomitant insulin resistance and impairment of secretion

- due to epigenetic factors
- typically in older adults

90% of subjects is obese – **metabolic syndrome!!!**

Insulin resistance

physiologic amount of insulin does not cause adequate response

compensatory hyperinsulinism

further worsening by down-regulation of insulin receptors

Clinical presentation of manifest DM

- due to the increase of blood osmolality, osmotic diuresis and dehydration

classical:

- polyuria
- thirst
- polydipsia
- weight loss
- temporary impairment of visus
- cutaneous infections

acute:

hyperglycemic coma

- ketoacidotic
- non - ketoacidotic

Complications of DM

microvascular

- diabetic retinopathy
- diabetic nephropathy
- diabetic neuropathy (sensoric, motoric, autonomic)

macrovascular

- atherosclerosis (CAD, peripheral and cerebrovascular vascular disease)

combined

- diabetic foot (ulcerations, amputations and Charcot´s joint)

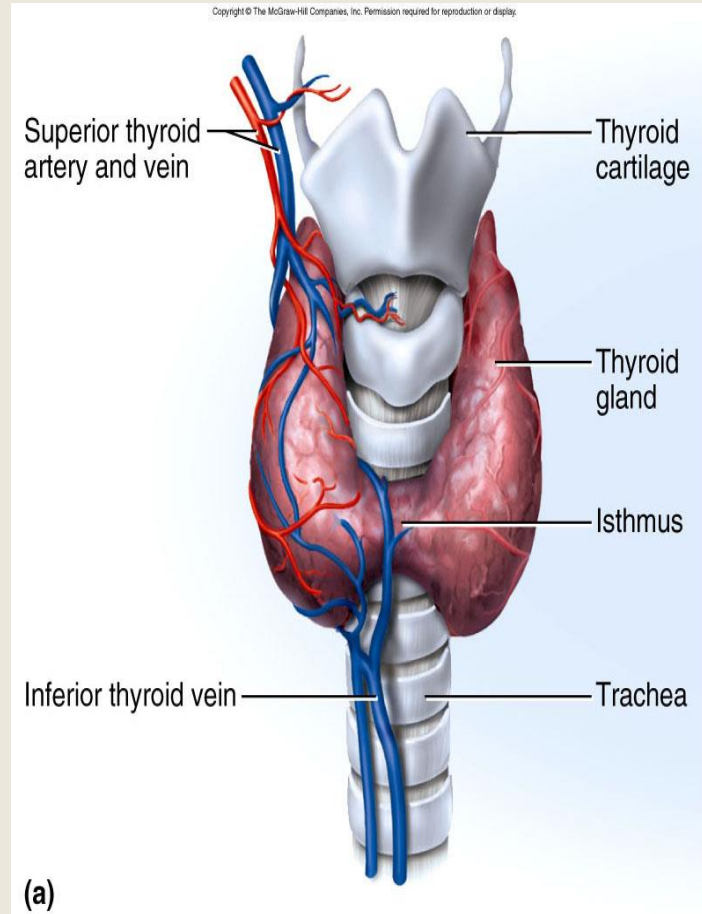
others

- periodontitis
- cataract
- glaucoma

Diabetic retinopathy

- Consequence of chronic progressive diabetic microvascular leakage and occlusion. Sight-threatening signs include macular oedema, ischaemia, or traction; vitreous haemorrhage; or retinal detachment. Main goals of therapy are to improve glycaemic, lipid, and blood pressure control and to ensure that disease is arrested before visual loss occurs.

Thyroid Gland Anatomy



- Largest endocrine gland; high rate of blood flow
 - *arises root of embryonic tongue*
- Anterior and lateral sides of trachea
 - *two large lobes connected by isthmus*

Thyroid Gland

- Thyroid follicles
 - *filled with colloid and lined with simple cubical epithelial (follicular cells) that secretes two hormones, T_3 and T_4*
 - *thyroid hormone*
 - \uparrow body's metabolic rate and O_2 consumption
 - calorogenic effect - \uparrow heat production
 - \uparrow heart rate and contraction strength
 - \uparrow respiratory rate
 - stimulates appetite and breakdown CHO, lipids and proteins
- C (calcitonin or parafollicular) cells
 - *produce calcitonin that \downarrow blood Ca^{2+} , promotes Ca^{2+} deposition and bone formation especially in children*

Hypothyroidism

this is not disease, but specific condition of organism, connected with reaction to low concentration of thyroid gland hormones.

Classification

There are different types of hypothyreosis:

- **Primary** (thyreogenic)
- **Secondary** (pituitary)
- **Tertiary** (hypothalamic)
- **Tissual** (transport, peripheral)

Also:

1. Hereditary;
2. Acquired, including postoperative (after resection of thyroid gland).

According severity primary hypothyreosis distinguish:

- Latent (subclinical) — increased level of TSH at normal T4
- Manifest — hyper secretion of TSH, at low level of T4, clinical manifestation
- Compensated
- Decompensated
- Severe clinical course (complicated)

Complications:

- cretinism
- cardiac insufficiency
- effusion into serous cavities
- secondary adenoma of hypophysis

As a rule, this is did not diagnosed cases, if stay without treatment can lead to myxedematous coma.

Typical syndromes

Metabolic – hypothermic syndrome:

- obesity
- low temperature, low resistance to cold
- hypercarotinememia, accompanies with icterus
(jaundice)

Mixedematous edema:

periorbital edema

- puffy face, big lips and tongue with imprints of teeth along lateral sides
- edema of legs
- difficult nasal breathing (connected with swelling of nasal mucosa)
- violation of hearing (edema of external auditory canal and middle ear)
- hoarse voice (edema and thickening of vocal cords)
- polyserositis

Syndrome of nervous system violation:

- somnolence
 - lethargy
 - reduction of memory
 - bradyphrenia
 - aching muscles with cramps
 - paresthesia
 - reduction of tendons reflexes
- polyneuropathy

Syndrome of CVS violation:

- Mixedematous heart (bradycardia, low voltage of ECG, negative T wave, circulatory insufficiency)
- hypotension
- polyserositis
- Possible atypical cases (with hypertension, without bradycardia, with constant tachycardia with circulatory insufficiency and paroxysmal tachycardia like simpatico-adrenal crisis at onset of hypothyreosis).

Syndrome of digestive system violation:

- hepatomegalia
- dyskinesia of bile ducts
- dyskinesia of large intestine
- inclination to constipation
- poor appetite
- atrophy of stomach mucosa
- nausea, sometimes vomiting.

Anemic syndrome:

anemia— norm chromic,
normoblasts

hypo chromic iron deficiency,
macroblasts B12-deficiency.

Syndrome of hyperprolactinemic hypogonadism

Ovarian dysfunction:

menorrhagia

oligomenorrhea or amenorrhea

fertility

galactorrhea

Syndrome of ectodermic violations:

Changes of skin, hair, nails:

- Hair on a head are sparse, thin, brittle, falls out from brows, head, extremities, grow slowly
- Dry skin
- Thin, brittle nails, with longitudinal or diametrical striation

Hypothyroid (myxedematous) coma.

- The most serious complication of hypothyreosis , sometimes mortal
- Characterize with progressive elevation of all symptoms of hypothyreosis.
- Mortality touch 40 %



600 x 579 - Pretibial Myxedema
woundsresearch.com



[myxedema.jpg](#)



The term **myxedema** is applied to hypothyroidism developing in the older child ...



Adult hypothyroidism: Pretibial
pharmacology2000.com



Hypothyroidism and **Myxedema** Coma

Iodine Deficiency Disorders



Goiter



Cretinism

Cretinism Vs **Myxedema**. Cretinism is the condition wherein the child has ...

Hyperthyroidism (toxic goiter)

- **syndrome**, caused of thyroid gland hyperfunction. Manifest of hormones elevation: Thriiodtironine (T3), thyroxin (T4). Hyperthyreosis, according level of damages,

distinguish:

- primary — thyroid gland
- **secondary** — hypophysis
- *tertiary* — hypothalamus.

Diffuse toxic goiter as one of
Grave disease display (Basedow
disease)

1835 year – described by
Grave

1821 year – described by Perry

1840 year - described by
Basedow

Germany doctor Basedow described in 1840 main triad of symptoms, typical for this disease:

1. goiter
2. exophthalmia
3. tachycardia

1. Diffuse toxic goiter (Basedow disease) — the most common reason of hyperthyreosis
2. Nodal toxic goiter (Plummer disease) present rare, then Graves disease, more typical for senile people
3. Sub acute thyreoiditis (thyreoiditis of de Quervain) is able provoke transient hyperthyreosis
4. Artificial hyperthyreosis can be result of uncontrolled use of thyroid hormones

Rare cases of hyperthyreosis:

1. Tumors of hypophysis with hyper secretion of TSH (for example, syndrome of Truel-JuneЖюне, or hyperthyreosis acromegalic with hyperostosis — combination of diffuse hyperostosis of skull arch , acromegaly and signs of hyperfunction of thyroid gland , as a result of increased secretion of adenohipophysis STH and TSH)
2. ovarian teratoma, produce thyroid hormones (ovarian struma)
3. Hyper production of thyroid gland hormones after over use of iodine (syndrome iodine - basedow).

- # ■ Pathogenesis
- Thyroid hormones increase tissue consumption of oxygen, increase mt production and energetic metabolism
 - Increase sensitivity of tissues to catecholamine and sympathetic stimulation
 - Increase transformation of androgens in estrogens in tissues and increase amount of circulative globulin, bind sexual hormones, as a result increase ratio estrogens to androgens. These hormonal changes can provoke gynaecomastia of men
 - Quick destruction of cortisol under influence of thyroids hormones stipulate clinical picture of hypocorticism (reversible suprarenal insufficiency).

Risk factors:

- complicated family anamnesis
- female gender
- autoimmune diseases

Clinical picture

1. Metabolic changes:

- Increased metabolic rate and weight loss, in spite of normal feeding and good appetite
- Sweating and heat intolerance display of increased heat production
- Not infrequently — reversible hyperglycemia

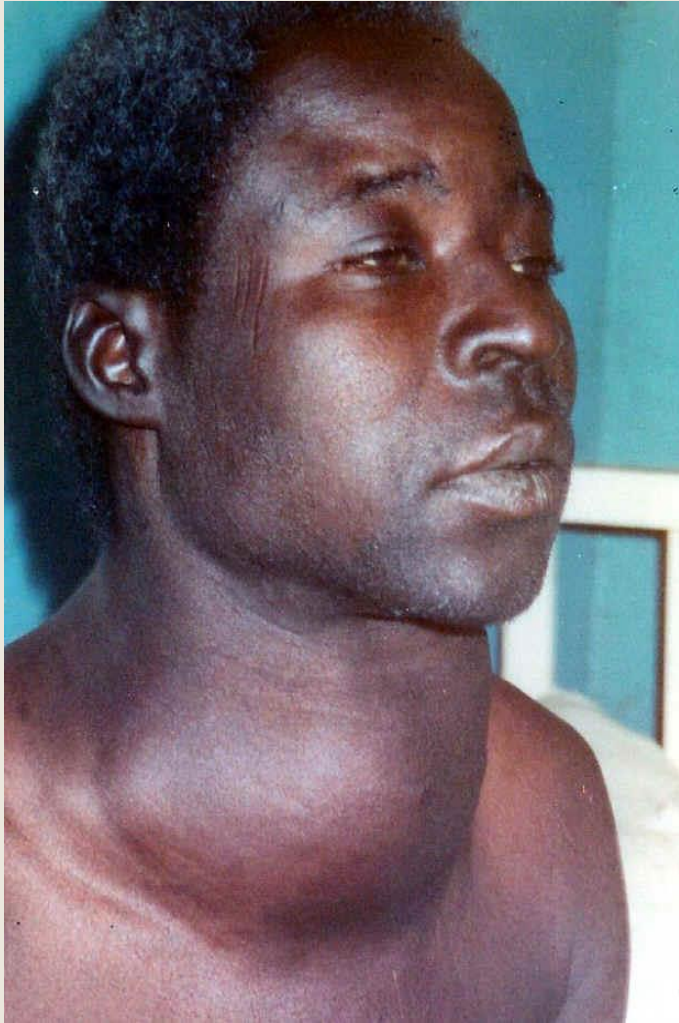
Classification of goiter (WHO, 2001)

There are III degrees of increase of the gland:

0 degree - no goiter (volume of shares does not exceed the size of the distal phalanx of the thumb of the subject).

Grade I - palpable increase in the proportion of thyroid, but it is not seen in the normal position of the neck. They also include nodules, which do not lead to an increase in the thyroid gland itself;

Grade II - enlarged thyroid gland visible in the normal position of the neck



endemic goiter



exophthalmia





There are typical ophthalmological symptoms:

- **exophthalmos**;
- **Upper lid lag from pupil at movement of eye downwards**. This white strip between upper lid and pupil not infrequently present at motionless eye ball (symptom of Graefe);
- **Lag of low lid from low margin of cornea at horizontal glance** (symptom of Dalrimple);
- **Tremor of lids** (symptom of Rosenbach);
 - «expression of astonishment».



Grave's disease

- violation of convergence, as a result is impossible fix vision on a tip of nose, one of eye deviate on some distance from fix point aside (symptom of Mebius);
- Disappeared or significantly decreased request of eye ball wet by means of wink. Glance of patient will be tense and like motionless (symptom of Stellwag).

Heart rate increase, and also arrhythmias
tachycardia **120 per minute and more**
(don't disappear at sleep and bad treat
with cardiac glycosides) — patient
feels palpitation at neck, head and
abdomen

- **Other arrhythmias due to increased excitability of myocardium**, for example, atrial fibrillation and flutter
- Tendency to elevation of systolic blood pressure and reduction of diastolic BP (big pulse pressure)
- **Symptoms of chronic cardiac insufficiency**

Digestive system symptoms:

- Increased appetite
- Constipation or diarrhea
- Attack of abdominal pain
- Sometimes vomiting
- In severe cases — reversible violations of liver (enlargement, tenderness or painfulness, possible jaundice)

1. Changes of skin and hair:

- Skin is warm, wheat due to vasodilatation of peripheral vessels and increased sweating.
- Typical thin, silky hair, possible early turn grey

2. CNS violations:

- Emotional liability, anxiety and tremor

1. Sexual disorders:

At women — violations of menstrual cycle up to amenorrhea

At man — reduction of libido, possible gynaecomastia

2. Muscles weakness and fatigue (due to concomitant hypocorticism)

Table 1. The most often signs of thyroid gland function violations after delivery

Toxic goiter	Myxedema
<ul style="list-style-type: none">• Anxiety• Tremor at all body• Palpitation• Feeling of heat• Difficult concentration of attention• Muscles weakness• Decrease of body mass	<ul style="list-style-type: none">• Quick tiredness• Weakness• Enlargement of body mass• Constipation• Worsening of memory• Cold intolerance• Muscles rigidity

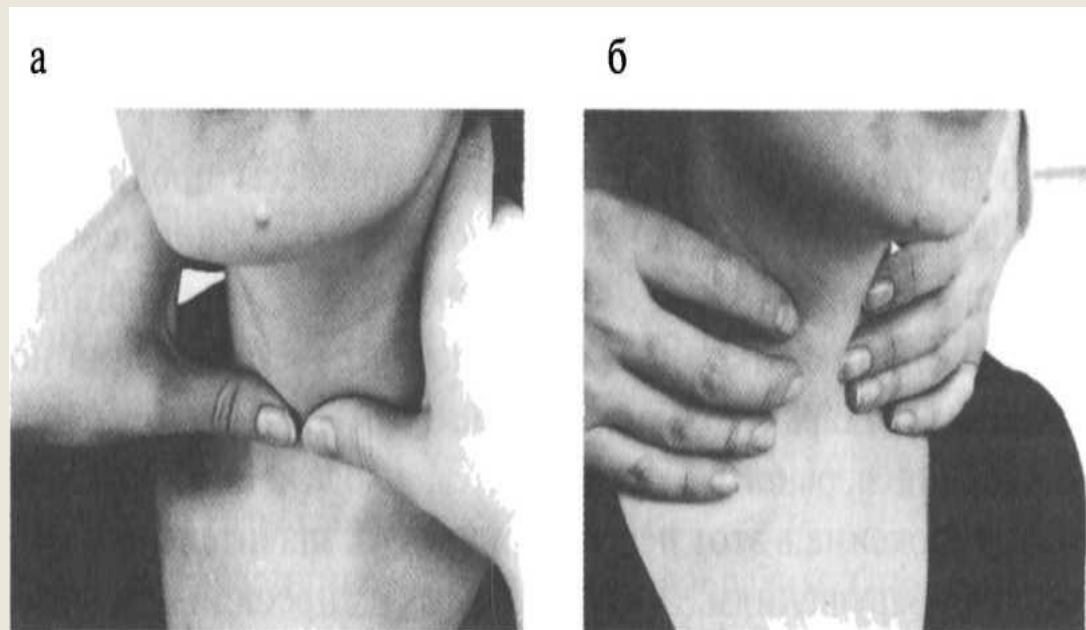
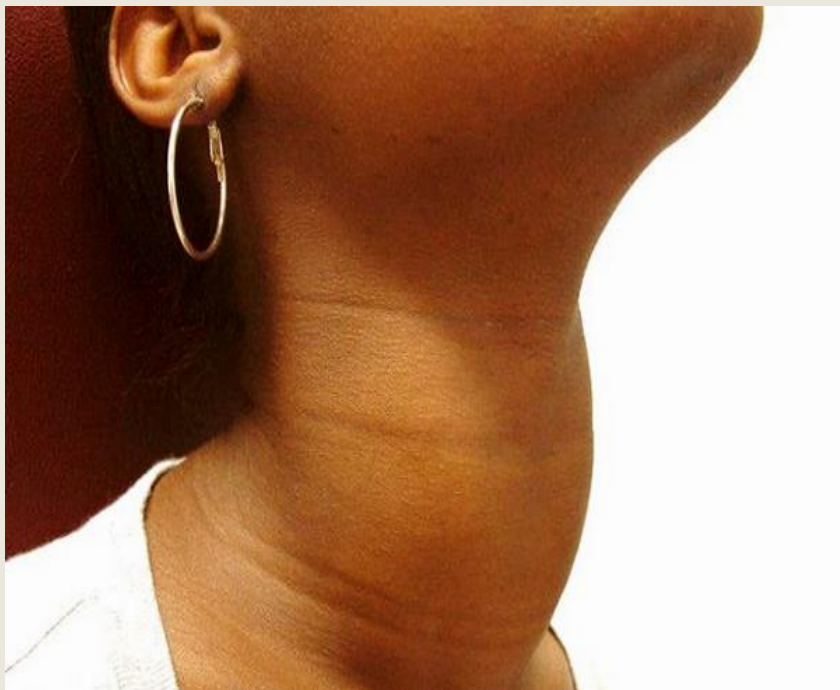


Рис. 3.5. Методы пальпации щитовидной железы



Базедова болезнь



- При избытке (гиперфункции) гормонов щитовидной железы освобождается много энергии. Процессы распада в организме идут быстро, человек худеет, развивается пучеглазие. Человек становится раздражительным.













Figure 4 - Bilateral, firm, non-pitting, asymmetrical yellow, pink to purple-brown plaques or nodules are early manifestations of pretibial myxedema.



Микседема



Сухие,
выпадающие
волосы

Отеки под
глазами

Отечное лицо,
сухая кожа



Будьте здоровы!

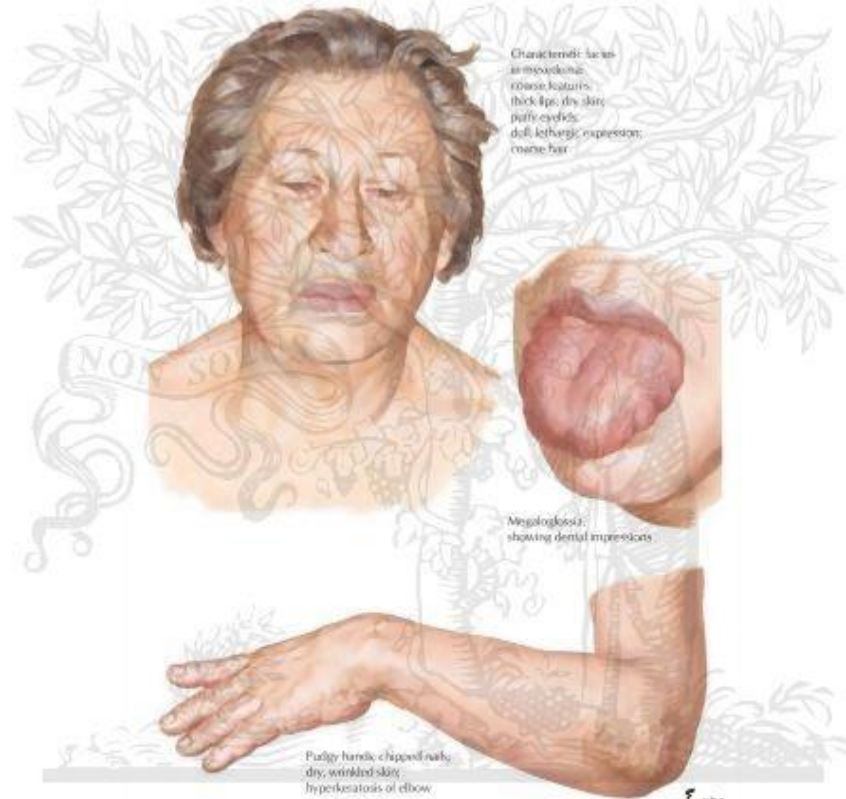
medinform-mos.ru







Adult Myxedema
Clinical Manifestations and Etiology



Characteristics:
facies
in myxedema;
coarse features;
thick lips, dry skin;
puffy eyelids;
dull, lethargic expression;
coarse hair.

Megalglossia:
showing dental impressions

Puffy hands; chipped nails;
dry, wrinkled skin;
hyperostosis of elbow.

ELSEVIER

F. Netter