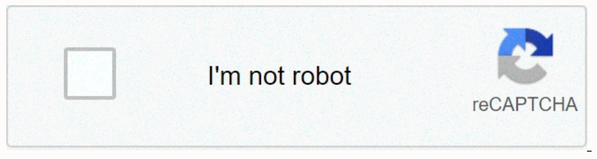

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Icd 9 graves disease

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ICD-9-CM 242.00 is a billable medical code that can be used to indicate the diagnosis of a refund claim, while 242.00 should only be used for service day billing before September 30, 2015. For charges that have a service date after October 1, 2015,ICD-10-CM code (or code). View latest version of ICD-9-CM 242.00.ICD-9-CM 242.00: Convert to 2013 2014 2015.ICD-10-CM: 242.00 Conversion Direct: 2015/1 6 ICD-10-CM E05.00 Thyroid toxicity crisis or thyroid poisoning of diffuse thyroiditis without storms Strabismosis due to grave disease Thyroid function Thyroiditis Toxic thyroiditis Toxic thyroiditistoxtoxtoxthiotoxic external eyesight with external eye ophthalmatitis (thyroid toxicity crisis or storm) However, gutter toxicity diffusion thyroid function unspecified acquired hypothyroidism Claimable until September 30, 2015 Date of use / Billable date after October 1, 2015 / 2015ICD-9-CM 244.9 is a billable medical code that can be used to indicate a diagnosis regarding a refund claim. 244.9 should only be used for charges with service days before September 30. , 2015.For charges made on or after October 1, 2015, use an equivalent ICD-10-CM code (or code). Converted to ICD-10-CM: 244.9 Direct conversion: 2015/16 ICD-10-CM E03.9 Hypothyroidism, Unspecified approximation synonym Hypothyroidism Hypothyroidism Due to hypothyroidism Hypothyroidism Pregnancy hypothyroidism (hypothyroidism) Pregnancy thyroid (hypothyroidism) pregnancy, pre-baby pregnancy hypothyroidism (hypothyroidism) hypothyroidism Post-thyroid hypothyroidism is a condition in which hypothyroidism (post-childbirth) hypothyroidism hypothyroidism Hypothyroidism Clinical hypothyroidism Thyroid hormone production decreases. Signs and symptoms of hypothyroidism include low metabolic rates, tendency to weight gain, somnolens and sometimes myoedema. The most common cause of hypothyroidism in the United States is Hashimoto's thyroiditis, an autoimmune disease in which the production of thyroid hormone by the thyroid gland decreases. Signs and symptoms of hypothyroidism include low metabolic rates, trends in weight gain, somnolens and sometimes myoedema. In the United States the most common cause of hypothyroidism is hashimoto's thyroiditis, which is an autoimmune disease. acinacylea disorder characterized by a decrease in the production of thyroid hormones due to glandular syndrome of the thyroid gland resulting from abnormally low secretion of thyroid hormones from the thyroid glandIn the most severe form, there is an accumulation of mucopolysaccharides in the skin and edema, known as myoedema of thyroid activity, which leads to a decrease in basal metabolic rate. It is characterized by a decrease in basal metabolic rate, fatigue and let energy, sensitivity to cold, menstrual disorders. Without treatment, it progresses to myoedema. In infants, severe hypothyroidism has too few thyroid hormones that lead to cretinism. Symptoms include weight gain, constipation, dry skin, and less sensitive thyroid hormones to cold. Symptoms include weight gain, constipation, dry skin and sensitivity to colds. Also applicable to hypothyroidism, hypothyroidism including reverse reference to 244.9 to primary or NOSICD-9-CM volume 2 index: anemia 285.9due anti-biopsychotic chemotherapy 285.3 blood loss (chronic 280.0acute 285.1 Chemotherapy, Anti-Reagino activity 285.3 Glycolysis of the Embuden Meyerhof pathway 282.3 Impaired glutathione metabolism 282.2 - See anemia. Type of chemotherapy (see also pharmaceutical and chemical tables) chemotherapy, anti-reeminant 285.3 Fetal blood loss 776.5 Fishworm (D. latam) infection 123.4 Glutathione metabolic disorder 282.2 Bleeding (chronic) 28 0.0Acute 285.1hexose single phosphate (HMP) shunt deficiency 282.2 Disability absorption 280.9 Atsemia (chronic) 280.0acute 285.1myxedema 244.9Nexatural Americanas 126.1 Immaturity 776.6 Selective Vitamin B12 Absorption due to proteinuria 281.1 Athyrea (see also Hypothyroidism) 244.9 Congenital hypothyroidism (postten) 244.9 Congenital 243 atrophy, Atrophic thyroid (gland) 246.8 Tinism 243 fibroids 244.9 Congenital 243 Brisseau infantile disease (Infant myelopathy) 244.9 Briscoe-Magege syndrome (infant myeloma) 244.9Cachexia 799.4strumipriva (see also hypothyroidism) 244.9 Deficient thyroid (gland) 244.9 Euthyroidism 244.9 Hypothyroidism (acquired) 244.9 Complex pregnancies, Childbirth, or Puerperium 648.1 Congenital 243 Goitros (dissipative) 246.1iatrogenic NEC 244.3iodine 244.2 Pituitary 244.8 Postative NEC 24 4.1 Post surgery 244.0primary 244.92 Next NEC 244.8 Specific Cause NEC 244.8sporadic goitrous 246.1In Efficiency Thyroid (Acquired) (Gland) 244.9 Infantism 259.9 Briscoe (Infant Bone Marrow) 244.9 > (Adult) (Stupidity) (Infant) (Infant) (Thyroid) (See also Hypothyroidism) 244.9 Assignment 2 42.9 Congenital 243cutis 701.8 Local condition (adibial) 242 .9madness (acute) 293.0subacute 293.1papular 701.8 pituitary 244.8 After delivery 674.8pretibial 242.9primary 244.9 Obesity (constitutional) (exogenous) (familial) (nutrition) (simple) 278.00 hypothyroidism (see also hypothyroidism) 244.9 Hypothyroidism (hypothyroidism) See also)) 244.9Strumipriva cachexia (see also Hypothyroidism) 244.9 Hypothyroidism (see also Hypothyroidism) 244.9 Congenital 243 Syndrome - See also Disease Brissault Mage (Infant)244.9 Autoimmune endocrine diseases Serious diseases and other name toxic diffuse goiter, Frajani-Rissou-Grave disease Grave disease Veneco disease Peculiar endocrinology of outer eyeball and lid pull-back classic discovery special Endocrinology symptoms Enlarge the thyroid gland, hypersensitivity, muscle weakness, sleep disorders, fast heartbeat, weight loss, poor resistance to heat[1] complications[1] risk factor history, other autoimmune diseases[1] diagnostic method blood tests Radioactive iodine uppicking[1][1] therapeutic radiation yodion therapy, medication, thyroid surgery[1] frequency[1] (male) 3% (female)[4] grape disease, also known as toxic bottle gwiter, is an autoimmune disease that affects the thyroid gland. [1] Frequently the most common causes of hyperthyroidism. [4] It also often results in hyperthyroidism. Signs and symptoms of hyperthyroidism include irritability, muscle weakness, sleep disturbances, fast heartbeat, poor heat tolerance, diarrhea and unintended weight loss. [1] Other symptoms include thickening of the skin of the shin, called a pre-tibia myoedema, and eye bulge, a condition caused by Graves ophthalmatosis. [1] About 25 to 80 percent of people with the condition develop eye problems. [1] [3] The exact cause is unknown. However, it is thought to include a combination of genetic and environmental factors. [2] If a person with a sick family is affected, the person is more likely to be affected. If one twin is affected, the probability of the other twin also getting sick is 30%. The onset of the disease can be caused by physical or emotional stress, infection or childbirth. [3] People with other autoimmune diseases, such as type 1 diabetes and rheumatoid arthritis, are more likely to be affected. Smoking can increase the risk of disease and exacerbate eye problems. [1] The disease arises from an antibody called thyroid-stimulating immunoglobulin (TSI), which has the same effect as thyroid stimulating hormone (TSH). These TSI antibodies cause the thyroid gland to produce excessive thyroid hormones. The diagnosis is suspected based on symptoms and is confirmed by blood tests and the upting of radioactive iodine. [1] [3] Typically, blood tests show an increase in T3 and T4 and increased upting of radioactive iodine in all areas of low TSH, thyroid and TSI antibodies. [3] The three treatment options are radioactive iodine therapy, medication and thyroid surgery. [1] Radioactive iodine therapy includes taking iodine-131 in the mouth, concentrating on the thyroid gland and destroying it for weeks or months. [1] Obtained hypothyroidism is treated with synthetic thyroid hormones. [1] Drugs such as beta blockers can control some of the symptoms, while antithyroid drugs such as metimazole can temporarily help people while other treatments have effects. [1] Surgery to remove the thyroid gland is another option. [1] Eye problemsAdditional treatment. [1] Grave disease develops in about 0.5% of men and 3% of women. [4] It occurs about 7.5 times more frequently in women than in men. [1] Often it starts between the ages of 40 and 60, but can start at any age. [5] It is the most common cause of hyperthyroidism in the United States [5] (about 50-80% of cases). [1] The condition was named after Irish surgeon Robert Graves, who he described in 1835. [5] There are also a number of pre-written descriptions. [5] Signs and Symptoms Main Articles: Symptoms and Symptoms of Serious Diseases Signs and symptoms of serious diseases arise virtually all from the direct and indirect effects of hyperthyroidism, but the main exceptions are significant ophthalmology, goiter, and a pre-tibial myoedema (caused by the autoimmune process of the disease). Symptoms of hyperthyroidism are mainly insomnia, hand-swinging, hyperhybolic, hair loss, excessive sweating, oligomenormeria, itching, heat insocia, weight loss despite increased appetite, diarrhea, frequent defecation, palpitations, periodic partial muscle weakness or paralysis are symptoms of a descent of asian descent in particular[6] and relief of skin and moisture. [7] Further signs that may be seen in physical examinations are most commonly diffuse enlargement (usually symmetry), non-tender thyroid, delayed lid, excessive plaques due to Graves ophthalmology, cardiac arrhythmia, adrenal tachycardia, atrial fibrillation, and early ventricular contraction, and hypertension. People with hyperthyroidism may experience behavioral and personality changes such as psychosis, mania, anxiety, agitation, and depression. [8] The exact cause is unknown. However, it is thought to include a combination of genetic and environmental factors. While the theoretical mechanisms by which exposure to severe stressors and high levels of subsequent distress, such as PTSD (post-traumatic stress disorder), increases the risk of autoimmune diseases and causes worsening of autoimmune responses that lead to serious illnesses, stronger clinical data is needed to draw firm conclusions. Genetics Genetic predisposition to serious diseases is seen, and some people are more prone to developing TSH receptor-activated antibodies due to genetic causes. Human leukocyte antigen DR (especially DR3) appears to play a role. [10] To date, no clear genetic defects have been found to refer to the cause of a single gene. Genes thought to be involved include tyrolovulin, tyrotropin receptors, protein tyrosine phosphatase nonreceptor type 22, cytachiotic T lymphocyte-related antigen 4, and the like. [11] Since infectious triggers are autoimmune diseases in which serious diseases appear suddenly, often in later life, viruses or bacterial infections can cause antibodies that cross-react with human TSH receptors, a known phenomenonAntigen imitation. It was assumed that it has structural similarities to thyroid stimulating hormone receptors[10] in humans and contributes to the development of thyroid autoimmunity that occurs for other reasons in genetically sensitive individuals. [12] Bacteria of the genus Elcinia Enterocolyci. In the 1990s, it was suggested that Y. engocolica may be a condition associated with both diseases that share heredity. More recently, the role of Y. Engocolytica has been disputed. [15] Epstein-Barr virus (EBV) is another potential trigger. [16] Thyroid-stimulated immunoglobulin recognizes and binds thyrotropin receptors (TSH receptors) that stimulate the secretion of thyroxine (T4) and triiodothyronine (T3). Thyroxine receptors in the pituitary gland are activated by surplus hormones to suppress the additional release of TSH in negative feedback loops. The result is very high levels and low TSH levels of circulating thyroid hormones. Histopathological image of hyperglytic hyperthyroidism (clinically presented as hyperthyroidism) of the thyroid gland (clinically presented as hyperthyroidism), an autoimmune disease in which the body produces antibodies to receptors of thyroid stimulating hormones. (Antibodies against tyrolobuline and thyroid hormones T3 and T4 can also be produced.) These antibodies bind to TSHr and chronically stimulate it, causing hyperthyroidism. TSHr is expressed on thyroid follicle cells (cells that produce thyroid hormones) in the thyroid gland, and the result of chronic stimulation is abnormally high production of T3 and T4. This causes clinical symptoms of hyperthyroidism and an enlarged thyroid gland that appears to be a goiter. Frequently encountered invasive outer eyes were explained by estimating that the thyroid and extraocular muscles share a common antigen recognized by antibodies. Antibodies that bind to the extraocular muscles will cause swelling behind the eyeball. The orange peel skin is explained by the infiltration of antibodies under the skin, causing an inflammatory reaction and subsequent fibrous plaques. The three types of autoantibodies against TSH receptors currently recognized are thyroid-stimulated immunoglobulin: these antibodies (mainly IgG) activate cells in a longer and slower way than TSH and have the effect of boosting the production of thyroid hormones. Thyroid growth immunoglobulin: These antibodies bind directly to TSH receptors and are involved in the growth of thyroid follicles. Psirotrophin binding inhibitory immunoglobulin: These antibodies inhibit the normal binding of TSH with its receptors. Some actually work as if TSH itself is bound to its receptors, thus inducing thyroid function.TSI and TSH are from binding and stimulating receptors. Another effect of hyperthyroidism is bone loss due to osteoporosis, caused by increased excretion of calcium and phosphorus in urine and stool. If hyperthyroidism is treated early, the effect can be minimized. Thyroid poisoning can also increase calcium levels in the blood by as long as 25%. This can cause stomach upset, excessive urination, and impaired renal function. [17] Diagnostic grave disease clinically presents one or more of these characteristic signs: rapid heartbeat (80%) diffuse and tactable goat sybring with audible brut (40%), extraorbital edema (25%) fatigue (70%), weight loss (60%) increased appetite in young people And other symptoms of anorexia, hyperthyroidism / hyperthyroidism in the elderly Heat insocia (55%) Severe (55%) Palpitations (50%) Two signs are really diagnosis of Graves disease (i.e., not seen in other hyperthyroidism): external eyeballs and non-exhaled edema (pre-tibial myoedema). Goiter is an enlarged thyroid gland and diffuse type (i.e., spreads throughout the glands). Isothyroiditis may be seen as other causes of hyperthyroidism, but Graves' disease is the most common cause of bottlegy gwiter. Large goiter is visible to the naked eye, while a small one (mild enlargement of the glands) can only be detected by physical examination. Occasionally, goiter is clinically undetectable, but can only be seen by computed tomography or ultrasound of the thyroid gland. Another sign of Graves' disease is hyperthyroidism, i.e. overproducial production of the thyroid hormones T3 and T4. Normal thyroid levels are also seen, sometimes hypothyroidism, which can also cause hypothyroidism (although that is not the cause of Graves' disease). Hyperthyroidism in Graves disease is confirmed by measuring elevated blood levels of free (unjoined) T3 and T4, as are other causes of hyperthyroidism. Other useful laboratory measurements are thyroid stimulating hormone (TSH, usually undetectable in critical diseases due to negative feedback from high T3 and T4), and protein-binding iodine (rise). Serologically detected thyroid-stimulating antibodies, radioactive iodine (RAI) upinture, or thyroid ultrasound with Doppler can all independently confirm the diagnosis of Graves disease. A biopsy is usually not required, but can be obtained if a thyroidectomy is performed. Thyroiditis of Graves disease is often not nodules, but thyroid nodules are also common. [18] It is important to distinguish common forms of hyperthyroidism, such as Graves' disease, monothyroidism, and toxic multiple goiter, in order to determine the appropriate treatment. [18] The digitization between these entities is based on imaging andTesting has been improved. Measuring TSH receptor antibodies using h-TBII assays has proven to be efficient and was the most practical approach in a single study. [19] Eye disease Details: Greves Ophthalmology Thyroid-related ophthalmology (TAO) or thyroid eye disease (TED) is the most common extrathyroid condition. It is a form of idiogenic lymphocytic orbital inflammation, and its etiology is not fully understood, but it is believed that autoimmune activation of orbital fibroblasts that express TSH receptors in TAO plays a central role. [20] Enlarged extraorbital muscles, fat production, and deposition of non-sulfatoized glycomoglycans and hyaluronic acid cause venous occlusion leading to intraocular neuropathy, increased intraocular pressure, proptosis, chemosis and peri-orbital edema, and progressive remodeling around the orbit, within the area around the bone, within the eye sockets of the eye. [21] [22] Other characteristic features of TAO include reticulion of the lid, restrictive myopathy, upper limb corneal conjunctivitis, and exposed keratosis. The severity of eye disease may be classified by mnmonic: NO SPECS:[24] Class 0: Signs and Symptoms Class 1: Signs (pulling and staring at the upper lid, with or without lid delay) Class 2: Involvement of soft tissue (conjunctival and lid edema, Conjunctival injections, etc.) Class 3: Extraocular muscle involvement (usually diproprecia) Class 5: Corneal involvement (mainly due to Ragov Talmos) Class 6 : Vision loss (due to optic nerve involvement) Typically, the natural history of TAO follows Rundle's curve describing rapid deterioration in the early stages, improving to the peak of maximum severity and to plate rest, however, returning to normal. [25] Treatment of serious diseases includes antithyroid drugs that reduce the production of thyroid hormones. Radioactive iodine (radioactive iodine I-131); thyroidectomy (surgical resection of glands) Surgery in patients with hyperthyroidism is dangerous, and before thyroidectomy, preoperation with antithyroid drugs is given to the patient to the euthyroid (i.e. normothyroid). Each of these treatments has its pros and cons. No one thinks a therapeutic approach is best for everyone. Treatment with antithyroid drugs must effectively be given from 6 months to 2 years. Still, hyperthyroidism can recur at the time of stopping the drug. The risk of recurrence is about 40-50%, and lifelong treatment with antithyroid drugs has side effects such as agranulocytosis and liver disease. [26] Side effects of antithyroid drugs include a potentially fatal reduction in white blood cells levels.Although it is the most common treatment in the United States along with radioactive iodine, antithyroid drugs and/or thyroidectomy are more frequently used in Europe, Japan, and most of the world. β blockers (such as propranolol) can be used to suppress sympathetic nervous system symptoms of tachycardia and nausea until antithyroid treatment is effective. Pure β blockers do not α eye lids, which are mediated by adrenergic receptors. Antithyroid drugs The main antithyroid drugs are carbimazole (UK), metimazole (USA), and propylthiouracil / PTU. These drugs block iodine binding and yodotyrocin coupling. The most dangerous side effects are agranulocytosis (1/250, more in PTU). Patients with these drugs should see a doctor if they develop a sore throat or fever. The most common side effects are rashes and peripheral neuritis. These drugs also cross the placenta and are secreted with breast milk. Lugol's iodine can be used to block hormone synthesis before surgery. A randomized controlled trial testing Graves' found single-dose treatment of metimazole achieved euthyroid status more effectively after 12 weeks than propylsuasil (methimazole 15 mg vs. 19.4% propyltiuracil 150 mg group). [27] Differences in results were shown to add thyroxine to antithyroid drugs and continue thyroxine vs. placebo after antithyroid drug withdrawal. However, two markers were found to help predict the risk of recurrence. These two markers are positive TSHr antibody (TSHR-Ab) and smoking. Positive TSHR-Ab at the end of antithyroid treatment increases the risk of recurrence to 90% (sensitivity 39%, specificity 98%), and negative TSHR-Ab at the end of antithyroid drug treatment is associated with a 78% probability of remaining in remission. Smoking was shown to have an independent effect on plus TSHR-Ab. [28] Radioactive iodine (radioactive iodine-131) before (top) and after (bottom) radioactive iodine therapy of the affected thyroid gland was developed at the Mallinglot General Clinical Research Center in the early 1940s. This modality is suitable for most patients, but some prefer to use it mainly for older patients. Indications for radioactive iodine are cases where medical or surgical therapy is contraindicated, failing medical therapy or surgery. Hypothyroidism may be a complication of this treatment, but if it appears it can be treated with thyroid hormones. The rationale for radioactive iodine is that it accumulates in the thyroid gland and irradiates the glands with its beta and gamma radiation, and about 90% of its total radiation is emitted by beta (electron) particles. The most common method of iodine-131 treatment is to administer a specified amount.Per gram of thyroid gland based on palpation or radiological imaging of glands over a 24-hour period. [29] Treated patients should be regularly monitored with thyroid blood tests to ensure they are treated with thyroid hormones before they become symptomatically hypothyroidism. Contraindications to RAI are pregnancy (absolute), ophthalmology (relative, which can exacerbate thyroid eye disease), or solitary nodal. [31] The disadvantage of this treatment is a high incidence of hypothyroidism (up to 80% requires final thyroid hormone replacement in the form of daily tablets. . .) Radioactive iodine treatment acts slowly (over months and years) to destroy the thyroid gland, and graves disease-related hyperthyroidism is not cured in everyone by radioactive iodine. It has a recurrence rate that depends on the dose of radioactive iodine administered. In rare doses, radiation-induced thyroiditis is associated with this treatment. [32] Surgery Details: Thyroid resection This modality is suitable for young and pregnant people. Indications for thyroidectomy can be told into absolute indications or relative indications. These signs help determine which people will benefit most from surgery. The absolute indication is if it is a large thyroid device (especially when compressing the trachea), suspected nodular or suspected cancer (pathologically examining the thyroid gland), a person with ophthalmology, and a person's preferred method of treatment, or refusing radioactive iodine treatment. Pregnancy is recommended to be delayed for 6 months after radioactive iodine treatment. [26] Both bilateral small-sided thyroidectomy and Hartley-Dunhill procedure (thyroidectomy on one side, partial lobectomy on the other) are possible. The benefits are immediate healing and the potential removal of cancer. Its risks are recurrent laryngeal nerve injury, hypothyroidism (due to removal of the parathyroid gland), hematoma (which can be life-threatening if the trachea is compressed), relapse after treatment, infections (less common), scarring. The increased risk of nerve damage can be attributed to an increase in blood vessels in the thyroid gland and the development of a link between thyroid capsules and surrounding tissue. According to reports, there is a 1% incidence of permanent recurrent laryngeal nerve palsy after a complete thyroidectomy. The removal of the glands allows you to perform a complete biopsy and have clear evidence of cancer anywhere in the thyroid gland. (Needle biopsies are not so accurate in predicting the normal state of the thyroid gland.) No further treatment of the thyroid gland is required unless cancer is detected. Radioactive iodine intake research is carried out after surgery to ensure that all remaining (potentially cancerous) thyroid cells (i.e., near nerves to the vocal cords) are destroyed.This means that the only remaining treatment is levothyroxine, or thyroid replacement drugs, to be taken for the rest of the patient. A 2013 review article concluded that surgery appears to be the most successful in managing Graves' disease, and that total thyroidectomy is the preferred surgical option. [33] Eye This section does not cite any source. Improve this section by adding citations to trusted sources. Unso supplied materials may be challenged and removed. (May 2014) Mild cases (see how and when to delete this template message) are treated with lubricant eye drops or nonsteroidal anti-inflammatory drugs. Severe cases are treated with vision-threatening (corneal exposure or optic nerve compression) steroids or orbital decompression. In all cases, smoking cessation is essential. Double vision can be corrected with prism glasses and surgery (the latter only if the process has been stable for a while). Difficult to close your eyes are lubricant gels at night, or tape to your eyes to allow for a full, deep sleep. Orbital decompression can be performed to cause bulging eyes to retreat to the head. The bones are removed from the skull behind the eyes, creating space for muscles and adipipipodic tissue to return to the skull. Eyelid surgery can go to the upper eyelids and/or lower eyelids to reverse the effects of Graves' disease on the eyelids. The eyelid muscles become tighter with Graves disease and cannot close the eyes the long time. Eyelid surgery involves a muscle scraping that cuts along the natural folds of the eyelids and keeps the eyelids open. This weakens the muscles and allows the eyelids to stretch more effectively over the eyeballs. Eyelid surgery can help reduce or eliminate dry eye symptoms. For the management of clinically active Graves disease, orbitalosis (clinical activity score >2) with at least mild to moderate severity is the treatment chosen by intravenous glucocorticoids, usually administered in the form of pulsed intravenous methylprednisolone. Studies have consistently shown that pulsed intravenous methylprednisolone is superior to oral glucocorticoids in terms of efficacy and reduced side effects for managing significant orbitalosis. .▲Prognosis If left unedited, birth defects in pregnancy, increased risk of miscarriage, mineral loss in bones [36], and, in extreme cases, death, more serious complications may occur. Serious illnesses are often accompanied by increased heart rate and can lead to further heart complications, including the loss of normal heart rhythms (atrial fibrillity), which can lead to stroke. If the eyes are lowered (bulging) so that they do not close completely at night, dryness can occur and the risk of secondary corneal infection can go into bed. Pressure on the optic nerveThe earth, likewise, can lead to visual field defects and loss of vision. Long-term, unedited hyperthyroidism can cause bone loss and may be resolved during treatment. Epidemiological grave disease occurs in about 0.5% of people[[3] about 7.5 times more frequently in women than in men.[3] 1] Often begins between the ages of 40 and 60. [5] It is the most common cause of hyperthyroidism in the United States [5] (about 50-80% of cases). [1] History's grave disease is named after Robert James Graves,[37], an Irish doctor who described the case of Gita with the outer eye in 1835. [38] Medical names are often styled non-exclusively. Therefore, grave disease and grave disease are the styling of variants of the same term. Germany's Karl Adolf von Bassedou independently

reported a constellation of the same symptoms in 1840. [39] As a result, in continental Europe, the terms Sebassou syndrome, Bassedow disease, or Morvas cesato[42] are more common than grave diseases[41][43] grave disease[41][42] also called external eye gug ITER. [41][42] Begbee's disease, Frajani's disease, Frajani-Sebatow syndrome, and Marsh's disease are generally unknown. The names of these diseases come from Caleb Hillier Parry, James Begbee, Giuseppe Frajani and Henry Marsh. Early reports of Gaiter's case with the outer eye were published in Italians Giuseppe Frajani[44] and Antonio Giuseppe Tester (1802 and 1810), respectively. [46] Before these, Caleb Hillier Parry, [47] a prominent provincial physician in late 18th-century England (and a friend of Edward Miller Garces), [48] described a case in 1786 that was not made public until 1825 and was still a decade earlier than Graves. However, fair credit for the first account of grave disease went to the 12th-century Persian doctor Sayyid Ismail al-Duljani[50] who pointed to the association between the gaiger and the outer eye in the Shah's thesaurus of Kwalasm, the main medical dictionary of the time. [41] [51] A remarkable case of society and culture Marty Feldman used his bulging eyes caused by grave disease, for the Gordick effect. Japanese singer Ayaka was diagnosed with Graves' disease in 2007. After her diagnosis and publicity in 2009, she took a two-year hiatus from music to focus on her treatment. [53] Susan Elizabeth Blow, an American educator and founder of the first publicly funded kindergarten in the United States, was forced to leave in 1884 and in 1884 was forced to seek treatment for grave disease. Former U.S. President George H.W. Bush developed new atrial fibrillosis and was diagnosed with hyperthyroidism for the disease in 1991 and treated with radioactive iodine. The president's wife, Barbara Bush, also developed the disease around the same time, in her case, inging severely invasive external eyeballs. [56]Dangerfield, American comedian and actor[57] Gail Devers, American sprinter: Doctors considered amputating her leg after she developed watering and swelling following radiation treatment for Graves disease, but she recovered and went on to win olympic medals. Missy Elliott, American hip-hop artist[58] Marty Feldman, British comedy writer, comedian and actor[59][60] Sia Furler, Australian singer-songwriter[61] Sammy Gravano, italian-American underboss of the Gambeano crime family. Scottish rugby player Jim Hamilton discovered Graves' disease shortly after retiring in 2017 [Heino, a German folk singer, whose dark sunglasses (worn to hide his symptoms) became part of his trademark look[64] Herbert Howells, an English composer. Yayoi Kusama, a Japanese artist, was the first person to be treated with radium injections. [66] Nadezhda Krupskaya, Russian Communist, wife of Vladimir Lenin[67] Barbara Lee, former American actress and fashion model, now a spokeswoman for the National Cemetery Disease Foundation[68] a Japanese singer and half of the duo Pink Lady. [69] [70] [71] [72] Yuko Miyamura, Japanese voice actor[73] Lord Monckton, a former UKIP and conservative politician, and a skeptical point on climate change. [74] Sofia Parnok, Russian poet[75][76][77] Sir Cecil Springlais, Britain's ambassador to the United States during World War I, died suddenly of the disease in 1918. [78] Christina Rossetti, English Victorian poet[79] Dame Maggie Smith, British actress[80] Mary Webb, British novelist and poet[81] Wendy Williams, American TELEVISION host[82] Literature in the conscience of Italo Svevo's novel Xeno, character Ada develops illness. [83] A research agent acting as an antagonist for thyroid stimulating hormone receptors is currently under investigation as a treatment for serious diseases. [85] See ^ a b c d e f g h i j k l n n o p p t u v w x y z Grave disease www.niddk.nih.gov. Archived from the original on August 10, 2012 and April 2, 2015. Acquired 2015-04-02. ^ a b c Menconi F, Markoch C, Marino M (2014). Diagnosis and classification of grave diseases. Autoimmune review. 13 (4–5): 398–402.Doi:10.1016/j.autrev.2014.01.013.PMID 24424182.^ a b c d d f g h Brent GA (June 2008). Clinical practice. serious illness. New England Journal of Medicine.358 (24): 2594–605.Doi: 10.1056/NEJMcp0801880. PMID 18550875.^ b c Birch HB, Cooper DS (December 2015). Management of grave diseases: a review. Jama. 314 (23): 2544–54.Doi:10.1001/jama.2015.16535.PMID 26670972.^ b c d e Nikiforf YE, Vidinter PW, Nikiforova LD, Vander PW (2012).Diagnostic pathology and molecular genetics of the thyroid gland (2nd) Philadelphia: Walters Kluwar Health/Lippincott Williams & Wilkins p. 69.Archived from the original to ISBN 9781451114553.2017-09-08. ^ N Barrow G, HJ, Volpe R (1989). 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