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Chapter 1 : Hossein Gharib - Wikipedia

This is the fatal error because these tests only pick up the most severe cases of hypothyroidism and miss virtually all of the milder cases that would respond favorably to thyroid hormone treatment. Cognitive, psychological and physical symptoms were measured. To qualify for the study, the 25 symptomatic patients had to have at least three symptoms from among a list that included: According to the researchers, thyroxine had no benefit in the symptomatic people. Of particular interest was the fact that the symptomatic group responded to placebo better than controls on 3 of 15 psychological measures, versus no improvement in the control group. Weetman concludes his commentary: But the Pollock study is not sufficient enough to rely on as a guide in any clinical decision making. Because, while Weetman may be satisfied that a normal TSH and free T4 rule out hypothyroidism or further treatment, he has overlooked a number of obvious issues that call the study results into question, as well as a number of unresolved questions that are at the real heart of the controversy. Dosage Size mcg. A key question is how was this dosage determined? According to many thyroid experts, hypothyroidism patients require on average a dose of approximately 1 microgram per pound of body weight. Would study results have been different had the dosage been mcg? How was this particular dosage determined, and how do the results at this dosage somehow preclude that another dosage -- perhaps a smaller or larger dose -- might not have different results? Without testing of T3 levels, there is no way to know if these patients had low or out of range T3 levels. Study Length It can take a long time to become hypothyroid, and get properly diagnosed, and as most thyroid patients know, it can take an equally long time to see any notable improvements in symptoms. After a diagnosis of hypothyroidism, treatment begins, and many patients find that blood levels return to normal, making them "euthyroid," weeks or months before symptoms resolve. So patients may be biochemically euthyroid, but it can be many months before any noticeable improvement in symptoms is felt. One endocrinologist regularly counsels her patients to wait at least four months after achieving euthyroidism before exploring additional options, supplemental or alternative drugs, or dosage changes to deal with their unresolved symptoms. This length of time seems utterly inadequate if even one endocrinologist will decide to make important diagnostic decisions based on the findings of this research. Compounded with the issue of a short duration, the extremely small size of this study calls the results of the study even further into question. In addition to discovering that nearly five percent of Americans suffer from often undiagnosed thyroid disease, the Centers for Disease Control and Prevention CDC report on the National Health and Nutritional Survey NHANES found that among the disease-free population those who did not have any presence of thyroid antibodies, or diagnosed thyroid disease , the mean TSH level was 1. This finding could bolster the assertions of some practitioners and many patients that the optimal TSH levels are between 1 and 2, and that levels above that may in fact represent an abnormality. It certainly points up the need to reconsider the basis for most U. Weetman himself appears to have overlooked his own study of the subject of TSH levels, which appeared in the 19 April issue of the British Medical Journal, in which he said: If relying on blood tests for diagnostic purposes, to fully assess whether or not someone should receive thyroid treatment, complete testing for thyroid antibodies should also be performed. After 1 year of therapy with levothyroxine, the antibody levels and lymphocytes evidence of inflammation decreased significantly only in the group receiving the medication. Among the untreated group, the antibody levels rose or remained the same. But this oversight is a major one, one that ignores some of the most cutting-edge research into the autoimmune process and its role in hypothyroidism, and continues to call his conclusions into serious question. If Weetman believes that the use of blood testing is in fact the gold-standard, then he would be doing a service to thyroid patients and the practice of endocrinology to call for a complete re-evaluation of the reference ranges for normal TSH levels, so that the numbers he and his

colleagues wish to rely so heavily on are in fact valid. Given the research, he should also be advocating the inclusion of antibodies tests as part of any standard thyroid panel to uncover thyroid disease and hypothyroidism. Because ultimately, the validity of research on the fundamental way hypothyroidism is treated rests on an outdated TSH reference range that calls out for a complete re-evaluation and revamping. This re-evaluation of the TSH reference range is the first priority among many much-needed and essential reforms to conventional thyroid doctrine and practice.

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Chapter 2 : Cate Pihoker, MD

The authors address potential conflicts of interest in the Endocrine Society's Clinical Practice Guidelines (CPGs) and outline the Society's ongoing efforts to enhance the value of its CPGs.

Endocrinology Conferences Diabetes Conferences Medical Conferences Medical doctors, patients and health care providers consider the prevention of endocrine disorders as an essential tool to improve the general health status of the population. According to a recent statistical survey, the proportions of people suffering from the disease are expected to increase in future. Realizing this imperative, Conference Series is set to organize 13th European Diabetes and Endocrinology Congress to be held during November , at Dublin, Ireland with a view to promote awareness and enhance research aiming in developing solutions for the challenges encountered. Diabetes professionals from around the world will gain unparalleled access to the best scientific research programs at Euro Endocrinology There are 3 types of diabetes: The body does not produce insulin. People usually develop type 1 diabetes before their 40th year, often in early adulthood or teenage years. The body does not yield enough insulin for appropriate function, or the cells in the body do not counter to insulin Gestational Diabetes: It is developed in women during pregnancy period. It is caused due to dysfunction of insulin receptors. The abstracts are peer reviewed prior to their acceptance for the conference. Diabetes is broadly acknowledged as one of the foremost causes of death and incapacity in the United States. It was the sixth leading cause of death in However, diabetes is likely to be underreported as the underlying cause of death on death certificates. About 65 percent of deaths among diabetic patients are ascribed to heart disease and stroke. Diabetes is accompanied with long-term complications that affect almost every part of the body. The disease often leads to blindness, heart and blood vessel disease, stroke, kidney failure, amputations, and nerve damage. Unrestrained diabetes can complicate pregnancy, and birth defects are more common in babies born to women with diabetes. Oral anti-diabetics were the leading category of drugs in and showed a growth rate of 6. The total sales for insulin products increased significantly as well. Endocrinology is a specialty of medicine; some would say a sub-specialty of internal medicine, which deals with the diagnosis and treatment of diseases related to hormones. Endocrinology covers such human functions as the coordination of metabolism, respiration, reproduction, sensory perception, and movement. Endocrinology also focuses on the endocrine glands and tissues that secrete hormones. Global endocrinology drugs market is expected to witness lucrative growth over the forecast period owing to rise in incidence rates of endocrine diseases. Growing adolescent populations which are at high risk of development of endocrine gland and associated disorders as a consequence of imbalance in hormones, and increasing prevalence of diabetes are also factors expected to drive growth of this market. Increasing disease inducing unhealthy lifestyle habits such as smoking, high intake of cholesterol, weight gain, and work stress which enhance the incidence rates of endocrine disorders are expected to fuel market growth throughout the forecast period. Major Diabetes Company List:

Chapter 3 : FDA Approved Drugs in Endocrinology | CenterWatch

The Journal of Clinical Endocrinology & Metabolism Sympathoadrenal Counterregulation in Patients with Hypothalamic 2, 1 February

Advanced Search In humans, the role of hypothalamic centers for activation of counterregulatory release of catecholamines and glucagon during hypoglycemia is unclear. To address this question, we investigated the counterregulatory response to acute insulin-induced hypoglycemia of glucagon, epinephrine, and norepinephrine in eight patients who had undergone transcranial surgery for a craniopharyngioma extending to the hypothalamic region. After the iv injection of 0. All subjects recovered spontaneously from hypoglycemia within 2 h. During hypoglycemia, virtually no adrenergic symptoms tremor, heart pounding, and anxiety were reported by these five patients, and changes in the heart rate were diminished. In three craniopharyngioma patients, the counterregulatory increase in catecholamines was unimpaired, adrenergic symptoms were reported and a rise in heart rate was observed during hypoglycemia. In all craniopharyngioma patients, the counterregulatory glucagon response to hypoglycemia was preserved and orthostasis increased both catecholamines and the heart rate similar to in the patients with hypopituitarism as well as in the healthy controls. Our results demonstrate selective impairment of counterregulatory sympathoadrenal activation in patients who had undergone surgery for a craniopharyngioma extending to the hypothalamic region. This strongly suggests the involvement of hypothalamic centers in hypoglycemia-induced activation of the sympathoadrenal axis in humans. It remains unclear as to whether hypoglycemia-induced glucagon secretion is also controlled by the hypothalamus. However, a common hypothalamic center controlling both counterregulatory catecholamine and glucagon release is unlikely, and sympathoadrenal activation is not required for hypoglycemia-induced glucagon secretion in humans. These hormones act in concert to prevent or correct hypoglycemia. The role and contribution of the individual counterregulatory hormones in the correction of hypoglycemia is relatively well understood 1 , 2. The mechanisms, however, and the loci responsible for activation of counterregulatory hormone release are less well defined. In various animal models, conflicting results were reported concerning the tissues that sense hypoglycemia and those which coordinate the counterregulatory endocrine responses 3 â€” More recent studies in rats suggested that nuclei in the ventromedial hypothalamus are involved in the activation of counterregulatory glucagon and catecholamine secretion 13 , In humans, however, the role of hypothalamic centers for activation of counterregulatory release of catecholamines and glucagon during hypoglycemia is poorly understood. Evidence that the hypothalamus is involved stems from a patient who suffered from neurosarcoidosis and infiltration of the hypothalamus, and who had a complete loss of the counterregulatory response to hypoglycemia To further explore this question, we investigated the counterregulatory response of glucagon, epinephrine, and norepinephrine in patients who had undergone transcranial surgery for a craniopharyngioma extending to the hypothalamic region. We compared them to a group of patients suffering from hypopituitarism and to a group of normal subjects. Subjects and Methods Subjects Eight patients who received transcranial surgery for a craniopharyngioma extending to the hypothalamic region group A , four patients with hypopituitarism group B , and six healthy volunteers group C were studied. Postoperatively, the patients required standard replacement therapy for hypopituitarism hydrocortisone, levothyroxine, and sexual hormones , and diabetes insipidus desmopressin. According to their adrenergic counterregulatory response, the patients were divided into groups A1 and A2 see Results. The BMI of patients in group A1 was Five craniopharyngioma patients suffered from postoperative weight gain four in group A1 and one in group A2. Causes of the hypopituitarism in these patients were due to transsphenoidal surgery of a chromophobe pituitary adenoma 1 patient , autoimmune hypophysitis one patient , and empty sella syndrome two patients. All patients received standard replacement therapy for hypopituitarism hydrocortisone, levothyroxine, and sexual hormones. Screening of the volunteers included a medical history, a physical examination, and routine

laboratory testing. None of the healthy volunteers had a history or showed signs of complete or partial hypopituitarism. Written informed consent was obtained from each subject, and the study was approved by the local ethical committee. Study design All subjects reported to the hospital at h after an overnight fast of 10â€”12 h, and the patients were advised to take their regular hormone replacement therapy hydrocortisone and levothyroxine between and h. An indwelling central venous catheter and an iv cannula in the opposite arm was put in place. Thereafter, the patients rested in a degree position for at least 1 h before the commencement of blood sampling and until the end of the insulin hypoglycemia test. Baseline sampling was started at h and human insulin 0. Samples for plasma glucose, plasma catecholamines, and glucagon were taken for 2 h thereafter. Blood samples were drawn every 2 min for determining plasma catecholamines and plasma glucose, and every 4 min for determining plasma glucagon. The samples were collected into prechilled EDTA-containing tubes. The protease inhibitor aprotinin Bayer Corp. The total volume of blood taken during the test was approximately ml. The venous catheter was kept open by a slow infusion of saline total volume ml. For subjects safety glucose levels were monitored in parallel by a glucose sensor Glucometer Elite, Bayer Corp. In accordance with Towler et al. Each symptom was given a score from 0 none to 5 severe. The mean of the two scores for each time segment was taken for further analysis. The neurogenic and neuroglycopenic symptom scores were obtained by adding the scores of the respective symptoms. To calculate the hypoglycemia-induced differences in the adrenergic or cholinergic symptom score, the incremental changes in the respective parameters from baseline to hypoglycemia were added. The heart rate was determined at baseline, during hypoglycemia and at the end of the test. After the insulin-hypoglycemia test, the subjects were allowed to stand-up, and after 10 min blood samples for the measurement of plasma catecholamines were taken. The heart rate was measured before orthostasis and once again after a min time lapse. Plasma epinephrine and norepinephrine were determined in duplicate by a single isotope COMT radioenzymatic assay combined with separation of the labeled metabolites by reverse-phase HPLC before scintillation counting 17 , The lower detection limit of this method was 5. The intra and interassay coefficients of variation at the relevant plasma concentrations were 5. The intra and interassay coefficients of variation were 4. All samples were assayed in duplicate and the samples of one subject were analyzed in the same run to avoid interassay variation. The two-tailed paired t test was used to analyze the effects of hypoglycemia and orthostasis on heart rate, epinephrine and norepinephrine within groups as indicated in the text. Results Insulin-induced hypoglycemia Plasma glucose. In the craniopharyngioma patients group A and in the patients with hypopituitarism group B , baseline plasma glucose concentrations were 4. In all three groups including the subgroups A1 and A2 see below , the plasma glucose concentrations declined to similar minimum levels within 30 min after the iv insulin bolus Fig. All subjects recovered spontaneously from hypoglycemia within a period of 2 h. For patients of group A and group B, the time course of recovery was slower and the plasma glucose concentrations reached at the end of the test were lower than in group C Fig. In healthy subjects, plasma glucose amounted to 4. Plasma glucose concentrations during insulin-induced hypoglycemia. The arrow indicates the iv administration of 0. In group C, hypoglycemia created a 9. In the eight craniopharyngioma patients, plasma epinephrine rose 4. In the other 3 patients group A2 plasma epinephrine concentrations rose 9. In group C, hypoglycemia caused a 2. In the eight craniopharyngioma patients, plasma norepinephrine rose 2. In the five patients of group A1, however, plasma norepinephrine levels rose only 1. Patients with hypopituitarism had a 2. View large Download slide Changes in plasma catecholamines and plasma glucagon during insulin-induced hypoglycemia. The arrow indicates the iv administration of human insulin. Data are expressed as a percentage of the mean baseline concentration of the respective hormone before the insulin bolus. The increases in plasma glucagon in response to hypoglycemia were comparable in all three groups Fig. In the eight craniopharyngioma patients, glucagon increased 2. In patients of group A1, who had an impaired catecholaminergic response to hypoglycemia, glucagon increased 1. Changes in heart rate during hypoglycemia.

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Chapter 4 : Issues | The Journal of Clinical Endocrinology & Metabolism | Oxford Academic

Identification of Bisphenol A as an endocrine disrupter based on its effects on the female reproductive system, the mammary gland, the metabolism and the neurodevelopment.

Early life[edit] Gharib grew up in Tehran, the son Dr. Mohammad Gharib â€" , a former professor and Chair of Pediatrics at Tehran University â€" , and Zahra Gharib â€" , daughter of Ostad Abdolazim Gharib â€" , a professor of Persian literature. Gharib attended Ferdowsi Grade School â€" and later Alborz High School â€" , graduating with honors in He traveled to the United States to study of medicine. He received a B. He went on to receive a medical degree from the University of Michigan Medical School in , and took his internship at Philadelphia General Hospital from to He completed an internal medicine residency â€" and fellowship in endocrinology and metabolism â€" at the Mayo Clinic in Rochester , Minnesota. Professional career[edit] In , he returned briefly to Iran, where he served on the faculties of the University of Tehran and the National University Medical Schools. He also teaches and conducts clinical research. Research In , Dr. Gharib and his colleagues developed the first radioimmunoassay RIA to measure triiodothyronine T3 in human serum. In the s, Dr. Gharib focused his attention on nodular thyroid disease NTD and thyroid cancer , making a number of important contributions to thyroid practice. For example, an early paper emphasized the importance of NTD in clinical practice New England Journal of Medicine, ; several studies illustrated the technique, accuracy, and impact of thyroid fine-needle aspiration FNA biopsy in the management of nodules Acta Cytol, ; Annals of Internal Medicine, ; and defined the limitations of FNA Annals, He gets credit for helping establish the accuracy and safety of thyroid FNA biopsy in the management of thyroid nodular disease. His seminal study published in NEJM in was a landmark report that challenged the conventional wisdom that long-term thyroid hormone therapy shrinks thyroid nodules. Additionally, Gharib and his colleagues described new algorithms for genetic testing in MTC syndromes Annals, Gharib in his Mayo Clinic Office, Publications, Lectures and Memberships Gharib co-edited the first[citation needed] evidence-based endocrinology textbook, originally published in , with the third edition printed in In , he and three colleagues edited and published the textbook Endocrinology: In he published the book Thyroid Nodule. Gharib has lectured at endocrine events, and has been a visiting professor at academic institutions. Other published works include more than peer-reviewed original papers, 50 review articles and 30 textbook chapters. Hossein, his siblings, and other members of the Gharib family are also portrayed in the film by Iranian actors.

Chapter 5 : Journal of Clinical Endocrinology and Metabolism

American Journal of Physiology-Endocrinology and Metabolism. February Pages EE March Pages EE

Chapter 6 : Theses on Endocrinology and Metabolism | International Library for Thesis - Photon eBooks

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Chapter 7 : Î£Ï…Î¼Î¼Î„Î¿Ï‡Î® Ïƒ Îµ Ï…Î½Î-Î´Ï•Î¹Î± - Endometabolism

The Journal of Clinical Endocrinology & Metabolism is the world"s leading peer-reviewed journal for endocrine clinical research and cutting edge clinical practice reviews. Each issue provides the latest in-depth coverage of new developments enhancing our understanding, diagnosis and treatment of endocrine and metabolic disorders.

Chapter 8 : Diabetes International Conferences | Diabetes Endocrinology Events

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Intern in Internal Medicine, Hospital of the University of Pennsylvania, Resident in Internal Medicine, Hospital of the University of Pennsylvania, Fellow, Endocrinology, Diabetes & Metabolism, Hospital of the University of Pennsylvania,

Chapter 9 : Christian L. Roth, MD

1 February | American Journal of Physiology-Endocrinology and Metabolism, Vol. , No. 2 Comparison of GH, IGF-I, and testosterone with mRNA of receptors and myostatin in skeletal muscle in older men.