PITUITARY STRUCTURE. The pituitary gland, located underneath the hypothalamus of the brain, is vital to body physiology because its hormones not only exert direct action on body organs (e.g., prolactin on mammary glands and antidiuretic hormone on the kidney) but also regulate the activity of several target endocrine glands (e.g., thyroid and gonads). The pituitary gland is controlled by the brain and mediates the effects of the central nervous system on hormonal activity in the body, which explains its critical anatomic position in relation to the brain.

The pituitary (hypophysis) is divided into an anterior lobe (adenohypophysis), a posterior lobe (neurohypophysis), and an intermediate lobe. In humans, the intermediate lobe either does not exist or is vestigial, consisting of a few cells with no known functions. The pituitary is connected
to the brain via the hypophyseal stalk. This plate focuses on the structure and functions of the posterior lobe, to illustrate the concept of neurosecretion. Neurosecretion is also essential for understanding of anterior lobe function, and is the cornerstone of the modern science of neuroendocrinology.

POSTERIOR PITUITARY AND HYPOTHALAMUS. The posterior lobe of the pituitary secretes two hormones, antidiuretic hormone (ADH) and oxytocin. The posterior pituitary is not an endocrine gland because it does not contain true secretory cells. In fact, the gland, being an extension of the brain hypothalamus, consists mainly of nerve fibers and nerve endings of the neurons of two hypothalamic nuclei. These neurons have their cell bodies in the hypothalamus and send their axons (hypothalamo-hypophyseal tract) to the posterior pituitary through the hypophyseal stalk.

NEUROSECRETION. These hypothalamic nuclei are the supraoptic and paraventricular. The neurons of these nuclei are typical examples of neurosecretory cells. The cell bodies of these special neurons are the site of the synthesis of the hormones which, in the case of the posterior pituitary, are synthesized as larger prohormone molecules. These molecules contain the true hormone and a nonhormonal portion called neurophysin, which may function in hormone transport. The prohormone complexes are packed within the vesicles (Herring bodies), which flow down the axon by rapid axoplasmic transport.

Before reaching the nerve terminals in the posterior lobe, the hormone is split off the larger prohormone and stored in the axon terminals, to be released into the blood capillaries and carried out to the target tissues. The stimulus for hormone release is the nerve impulse arriving from the cell body down the axon membrane to the terminal, which causes calcium ions to flow into the
terminal. This leads the secretory vesicles to fuse with the terminal membrane and the hormone to be released into the extracellular fluid and blood capillary.

ANTIDIURETIC HORMONES (VASOPRESSIN). The cells of the supraoptic nucleus make and secrete principally the antidiuretic hormone (ADH, also called vasopressin). Involved in regulating body water, ADH is secreted whenever the amount of water in the blood is decreased, as in dehydration due to excessive sweating or osmotic diuresis (caused by an increase in glucose or ketone bodies or sodium loss in the urine), as well as during hemorrhage and blood loss. The signal for ADH release is believed to be an increase in the osmolarity of the blood mediated by an increase in the concentration of sodium ions in the plasma. The sodium elevation is sensed by specific osmoreceptor neurons in the hypothalamus, which in turn stimulate the supraoptic neurons to release ADH from the posterior pituitary. ADH acts principally on the collecting ducts in the kidney, by increasing their permeability to water. Water moves by osmosis from the kidney ducts to the plasma, decreasing plasma osmolarity. (See plate 62.) ADH is also secreted when mechanoreceptors (volume receptors) in the heart, and pressure receptors in the vasculature, are stimulated after hemorrhage and blood loss. After a hemorrhage, ADH causes vasoconstriction, leading to an increase in blood pressure (vasopressive action).

OXYTOCIN HORMONE. Oxytocin is secreted principally by the cells of paraventricular nuclei, stimulated by sensory mechanoreceptors in the nipples of the breasts and cervix of the uterus, as part of neurohormonal reflex arcs. Sensory nerves convey the signals from the sensory receptors to the hypothalamus, leading to the secretion of oxytocin from the posterior pituitary. During labor, oxytocin acts on the myometrium of the uterus to cause massive contractions, eliciting the expulsion of the fetus (oxytocin = swift birth). During lactation, oxytocin acts on the myoepithelium of the mammary glands to elicit their contraction and cause the ejection of milk. (See also plates 150, 151.) There are no known functions for oxytocin in the male.
Oxytocin and ADH are both polypeptides containing nine amino acids. Their structures are identical except for the substitution, in ADH, of phenylalanine and arginine in place of one of the tyrosine and the leucine found in oxytocin.
EXCESS GROWTH HORMONE

**Acromegaly** is a hormonal disorder that results when the pituitary gland produces excess growth hormone (GH). It most commonly affects middle-aged adults. Once recognized, acromegaly is treatable in most patients, but because of its slow and often insidious onset, it frequently is not diagnosed correctly.

The name acromegaly comes from the Greek words for “extremities” and “enlargement” and reflects one of its most common symptoms, the abnormal growth of the hands and feet.

Soft tissue swelling of the hands and feet is often an early feature, with patients noticing a change in ring or shoe size. Gradually, bony changes alter the patient’s facial features: the brow and lower jaw protrude, the nasal bone enlarges, and spacing of the teeth increases.

Overgrowth of bone and cartilage often leads to arthritis. When tissue thickens, it may trap nerves, causing carpal tunnel syndrome, characterized by numbness and weakness of the hands. Other symptoms of acromegaly include thick, coarse, oily skin; skin tags; enlarged lips, nose and tongue; deepening of the voice due to enlarged sinuses and vocal cords; snoring due to upper airway obstruction; excessive sweating and skin odor; fatigue and weakness; headaches; impaired vision; abnormalities of the menstrual cycle and sometimes breast discharge in women; and impotence in men. There may be enlargement of body organs, including the liver, spleen, kidneys and heart.
The most serious health consequences of acromegaly are diabetes mellitus, hypertension, and increased risk of cardiovascular disease. Patients with acromegaly are also at increased risk for polyps of the colon that can develop into cancer.

When GH-producing tumors occur in childhood, the disease that results is called gigantism rather than acromegaly. Fusion of the growth plates of the long bones occurs after puberty so that development of excessive Growth Hormone production in adults does not result in increased height. Prolonged exposure to excess Growth Hormone before fusion of the growth plates causes increased growth of the long bones and increased height.

Acromegaly is caused by prolonged overproduction of Growth Hormone by the pituitary gland. The pituitary is a small gland at the base of the brain that produces several important hormones to control body functions such as growth and development, reproduction, and
metabolism. GH is part of a cascade of hormones that, as the name implies, regulates the physical growth of the body. This cascade begins in a part of the brain called the hypothalamus, which makes hormones that regulate the pituitary. One of these, growth hormone-releasing hormone (GHRH), stimulates the pituitary gland to produce GH. Another hypothalamic hormone, somatostatin, inhibits GH production and release. Secretion of GH by the pituitary into the bloodstream causes the production of another hormone, called insulin-like growth factor 1 (IGF-1), in the liver. IGF-1 is the factor that actually causes the growth of bones and other tissues of the body. IGF-1, in turn, signals the pituitary to reduce GH production. GHRH, somatostatin, GH, and IGF-1 levels in the body are tightly regulated by each other and by sleep, exercise, stress, food intake and blood sugar levels. If the pituitary continues to make GH independent of the normal regulatory mechanisms, the level of IGF-1 continues to rise, leading to bone growth and organ enlargement. The excess GH also causes changes in sugar and lipid metabolism and can cause diabetes.

In over 90 percent of acromegaly patients, the overproduction of Growth Hormone is caused by a benign tumor of the pituitary gland, called an adenoma. These tumors produce excess Growth Hormone and, as they expand, compress surrounding brain tissues, such as the optic nerves. This expansion causes the headaches and visual disturbances that are often symptoms of acromegaly. In addition, compression of the surrounding normal pituitary tissue can alter production of other hormones, leading to changes in menstruation and breast discharge in women and impotence in men.

There is a marked variation in rates of GH production and the aggressiveness of the tumor. Some adenomas grow slowly and symptoms of GH excess are often not noticed for many years. Other adenomas grow rapidly and invade surrounding brain areas or the sinuses, which are located near the pituitary. In general, younger patients tend to have more aggressive tumors. Most pituitary tumors arise spontaneously and are not genetically inherited.

In a few patients, acromegaly is caused not by pituitary tumors but by tumors of the pancreas, lungs, and adrenal glands. These tumors also lead to an excess of Growth Hormone, either because they produce Growth Hormone themselves or, more frequently, because they produce GHRH, the hormone that stimulates the pituitary to make Growth Hormone. In these patients, the excess GHRH can be measured in the blood and establishes that the cause of the acromegaly is not due to a pituitary defect. When these non-pituitary
tumors are surgically removed, Growth Hormone levels fall and the symptoms of acromegaly improve.

If a doctor suspects acromegaly, he or she can measure the GH level in the blood after a patient has fasted overnight to determine if it is elevated. However, a single measurement of an elevated blood Growth Hormone level is not enough to diagnose acromegaly, because Growth Hormone is secreted by the pituitary in spurts and its concentration in the blood can vary widely from minute to minute. At a given moment, a patient with acromegaly may have a normal Growth Hormone level, whereas a Growth Hormone level in a healthy person may be five times higher.

Because of these problems, more accurate information can be obtained when Growth Hormone is measured under conditions in which Growth Hormone secretion is normally suppressed. Physicians often use the oral glucose tolerance test to diagnose acromegaly, because ingestion of 75 g of the sugar glucose lowers blood Growth Hormone levels less than 2 ng/ml in healthy people. In patients with GH overproduction, this reduction does not occur. The glucose tolerance test is the most reliable method of confirming a diagnosis of acromegaly.

Physicians also can measure IGF-1 levels in patients with suspected acromegaly. As mentioned earlier, elevated GH levels increase IGF-1 blood levels. Because IGF-1 levels are much more
stable over the course of the day, they are often a more practical and reliable measure than GH levels. Elevated IGF-1 levels almost always indicate acromegaly. However, a pregnant woman’s IGF-1 levels are two to three times higher than normal. In addition, physicians must be aware that IGF-1 levels decline in aging people and may be abnormally low in patients with poorly controlled diabetes mellitus.

After acromegaly has been diagnosed by measuring Growth Hormone or IGF-1, imaging techniques, such as computed tomography (CT) scans or magnetic resonance imaging (MRI) scans of the pituitary are used to locate the tumor that causes the Growth Hormone overproduction. Both techniques are excellent tools to visualize a tumor without surgery. If scans fail to detect a pituitary tumor, the physician should look for non-pituitary tumors in the chest, abdomen, or pelvis as the cause for excess GH.

The goals of treatment are to reduce Growth Hormone production to normal levels, to relieve the pressure that the growing pituitary tumor exerts on the surrounding brain areas, to preserve normal pituitary function, and to reverse or ameliorate the symptoms of acromegaly. Currently, treatment options include surgical removal of the tumor, drug therapy, and radiation therapy of the pituitary.

**Surgery**

Surgery is a rapid and effective treatment. The surgeon reaches the pituitary through an incision in the nose and, with special tools, removes the tumor tissue in a procedure called transsphenoidal surgery. This procedure promptly relieves the pressure on the surrounding brain regions and leads to a lowering of GH levels. If the surgery is successful, facial appearance and soft tissue swelling improve within a few days. Surgery is most successful in patients with blood GH levels below 40 ng/ml before the operation and with pituitary tumors no larger than 10 mm in diameter. Success depends on the skill and experience of the surgeon. Complications of surgery may include cerebrospinal fluid leaks, meningitis, or damage to the surrounding normal pituitary tissue, requiring lifelong pituitary hormone replacement.

Even when surgery is successful and hormone levels return to normal, patients must be carefully monitored for years for possible recurrence. More commonly, hormone levels may improve, but not return completely to normal. These patients may then require additional treatment, usually with medications.
Conventional Drug Therapy

Two medications currently are used to treat acromegaly. These drugs reduce both GH secretion and tumor size. Medical therapy is sometimes used to shrink large tumors before surgery. Bromocriptine (Parlodel®) in divided doses of about 20 mg daily reduces GH secretion from some pituitary tumors. Side effects include gastrointestinal upset, nausea, vomiting, light-headedness when standing, and nasal congestion.

The second medication used to treat acromegaly is octreotide (Sandostatin®). Octreotide is a synthetic form of a brain hormone, somatostatin, that stops GH production. This drug must be injected under the skin every 8 hours for effective treatment.

Because octreotide inhibits gastrointestinal and pancreatic function, long-term use causes digestive problems such as loose stools, nausea, and gas in one third of patients. In addition, approximately 25 percent of patients develop gallstones. In rare cases, octreotide treatment can cause diabetes.

Radiation Therapy

Radiation therapy has been used both as a primary treatment and combined with surgery or drugs. It is usually reserved for patients who have tumor remaining after surgery. These patients often also receive medication to lower GH levels. Radiation therapy is given in divided doses over four to six weeks. Radiation therapy causes a gradual loss of production of other pituitary hormones with time. Loss of vision and brain injury, which have been reported, are very rare complications of radiation treatments.

No single treatment is effective for all patients. Treatment should be individualized depending on patient characteristics, such as age and tumor size.

Homeopathy treats the person as a whole. It means that homeopathic treatment focuses on the patient as a person, as well as his pathological condition. The homeopathic medicines are selected after a full individualizing examination and case-analysis, which includes the medical history of the patient, physical and mental constitution etc.
Following homeopathic medicines have been found effective in many cases of Acromegaly:

- Thyroidinum
- Chrysarobinum
- Baryta carb
- Carcinocin
- Pitutrinum

WHOLE HEALING with HOMEOPATHY

IMUNE e-training anywhere, anytime
Sarcodes
"Using Healthy tissue in dilute form to treat an organ or Patient"

Use Sarcodes Panels to Treat Organs that are Hyper or Hypo Active
New guidelines for the treatment of acromegaly, a serious growth hormone disorder

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By Jon Danzig, award-winning medical journalist [email Jon Danzig]

Introduction

It’s said a lot can happen in seven days. In seven years, a lot has happened in the understanding of acromegaly, a debilitating condition that causes a patient to have too much growth hormone.

It’s seven years since the American Association of Clinical Endocrinologists last produced guidelines for the diagnosis and treatment of acromegaly. Their 2004 guidelines were just 13 pages long. Their latest, the 2011 guidelines, have grown – to 44 pages.

In 2004, the Association reported acromegaly as an “uncommonly diagnosed” disorder with an annual estimated incidence of 3-4 cases per 1 million people. In the 2011 guidelines, it’s added that newer studies suggest a much higher incidence (although I believe the authors meant prevalence, not incidence). A Belgian study proposing 130 cases per million; a German study concluding 1,034 cases per million. This, reports the Association, suggests that acromegaly “may often be under-diagnosed”.

In 2004, the guidelines made no mention of the psychological damage caused by acromegaly. In contrast, the new 2011 guidelines discuss possible irreversible “psychological alterations” associated with the disease, including depression, mood swings and personality changes. Similarly, the 2004 guidelines made no reference to acromegaly patients ‘quality of life’. By comparison, the new guidelines acknowledge that patients with active acromegaly, and even those in remission, can have significant quality-of-life issues that it recommends should be addressed.

The 2004 guidelines agreed that the gold standard check for acromegaly was an oral glucose tolerance test, with a normal result being a growth hormone level of less than 1 ng/mL. The Association now suggests that be changed – to less than 0.4 ng/mL.

Although the 2011 guide doesn’t report any new drugs since 2004, novel ways of combining the existing drugs are featured, with efficacious and cost benefits.

And yet...
• Both the 2004 and the 2011 guidelines report no change in the delayed diagnosis for acromegaly – it’s still up to ten years.
• Both the 2004 and 2011 guidelines report no change to the proportion of patients found with large tumours – it’s still around 80%. For them, surgical cure rate, even in the best hands, is still 50% or less.
• Many patients with acromegaly still have uncontrolled disease.
• Even those in remission can suffer “quality-of-life” issues years later.
• Most people with acromegaly, of which there may be many more thousands than previously realised, remain undiagnosed.

So, despite the impressive increase in size of the growth-disorder-guidelines over seven years, the improvements seem to have been more subtle and slow.

Below I have presented my own stylised summary of the 2011 guidelines. This I’ve put together mainly for patients, their families and friends, and primary care attendants, all of whom can play a vital role in the earlier detection of this potentially life-threatening condition.

Defining

The definition of acromegaly is clear enough: it’s the excess secretion of growth hormone, causing “multi-system associated morbidities” and “increased mortality.” In almost all cases, the cause is a non-cancerous tumour of the pituitary, a pea-sized gland that’s situated at the front base of the brain and responsible for producing hormones that drive many vital functions of the body.

There’s no doubt that acromegaly is a serious illness, with a long list of debilitating and often disfiguring symptoms. (Hardly “relatively symptom-free” – the claim made by one ‘expert’ doctor that caused me considerable problems when I objected. See The Guardian newspaper report: ’Charity accused of mistreating its members.’)

The Association’s new guide reports that acromegaly can lead to “a myriad of soft tissue and bone overgrowth” problems. Most patients (86%) will present with enlargement of their extremities (hands, feet, nose), excessive perspiration (up to 80%), thyroid nodules (73%), joint pains (75%), facial changes (74%), sleep apnoea (70%), carpal tunnel syndrome (up to 64%), type 2 diabetes mellitus (56%), and headaches (55%).

About half of patients will have a pituitary tumour that also secretes excess prolactin, a hormone primarily responsible for stimulating milk production after childbirth. This hormone, in surplus, contributes to menstrual problems in women and testosterone deficiency and sexual dysfunction in men.

Almost half of patients will present with high blood pressure, impaired glucose tolerance and heart disease. Many acromegaly patients commonly report fatigue and weakness. And new for the 2011 guidelines, the Association reports that acromegaly appears to be associated with psychological changes and alterations in personality. Patients often have depression, apathy and considerable mood changes. One study suggested that acromegaly could cause
cognitive impairment, but the Association advises that further investigations need to be undertaken.

Other factors include an increased risk of cancer, although a possible connection with colon cancer remains unclear. The tumour itself can also cause visual defects. My summary isn’t exhaustive. The list of recognised symptoms associated with acromegaly has grown since the 2004 guidelines. I suspect even more will be discovered in the next seven years.

The biggest risk of all, however, remains the same: untreated acromegaly is associated with a 2 to 2.5 times increased mortality compared to healthy people. Fortunately, this risk is abrogated – or cancelled – once acromegaly is properly controlled. What is the correct definition of ‘properly controlled’ is still being continuously debated and refined.

**Finding**

Finding and diagnosing acromegaly patients as early as possible is still the best way to achieve an outright cure and avoid the long-term disabilities associated with the disease’s progression. Yet the 2011 report states that diagnosis is typically delayed by 7 to 10 years in most patients. By then, the pituitary tumour is usually large and more difficult to completely remove with surgery.

Even in the best hands, surgical cure rates of patients with a large pituitary tumour are only between 40% and 50% (and surgery is usually considerably less successful if the tumour is very large and/or the growth hormone levels are very high).

There’s also a financial incentive to ‘find and fix’ acromegaly patients as soon as possible. Acromegaly is a disease “with a substantial financial economic burden”. In Canada, ongoing treatment for patients who had large tumours cost on average CAN $11,425 per year (about £7,000 per year; 1998 figures, no doubt higher now).

Early diagnosis of acromegaly is rarely achieved, however. That’s because, states the 2011 report, the onset of acromegaly is insidious and often non-specific, with symptoms such as lethargy, headache and increased sweating – signs often mistaken for ageing.

Surprisingly, only a fraction of patients are discovered because of the classic signs of acromegaly, that slowly develop over years (enlarged feet, hands, lips, nose and jaw; often protruding forehead and rough, pronounced facial features). Most often patients themselves are not even aware of the harmful changes happening to them, because they are so gradual.

There’s a need, reports the 2011 guidelines, for primary care physicians and other medical groups to be better educated to watch out for the signs and symptoms of acromegaly so that earlier diagnosis can be achieved.

The dentist, for example, could be suspicious of a patient whose lower jaw is protruding further than the upper jaw – a typical symptom of acromegaly patients. The optician should
be alerted by a visual field defect that might be caused by a pituitary tumour. Rheumatologists often test for disorders that might also lead to a diagnosis of acromegaly.

The new guidelines propose that doctors should consider acromegaly when two or more of the following 12 symptoms are present:

1. New onset diabetes
2. Wide spread joint pains
3. New or difficult to control high blood pressure
4. Heart disease
5. Fatigue
6. Headaches
7. Carpal tunnel syndrome (pain in hand and fingers)
8. Sleep apnoea (snoring with breathing difficulties)
9. Excessive sweating
10. Loss of vision
11. Colon polyps
12. Increasing difficulties in closing the jaw

Once suspected, acromegaly is easy to diagnose. A simple and quick blood test to check for abnormally high serum IGF-1 levels is reported in the new guidelines to be “excellent for diagnosis, monitoring and especially screening” for acromegaly. It’s vital, however, that the IGF-1 test is age and sex matched to normal subjects.

IGF-1 (insulin-like growth factor 1) is produced by the liver in response to growth hormone secreted by the pituitary gland. IGF-1 then circulates in the body and stimulates cell growth. In acromegaly, excessive growth hormone generated by the pituitary tumour leads to the liver over-producing IGF-1.

A random one-off measurement of growth hormone itself is not helpful as it’s too variable. Measuring growth hormone levels to diagnose acromegaly requires a more specialist procedure, called an Oral Glucose Tolerance Test, and is still considered the ‘gold standard test’ for acromegaly.

After drinking the glucose following over-night fasting, blood is taken every half-an-hour for two hours. In patients without acromegaly, serum growth hormone levels will fall to 1 ng/mL or less (although the Association is now considering a new cut-off point of 0.4 ng/mL). In patients with acromegaly, the glucose fails to suppress growth hormone levels and they remain above 1 ng/mL.

However, the new guidelines also state that this ‘gold standard test’ can be skipped altogether if IGF-1 is elevated and there are signs and symptoms of acromegaly. That certainly makes diagnosis simpler, quicker and cheaper.

Following confirmation of acromegaly, an MRI-scan should be ordered to check the size and exact location of the tumour attached to the pituitary gland.
Patients diagnosed with acromegaly need to be regularly re-tested for the rest of their lives. The pituitary tumour has been known to recur, sometimes many years later.

‘Fixing’

Most patients with acromegaly are never fully ‘fixed’ or entirely free of the disease and its long term damage. Some may disagree, but I believe it can be postulated. After all, it’s rare for chronic internal medical diseases to be 100% ‘fixed’; more usually it’s hoped that they can be improved or put into remission or controlled.

As Professor Laurence Katzenelson, Chair of the committee that drew up the new guidelines, wrote to me, “Many patients with acromegaly are left with residual difficulties.”

Subsequently, some acromegaly patients have also written to me saying it’s inappropriate for doctors to use the term ‘biochemical cure’ - meaning their blood test results no longer show signs of acromegaly – when they continue to suffer debilitating symptoms.

Out of 100 patients discovered with acromegaly, about 20 will have a small pituitary tumour and about 80 a large one. Depending on which surgeon operates, surgery alone will result in a ‘biochemical cure’ in only about half, more or less, of the 100 patients. Medication or radiation will achieve ‘biochemical cure’ in many, but not all, of the rest.

Although ‘biochemical cure’ is now much more achievable than previously, and can result in considerable improvements for patients, it’s agreed that it doesn’t necessarily equate to satisfactory elimination of the disease and its consequences in many patients.

This is discussed in the new guidelines, which report that many patients who have been in ‘biochemical remission’ for years continue to suffer quality-of-life issues, especially relating to musculoskeletal complications resulting in significant joint pains. Adverse changes to appearance caused by the disease can also cause profound difficulties. Unlike soft tissue changes in acromegaly, bone enlargement caused by the disease is permanent.

Significant psychological issues can also persist despite the biochemistry apparently being in normal range. The new guidelines raise the possibility that acromegaly can cause irreversible changes to mood and behaviour. The authors recommend that all acromegaly patients, whether with active disease or in remission, have attention to quality-of-life issues.

New theory on why ‘cured’ acromegaly patients still suffer

New research conducted jointly in the Netherlands, Denmark and the USA proposes a novel theory as to why so many acromegaly patients apparently under ‘biochemical control’ still have ongoing symptoms. If the theory transpires to be true, it could radically change the medical treatment for all acromegaly patients who fail to be cured by surgery alone.

The international team of doctors put forward a new paradigm – or model – for ‘systematic
acromegaly’ that affects patients who are treated with an acromegaly medicine that’s widely used called ‘long acting somatostatin analogues’. According to the new theory, these patients could still have acromegaly in many parts of the body, other than the liver, because of the way this particular medicine works. Consequently, this ‘remaining acromegaly’ is hidden as it doesn’t show up in IGF-1 testing. It means, say the researchers, that these patients continue to suffer the damaging effects of excess growth hormone, even though their biochemistry indicates that they are in ‘normal range.’

The research team, led by Dr Sebastian Neggers of Erasmus University in the Netherlands, has named this ‘peripheral’ or ‘extra-hepatic acromegaly’ (i.e. acromegaly outside the liver). He claims that this ‘peripheral’ form of acromegaly has, “a significant negative impact on the quality of life of many patients who were previously considered to be biochemically cured.”

The authors of the concept claim that, if their hypothesis is correct, it could mean that the medical treatment for acromegaly patients will require “a significant update”. In particular, their theory challenges whether IGF-1 is a reliable marker of disease activity in acromegaly patients. There is a need, the researchers believe, for newer measures of acromegaly to be developed, “either genomic, metabolomic, proteomic, or others” which integrate both ‘hepatic’ and ‘peripheral’ or ‘non-hepatic’ forms of the disease. Such new markers of the disease could also help to optimise treatment on a more individual basis, especially with regard to ‘quality of life’ issues.

Dr Neggers and his co-workers also conducted additional research indicating that if treatment with ‘long-acting somatostatin-analogues’ is combined with another drug, called pegvisomant, this can successfully resolve the “remaining, peripheral form of acromegaly.” (See section on ‘medication’ below).

However, the new theory does not appear in the new American guidelines for acromegaly, and Dr Neggers admits that, “around the world there are some non-believers.” One leading professor of endocrinology told me he remained “agnostic” about the hypothesis. The American Association of Clinical Endocrinologists advised me that they only included data in their latest guidelines that had achieved “general consensus”, but if the new theory proved to be accurate, it could appear in the Association’s next guidelines.

Dr Neggers and his team are now calling for other scientists, clinicians and pharmaceutical companies to conduct further studies to test whether their theory is correct.

New research in Germany indicates that acromegaly might affect millions more people than previously thought. In the past, it was estimated that the world-wide prevalence of acromegaly was only about half-a-million. But if the German study quoted in the new American guidelines is correct, the worldwide prevalence of acromegaly might be as high as 7 million.

Also of interest, acromegaly appears to affect both sexes and all races in equal proportions.
There’s no known way to avoid getting acromegaly, since so far we are not even sure what causes it in the first place. The best chance for patients is to be discovered in the very early stages of the disease, when the tumour is small and there’s the highest chance of a real cure through surgery alone. For the rest, the majority, the doctor’s toolbox is limited: to surgery, medication or radiation.

The new guidelines report five goals in the treatment of acromegaly:

1. **To bring the chemical measures of acromegaly to normal**
2. **To control the size of the tumour**
3. **To reduce the signs and symptoms of the disease**
4. **To prevent or improve medical conditions related to the disease**
5. **To prevent premature mortality**

The following methods can be used alone or in combination to try and achieve these goals:

**Surgery** – it remains the most effective option to achieve rapid and complete cure for all patients who can have surgery. Even if cure doesn’t occur, the reduction in tumour mass can result in considerable recovery and also improve the response of subsequent medication. These days most pituitary surgery is endoscopic transsphenoidal through the nose, which is minimally invasive. The most experienced surgeons – those performing at least 50 transsphenoidal procedures a year – have the best outcomes with low mortality and morbidity.

**Medication** – for those who cannot have surgery, and for those for whom surgery did not result in a ‘cure’. There is also some evidence, according to the new guidelines, that medication taken prior to surgery might result in a better post-operative outcome.

There are three classes of medical therapy: dopamine agonist, somatostatin analogues and a growth hormone receptor antagonist:

* **Dopamine agonist** – (tablets) usually cabergoline. Sometimes used as a first-line medical therapy because it’s taken orally and inexpensive. However, it’s only effective in a minority of patients, and usually only considered for patients with ‘mild’ acromegaly.

* **Somatostatin analogues** – (injections) usually octreotide LAR or lanreotide autogel. The new guidelines report that with this medical therapy, about 55% of patients achieve normal growth hormone and IGF-1 levels. The medication can also result in modest or significant reduction in tumour size in some patients. There have been mixed studies of whether treating with somatostatin analogues prior to surgery improves the results, and the guidelines authors state that further study is needed on this.

* **Growth hormone receptor antagonist** – (injection) known as pegvisomant, this is the most efficacious but unfortunately the most expensive of the drugs available. It works in a completely different way to the other medications. In patients treated with pegvisomant for a year, the new guidelines report that IGF-1 levels were normalised in 97% of patients, confirming it to be the most effective drug currently available. Patients also reported
improvements in their signs and symptoms of acromegaly. To be cost effective, however, the guidelines report that the price of pegvisomant needs to be reduced by one third.

*Combination therapy:* the new guidelines describe some success with combining the medications when one alone didn’t work sufficiently. For those who only partially responded to somatostatin analogue treatment, the addition of cabergoline helped 42% of them to achieve normal IGF-1 levels. The guidelines also reported that the combination of somatostatin analogues with pegvisomant often appeared to be more effective in normalising IGF-1 levels than either drug used on its own. In one study (featured in the guidelines) the addition of weekly pegvisomant to somatostatin analogue treatment resulted in IGF-1 levels becoming normal in 95% of patients. Another study (not in the new guidelines) demonstrated that this combination resulted in significant improvements to patients’ quality-of-life. Since this combined therapy usually involves lower doses of both drugs, it’s been argued that this can result in cost savings over the use of one drug alone.

Radiotherapy – usually used as a last resort, when surgery and medication haven’t worked. However, the guidelines report that with the availability of effective medication, the role of radiotherapy has subsequently diminished. It is, though, still used to reduce the need for (expensive) lifelong medication and with a goal of ‘disease cure’.

One downside is that it takes a long time for the radiation to work – from several years to over a decade. The guidelines state that techniques for radiotherapy have improved in recent years, with better targeting to the tumour and subsequently less radiation exposure to surrounding tissue.

The results of the more old fashioned ‘conventional radiotherapy’ have recently been reassessed to take account of the stricter criteria of ‘biochemical cure’ for acromegaly. Whereas previously it was thought that conventional radiotherapy resulted in a remission rate of over 80%, that’s now been revised considerably downwards to just 10 to 60%. Furthermore, conventional radiotherapy can take about ten years to be effective – even longer in patients with initially higher IGF-1 and growth hormone levels.

The more modern radiotherapy is called stereotactic radiosurgery, of which there are several versions, but the new guidelines concentrate on reporting ‘gamma knife’ radiosurgery, because it’s the one most referred to in the medical literature for acromegaly. With this newer, more precise form of radiation, remission can sometimes be achieved in two years, rather than ten for the conventional form of radiotherapy. Earlier studies reported remission rates of 90% for gamma knife radiosurgery, but again, with the stricter definitions of cure, this has now been revised to just 17 to 50% remission rates during two to five years of follow-up. The guidelines state that further studies are needed over a longer time-frame.

There are, however, significant drawbacks to radiotherapy, report the 2011 guidelines. One is the development of hypopituitarism – or failure of the pituitary gland – in more than half of patients after five to ten years. Hypopituitarism has been linked to increased mortality. The authors also point out that similar prevalence of hypopituitarism has been found with
the more modern stereotactic radiosurgery, although most studies so far have only reported on less than six-years average follow up for gamma knife radiosurgery.

In a recent talk to doctors (not mentioned in the new guidelines) by Professor John Wass, one of the world’s foremost experts in acromegaly, he said, “In the olden days people gave radiotherapy, really without thinking, to patients with pituitary tumours.” He added that the side effects of conventional radiotherapy include hypopituitarism; some patients develop tumours in the field of the radiotherapy; mental agility was also thought to be interfered with; and radiotherapy may also cause acromegaly patients, ironically, to become growth hormone deficient.

Regarding the newer form of radiotherapy, Professor Wass commented, “I don’t think that the data that have been provided for gamma knife radiotherapy are particularly good.”

The new guide also raises similar concerns about radiotherapy, and points out that acromegaly patients who received conventional radiotherapy were at significant greater risks of “all-cause mortality” than those who did not receive the treatment. The 2011 guidelines advise that long-term data of such risks for patients undergoing the more modern gamma knife radiosurgery are not yet available, since most of the data relates to the older forms of radiation delivery. The newer systems may potentially yield better results, but we will not know until longer-term data become available.

**My conclusions**

The goal of the new guidelines is to, “update clinicians regarding all aspects in the current management of acromegaly...” Patients also need updating, and I’ve tried my best to summarise the latest recommendations primarily for the benefit of patients, although hopefully physicians will find this summary helpful too.

Given a choice, this is actually the best time ever to have acromegaly. You only have to go back to the middle of the last century – within the lifetimes of many of us – when the prospects for acromegaly patients were much grimmer, with fewer therapeutic options and more premature deaths. We’ve come a long way, but not far enough. Most acromegaly patients remain undiagnosed, and most of those who have been diagnosed had to wait an awful long time, and still suffer long term symptoms.
Acromegaly and Yoga

Yoga helps to control Pituitary Gland disorders. If you are suffering from the chronic disease Acromegaly (Over secretion of growth hormones). You can get rid of this disease through Yoga practices.

People suffering from acromegaly, a chronic metabolic disorder will experience rapid increase in growth hormone and gradually they will notice enlargement of their body tissues. Other symptoms that point to a dysfunction of the pituitary gland include decreased muscle strength, body odor, carpal tunnel syndrome, thickened lips and tongue, widening space between your teeth and so on.

The pituitary gland, a pea shaped gland, located at the base of your brain houses all the hormones secreted in your body. It also controls and regulates the function of your growth hormones, which play an important part in your physical growth. A benign pituitary tumor can cause acromegaly as it presses on delicate brain tissue. While a malignant tumor is fatally dangerous and needs to be surgically removed, a doctor may suggest that individuals recovering from pituitary tumor try Yoga as part of therapy along with hormone regulating medicines.

Treatment and exercise needs to focus on lowering the production of growth hormones and negating the effects of the tumor on the surrounding tissues. Exercises beneficial for the pituitary gland include Yoga poses such as Halasana or the Plow Pose, Janu Shirsasana or Head to Knee pose and Shirsasana or Head Stand Pose.
To improve the functioning of the pituitary gland, Yoga poses, which emphasize extending your neck and spine, are beneficial. Although difficult, the Head Stand is an excellent pose for both the pituitary and pineal gland. Yoga can help regulate the function of pineal glands, which include sleep/wake patterns, and seasonal functions allowing you periods of alertness and restfulness.

To improve the functioning of the adrenal gland, try Yoga poses like Ardha Matsyandrasana or Half Spinal twist and Marichyasana or Sage twist. These and other torso twisting poses will benefit the adrenal gland, which regulates the functions of your sex hormones. The poses must be done slowly without any sudden twists or jerking motions.
To regulate the function of the thymus gland, Yoga breath techniques like Kapalbhatti or Forced breathing can be practiced everyday. This is especially beneficial for people suffering from seasonal allergies as it helps to clear your nasal pathway from mucous. Since its basic function is to build immunity in our body, the thymus gland benefits from Yoga poses, that support and strengthen the immune system.

If you need to improve the functioning of the pituitary gland, practice Yoga poses and Pranayam at least for 30 minutes daily. It is a gentle workout routine with no side effects. Ensure that if you suffering from any disorder or dysfunction of the pituitary glands, Yoga should be practiced under strict medical advice and with the help of a qualified Yoga instructor. For chronic conditions like
acromegaly or other complaints of the pituitary gland, Yoga alone may not suffice. Consult your doctor immediately if any symptoms disrupt your daily life.

**Alternate Channel Breathing**

*Nadi Shodhana*

While your condition can range from uncomfortable to painful, Yoga can help with relieving stress, calming your nerves and regulating your body functions.

**7 Reasons Pranayama Makes You Younger**

Pranayama is the practice of cultivating life-force or pranic energy through breathing. It is practiced as a means of developing greater lung-capacity, but also to eventually control the breath in a way that is incredibly beneficial for the body and mind.

Here are seven reasons that pranayama makes you younger:
Look Younger

While pranayama can’t do it alone, when added to meditation, and asana, it makes your complexion gorgeous. The added oxygenation of your blood makes your whole body more vital, so it shows in your skin. You look more radiant, and experience fewer breakouts and a reduction in other signs of aging like wrinkles and dark circles under the eyes. The additional prana you develop in your will also inspire you to be more active. Once you add a healthy diet, you will look years younger than your friends who don’t practice pranayama and yoga.

Live longer

Although western scientists aren’t exactly sure how it happens, pranayama tends to lengthen your life span. Maharishi Raghuvacharya lived to the age of 115. This is not unusual for many yogis. Pranayama practices helps to maintain circulatory health, and often, since you are filling the body with energy that doesn’t involve caloric intake, you live longer as well. There is scientific evidence that a restricted caloric intake can lengthen life expectancy.

Be Smarter

Pranayama practice makes you smarter, so you are less likely to suffer from age-related dementia. The practice of yogic breathing increases neuronal activity and health. The more you breath like a yogi, the more vital your brain becomes.

Vital Immunity System

Another common sign of aging is a reduced immune function. Pranayama practice reverses this otherwise ‘normal’ degeneration. Pranayama makes the immune system more vital and increases the lymphocyte health in the body, which is the primary way our bodies fight foreign invaders like viruses and bacteria.

Better Sleep
People who don’t sleep well age faster. Pranayama practice has been proven to increase restful sleep.

**Improving Sight & Hearing**

Pranayama practice can make your eye-sight and hearing better. Often aging populations experience a reduction in hearing and sight. This is often due to poor blood flow to these important organs. Pranayama helps to increase blood flow to the eyes and ears. If you add asana like shirshasana (headstand) or other inversions, this effect multiples significantly.

**Neutralizing Radicals**

Pranayama helps the body eliminate the destructive action of free radicals. While free radicals are the normal effects of metabolism, pranayama can help neutralize the
DEFICIENT GROWTH HORMONE

Enhancing Growth Hormone Naturally

During our youth, abundant levels of growth hormone (GH) promote an energetic physiology essential for healthy metabolism and an optimal ratio of lean muscle tissue to body fat.

By the time we reach middle age, however, levels of essential hormones such as testosterone and DHEA decline, while age-associated decreases in muscle mass and increases in body fat become noticeable.

Furthermore, research shows that in aging men, the amplitude of pulsatile GH release (the magnitude of the GH pulse) declines by 50% every seven years after 18-25 years of age. Exogenous subcutaneous injection of human recombinant growth hormone is expensive and still controversial. Fortunately, studies have shown that there are strategies that may naturally boost the endogenous production of growth hormone and thus provide a viable alternative to expensive injections. In particular, exciting research suggests that the growth hormone-blocker somatostatin can itself be inhibited with a nutrient called CDP-choline, thus slowing the rate at which growth hormone declines.

Naturally supporting the body’s own endogenous growth hormone production using targeted lifestyle and nutritional strategies may provide a safe method of harnessing the vigor and vitality associated with youthful growth hormone levels.

Growth Hormone Basics

Growth hormone (GH), also known as somatotropin, is a peptide hormone produced by the anterior lobe of the pituitary gland. Growth hormone secretion occurs in a pulsatile fashion following a circadian (daily) rhythm, which is controlled by a central area of the brain known as the hypothalamus. The hypothalamus regulates serum GH levels through the release of two functionally opposing hormones: growth hormone-releasing hormone stimulates GH release, while somatotropin release-inhibiting hormone reduces it.
Endogenous (made within the body) GH exerts its actions by binding directly to specific receptors on target tissues including muscle, connective tissue (tendons, ligaments, bone, and fat), as well as every major organ. Growth hormone also works indirectly by stimulating liver cells to produce and secrete polypeptide molecules known as somatomedins, the best studied of which is insulin-like growth factor-1 (IGF-1). Like GH, IGF-1 boasts receptors throughout the body and serves many functions. Together, GH and IGF-1 play influential roles in virtually every system—from muscle, bone, and connective tissue growth and repair, to the selective regulation of various aspects of metabolism, as well as helping maintain normal brain function and cardiac health.

However, GH secretion falls precipitously with advancing age. Furthermore, research shows that in aging men, the amplitude of pulsatile GH release (the magnitude of the GH pulse) declines by 50% every seven years after 18-25 years of age.\(^1\)

This decline is also mirrored by diminishing IGF-1 levels. The decrease in the secretory activity of the GH/IGF-1 axis, commonly referred to as somatopause, correlates with a number of undesirable symptoms generally associated with aging. Most notably, diminishing GH/IGF-1 has been shown to reflect disordered sleeping patterns, bone frailty, increases in central adiposity (fat accumulation around the middle of the body including the abdomen), as well as decreases in cognition and muscle mass, strength, and conditioning.\(^2\)-\(^9\)

Is Synthetic GH Replacement Therapy Beneficial? Answer NO
Since the decline of GH correlates with the onset of aging-related symptoms, scientists have investigated whether synthetic GH replacement may prove beneficial.

Some of the most compelling evidence that somatopause may respond favorably to synthetic GH replacement therapy comes from investigations involving patients suffering from total or near total absence of GH secretion as a result of pituitary disease. Without treatment, adults suffering from pituitary disease are both physically and psychologically less healthy than their age-matched peers, demonstrating significantly reduced muscle mass, bone density, exercise performance, thyroid function, and collagen production, with a concurrent escalation of central fat mass (especially fat accumulation in the abdominal organs) and insulin resistance, as well as an increased risk for cerebrovascular accidents (strokes) and cardiac events. Psychologically, they tend to experience more emotional lability (abrupt changes in mood in response to everyday events), depression, and social isolation, and their average life expectancy is measurably reduced.

In the late 1980s and early 1990s, GH replacement studies in adults with poor pituitary function were designed with the goal of restoring normal GH levels. However, the doses used in these chronically GH-deficient individuals produced IGF-1 concentrations that greatly exceeded the expected range, resulting in unacceptably high rates of adverse reactions. In subsequent work, with GH doses adjusted to produce age-appropriate IGF-1 concentrations, negative side effects were largely eliminated or reduced to tolerable levels. Study subjects demonstrated significant and sustained improvements in body composition, physical performance, bone density, and psychological well-being, as well as substantial reductions in biomarkers for cardiac disease.

In light of these results, researchers felt cautiously optimistic that men and women with partial GH deficiency secondary to advancing age might also reap the benefits of GH replacement therapy. However, following a landmark study by Rudman and colleagues in 1990, which provided the first evidence that GH supplementation in the elderly could diminish—and potentially reverse—some of the physical symptoms associated with somatopause, exogenous GH therapy has been controversial and associated with high costs.

Fortunately, scientists are discovering that the benefits of youthful GH levels can also be harnessed safely by naturally increasing the body’s own hormone levels with the right nutrients and lifestyle practices.

### Nutritional Strategies for Optimizing GH

Nutritional strategies can offer targeted support for individuals seeking to enhance their endogenous production of GH. Such nutrients may work either by directly enhancing GH release from the pituitary gland or by enhancing the efficacy of sleep or exercise, the two activities that best support GH secretion.
CDP-Choline Boosts GH, Supports Brain Health

A growing body of research suggests that the compound cytidine-5’-diphosphate choline (CDP-choline) may boost GH secretion while conferring an array of brain health benefits for aging adults.

As adults grow older, GH secretion from the anterior pituitary gland declines precipitously. Exciting scientific research suggests that decreased GH release results in part from increasing levels of somatostatin with aging. Somatostatin produced by the hypothalamus inhibits the release of GH from the anterior pituitary.

This innovative idea has led researchers to search for agents that inhibit somatostatin and thus potentially increase the release of GH. Experimental research shows that treatment with cholinergic agonists increases GH release by inhibiting somatostatin release from the hypothalamus.38

These compelling findings were soon supported by a human study. This investigation showed that administration of CDP-choline to healthy elderly adults resulted in a dramatic four-fold increase in serum GH levels, compared with baseline values.39 These findings build upon evidence from an earlier study showing that CDP-choline administration in healthy men increased serum GH levels.40

In addition to its effects on GH release, CDP-choline acts through other mechanisms to promote brain cell integrity and health. CDP choline acts as an intermediate in the synthesis of neuronal membranes, promoting healthy brain cell membrane structure and function. CDP-choline counteracts the deposition of amyloid-beta, a pathological protein found in the brains of patients with Alzheimer’s disease. Human research suggests that CDP-choline supports release of the essential neurotransmitter norepinephrine, while animal studies show that CDP-choline increases brain levels of key neurotransmitters including dopamine and serotonin.41

In clinical trials, CDP-choline has shown promise in improving age-associated memory impairment, boosting cognitive performance in the early stages of Alzheimer’s disease, and supporting recovery from both ischemic and hemorrhagic stroke.41

These findings combine to suggest a powerful role for CDP-choline in supporting healthy GH levels and in optimizing brain health with aging.

Protein, Amino Acids Enhance GH Release, Lean Body Mass

Protein (especially protein derived from animal sources) provides important essential and
conditionally essential amino acids known to assist endogenous GH secretion. An added bonus: these same essential amino acids are vital for supporting muscle growth and recovery in active men and women.

The most abundant amino acid in the body is glutamine. Consuming even a relatively small amount of glutamine (2,000 mg) has been shown to increase plasma GH levels. Glutamine has also been shown to help preserve muscle mass in individuals vulnerable to losing lean body mass due to inactivity following surgery. This suggests that glutamine may provide important benefits in maintaining lean body tissue.

Like glutamine, oral intake of the amino acid arginine increases the release of GH at rest. The combination of arginine intake with exercise produces even greater increases in GH levels. In addition to its anabolic (tissue-building) effects, ornithine alpha-ketoglutarate has also been reported to increase GH secretion.

Compelling evidence demonstrates that the combination of arginine and ornithine augments the results of resistance training by helping to increase lean body mass and strength. The investigation also indicated that oral doses as relatively small as 1 gram of ornithine and arginine were effective in enhancing strength and lean tissue mass.

**WHAT YOU NEED TO KNOW: ENHANCING GROWTH HORMONE NATURALLY**

- Growth hormone (GH) is a peptide hormone that is intimately involved in tissue growth and repair. Together with insulin-like growth factor 1 (IGF-1), GH helps regulate metabolism and maintain normal brain and cardiac function.

- Secretion of GH falls dramatically with aging, correlating with age-related symptoms such as disordered sleep patterns, fragile bones, cognitive decline, and decreased muscle mass and strength.

- Studies examining exogenous GH therapy in elderly adults with declining GH levels have yielded mixed results.

- Given the mixed results and the high cost of subcutaneous injection of human recombinant GH therapy, a more natural approach to maintaining youthful health and vigor is to employ lifestyle choices that optimize the endogenous production of GH.

- Safe methods for enhancing endogenous GH production include: losing excess body fat, particularly abdominal fat; avoiding high-glycemic load carbohydrates; optimizing sleep habits; eating a high-protein, low-carbohydrate snack before bedtime; and exercising regularly to your lactate threshold. Targeted nutrients including CDP-choline, arginine, ornithine, glycine, glutamine, and niacin (vitamin B3) can help support endogenous GH secretion, assist muscle growth and recovery from exercise, and promote healthy sleep.
Enhancing Growth Hormone Naturally

Glycine Supports Healthy Sleep, GH Release

Since GH secretion occurs primarily at night, ensuring good sleeping habits is essential for individuals seeking to optimize their natural levels of GH. Unfortunately, one-third of adults report at least occasional bouts of insomnia, and about one-third of them suffer from sleeplessness or disturbed sleep on a more chronic basis, to the point that it regularly impairs daytime functioning. For the millions of sleepless among us, there may be good news—in the form of an inexpensive, naturally occurring amino acid known as glycine.

Within the central nervous system, glycine functions as an inhibitory neurotransmitter, playing a well-documented and critical role in initiating normal patterns of REM sleep. Now, a new study of chronic insomniacs demonstrates that glycine administered orally just prior to bedtime significantly improves sleep quality, shortening the latency between sleep onset and initiation of slow-wave (deep) sleep as measured by polysomnography. Volunteers also reported less daytime sleepiness, a subjective finding that was objectively corroborated by improved performance on cognitive tasks testing memory recognition.

DECLINING GH LEVELS AND POOR HEALTH

There are a number of lifestyle factors that lead to decreased GH and IGF-1 secretion. For example, multiple studies indicate that central adiposity (the accumulation of central body fat) accurately predicts GH decline. In addition, it is well established that poor nutritional status, inadequate sleep, and lack of physical fitness can all contribute to decreases in circulating GH and IGF-1, regardless of age.

Individually or in combination, poor nutritional status, inadequate sleep, and lack of physical fitness negatively impact body composition, bone strength, athletic conditioning, and cognition—indeed, independent of their effects on serum GH levels.

It seems clear that an unhealthy lifestyle contributes to somatopause both directly, by causing profound reductions in GH secretion, as well as indirectly, by promoting the physical and psychological symptoms of accelerated aging.

These findings build on previous work showing that a supplement cocktail containing glycine, glutamine, and niacin (vitamin B3) significantly increases endogenous GH secretion in healthy, middle-aged men and women. Individual test subjects in the study who demonstrated a concomitant increase in IGF-1 also exhibited improved memory and vigor.
Lifestyle Techniques to Naturally Boost Endogenous GH Secretion

Healthy lifestyle practices are an essential component of a program to enhance endogenous GH production. The most important techniques for optimizing GH levels include:

1. **Deflate the spare tire.** If you happen to suffer from fat stores concentrated centrally around the organs of the abdominal region, GH secretion will be even more impaired. Fortunately, research indicates that declining GH due to body fat gain is partially reversible with weight loss. Unfortunately, visceral adiposity is often an indicator of both insulin and leptin resistance and, as a result, can be very difficult to shed permanently. Fortunately recent work has led to the discovery of effective, natural methods for combating leptin resistance. For more information, see: “Deflating your spare tire for a longer, leaner life…Understanding the risks of leptin resistance” *Life Extension*, February 2009, and “Vindication” (How correcting a testosterone deficit can reduce abdominal adiposity), *Life Extension*, December 2008.

2. **Avoid high-glycemic-load carbohydrates.** Insulin is a powerful, direct inhibitor of GH secretion. To prevent the unhealthy surges of insulin or “insulin spikes” that decrease endogenous GH levels and increase your risk for type 2 diabetes, avoid highly processed carbohydrates like refined white bread and sugary cereal, as well as high-glycemic-load foods such as white rice, potato chips, cookies, soda, and commercially processed fruit juices (high in fructose and devoid of fiber). Instead, emphasize nutrient and fiber-rich whole fruits, vegetables, nuts, and legumes (beans).

3. **Insist on a good night’s sleep.** The majority of GH secretion occurs at night during slow-wave (deep) sleep. Along with high-intensity exercise, another natural stimulus of endogenous GH secretion is sleep itself. It is well documented that inadequate sleep, irregular sleeping patterns, and poor quality sleep can substantially inhibit GH secretion. To optimize sleep, maintain good sleep hygiene habits: keep to a regular bedtime and wake up time; do not consume alcohol or caffeine 4-6 hours before bedtime; and keep excess light and noise out of the bedroom.

4. **Plan your last meal of the day carefully.** Your last meal of the day is the most important for maintaining a robust GH/IGF-1 axis. A high-protein, low-carbohydrate snack before bedtime serves a dual purpose. First, it helps minimize insulin release and allows for maximum endogenous GH secretion. Second, important essential and conditionally essential amino acids found in protein assist endogenous GH secretion.

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**Harnessing Your Lactate Threshold**

At some point, depending on exercise duration and intensity, the rate of lactic acid formation in your muscles becomes greater than the rate of dispersion. This is known as the lactate threshold, and can usually be elicited at activity levels that demand between 80% and 90% of a trained athlete’s maximum heart rate.

One of the easiest ways to surpass your lactate threshold is through resistance training, but you
do not have to lift heavy weights to take advantage of increased endogenous GH. Several studies have shown that circuit training, which utilizes relatively light resistances, can be just as effective at driving GH release as a more strenuous workout.\textsuperscript{64,69,70} Intuitively, circuit training might seem less intense than power training. However, circuit training calls for an increased number of repetitions per set and the rest periods between consecutive sets are often considerably shorter, typically on the order of zero to 30 seconds, versus a minute or two for heavier lifting.

If longer-duration, lower-intensity “cardio”-type activities such as running, cycling, or swimming are more your cup of tea, you may need to enhance your workouts to generate optimal GH secretion during exercise. Punctuating your usual, lower-intensity cardio routine with brief, all-out sprints every three to five minutes will rapidly push you to your lactate threshold.

5. **Stay active!** Exercise is a significant, natural optimizer of GH secretion.\textsuperscript{62} The type of exercise you do, as well the intensity and duration of your workouts, all play an important role in determining to what degree your training regimen contributes to GH secretion. A number of studies have suggested that the intensity necessary to trigger exercise-induced GH release corresponds to the lactate threshold—the exercise intensity at which lactic acid accumulates in the blood.\textsuperscript{63} Exercise training above the lactate threshold appears to amplify the pulsatile release of endogenous GH at rest, increasing total secretion for at least 24 hours.\textsuperscript{64}

**Conclusion**

The plentiful growth hormone levels of youth are associated with strength, good health, and vitality. However, given the high cost and mixed study results associated with recombinant GH injections, optimizing lifestyle choices to enhance endogenous GH production may represent the most intelligent way to benefit from this youthful hormone. Through weight management, exercise, healthy sleep habits, minimizing intake of high-glycemic-load carbohydrates, and consuming targeted nutrients such as CDP-choline, niacin, glycine, glutamine, arginine, and ornithine, you may safely and cost-effectively capture the many benefits of naturally high GH levels.

If you have any questions on the scientific content of this article, please call a Life Extension Health Advisor at 1-800-226-2370.

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Super ANTI-AGING
WHAT TO EAT

1. Eat Natural Foods with little preservatives
2. Eat more fruits, seed products, leafy greens, salads
3. Let Fruit be your Sweetener,
4. Drink ONLY 100% Fruit juice diluted with water
5. Boil foods in WATER, NOT OIL
6. Use fresh, cold processed UNHEATED olive oil, sunflower oil, safflower oil etc.
7. Less Cooking, Use stir fry well washed veggies
8. Foods made with Love and Nature is Blessed Nutrition, Foods made and eaten with Hate and Anger are poisons.
9. Celebrate each meal with friends, family or at least your joyous self. Celebrate
10. Listen to your inner self what to eat, and when to stop, do not eat with your eyes
<table>
<thead>
<tr>
<th>No.</th>
<th>Hormone</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Bradykinin</td>
<td>Bioactive peptide, plasma kallikrein that stimulates inflammation</td>
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<tr>
<td>2</td>
<td>Endorphin</td>
<td>Opioid peptide for pain control</td>
</tr>
<tr>
<td>3</td>
<td>Cholecystokinin</td>
<td>Stomach hormone that stimulates the gallbladder</td>
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<tr>
<td>4</td>
<td>Oxytocin</td>
<td>Opioid peptide, stimulates lactation and emotional bonding</td>
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<tr>
<td>5</td>
<td>Leucine enkephalin</td>
<td>Brain hormone for pain and emotional control</td>
</tr>
<tr>
<td>6</td>
<td>Morphine</td>
<td>Opioid peptide</td>
</tr>
<tr>
<td>7</td>
<td>Noradrenalin</td>
<td>Adrenaline hormone for action &amp; activity, cortisol</td>
</tr>
<tr>
<td>8</td>
<td>Serotonin</td>
<td>Happiness hormone that stimulates repair and recovery</td>
</tr>
<tr>
<td>9</td>
<td>Glutamine (Glutamic Acid)</td>
<td>Amino acid balances cerebral neurotransmitters</td>
</tr>
<tr>
<td>10</td>
<td>Dopamine</td>
<td>Hormone for starting action &amp; regulating many functions</td>
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<tr>
<td>11</td>
<td>Acetylcholine</td>
<td>Cholinergic neuronal transmitter for stimulating neurotransmission</td>
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<td>12</td>
<td>Substance P</td>
<td>Responsible for communicating pain sensations</td>
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<td>13</td>
<td>Aspartate</td>
<td>Food additive used in aspartame, can cause headaches</td>
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<tr>
<td>14</td>
<td>GABA (Gamma Amino Butyric Acid)</td>
<td>Brain factor that controls electric activity</td>
</tr>
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<td>15</td>
<td>Histamine</td>
<td>Amino acid used for inflammation and allergy control</td>
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<td>16</td>
<td>Vasopressin</td>
<td>Opioid peptide, antidiuretic, blood pressure regulator</td>
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<td>Glycine</td>
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<td>18</td>
<td>Carnosine</td>
<td>Amino acid used for energy control and heart stability</td>
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<td>19</td>
<td>Taurine</td>
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<td>20</td>
<td>All Transmitters Opioid Type</td>
<td>Opioid receptors in the brain</td>
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<tr>
<td>21</td>
<td>Insulin</td>
<td>Allows glucose to penetrate cell membranes for energy</td>
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<td>22</td>
<td>Somatostatin</td>
<td>Opioid peptide, part of growth hormone, stimulates growth</td>
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<td>23</td>
<td>Thyromosin</td>
<td>Stimulates white blood cell for immune system defense</td>
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<td>24</td>
<td>DL-Thyroxine</td>
<td>Major thyroid hormone for energy and metabolism</td>
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<td>25</td>
<td>Proenkephalin</td>
<td>Hormone for pain and emotion control</td>
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<tr>
<td>26</td>
<td>Enterokinase</td>
<td>Digestive enzyme hormone for digestive regulation</td>
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<td>27</td>
<td>GABA (Gamma Amino Butyric Acid)</td>
<td>Brain factor that controls electric activity</td>
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<td>28</td>
<td>Prostaglandin</td>
<td>Aids inflammation disease</td>
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<tr>
<td>29</td>
<td>Thyrotropin Releasing Hormone (TSH)</td>
<td>Aids thyroid disorder</td>
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<tr>
<td>30</td>
<td>Proenkephalin</td>
<td>Hormone for pain and emotion control</td>
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<tr>
<td>31</td>
<td>Cortisol</td>
<td>Catabolic hormone made in mid adrenal, destroys immune</td>
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<td>32</td>
<td>Beaurerkin</td>
<td>Biactive peptide, shows emotional problems of a person</td>
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<td>33</td>
<td>Neurotensin</td>
<td>GI peptide, affects hypertension, hyperglycemia</td>
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<td>34</td>
<td>Secretin</td>
<td>Gastrointestinal peptide, aids gastrointestinal disease</td>
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<td>Anti Pain Enzyme Inhibitor</td>
<td>For pain regulation</td>
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<td>36</td>
<td>Renin Biactive Peptide</td>
<td>Kidney hormone precursor of angiotension</td>
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<td>37</td>
<td>Adrenocorticotropic Hormone</td>
<td>ACTH Corticotropin A</td>
</tr>
<tr>
<td>38</td>
<td>Angiotensin</td>
<td>Biactive peptide, hormone related to stress</td>
</tr>
<tr>
<td>39</td>
<td>All Hypothalamic Hormones</td>
<td>For all homeostatic regulation</td>
</tr>
<tr>
<td>40</td>
<td>Erythropoietin</td>
<td>Biactive peptide that helps control anemia and blood clotting</td>
</tr>
<tr>
<td>41</td>
<td>Testosterone</td>
<td>Male sex hormone, aids aggression &amp; irritability</td>
</tr>
<tr>
<td>42</td>
<td>Progesterone</td>
<td>Hormone made in the corpus luteum during pregnancy</td>
</tr>
<tr>
<td>43</td>
<td>Gamma globulin</td>
<td>Used to increase the immune system</td>
</tr>
<tr>
<td>44</td>
<td>Love</td>
<td>153</td>
</tr>
<tr>
<td>45</td>
<td>Human Growth Hormone</td>
<td>158</td>
</tr>
<tr>
<td>46</td>
<td>Human Chorionic Gonadotropin</td>
<td>175</td>
</tr>
</tbody>
</table>
Thyroid system

Hypothalamus

Anterior pituitary gland

Thyrotropin-releasing hormone (TRH)

Negative feedback

Thyroid-stimulating hormone (TSH)

Thyroid gland

Thyroid hormones (T3 and T4)

Increased metabolism

Growth and development

Increased catecholamine effect
"Although our intellect always longs for Clarity, Predictability and Certainty, True Intellect respects Uncertainty, Synchronicity and the Awe of Nature. And by Embracing Chaos as Gentle Process the Intellect can be Expanded."

Desire 'Dubouet