WHAT IS ALOPECIA AREATA OR WHY SAMSON WAS NOT ONE OF US?

http://npntserver.mcg.edu/html/alopecia/AlopeciaFAQ(part01).html#AlopeciaFAQ-AA.0

Patient: "I have had alopecia areata since '91. That was the first time I noticed it anyway, it
could have been earlier. I had taken a 10 speed bicycle ride from
Florida to Texas a year earlier and really don't remember any hair loss."

Doctor: "AHA! Found what caused your aa! You rode that bike sooo
fasssst your hair blew off! Advise.....peddling backwards fast so you can catch
up to it!!!!!

Alopecia areata is a fairly common form of hair loss. Approximately 1% of the
population will develop alopecia areata at a certain point in their lives.
At any given time approximately .1 to .2% of the general population will exhibit the
symptoms. This estimate is based on prevalence studies such as the First National Health
and Nutrition Examination Survey, conducted from 1971 to 1974 and published in May, 1992. The survey found that of 20,749 individuals studied, 37 could
be confirmed as having alopecia areata. Thus, a nationwide prevalence of
about 306,000 cases is projected, with estimates ranging from 218,000 to
426,000 (Safavi, Arch Derm 128:702, 1992). In Britain and the United States the sexes are equally affected at all ages.

Clinically alopecia areata is characterized by the sudden appearance of a round or oval patch of
non-scarring and painless hair loss with spontaneous remissions and exacerbations.
A few patients complain of itching in the scalp before or during the loss of hair.
The patches are well-circumscribed and occasionally exhibit the presence of
"exclamation-mark" (see the Dictionary for definition) hairs around their margin.
Most cases (95%) involve the scalp. Loss of hair from the trunk and extremeties is associated with a more severe course.

The involved skin in alopecic patches remains soft, smooth and almost totally devoid
of hair. Patches may appear in analogous fashion to dropping a number of stones at
the same time in a clear pool of water. The ripples reverberate around and

meet, mix, or bounce off one another. This circular "growth process" has given the idea to a few researchers that alopecia areata is of infectious origin but most believe in an autoimmune etiology (more about this later). Usually the earlier the onset of symptoms the worse the prognosis. Five to ten percent of patients, especially children, end up losing all of their scalp hair (alopecia totalis). When hair is lost throughout the whole body the disorder is termed alopecia universalis. Although the disease itself is non-life threatening, the cosmetic and psychological impact on both patients and parents is tremendous and may lead to a high lifetime prevalence rate of major depression and/or generalized anxiety disorders.

Alopecia areata is unusual during the first year of life, one third to one-half of all cases start by age 20, and only 25% of the cases occur after age 40. Twenty to thirty percent of patients develop a single episode. The statistics show that some optimism will not hurt since in 60% of cases the first patch exhibits hair regrowth during the first year. In one third of patients the initial patch never exhibits evidence of regrowth. Some studies suggest that the percent of patients who develop extensive disease (alopecia areata to either totalis or universalis) is anywhere from 16.7% to 19%. Recurrences are possible even after a 20 year remission.

It is currently accepted that alopecia areata is caused by an auto-immune response of your own body. Research yields the theory that a gene or genes from our parents gives us a pre-disposition for this condition, yet it doesn't appear at birth. Rather, the theory holds that sometime after birth, we are exposed to an "insult" (Fortune favors the brave!). This insult may be a virus or disclosure of a previously secluded protein for which our immune system defends itself. Here's the problem: the portion of protein exposed by this insult closely resembles an intrinsic protein sequence within the hair follicles' cells. The end result is that the immune system attacks the hair follicle in a case of mistaken identity. The resultant inflammatory reaction is non-specific and other structures surrounding the hair follicle also die. This includes sweat glands as well as the cells that provide the pigment to the hair. Surrounded by more and more inflammatory cells, the

hair follicle retreats into the higher layers of skin. It then starves itself
by not being able to get nutrition, and sheds its shaft (the hair) thus entering
an extended period of dormancy.

Some believe the predisposition to the disease and the subsequent physical insult
is not always enough. These people believe that emotional or stress problems can
trigger the disease. This may offer an explanation as to why stress reduction,
hypnotherapy, etc., have been of some help to a few patients. I am personally
skeptical. There seems no doubt that emotions can affect the immune system, but
perhaps after being triggered, the condition runs freely on its own. Sometimes
this process abates temporarily and hair regrows, sometimes not.

There is an overlap between alopecia areata and other organ specific autoimmune
disorders. Some of these include atopy (e.g., seasonal rhinitis, bronchial
asthma, and atopic dermatitis), thyroiditis, Addison's disease and vitiligo.
Case reports have also claimed an association with pernicious anemia and
myasthenia gravis. The generation of these auto-antibodies appears to be gender
related. It is now known that females with antithyroid antibodies are at
increased risk for developing severe hair loss. There is undoubtedly an
increased familial incidence of the disorder (about 20% of cases) which is more
likely genetic rather than environmental. In familial cases the severity of
involvement in relatives bears no prognostic significance to individual
patients. Current therapy relies on attempts to modulate the immune system.
Thus far, corticosteroids, contact sensitizers and UV radiation have failed to
alter the course of the illness.

ARE THERE SUBTYPES OF ALOPECIA AREATA?

> "I've been through just about all degrees of (hair) loss over the years, ... there's probably some colloquialism about "tempting fate" that would apply here, so maybe I should get back to counting my blessings instead of indulging in fear."

> "The chemical shorthand for gold (and for alopecia universalis) is AU.
> Surely, there must be some value in that .... beyond that I shine."

Alopecia areata is the 'umbrella term' that refers to patchy hair loss, total (or near total) loss on the scalp (a. totalis) and combined loss of scalp, facial and body hair (a. universalis). These terms are useful to indicate the extent of hair loss but they are more descriptive than clinical.

Using these terms can sometimes be difficult - it is not always clear in which category a person belongs. It really isn't important to force a definition when one is in that vague in-between area. Some of our patients have made transitions from patchy to totalis followed by regrowth. Similarly, some patients with totalis have patches of hair loss elsewhere in the body while others with "universalis" have scattered hairs still present in some parts of their body. At what point did they change from areata to totalis, or from totalis back to areata or universalis? Technically it is a matter of percentages and that is really not important to us. Like one of our members said, "I have one hair that periodically grows on my chin (of all places) and another that mysteriously appears on my knee. And yet another single hair periodically grows on my left big toe. Does this make me AT as opposed to AU?" The distinction is sort of like splitting "hairs" (or hair as the case may be!).

For clinical research purposes it is very necessary to define the group being studied. It is a great concern of researchers to have uniform standards when studying alopecia areata so that results from one study can be relevant when comparing results from another study. One of these parameters is severity or extent and clinicians have often taken an arbitrary cut-off when designing their studies- those that have less than 25% hair loss are either mild or moderate, those that have more than 25% hair loss are staged as severe. In the future such criteria will be superseded by factors that are at the molecular level such as HLA markers.

Other terms used in describing different subtypes of alopecia areata include:

OPHIASIS, which literally translated from the Greek means snake and referes to the winding or bow-like spread of hair loss around the margins of the

scalp. It is most commonly seen as a band affecting the back and sides of the head (otherwise known as the occipital and temporal regions).

RETICULAR, describing a reticular or grid-like hair loss. Patients with reticular alopecia have many patches involving most of the scalp. Due to the infrequency of this subtype and its similarity to hair loss of different causation a skin biopsy is usually ordered to clarify the diagnosis.

A DIFFUSE form is seen after recurrent bouts of hair loss followed by partial regrowth. In affected regions, short hair often intermixes with longer hair. The appearance of the scalp is one of thinning rather than a bald patch. Again, because of similarities with other types of hair loss, a skin biopsy may be ordered to clarify the diagnosis.

(Note: An interesting case report appeared published in the Am J Dermatopathol 1999 Feb; 21(1):46-50. The article written by S. Kossard was entitled "Diffuse alopecia with stem cell folliculitis: chronic diffuse alopecia areata or a distinct entity?:

> A 34-year-old woman presented with an 8-year history of slowly progressive diffuse nonscarring alopecia with loss of hair density. Scalp biopsy specimens showed increased miniaturized follicles and an asymmetric wedge-shaped lymphocytic infiltrate concentrated on the stem cell-rich region at the point of entry of sebaceous ducts and at bulge-like regions of multiple follicles. Several hair bulbs emerging at the stem cell compartment also were inflamed, but the hair bulbs in the deeper dermis and subcutis were spared. I speculate whether these findings may represent a stem cell folliculitis similar to the reaction pattern previously observed in graft versus host disease and in androgenetic alopecia. The additional presence of peribulbar lymphocytic inflammation could indicate that the patient had a variant of alopecia areata. The clinical presentation of a slowly progressive diffuse alopecia without progression to clinically recognizable alopecia areata and the prominent lymphocytic inflammation involving the stem cell compartment may prompt a reexamination of similar cases currently classified as chronic diffuse alopecia areata. The concept that lymphocytes can inhibit stem cell function without destroying the stem cells themselves needs consideration.)

TRIANGULAR describes a type of hair loss that resembles a triangle.

A rare PERINEVOID variant is diagnosed when the hair loss surrounds a birthmark.

When the condition affects the beard it is known as alopecia BARBAE. As one of our members said, "Sometimes I look like I have shaved while totally drunk." The beard is involved in about 10% of men.

In biblical times a name was supposed to say a little of who you were. A name even defined a person's destiny. Take for example the name Jesus, it is synonymous with Savior. -Although somewhat unrelated and jokingly, I always thought that the name itself (Jesus) gave away the punch line to the story.- I think there is some truth to the fact that names provide a little about a person's destiny. You see, once a diagnosis (a name for a medical condition) is received it is repeated time and time again. The name dwells within you, the diagnosis becomes a Mantra, an exercise in self-hypnosis. Names are definite and don't necessarily adapt to many of the things we learn or have learned about the condition. Once you give or receive a name, it may mark the beginning of the end. It is unfortunate that many patients are slaves to the power of the word. It shouldn't come as a surprise that these same patients accept what others say of them rather than who they are. One of the things that is hard for some people to understand is that contrary to names, people have the capacity to progress in who they are. Are you always an alopeciac? Do you TAKE TIME to enjoy being a good mother/father, friend, cook, golfer, etc.? Given enough time (aging) anybody can qualify as being an alopeciac, but how many will qualify as having been a good parent or a good friend? I think that splitters, those that believe in areata/totalis/universalis are the gnostics of the world. When you put "medical truth" above faith, you deny a person of his/her humanity. In our case salvation is attained by accepting who we are rather than reading labels.

In summary,the term alopecia areata has survived most attempts by the medical community to broaden it almost to the point of non-existence. Not only do we have alopecia areata but we must consider totalis, universalis, ophiasis, reticular, diffuse, triangular, perinevoid, barbae, etc. In a certain sense alopecia universalis stands for a severe form of the

condition, while alopecia areata is a less severe condition. There are studies
suggesting that certain people may have a selective vulnerability to the
more severe forms of the disorder. I personally believe that there are no
real points of demarcation. Transitional forms can regularly be seen and
outcome is the same for all of them (mainly unpredictable). Usually there
are periods of clinical standstills followed by sudden exacerbations. If anything,
the glorified latin nomeclature does justice to the marked variability of the
disorder especially in chronic cases. Nothing in the medical terminology
is sacrosant or distinctly procrustean. As a matter of fact we can create
our own terminology. How about dividing the condition into positive and
negative symptoms? Positive symptoms would be those distinguished by the
appearance of an abnormal phenomenon (e.g., inflammatory cells in a biopsy,
abnormal CD4/CD8 lymphocyte ratio, hyperactive thyroid, atopy), while
negative symptoms are those characterized by absence or diminution of
normal function (e.g., lack of skin pigmentation, depression, social
[mal]adjustment, amount of hair loss?). We could probably say that the
negative symptoms are the more enduring and those that underlie most of the
"disability". In essence I don't believe in merely attaching labels that
pan out to be meaningless or worse, pejorative. If this has been the case
in alopecia areata it is because the medical profession has found that the
presentation of the condition is diverse, its cause unknown, and response
to treatment unsatisfactory. If anybody believes that they can dissect
apart AA from AU solely on the amount of hair loss, HLA subtyping, etc.,
they should perform a good epidemiological study with statistical methods
based on cluster or discriminative function analysis to see whether their
populations are trully heterogeneous. In the discussion of
lumpers vs. splitters I get off my soapbox by reminiscing that in all of
my medical readings I have learned more about what alopecia is not rather
than what it is. I now understand a lot better the condition in social
and cultural terms than from the medical standpoint. Maybe alopecia areata
is not a disease and that's the reason we haven't been able to find a cure.
If anything we should find a cure for our society. Maybe people should stop

victimizing and persecuting others simply because they are different.

WHAT IS THE DIFFERENTIAL DIAGNOSIS OF BALDNESS?

Clinicans usually classify hair loss as scarring and nonscarring. In scarring alopecia, the hair follicles are involved by inflammation. The attendant fibrosis and loss of hair follicles prevents any potential regrowth. Scarring is manifested by a pale, glazy and smooth scalp. Scarring types of alopecia include infections, systemic diseases (e.g., lupus erythematosus, sarcoidosis, scleroderma, dermatomyositis), and physical or chemical trauma (e.g., freezing, burns, trauma, radiation, acids, alkali). In noscarring alopecia, the hair follicles are preserved or resting. Snoozzzzzzzze. This explains the reversible nature of many of these disorders. Among the nonscarring types of alopecia (accounting for over 90% of cases) that produce localized hair loss are alopecia areata, telogen effluvium, androgenetic alopecia (male pattern baldness), tinea capitis/scalp ringworm, and trauma (hair pulling and traction). Other causes of nonscarring alopecia with diffuse involvement include systemic diseases like lupus, hypo and hyperthyroidism, and nutritional deficiencies (e.g., iron, protein, biotin, zinc).

Telogen effluvium:

The normal hair undergoes a growth cycle with several phases. The active growth phase is called anagen and the resting phase is called telogen. Normally 80 to 90% of your hair is in the anagen stage. People with telogen effluvium have shifted their growth cycle in such a way as to precipitate many of the anagen hairs into the telogen phase. If you keep track of the amount of hair lost in your hair brush, pillow, and bathtub a person with telogen effluvium will probably lose some 400 per day. This amount is 4 to 5 times what is considered normal. The hair loss in telogen effluvium is diffuse and often barely perceptible to the clinican. Usually it takes for a 25% loss of hair (diffuse) in a normal individual before hair loss is noticeable.

In alopecia areata, hairs have similarly been shifted to the telogen resting stage. Contrary to the case of telogen effluvium, the shift occurs in a well circumscribed area. A biopsy specimen will also show that the

trigger mechanism for both conditions is different. The base of the hair follicle in alopecia areata will show inflammatory cells, but the same will be absent in the telogen effluvium biopsy.

It may be noteworthy to point out that stress may be a the precipitating factor for the shift of anagen hairs to their telogen phase. Two to three months after a severe illness (especially one with bouts of fever) or childbirth, scalp hair may be shed in abundance. Other reported stressors include hemorrhage (including blood donation), crash dieting or malnutrition, emotional stress and certain drugs. Among the drugs associate to telogen effluvium are aminosalicylic acid, amphetamines, bromocriptine, capatopril, carbamazepine, cimetidine, coumadin, danazol, enalapril, etretinate, levodopa, lithium, metoprolol, propanolol, pyridostigmine, and trimethadione. Unless these drugs or stressor are repeated, hair tends to grow back.

Telogen effluvium is more commonly seen in women than in men. It is classically described following childbirth or withdrawal from the pill. It begins from less than a month to 4 months post partum. Hair shedding may last for as long as a year. Treatment with different hormones (e.g., estrogen, progesterone), zinc, biotin, and even minoxidil 5% have provided no consistent effect.

Women may also complain that they never recover their full length of hair but this may be due to the normal aging process. As you get older the time your hair spends in the anagen phase seems to shorten. Consequently the length of your hair will shorten with ageing.

Other causes of diffuse hair loss include;

endocrine conditions (hypo- and hyperthyroidism, hypopituitarism, and hypoparathyroidism)
drug-induced (oral contraceptives, antibiotics, vitamin A excess, anticoagulants)
some cases of androgenetic alopecia (male pattern baldness, especially in women)
iron deficiency
malnutrition
severe chronic illnesses
diffuse type of alopecia areata

If no cause is immediately obvious, the work-up should include a skin biopsy, CBC (complete blood cell count) (with hemoglobin), ESR (erythrocyte sedimentation rate), ANA (antinuclear antibody), serum iron, and thyroid

function tests. The thyroid panel should include total and free T4, T3 and thyroid stimulating hormone (TSH). (See the Dictionary for definitions).
For females with suspected androgenetic alopecia levels of DHE-SO4 and free testosterone are included. Theodore J. Daly of Garden City Dermatology, New York routinely orders the following laboratory tests for patients where he suspects alopecia areata: TSH, T4, antithyroid antibody, antimicrosomal antibody, ASO, anti DNAse B titer, anti-reticulin antibodies, antigliadin antibodies, ANA, ds-DNA.

Androgenetic alopecia (also known as common baldness, male pattern baldness, androgen-dependent alopecia, premature baldness, diffuse alopecia of females).

This is the most common type of alopecia with an onset after puberty. Over the age of 40 it affects some 50% of men and roughly an equal number of women. It is said to follow an autosomal dominant inheritance pattern but some clinicians differentiate between patients with an early onset of symptoms (less than 30 years of age) and those in which the same pattern develops later in life. Usually the younger the patient is at the onset of symptoms, the more severe the eventual loss of hair. Certain racial groups have a much lower incidence of androgenetic alopecia, e.g., Chinese, Japanese, Native Americans, and some black Africans.

While hair loss in men tends to be manifested at the vertex, in women it tends to be more diffuse. Women with androgenetic alopecia rarely become complete bald. Hormonal factors appear to play a major role in its development. Men castrated before puberty do not develop androgenetic alopecia regardless of any genetic risk factors. They only develop the baldness if given testosterone. Then, if the hormone (testosterone) is discontinued, the baldness does not progress, but unfortunately, it does not reverse either. In women there is usually a delay until after a period of hormonal imbalance (e.g.,menopause, institutions or discontinuation of oral contraceptives, the postpartum period),thus suggesting a protective effect for estrogen. Some women also tend to have greasy skin, acne, and sometimes, evidence of

polycystic ovarian disease. Sometimes androgentic alopecia can be initiated at times of hormonal change (e.g., institution or descontinuation of hormonal contraceptives). Still results of tests measuring testosterone, gonadotropins, and estradiol in women with androgenetic alopecia have been quite variable (most results show normal levels).

Clinically, androgenetic alopecia is characterized by a miniaturization of hair and its follicle. Individual hairs are reduced in diameter or thinned out. The hairs become smaller and eventually unpigmented. Skin biopsies have shown an abnormality of the growth cycle, with a large proportion of individual hairs in the resting (telogen) phase. In addition, some biopsies reveal a moderate chronic inflammatory reaction around the hair follicle and its surrounding structures. The condition usually manifests itself as a bitemporal recession of the hair line in males. The remaining hairs become finer and less manageable for the patient. Some patients may also notice increased oiliness and a tingling sensation of the scalp.

Several medical conditions have been associated to androgentic alopecia. A thorough work-up for an endocrine type abnormality may be necessary if patients exhibit irregularities in either menstruation or lactation, acne, or virilizing hair growth. Laboratory work up may disclose pathological ovaries (polycistic ovary syndrome), adrenals, or pituitary. Among the laboratory tests recommended for all balding females are thyroid function tests, serum levels of dehydroepiandrosterone sulfate, testosterone, androsteneidione, and sex hormone-binding globulin.

In some women with abnormal androgen metabolism, antiandrogen drugs (e.g., cyproterone acetate) have been prescribed with some success. The drugs prevent the progression of the hair loss but only produce mild to moderate regrowth. Side effects prevent the use of hormonal therapy in males. Surgical intervention is directed at redistributing hair from the side and back of the head into the balding areas. This procedure entails multiple punch biopsies with removal of hair plugs and their transplantation to affected areas. Other surgical techniques attempt to reduce the size of the bald area by excising an elliptical area of skin from the vertex and stretching the adjacent skin. Repeated interventions may be necessary. Some information regarding wigs are provided in other

sections of this document.

Hypothyroidism:

"Last Saturday I was in the library working on a midterm when I almost passed out (2x in a row). It was unexpected and severe, and a woman at the library helped me get to a doctor. To make a long story longer...I have no family history on either side of thryoid disorder. My mother, however, told me tonight that she has known her thyroid has had *low* activity levels that she has never been treated for in the past. I also want to mention now that I had my thyroid tested (during a CBC--complete blood count) about six years ago, and received no callback that anything was wrong. Well...today I saw my doctor for a follow-up appointment and she said that my thyroid function is indeed low. I have been prescribed medicine that I will have to take for the rest of my life."

"After researching hypothyroidism (as opposed to hyperthyroidism) via the web, I made some startling discoveries. The symptoms vary, and often go unnoticed, ranging from dry skin and nails, losing hair in patches or all over, and constipation, to weight gain, tiredness, fatigue, and sometimes even mental illness and psychosis. This condition is so often overlooked, and perhaps because it occurs most often in women, it is misdiagnosed as a mental condition."

"I have had all of these symptoms to one degree or another at various stages of my life. Also included as symptoms are bouts of severe weakness (which go away within minutes or hours, and may leave one feeling like it's all mental, ie. crazy), a feeling of being *unbalanced*, forgetfulness, and cold sensitivity. I was treated by two different doctors over the past few years, and one even prescribed an antidepressant (which I later flushed down the toilet because it made me sick). Both doctors were male (but this may not mean anything) and treated me like a *stressed out female*."

Approximately half of hypothyroid patients suffer from hair loss. The

alopecia is generalized and characterized by thinning of hair rather than bald patches. The hair itself becomes dull, coarse, and brittle. This is a systemic disorder and hair all over the body appears to be affected; including the beard, pubic hair, etc. Some researchers have reported that the hair cycle of hypothyroid patients is shifted to the telogen (resting) phase- a phenomenon similar to that observed in alopecia areata and telogen effluvium. Another similarity betwen these conditions resides in the fact that these patients exhibit brittle nails with longitudinal and transverse ridges. Many years ago researchers thought that loss of the lateral third of the eyebrows (Hertoghe's symptom) was characteristic of the condition. This change has now been reported in thallium toxicity, lupus, seborrheic dermatitis, and some patients with alopecia areata.

The most common cause of primary hypothyroidism is autoimmune in origin.
Laboratory tests have shown the presence of a circulating antithyroid antibody (see antimicrosomal and antithyroglobulin antibodies defined in the Dictionary section). This antibody can be found in some patients with alopecia areata.
The end result is a more severe type of hair loss with a poorer prognosis.
Besides loss of hair, some of the manifestations of hypothyroidism include;
fatigue, lethargy, cold intolerance, cramps, muscle stiffness, weight gain, cold hands and feet, dry skin, low body temperature (below 97.8F) --sufferers feel the cold deep inside, low blood pressure, constipation, and unexplained fatigue (particularly upon rising and in the afternoon; more energy at night).

Thyroid effect many body functions. Many women have a family history of thyroid disease, particularly on their maternal side. Menstrual problems are common among low-thythroid patients; irregular cycles, painful cramps, heavy bleeding, PMS, endometriosis, infertility, ovarian cysts, etc. Some clinicians suggest that PMS may be a form of thyroid disease.

The diagnosis is based on thyroid function tests primarily the measuring the levels of TSH total T4, free T3, and T3. However, blood tests are not always reliable.

For instance, the Center for Disease Control regularly sends out blood samples oto 7 per cent of all laboratories in the U. S. Between 8 and 25 per cent of tests yield erroneous results. If you have the symptoms but not the laboratory results, have them done again at another laboratory!

The following is an instructive story from one of our participants;

"My 13 year old son was tested twice for his thyroid by two different dermatologist and was told :"nothing wrong". This really bugged me as he had all the classic symptoms of hypothyroidism. We are currently with an immunologist and he also deals with people with chemical sensitivities. We took my sons basal temperature for a week every morning same time before he got out of bed and found that his temp was between 94 and 97 which is very low. He had his cortisol tested which was 3.2 (very low) should be 4.2 to 10. He is currently on a low thyroid hormone (1mg). I can see a marked improvement in his well being. His skin is not dry anymore. The eczama on his arms and legs have gone and he is not as sluggish anymore."

Before the advent of sophisticated laboratory tests for thyroid hormones, physicians commonly diagnosed hypothyroidism based on two things -- basal body temperature (that is the temperature of the body at rest) and Achilles reflex time (reflexes are slowed in hypothyroidism). The problem with relying totally on thyroid hormone levels is that *mild hypothyroidism* is the most common form and doesn't show up in the blood tests so it goes undetected until it becomes moderate to severe.

Basal body temp should be between 97.6 - 98.2 (or 36.4 - 36.7 celsius). Women who still have *periods* must specifically tests themselves on days 2/3/4 during their menstrual cycle. For men and postmenopausal women any days are fine.
In contrast, high basal body temperatures (above 98.6 F or 37.0 C) are less common, but "may" be evidence of hyperthyroidism.

When testing basal temperature;

1) Use a glass (not digital - sorry) mercury thermometer.
2) Shake the thermometer "down" to below 95.0 F (or 35.0 C) and place it next to your bed BEFORE going to sleep.
3) As soon as you wake up, place the thermometer under your armpit for a FULL 10-minutes. Try not to move and keep your eyes closed. It might

help you to keep your eyes closed if you can set a timer to go off in 10-minutes (egg timer, snooze button).
4) AFTER 10-minutes, read and write down the temperature and date. If you are a woman having a period, also write down which day it was in your
cycle -- day-2, etc. (Day-1 being the first day of menstruation.)
5) Record your temperature for at least 3 mornings, preferably at the same time of day. If you happen to be a person who works nights, and/or
typically is "up all night", then take your temp when you wake up. *Morning* is not a key word -- *resting temperature* IS. But, it should
be at near the same time of "day".

Treatment is directed at replenishing those thyroid hormones that are scarcely available.
Thyroxine usually forestalls thyroid gland enlargement making surgery (to
relieve obstructive symptoms) unecessary. Especially in the elderly or patients with
heart disease, hormonal therapy should be instituted quite cautiously in order not
to tax the function of the heart. Unfortunately, replacement therapy while preventing
the further loss of hair, often fails to reverse the original hair loss or
provides for incomplete regrowth. One final caveat regarding replacement
therapy is that Synthroid is the commercial compound that is sold as an identical
hormone to that produced by the thyroid gland. In the information packet
for the patient that accompanies the Synthroid pills the following statement is
provided, "Partial hair loss may occur rarely during the first few months of
Synthroid therapy, but it is usually temporary."

Some alternative approaches to a healthy thyroid gland include supplementing your diet with up to 12 kelp tablets a day (or eat sea vegetables (in
particular, kombu, arame and dulse) and use iodized salt when you cook. Also, go easy on foods that suppress thyroid function (goitrogens) which
can interfere with the production of thyroid hormones, particularly when
eaten raw (cabbage, radishes, rutabagas, turnips and others such as peaches, peanuts, soybeans, and spinach).

There is some anecdotal evidence that the need for thryoid supplementation
may be diminished in women taking progesterone. According to Jonh Lee, MD
in "What Your Doctor Didn't Tell You About Menopause" estrogen may act as
a competitive inhibitor for the binding of thyroid hormone to its receptor

as both compounds have phenol rings.

You may obtain more information on hypothyroidism from;

The Broda O. Barnes, M. D., Research Foundation
P. O. Box 98
Trumbull, Connecticut 06611
(203) 261-2101
FAX (203) 261-3017

Although the Barnes Foundation provides for education and distribution of
information regarding thyroid conditions, they have a vested interest in
using their own diagnostic urine test and in networking patients to physicians using their methods.

You may also obtain information from the net by going to
http://wellweb.com/altern/diseases/thyroid.htm or from The Thyroid Foundation of
America at phone #800-832-8321 or fax 617-726-4136.