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# Alopecia: A New Approach to Treatment

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The baffling pathogenesis of hair loss has hindered the development of therapies. A new modality using low-dose corticosteroids plus an anabolic agent has, in a small study, produced good to excellent results. This method, plus tranquilizers, estrogens, and TLC by the physician holds promise.

The female patient views hair loss, or alopecia, as a source of embarrassment and even as a sign of disfigurement. Thus, although it does not endanger her physical well-being, hair loss is a significant threat to the patient's sense of femininity and self-esteem, as well as a frustrating challenge to the clinician.

Why the normal cycle of hair growth (anagen), rest (telogen), and loss (catagen) undergoes alteration or disruption is still poorly understood. Heredity, autoimmune disease, stress, hormonal changes associated with pregnancy and childbirth, certain febrile illnesses, and chemical agents have all been implicated in the various forms of hair loss, but many questions remain unanswered. What controls the cyclicity of the hair follicle? How does testosterone act on the follicle via the skin? Why does hair proliferate in one area and regress in another?

Because the etiologies of alopecia are largely obscure, treatment of this condition has met with only

moderate success. Thus, a new approach using low-dose corticosteroids combined with an anabolic agent merits consideration, even though the clinical study group was small.

## Classification

Alopecia is divided into three categories, based on the degree and location of hair loss: alopecia areata, alopecia totalis, and alopecia universalis.

**Alopecia Areata:** This form of hair loss occurs in 17.2/100,000 of the population and usually presents with a well-circumscribed oval-shaped area of hair loss that spreads centrifugally with some degree of rapidity. Occasionally, the initial patches lack a regular outline, and scattered long hairs may be found within the bald areas. Characteristically, the skin of these patchy bald spots is ivory-white and completely hairless; rarely, there may be some erythema or edema. The occurrence

of short ("exclamation point") hairs is a pathognomonic finding in alopecia areata. A not infrequently predominant feature of the anagen phase of the hair cycle is the cessation of hair growth.



**Alopecia** of short the so-called finding classical circumscript or scaling dermat

lesions, stage, 10 of the p telogen Thus, a alopecia matrix, hair.

**Alopecia** alopecia develop alopecia children

**Alopecia** all body worst m Progr hair reg few, wh manifests relapses worst p

**Diagnosis** The diagnosis of alopecia areata is based on the clinical findings of baldness easily diagnosed disorder

of short, protruding club hairs with frayed points ("exclamation-mark" hairs) in the affected area is pathognomonic of the condition. Under microscopic examination, these hairs have an irregular diameter and a poorly pigmented attenuated bulb (due to atrophy of the hair root), which is unlike the club-shaped tips of telogen hairs.

Another important feature of alopecia areata is the predominance of hair follicles in a state of arrested anagen. In an interesting study using hairs plucked successively from concentric rings surrounding developing

### fast take

**Alopecia areata** is characterized by the presence of short, protruding club hairs with frayed points, the so-called "exclamation mark" hairs. This finding, combined with rapid onset and a classical clinical appearance of well-circumscribed bald areas, without inflammation or scaling, easily distinguish alopecia from other dermatologic disorders.

lesions, Eckert and colleagues found that, at an early stage, 100 per cent of the hairs removed from the center of the patch were club hairs, and that the number of telogen hairs increased outwardly from the epicenter. Thus, a simple hypothesis for the pathogenesis of alopecia areata may be a transitory failure of the hair matrix, resulting in the premature shedding of anagen hair.

**Alopecia Totalis:** This condition, a severe form of alopecia areata, results in total loss of scalp hair. It develops slowly in 5-10 per cent of patients with alopecia areata and is observed more frequently in children.

**Alopecia Universalis:** This virtually complete loss of all body hair, including eyebrows and eyelashes, is the worst manifestation of the disease.

Prognosis depends on the extent of the disease: full hair regrowth may be expected when the lesions are few, while only 2.5 per cent of alopecia totalis patients manifest even short periods of complete hair regrowth; relapses are frequent. Alopecia universalis carries the worst prognosis.

### Diagnosis

The diagnosis of alopecia is based mainly on clinical appearance. Rapid onset, well-circumscribed areas of baldness, and the absence of inflammation or scaling easily differentiate alopecia from other dermatologic disorders.

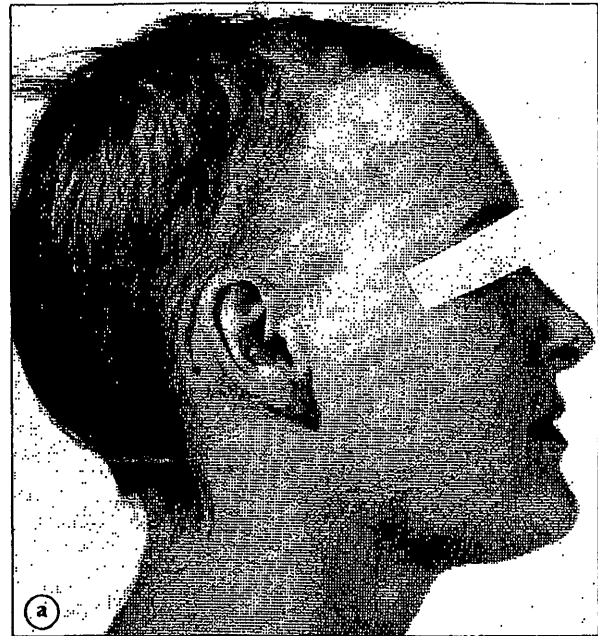


Fig. 1a. Alopecia universalis patient after 1 year of therapy. Before treatment there was a 90 per cent loss of head hair, and complete absence of eyebrows and body hair. Her eyelashes, axillary and pubic hair were scanty.



Fig. 1b. The patient, 21 years later. She received cortisone, 50 mg/day, which was gradually reduced to 25 mg/day; prednisone, 7.5 mg/day, was then substituted and gradually reduced to 2.5 mg/day. Corticoid therapy was discontinued 5 years ago. Concomitant therapy, from the beginning to the present, included subcutaneous pellet implantation of 50 mg of estradiol, and 75 mg of testosterone, with cyclic 7-day courses of a progestin to assure regular withdrawal of menstrual periods.

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# SINEQUAN (doxepin HCl)

Reference: 1. Barranco SF Thrash ML, Hackett E, Frey J, et al (Pfizer Pharmaceuticals, Pfizer Inc., New York, N.Y.): Early onset of response to doxepin treatment. *J Clin Psychiatry* 40:265-269, 1979.

## BRIEF SUMMARY

### SINEQUAN® (doxepin HCl) Capsules/Oral Concentrate

**Contraindications.** SINEQUAN is contraindicated in individuals who have shown hypersensitivity to the drug. Possibility of cross sensitivity with other dibenzoxepines should be kept in mind.

SINEQUAN is contraindicated in patients with glaucoma or a tendency to urinary retention. These disorders should be ruled out, particularly in older patients.

**Warnings.** The once-a-day dosage regimen of SINEQUAN in patients with intermittent illness or patients taking other medications should be carefully adjusted. This is especially important in patients receiving other medications with anticholinergic effects.

**Usage in Geriatrics:** The use of SINEQUAN on a once-a-day dosage regimen in geriatric patients should be adjusted carefully based on the patient's condition.

**Usage in Pregnancy:** Reproduction studies have been performed in rats, rabbits, monkeys and dogs and there was no evidence of harm to the animal fetus. The relevance to humans is not known. Since there is no experience in pregnant women who have received this drug, safety in pregnancy has not been established. There are no data with respect to the secretion of the drug in human milk and its effect on the nursing infant.

**Usage in Children:** The use of SINEQUAN in children under 12 years of age is not recommended because safe conditions for its use have not been established.

**MAO Inhibitors:** Serious side effects and even death have been reported following the concomitant use of certain drugs with MAO inhibitors. Therefore, MAO inhibitors should be discontinued at least two weeks prior to the cautious initiation of therapy with SINEQUAN. The exact length of time may vary and is dependent upon the particular MAO inhibitor being used, the length of time it has been administered, and the dosage involved.

**Usage with Alcohol:** It should be borne in mind that alcohol ingestion may increase the danger inherent in any intentional or unintentional SINEQUAN overdose. This is especially important in patients who may use alcohol excessively.

**Precautions.** Since drowsiness may occur with the use of this drug, patients should be warned of the possibility and cautioned against driving a car or operating dangerous machinery while taking the drug. Patients should also be cautioned that their response to alcohol may be potentiated.

Since suicide is an inherent risk in any depressed patient and may remain so until significant improvement has occurred, patients should be closely supervised during the early course of therapy. Prescriptions should be written for the smallest feasible amount.

Should increased symptoms of psychosis or shift to manic symptomatology occur, it may be necessary to reduce dosage or add a major tranquilizer to the dosage regimen.

**Adverse Reactions. NOTE:** Some of the adverse reactions noted below have not been specifically reported with SINEQUAN use. However, due to the close pharmacological similarities among the tricyclics, the reactions should be considered when prescribing SINEQUAN.

**Anticholinergic Effects:** Dry mouth, blurred vision, constipation, and urinary retention have been reported. If they do not subside with continued therapy, or become severe, it may be necessary to reduce the dosage.

**Central Nervous System Effects:** Drowsiness is the most commonly noticed side effect. This tends to disappear as therapy is continued. Other infrequently reported CNS side effects are confusion, disorientation, hallucinations, numbness, paresthesias, ataxia, and extrapyramidal symptoms and seizures.

**Cardiovascular:** Cardiovascular effects including hypotension and tachycardia have been reported occasionally.

**Allergic:** Skin rash, edema, photosensitization, and pruritus have occasionally occurred.

**Hematologic:** Eosinophilia has been reported in a few patients. There have been occasional reports of bone marrow depression manifesting as agranulocytosis, leukopenia, thrombocytopenia, and purpura.

**Gastrointestinal:** Nausea, vomiting, indigestion, taste disturbances, diarrhea, anorexia, and aphthous stomatitis have been reported. (See anticholinergic effects.)

**Endocrine:** Raised or lowered libido, testicular swelling, gynecomastia in males, enlargement of breasts and galactorrhea in the female, raising or lowering of blood sugar levels have been reported with tricyclic administration.

**Other:** Dizziness, tinnitus, weight gain, sweating, chills, fatigue, weakness, flushing, jaundice, alopecia, and headache have been occasionally observed as adverse effects.

**Dosage and Administration.** For most patients with illness of mild to moderate severity, a starting daily dose of 75 mg is recommended. Dosage may subsequently be increased or decreased at appropriate intervals and according to individual response. The usual optimum dose range is 75 mg/day to 150 mg/day.

In more severely ill patients higher doses may be required with subsequent gradual increase to 300 mg/day if necessary. Additional therapeutic effect is rarely to be obtained by exceeding a dose of 300 mg/day.

In patients with very mild symptomatology or emotional symptoms accompanying organic disease, lower doses may suffice. Some of these patients have been controlled on doses as low as 25-50 mg/day.

The total daily dosage of SINEQUAN may be given on a divided or once-a-day dosage schedule. If the once-a-day schedule is employed the maximum recommended dose is 150 mg/day. This dose may be given at bedtime. The 150 mg capsule strength is intended for maintenance therapy only and is not recommended for initiation of treatment.

Anti-anxiety effect is apparent before the antidepressant effect. Optimal antidepressant effect may not be evident for two to three weeks.

## Overdosage.

### A. Signs and Symptoms

1. Mild: Drowsiness, stupor, blurred vision, excessive dryness of mouth.  
2. Severe: Respiratory depression, hypotension, coma, convulsions, cardiac arrhythmias and tachycardias.

Also: urinary retention (bladder atony), decreased gastrointestinal motility (paralytic ileus), hyperthermia (or hypothermia), hypertension, dilated pupils, hyperactive reflexes.

### B. Management and Treatment

1. Mild: Observation and supportive therapy is all that is usually necessary.  
2. Severe: Medical management of severe SINEQUAN overdosage consists of aggressive supportive therapy. If the patient is conscious, gastric lavage, with appropriate precautions to prevent pulmonary aspiration, should be performed even though SINEQUAN is rapidly absorbed. The use of activated charcoal has been recommended, as has been continuous gastric lavage with saline for 24 hours or more. An adequate airway should be established in comatose patients and assisted ventilation used if necessary. EKG monitoring may be required for several days, since relapse after apparent recovery has been reported. Arrhythmias should be treated with the appropriate antiarrhythmic agent. It has been reported that many of the cardiovascular and CNS symptoms of tricyclic antidepressant poisoning in adults may be reversed by the slow intravenous administration of 1 mg to 3 mg of physostigmine salicylate. Because physostigmine is rapidly metabolized, the dosage should be repeated as required. Convulsions may respond to standard anticonvulsant therapy, however, barbiturates may potentiate any respiratory depression. Dialysis and forced diuresis generally are not of value in the management of overdosage due to high tissue and protein binding of SINEQUAN.

More detailed professional information available on request.

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On histologic examination, lymphocytic cell infiltration surrounding the dermal papilla is a prominent feature. The affected area is characterized by a non-specific inflammatory infiltrate of the hair follicle, and a round-cell "swarm of bees" type of infiltration around the hair bulb. The sebaceous glands are unaffected, and the surrounding skin is histologically normal.

Another type of hair loss, male-pattern baldness, should not be confused with alopecia. In the former, heredity and an inappropriate response to circulating androgen play a primary role; in the latter, the causes are largely unknown. The predominant role of testosterone in male-pattern baldness is manifested in women suffering from congenital adrenal hyperplasia and androgen-producing tumors.

## Possible Causes

**Autoimmune Disease:** This condition is most frequently suggested as a cause of hair loss. High or low titers of circulating autoantibodies and anti-smooth-muscle antibodies have been reported in several studies of patients with alopecia. Kern and co-workers found the same type of histologic abnormalities: perifollicular infiltrates and thickened connective tissue sheaths around the follicles. They also measured higher antibody levels for thyroglobulin, parietal cells, adrenal cells, and thyroid cells. No antibodies against hair follicle cells were found. The leukocyte count was normal in most patients, and neither thyroid nor adrenal disease was present at the time. However, according to Van Scott, the primary immunologic mechanism was degenerative change in the connective tissue surrounding the blood vessels that supply the dermal papilla, leading to a decrease in hair matrix volume.

## fast take

**Causes:** Autoimmune disease is named more often than any other factor as a cause of hair loss. Cancer chemotherapeutic drugs, stress, high doses of vitamin A and hormonal changes postpartum are also implicated. Most forms of alopecia are reversible with treatment.

**Psychogenic or Emotional Stress:** Frequently, patients correlate the onset of alopecia with a stressful situation or an emotionally upsetting event. There is little doubt that in susceptible individuals emotional disturbances are strong promoters, if not initiators, of excessive hair loss. A condition, sometimes referred to as telogen alopecia, (the loss of telogen hair) has been shown to be stress related. Although there is usually a latent period

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## ALOPECIA

**Postpartum Hair Loss:** Temporary alopecia occurs with varying degrees of severity in most parturient women. There is typically a latent period of 4-16 weeks, and onset within a few days of delivery strongly suggests a preexisting cause. In these patients, alopecia represents a delay in the normal anagen/telogen conversion, resulting in a temporary increase in the number of anagen hairs changed to telogen hairs in the postpartum period. The precise hormonal mechanism is unknown. There are conflicting data concerning the reoccurrence of alopecia with consecutive pregnancies, but the majority of investigators find no consistent relationship. Complete hair regrowth has been observed in about 50 per cent of patients.

**Drug-Induced Alopecia:** Hair loss associated with chemical agents is being seen with increasing frequency. Since these drugs are designed to attack cells indiscriminately in their most active phase of replication, cells of bone marrow, gastrointestinal and germinal epithelium, hair follicles, skin and embryonic tissue may be equally affected. However, not all chemotherapeutic agents cause alopecia, and the reasons for this are unclear. Doxorubicin leads to hair loss within 3 weeks in more than 80 per cent of patients. Cyclophosphamide, vincristine, methotrexate, and 5-fluorouracil also induce significant hair loss. Since anagen hair is chiefly affected, hair loss is observed where the anagen count is highest. The affected hair is irregularly narrowed, fragile, and breaks off at its narrowest point as soon as it emerges from the follicle, giving the characteristic appearance of "chemotherapy hair." The observed hair loss is temporary and regrowth occurs when therapy is discontinued.

A significant number of nonchemotherapeutic drugs, such as heparin and the coumarin derivatives, also cause telogen alopecia. Thallium used in treating tinea capitis, colchicine for hyperuricemia, as well as propranolol, trimethadione, methysergide, clofibrate, and lithium carbonate cause a moderate degree of hair loss. Megadoses of vitamin A and antithyroid drugs may lead to diffuse, but reversible, hair loss. The only drug-induced irreversible telogen alopecia is caused by high doses of androgens given to genetically predisposed individuals.

### Treatment

Since alopecia requires long-term systemic treatment before results are evident, hair pieces or wigs are recommended to minimize the psychic trauma.

A number of specific treatment regimens (Table 1) have been used for alopecia, with varying degrees of success. Phenol, rubefacients (e.g., tincture of cantharides or nicotinic acid derivatives), ultraviolet light, and dinitrochlorobenzene (DNCB) sensitization all appear



Fig. 2a. Alopecia areata in a 31-year-old hysterectomized woman.

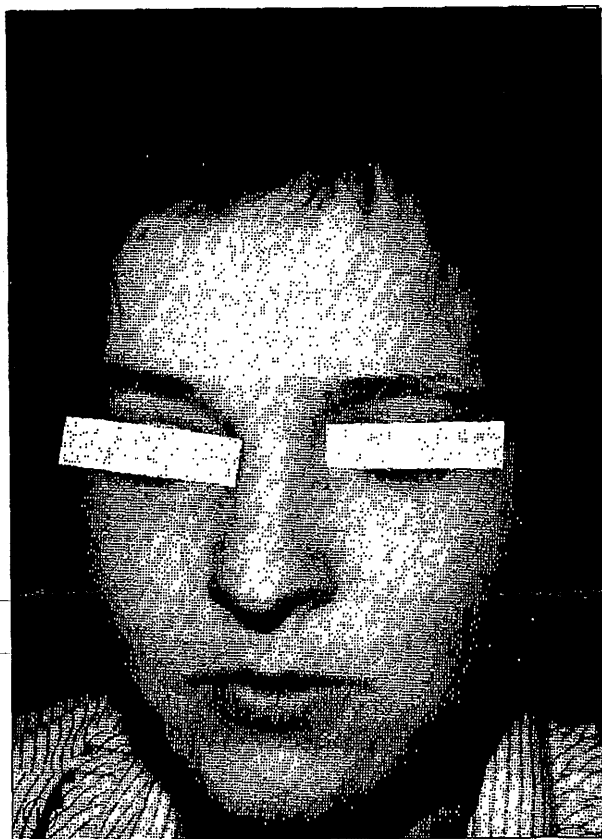


Fig. 2b. Same patient 1 year later. There is marked hair regrowth, which continued to improve with 4-6 mg/day of methylprednisone, and concomitant subcutaneous pellet implants of 25 mg estradiol and 75 mg of testosterone.

of 2-4 months, hair loss may be obvious earlier. Whether alopecia following serious febrile illnesses, such as typhoid, is caused by stress or an agent associated with the illness is not yet known.



Fig. 3a. Alopecia areata in a 25-year-old woman.



Fig. 3b. Same woman 9 months later, following treatment with 4 mg/day of methylprednisone.

to act via local vasodilation, followed by tissue hyperemia, which is believed to stimulate hair growth. Corticosteroids and psoralen (PUVA) probably exert their therapeutic effect via the autoimmune system. As for the other treatments listed in the Table, there is no universally accepted explanation for their mode of action.

Corticosteroids are currently the most frequent treat-

## ALOPECIA

ment method. One of the modes of administration, intralesional corticosteroid injection, is too painful for routine use. Systemic administration of high dosages, alone or with simultaneous local applications, has been fairly successful, although relapses are frequent. The duration and type of corticosteroid used have varied. Prednisone is generally recommended: 20–30 mg/day, for 4–6 weeks, followed by alternate-day therapy and/or treatment-free periods. Some clinicians, however, use comparatively short courses of prednisone at dosages rarely exceeding 30–40 mg/day, in combination with local treatment.

### Table 1 Treatments for Alopecia

Corticosteroids: Local  
Intralesional  
Systemic  
In conjunction with anabolic agent

Phenol  
Rubefacients  
Ultraviolet light  
DNCB sensitization (dinitrochlorobenzene)  
Psoralen (PUVA)  
Diaminodiphenylsulfone  
Zinc sulfate  
Penicillin  
Aloe  
Topical estrogens  
Tranquilizers

**A New Approach:** We believe treatment with chronic and/or high doses of corticosteroids carries many risks; therefore, we have employed a much lower dosage regimen along with anabolic agents.

Our study consisted of 13 female patients: 8 with alopecia areata, 2 with alopecia totalis, and 3 with alopecia universalis in the incipient stage. Treatment was started at the first visit, and patients were seen at 6-month intervals thereafter.

Oral systemic and local corticosteroids in ointment form were used as treatment modalities; intralesional corticosteroid injections were employed in an occasional patient, but later abandoned because of pain or poor results. Oral preparations included cortisone in 1 patient (50 mg/day, gradually reduced to 25 mg/day), prednisone in 5 patients (5–10 mg/day), and methylprednisolone in 7 patients (4–6 mg/day). The duration of therapy varied from patient to patient, but was continued until satisfactory hair growth was achieved. Then the medication was gradually withdrawn.

Corticosteroids were administered with an anabolic

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**ALDOMET® (Methyldopa) (MSD)**

Tablets, containing 125, 250, or 500 mg methyldopa; Oral Suspension, containing 250 mg methyldopa per 5 ml and alcohol 1%.

**Contraindications:** Active hepatic disease, such as acute hepatitis and active cirrhosis; if previous methyldopa therapy has been associated with liver disorders (see Warnings); hypersensitivity.

**Warnings:** It is important to recognize that a positive Coombs test, hemolytic anemia, and liver disorders may occur with methyldopa therapy. The rare occurrences of hemolytic anemia or liver disorders could lead to potentially fatal complications unless properly recognized and managed. Read this section carefully to understand these reactions. With prolonged methyldopa therapy, 10% to 20% of patients develop a positive direct Coombs test, usually between 6 and 12 months of therapy. Lowest incidence is at daily dosage of 1 g or less. This on rare occasions may be associated with hemolytic anemia, which could lead to potentially fatal complications. One cannot predict which patients with a positive direct Coombs test may develop hemolytic anemia. Prior existence or development of a positive direct Coombs test is not in itself a contraindication to use of methyldopa. If a positive Coombs test develops during methyldopa therapy, determine whether hemolytic anemia exists and whether the positive Coombs test may be a problem. For example, in addition to a positive direct Coombs test there is less often a positive indirect Coombs test which may interfere with cross matching of blood. At the start of methyldopa therapy, it is desirable to do a blood count (hematocrit, hemoglobin, or red cell count) for a baseline or to establish whether there is anemia. Periodic blood counts should be done during therapy to detect hemolytic anemia. It may be useful to do a direct Coombs test before therapy and at 6 and 12 months after the start of therapy. If Coombs-positive hemolytic anemia occurs, the cause may be methyldopa and the drug should be discontinued. Usually the anemia remits promptly. If not, corticosteroids may be given and other causes of anemia should be considered. If the hemolytic anemia is related to methyldopa, the drug should not be reinstituted. When methyldopa causes Coombs positivity alone or with hemolytic anemia, the red cell is usually coated with gamma globulin of the IgG (gamma G) class only. The positive Coombs test may not revert to normal until weeks to months after methyldopa is stopped.

Should the need for transfusion arise in a patient receiving methyldopa, both a direct and an indirect Coombs test should be performed on his blood. In the absence of hemolytic anemia, usually only the direct Coombs test will be positive. A positive direct Coombs test alone will not interfere with typing or cross matching. If the indirect Coombs test is also positive, problems may arise in the major cross match and the assistance of a hematologist or transfusion expert will be needed.

Fever has occurred within first 3 weeks of therapy, occasionally with eosinophilia or abnormalities in liver function tests, such as serum alkaline phosphatase, serum transaminases (SGOT, SGPT), bilirubin, cephalin cholesterol flocculation, prothrombin time, and bromsulphalein retention. Jaundice, with or without fever, may occur, with onset usually in the first 2 to 3 months of therapy. In some patients the findings are consistent with those of cholestasis. Rarely fatal hepatic necrosis has been reported. These hepatic changes may represent hypersensitivity reactions; periodic determination of hepatic function should be done particularly during the first 6 to 12 weeks of therapy or whenever an unexplained fever occurs. If fever and abnormalities in liver function tests or jaundice appear, stop therapy with methyldopa. If caused by methyldopa, the temperature and abnormalities in liver function characteristically have reverted to normal when the drug was discontinued. Methyldopa should not be reinstituted in such patients. Rarely, a reversible reduction of the white blood cell count with primary effect on granulocytes has been seen. Reversible thrombocytopenia has occurred rarely. When used with other antihypertensive drugs, potentiation of antihypertensive effect may occur. Patients should be followed carefully to detect side reactions or unusual manifestations of drug idiosyncrasy.

**Pregnancy and Nursing:** Use of any drug in women who are or may become pregnant or intend to nurse requires that anticipated benefits be weighed against possible risks; possibility of fetal injury or injury to a nursing infant cannot be excluded. Methyldopa crosses the placental barrier, appears in cord blood, and appears in breast milk.

**Precautions:** Should be used with caution in patients with history of previous liver disease or dysfunction (see Warnings). May interfere with measurement of: urinary uric acid by the phosphotungstate method, serum creatinine by the alkaline picrate method, and SGOT by colorimetric methods. Since methyldopa causes fluorescence in urine samples at the same wavelengths as catecholamines, falsely high levels of urinary catecholamines may be reported. This will interfere with the diagnosis of pheochromocytoma. It is important to recognize this phenomenon before a patient with a possible pheochromocytoma is subjected to surgery. Methyldopa is not recommended for patients with pheochromocytoma. Urine exposed to air after voiding may darken because of breakdown of methyldopa or its metabolites.

Stop drug if involuntary choreoathetoid movements occur in patients with severe bilateral cerebrovascular disease. Patients may require reduced doses of anesthetics; hypotension occurring during anesthesia usually can be controlled with vasopressors. Hypertension has recurred after dialysis in patients on methyldopa because the drug is removed by this procedure.

**Adverse Reactions:** *Central nervous system:* Sedation, headache, asthenia or weakness, usually early and transient; dizziness, lightheadedness, symptoms of cerebrovascular insufficiency, paresthesias, parkinsonism, Bell's palsy, decreased mental acuity, involuntary choreoathetoid movements; psychic disturbances, including nightmares and reversible mild psychoses or depression. *Cardiovascular:* Bradycardia, prolonged carotid sinus hypersensitivity, aggravation of angina pectoris. *Orthostatic hypotension* (decrease daily dosage). *Edema* (and weight gain) usually relieved by use of a diuretic. (Discontinue methyldopa if edema progresses or signs of heart failure appear.) *Gastrointestinal:* Nausea, vomiting, distention, constipation, flatulence, diarrhea, colitis, mild dryness of mouth, sore or "black" tongue, pancreatitis, sialadenitis. *Hepatic:* Abnormal liver function tests, jaundice, liver disorders. *Hematologic:* Positive Coombs test, hemolytic anemia. Bone marrow depression, leukopenia, granulocytopenia, thrombocytopenia. Positive tests for antinuclear antibody, LE cells, and rheumatoid factor. *Allergic:* Drug-related fever, lupus-like syndrome, myocarditis. *Dermatologic:* Rash as in eczema or lichenoid eruption; toxic epidermal necrolysis. *Other:* Nasal stuffiness, rise in BUN, breast enlargement, gynecomastia, lactation, hyperprolactinemia, amenorrhea, impotence, decreased libido, mild arthralgia, myalgia.

**Note:** Initial adult dosage should be limited to 500 mg daily when given with antihypertensives other than thiazides. Tolerance may occur, usually between second and third months of therapy; increased dosage or adding a diuretic frequently restores effective control. Patients with impaired renal function may respond to smaller doses. Syncope in older patients may be related to increased sensitivity and advanced arteriosclerotic vascular disease; this may be avoided by lower doses.

For more detailed information, consult your MSD Representative or see Prescribing Information. Merck Sharp & Dohme, Division of Merck & Co., Inc., West Point, PA 19486

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**Fig. 4. Alopecia in a 4-year-old girl with almost total loss of head hair, and complete absence of eyebrows, eyelashes, and body hair.**

steroid, either in an oral form, such as stanozolol or by subcutaneous implantation of a 75 mg pellet of pure crystalline testosterone, and 1 or 2 pellets of estradiol (25 mg) at 6-month intervals. Anabolic agents were chosen for their psychotonic action as a mood elevator, helpful in women experiencing the emotional upheaval following hair loss, and for their ability to counterbalance the catabolic effects of long-term corticosteroid therapy. It should be emphasized, however, that corticosteroids are the primary method of treatment, since they apparently suppress the underlying autoimmune disorder.

### fast take

**An alternative therapy was evaluated in a study of 13 women using low-dose corticosteroids administered with an anabolic agent. The latter was chosen for its action as a mood elevator, and for its ability to counterbalance the catabolic effects of long-term corticosteroid therapy. There were no adverse reactions to corticoids or anabolic steroids: results were comparable, and probably better, than with other modalities.**

Treatment was assessed on a scale of 100 per cent improvement by both patient and examiner. Of 8 patients afflicted with alopecia areata, results were excellent in 1 (95 per cent improvement), moderately good in 5 (about 50 per cent improvement), and poor in 1 (25 per cent improvement). One patient relapsed, although

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moderately good results were produced by further therapy.

Of the 2 patients with alopecia totalis, 1 had excellent results and the other, a recent patient, has shown decided improvement in a few months. Of the 3 patients with incipient alopecia universalis, 1 had 100 per cent hair regrowth; another did not respond until after the simultaneous local application of 10 mg of prednisone and 2 mg of an oral anabolic agent (stanozolol); the third, a young child, responded well over a 5-year period while on therapy but relapsed within a year after corticosteroids were discontinued. No untoward effects occurred from the dosages of corticoids and anabolic steroids used in this study.

The simultaneous use of oral corticosteroids and anabolic steroids achieved results that were at least comparable to, and probably better than, most other approaches.

Treatment is not too complex. Family physicians, general practitioners, and gynecologists with endocrinologic orientation may readily undertake the long-term management of alopecia. Administration of relatively small doses of corticosteroids, mild tranquilizers, tender loving care, and supportive therapy with estrogens and/or mild anabolic agents have proved rewarding for many victims of alopecia. □

## Hair Physiology

Hair is embryologically derived from the mechanoreceptor units in the hinge regions between the scales of certain reptilian species. It is composed of fibrous protein, which undergoes rapid keratinization and intracellular coherence to form hair fiber. Hair follicles, like other rapidly proliferating tissues, are constantly cycling.

### Anagen

This is the first phase of the cycle, when hair is synthesized from a follicle during a period ranging from a few weeks to several months. Approximately 85 per cent of scalp hair follicles are in anagen.

### Telogen

The next phase is telogen, the resting period, which lasts several weeks and may be identified by its typical clubbed hair root. Unlike scalp hair, most follicles from other areas of the body are in telogen.

### Catagen

Catagen is the final phase of the cycle during which telogen hair is shed.

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