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# Telogen Effluvium

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Additional information is available at the end of the chapter

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## Abstract

Telogen effluvium (TE) is a noninflammatory disease characterized by diffuse loss of telogen hair. It is the most frequent cause of diffuse hair loss and the actual incidence of the disease is not known. According to the underlying etiology, TE could be physiologically and pathologically classified. The evaluation of a patient with TE includes a detailed history, physical examination, and laboratory tests. The patients should be questioned in terms of TE subtype, duration, and clinical course of hair loss. The most important point in the treatment of TE is to consult about the natural course of the disease.

**Keywords:** hair loss, iron deficiency, management, treatment, telogen hair

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## 1. Introduction

Hair is important for social communication and healthy appearance, and acts as marker for identity of one's personal image. It can be, indeed, directly related to a feminine appearance, sexuality, attractiveness, and the concept of personality in females. In addition, hair is the second fastest growing tissue of the body, followed by bone marrow. Therefore, many metabolic derangements can be manifested with alopecia, and hair loss may be the initial clinical sign of a systemic disease [1]. Although hair loss may cause anxiety in individuals, irrespective of age and sex, and results in reduced quality of life and restriction of social relations in females, more than males. As a result, hair loss is the most frequent cause of admission to dermatology clinics [2].

## 2. Definition

Telogen effluvium (TE) was first termed by Kligman to define an increased shedding of normal club hairs based on the hypothesis that, irrespective of the cause, the follicle tends to act in a similar manner undergoing a premature termination of anagen and precipitating telogen [3].

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Telogen effluvium is a noninflammatory disease characterized by diffuse loss of telogen hair, caused by any disruption of hair cycle which leads to increased and synchronized telogen shedding [4]. It is the leading cause of diffuse hair loss. However, the actual incidence of the disease remains unknown [2]. It has been suggested to result from an abrupt shift of large numbers of anagen hairs to telogen hairs on the scalp with altered ratio of anagen hair to telogen hair from the normal ratio of 90:10–70:30 [1]. The degree of telogen effluvium depends on the severity and duration of exposure, but not the type of the agent [5].

Although the effect of age also remains unclear; elderly women are reported to be more vulnerable to acute TE following high fever, surgical trauma, severe hemorrhage, or immense psychological stress. In children, TE is responsible for only a minority of cases with hair loss [6, 7].

Telogen effluvium can be classified into two groups, according to the duration of disease, as acute and chronic. The duration of disease is shorter than 6 months in acute TE, while it takes more than 6 months in chronic state. In acute TE, hair loss typically begins 2–3 months later than the triggering event, although the triggering factor is unknown in 33% of the cases. However, chronic TE is frequent in healthy females in the fourth to fifth decades [7]. In addition, telogen gravidarum is a variant of TE which follows childbirth. Significant telogen gravidarum can affect a third to a half of all women following childbirth [8]. In postpartum TE, follicles remain in a prolonged anagen phase, rather than cycling into the telogen phase. Once released from anagen, an increased shedding of telogen hair is one of the clinical signs of the disease [9].

### 3. Epidemiology

The majority of TE is subclinical; however, its actual incidence or prevalence still remains unclear. It has no predilection for particular racial or ethnic groups. Although it affects both sexes, women are more likely to present for the evaluation of acute TE than men [10]. A higher number of women also suffer from chronic TE than men. Chronic TE, which is less common than the acute variant, mostly affects women between the ages of 30 and 60 years [11, 12].

### 4. Etiology and pathogenesis

The cycle of hair follicle includes anagen, catagen, and telogen phases. In a normal scalp, about 90–95% of the hair follicles are in the anagen phase, 5–10% are in the telogen phase, and a loss of 100–150 hairs per day is accepted as normal. Telogen effluvium is an abnormality of hair cycling during which a higher percentage of the scalp hairs are in the telogen phase. In TE, the number and ratio of hair follicles in the telogen phase increase. The physiological daily shedding of about 100–150 telogen club hair from the scalp is a usual nature of the hair cycle. In addition, follicles normally retain telogen hair, until the reentry to the anagen phase. Eventually, a new anagen hair pushes the old telogen hair out. It is unlikely to result in visible alopecia and to alter the trichogram [13]. Temporary alopecia may develop, since the shorter telogen hair is replaced by the long telogen hair. When the new anagen hair grows within 3–6 months, alopecia resolves [7]. In addition, no genetic cause for TE has been proposed to date [2, 9].

According to the underlying etiology, TE can be physiologically and pathologically categorized. Physiological causes include neonatal and physiological TE. However, pathological causes of TE include inflammatory diseases, stress, drugs, endocrine disorders, organ dysfunctions, nutritional causes, exogenous factors, syphilis, and systemic lupus erythematosus [2]. The main causes of TE are shown in **Table 1** [2, 10].

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**Acute telogen effluvium**

Acute or chronic major illness  
 Collagen vascular disease  
 Febrile illness:  
 -HIV infection  
 -Malaria  
 -Tuberculosis  
 -Typhoid  
 Major surgery  
 Endocrine disorders:  
 -Hypothyroidism  
 -Hyperthyroidism  
 Nutritional:  
 -Protein or caloric dietary restriction  
 -Nutritional deficiencies  
 -Iron deficiency anemia  
 -Congenital or acquired zinc deficiency  
 -Rapid weight loss  
 Drugs, supplements, or toxins  
 Physiological:  
 -Telogen gravidarum  
 -Physiological effluvium of newborn  
 Significant emotional stress  
 Inflammatory conditions of the scalp (e.g., seborrheic dermatitis)  
 Infectious conditions that affect the scalp (e.g., fungal, bacterial, or spirochetal)

**Chronic telogen effluvium**

Shortened anagen  
 May follow acute TE or telogen gravidarum

**Chronic diffuse telogen hair loss**

Thyroid disorders  
 Iron deficiency anemia  
 Acrodermatitis enteropathica  
 Crash dieting  
 Chronic starvation  
 Hypoproteinaemia  
 Metabolic disturbances  
 Advanced malignancy  
 Senility  
 Systemic lupus erythematosus  
 Dermatomyositis  
 Syphilis  
 Drugs  
 HIV infection  
 Anorexia nervosa

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HIV: human immunodeficiency virus, TE: telogen effluvium

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**Table 1.** Causes of telogen effluvium [2, 10].

## 5. Functional types of telogen effluvium

Headington defined five types of functional TE, according to the different follicular cycles. These include the immediate anagen release, delayed anagen release, immediate telogen release, delayed telogen release, and short anagen phase [2].

### 5.1. Immediate anagen release

It is a common form of TE which is related to physiological stress, severe illness, and drug use. During stress, the cytokines induce apoptosis of hair follicle keratinocytes, first with catagen, followed by telogen [2]. Therefore, follicles, which are induced to leave the anagen, enter telogen early [14].

### 5.2. Delayed anagen release

It typically occurs in women with postpartum hair loss, and when the oral contraceptives are discontinued. It is also known as telogen gravidarum. It is caused by high levels of circulating placental estrogen, which prolongs the anagen phase, result in a full head of hair during pregnancy. The withdrawal of these hormones during delivery stimulates the overdue anagen hair to enter into the catagen phase simultaneously. As a result, an increased shedding of telogen hair can be seen after a couple of months of delivery [2, 15].

### 5.3. Immediate telogen release

Drug-induced shortening of telogen results in follicles with the reentry of the anagen prematurely [14]. Hair follicles typically release the club hair 100 days later. It is caused by a shortened normal telogen cycle. This type of hair shedding usually occurs 2–8 weeks, following therapy with topical minoxidil [16]. As the exogen hair at resting is released, this paradoxical phenomenon occurs, by stimulated anagen phase [2].

### 5.4. Delayed telogen release

Hair follicles do not shed or recycle into anagen, but remain in prolonged telogen. When teloptosis defined as the termination of telogen phase with hair shedding occurs, the main clinical manifestation of increased shedding of the club hair presents. In such cases, the major cause is seasonal hair loss [2, 16].

### 5.5. Short anagen phase

Idiopathic shortening of anagen duration results in persistent telogen shedding. The condition is not associated with the hair shaft fragility or hair unruliness. It leads to resistant and chronic TE. It is common in hereditary hypotrichosis and ectodermal dysplasia and as an isolated disorder in otherwise healthy children [2, 16].

## 6. Clinical findings

### 6.1. Acute telogen effluvium

Women with acute TE are usually admitted with complaints of increased hair loss while washing hair and combing or brushing hair. These patients often have a concern of baldness. Despite excessive hair shedding, the density of the hair is visible [14]. If the main triggering cause of TE is eliminated, hair loss lasts for up to 6 months [1].

In the absence of a concomitant hair or scalp disorder, the scalp and hair shafts seem normal without any symptoms. In TE, the distribution of the scalp hair loss is diffuse; however, the bitemporal area may be the most affected area [10, 11]. In general, patients do not relate these events to their recent illness and have a concern of baldness [7]. In addition, no scarring or inflammation is present [2, 13].

To date, several factors have been suggested to be associated with the induction of TE, depending on clinical observations. However, there is no data to confirm and define the level of risk for TE related to these factors. It is estimated that an inciting factor is unable to be detected in about one-third of patients with acute TE [10].

In postpartum TE, time to hair loss often takes 2 or 3 months after delivery, although it can be delayed up to 6 months, depending on the length of the telogen phase. More interestingly, TE can be more pronounced, if delivery occurs in the fall, as the time of postpartum TE coincides with an increased seasonal hair shedding during the winter. In addition, breastfeeding may partially reduce TE with the effects of prolactin, since lactating women have an increased anagen-to-telogen ratio at 4 months in the postpartum period, compared to the nonlactating women. The condition often resolves by 12 months after delivery, even if breastfeeding continues [14, 17].

### 6.2. Chronic telogen effluvium

Women with chronic TE usually suffer from a prolonged, fluctuating course of TE for more than 6 months. In general, there is no trigger factor; however, some patients may have a continuation of acute TE with a shortened anagen phase, underlying a complaint of shortened hair as well as hair shedding seen in all patients with TE [13, 14].

In some cases, this type of hair loss may last for several years. Prolonged TE may be caused by multiple sequential triggers, although no trigger is identified in certain cases [1, 12].

In primary chronic TE, there is no specific triggering agent. Chronic TE can be induced by an acute TE [16]. Both hypothyroidism and hyperthyroidism are associated with chronic diffuse telogen hair loss (CDTHL). This is usually reversible upon reestablishment of the euthyroid state, although at times, longstanding hypothyroidism may cause hair follicle atrophy [18].

Iron deficiency anemia is also a causative factor of CDTHL, since follicles need iron to stimulate the anagen phase of the hair cycle [19]. The hair loss can be reversed with the iron supplementation. Iron deficiency without anemia is more controversial, as it has a potential relationship with CDTHL [20]. In the majority of cases, drug-induced CDTHL occurs mechanistically via the immediate anagen release [19]. It typically occurs within 6–12 weeks of treatment and progresses while on the medication. It, then, begins to resolve after the discontinuation of the drug [21]. To the best of our knowledge, no controlled trials showing a causal relationship for specific medications have been conducted; however, if a medication is suspected, it should be discontinued for a period of at least 3 months to examine its possible link to the hair loss [4, 14].

Moreover, diet is associated with the hair condition. Therefore, each patient should be questioned for the protein intake. Eating disorders can also lead to hair loss. In a study, 67% of the patients with TE had bulimia, while 61% had anorexia nervosa [22].

## 7. Diagnosis

In the presence of hair loss, the key step is taking a thorough history of the patient. In addition, the affected areas of the scalp should be examined to determine the presence or absence of follicular orifices. It helps to distinguish the scarring and nonscarring alopecia [23]. In the presence of follicular orifices, nonscarring alopecia can be suspected, although further history findings are useful to unveil the main cause [14].

The diagnosis of TE is usually based on the patient's history, physical examination findings, and hair pull test results (**Table 2**) [10]. The recognition of diffuse, noninflammatory, nonscarring hair loss should raise the clinical suspicion for TE, when it occurs acutely and is preceded by a physiological or psychological stressor, in particular. In the majority of cases, scalp biopsy is not required [10].

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Anamnesis, clinical diagnosis

Hair pull test

Trichogram

Light microscopy

Biopsy

Laboratory evaluation

Wash test

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**Table 2.** Diagnosis of telogen effluvium [10].



## 8. Histopathology

With the exception of TE induced by inflammatory disorders affecting the scalp, scalp biopsies of TE not show an inflammation. On the other hand, increased telogen follicles are the main histopathological finding [24]. Unlike androgenetic alopecia, the rate of vellus hair follicles does not increase [12].

## 9. Evaluation

Patients with TE should be evaluated with a detailed history, physical examination, and laboratory tests (**Table 3**) [1]. In addition, TE subtype, duration, and clinical course of hair loss should be also questioned. In particular, possible triggering factors within 2–5 months before hair loss begins should be addressed [7]. In the absence of no causative factor, a complete blood count, serum ferritin, biochemical markers, and thyroid function tests should be performed [2].

In addition, hair-care practices of patients which may damage hair such as braiding leading to traction alopecia, and the loss of eyelashes, eyebrows, and axillary, pubic, or body hair should be questioned, as alopecia areata or trichotillomania may affect any hair-bearing area. A detailed history, childbirth, prior surgeries, and psychosocial stress should be also assessed. Furthermore, drugs that may cause TE should be examined (**Table 4**) [1, 2]. Additionally, acne, irregular menstrual cycles, or hirsutism may indicate androgen excess, which contributes to female pattern hair loss. Symptoms of hyperthyroidism or hypothyroidism should be also evaluated and current and previous medications should be carefully reviewed. A history of following a strict vegetarian diet or heavy menses may suggest iron deficiency anemia [1].

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Duration

Thinning, shedding

Localization: localized diffuse

Associated symptoms: itching, pain, burning

Systemic symptoms, personal history

Nutritional history

Drug history

Psychosocial history

Hair care products-cosmetics history

Family history

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**Table 3.** History of hair loss checklist [1].



More than 5%:	1–5%:	Less than 1%
Mood stabilizers (lithium and valproic acid): <i>the most common</i>	Oral contraceptives	Amiodarone
Antidepressants (fluoxetine)	Acyclovir	Amitriptyline
Oral retinoids (acitretin, isotretinoin)	Allopurinol	Azathioprine
Anticoagulant (heparin, enoxaparin, warfarin)	Buspirone	Dopamine
Antimicrobial (isoniazid)	Captopril	Naproxen
Antiviral (indinavir)	Carbamazepine	Omeprazole
Interferon alpha	Cyclosporine	Paroxetine
Terbinafine	Lamotrigine	Sertraline
Beta-blockers (metoprolol, propranolol)	Nifedipine	Verapamil
	Hypolipidemic drugs	Venlafaxine
	Cetirizine	Vinblastine
	Gold	Vincristine

**Table 4.** Drugs associated with telogen effluvium [1, 2].

## 10. Laboratory

Although the majority of female patients with hair loss have normal laboratory test results, a complete blood count, ferritin, thyroid-stimulating hormone, antinuclear antibody titer, and vitamin D level can be studied, as abnormal levels of these parameters are likely to be associated with distinct forms of alopecia. In addition, serum and free testosterone, dehydroepiandrosterone sulfate, and prolactin should be analyzed, in the presence of any signs of potential endocrine abnormalities including severe acne, hirsutism, virilization, galactorrhea, menstrual irregularities, or infertility [14]. In case of any risk factors for syphilis, the venereal disease research laboratory test is recommended [1].

The link between serum levels of ferritin or vitamin D and TE is controversial. To date, studies investigating the relationship between serum ferritin levels and TE have shown controversial results [20, 25–27].

Iron deficiency anemia and thyroid disorders are the common conditions associated with TE. However, in the majority of cases, no apparent clinical features suggesting these conditions are observed [2].

A strict vegetarian diet or heavy menses may be suggestive for iron-deficiency anemia. Iron supplementation is recommended for TE patients who have had a serum ferritin level less than 70 ng per milliliter [20]. However, the effects of iron supplementation for TE have not been extensively investigated in controlled trials. The efficacy data are limited to case series, indicating cessation of hair loss and new hair growth with iron supplementation in women with low ferritin levels. On the other hand, the beneficiary effect of iron supplementation has not been established in all cases [27, 28].

Furthermore, perimenopausal symptoms such as hot flashes and irregular bleeding should be evaluated in older women. In this age group, starting or interrupting hormonal replacement therapies should be ruled out as a possible cause of TE [29].

## **11. Physical examination**

The scalp hair and scalp skin should be carefully examined in all patients. Scale, inflammation, pustules, and scarring or abnormalities of the hair shaft should not be evaluated as the manifestations of an isolated TE. On physical examination, the entire skin surface should be examined to identify the extent of hair loss and to detect the main features of other hair or scalp disorders [10].

### **11.1. Hair pull test**

A hair pull test should be performed as part of the physical examination in patients with suspected TE. The test is helpful to detect active hair shedding. About 50–60 hair fibers close to the skin surface are grasped and the hairs from the proximal to distal ends are tugged. Normally, only two or three hairs are pulled out by this method. In the presence of abnormal shedding, more than 10% hair (6–10 hair) can be easily pulled out from any part of the scalp, if the patient has not shampooed for more than 24 h [2, 30]. The test should be performed in four regions of the scalp: the frontal, occipital, and both temporal regions. The hair should not be shampooed for at least a day [29]. Light microscope is used to examine the hair shafts and to confirm that the loose hairs are telogen hairs [10].

Of note, the hair pull test may produce a false-negative result, if the patient has shampooed or vigorously groomed the hair on admission. In addition, if the patient has not shampooed or combed the hair for several days, the test may yield false positivity [10].

### **11.2. Trichogram (hair pluck test)**

From a hair pluck, sample is abnormal, which indicates higher than 25% telogen hair [13]. Since telogen rate in this test is not associated with the severity of the hair loss, the sensitivity of the hair pull test is low [2].

### **11.3. Wash test**

As daily hair count is troublesome, the wash test has been proposed. In wash test, the patient is instructed to wash hair after 5 days of last shampoo in a sink with its drain covered by gauze. The hair entrapped in the gauze is, then, counted [2].

### **11.4. Dermoscopy**

Data relating to the dermoscopic findings of TE are limited. Acute TE may indicate empty follicles and regrowing hairs of normal thickness ( $>0.03$  mm). Dermoscopic findings are useful to distinguish chronic TE from female pattern hair loss (female androgenetic alopecia). The latter variably exhibits a greater hair diameter [31].

### 11.5. Wood's light examination

TE can be due to seborrheic dermatitis of the scalp. On physical examination, a greasy scale and erythema on the scalp can be seen with a characteristic distribution. In addition, examination with a Wood's lamp (a source of ultraviolet A light) can be useful for the definite diagnosis of seborrheic dermatitis, which unveils the scale [32].

### 11.6. Procedures

In the majority of cases, further investigation is not required, beyond the clinical history and physical examination. However, additional diagnostic tools can be useful in patients in whom the diagnosis remains unclear [10].

### 11.7. Scalp biopsy

In most cases, scalp biopsies are not required and are only reserved for certain patients with an obscure diagnosis. Although scalp biopsy is not mandatory, it helps to exclude female pattern hair loss and alopecia areata. In general, biopsy results are normal, except increased telogen follicles (normal telogen counts vary between 6 and 13%). The rate of telogen follicles more than 15% indicates TE, while more than 25% is the major manifestation of TE [2, 30].

A 4-mm punch biopsy is sectioned horizontally for each specimen, and a second specimen is sectioned vertically. In general, we perform biopsy in an area outside of predilection for androgenetic alopecia to reduce the possibility of diagnostic uncertainty; therefore, we avoid bitemporal, frontal, and vertex areas of the scalp, if applicable. We usually select the leading edge of the alopecic area and avoid completely bald areas [10].

### 11.8. Trichograms and phototrichograms

Although these techniques are less common, they may be helpful to confirm the diagnosis of TE. With the use of these techniques, the rate of telogen and anagen hair follicles on the scalp can be evaluated. Currently, trichograms and phototrichograms are mostly used in specialized clinical hair centers and research studies. These procedures are described in detail in a separate section [10].

## 12. Differential diagnosis

The pattern of hair thinning or shedding can be helpful in the differential diagnosis. Diffuse thinning of the scalp hair in both temporal regions is highly suggestive of TE [33]. Frontal fibrosing alopecia almost particularly affects the frontal and frontotemporal hairlines. In case of traction alopecia, the periphery of the scalp is usually affected. Central centrifugal cicatricial alopecia (CCCA) typically begins at the vertex of the scalp, expanding centrifugally [34]. Alopecia areata may present in varying patterns. The patchy type is usually localized, whereas a more diffuse pattern has also been described [29, 35].

### 13. Treatment

Consulting on the natural course of the disease is the mainstay of the treatment of TE. A detailed evaluation should be performed to identify the underlying cause. In general, hair loss halts within 3–6 months in patients in whom a triggering factor is identified and eliminated [2].

Although spontaneous improvement is expected for patients with TE related to an isolated event such as childbirth, those related to a persisting insult should have the cause eliminated or treated, if applicable. In case of drug-induced TE, the suspected drug should be discontinued for at least 3 months to identify whether hair loss improves with the discontinuation of therapy. In addition, concomitant hair or scalp disorders such as seborrheic dermatitis should be simultaneously treated [10].

Furthermore, hair loss may profoundly affect the psychosocial status of the patient, irrespective of the degree of hair loss. Therefore, emotional well-being of the patient is critical in the management. All concerns of the patient should be sensitively addressed by the clinician. In addition, patients should be educated on the hair growth cycle and the expected course of TE, including an explanation that complete hair loss is not expected to occur, to reassure patients. Follow-up is also helpful both to encourage the patient and to identify those requiring further evaluation for persistent TE [10].

Moreover, the diagnosis and treatment of TE should be briefly discussed with the patient. Potential therapeutic options include the followings, based on the pathogenesis of TE:

1. Inhibition of catagen.
2. Induction of anagen in telogen follicles.
3. Inhibition of exogen.

Currently, no potent, FDA-approved catagen inhibitors or anagen inducers are commercially available. However, catagen-inducing drugs such as beta-blockers, retinoids, anticoagulants, or antithyroid drugs should be avoided and catagen-inducing endocrine disorders including thyroid dysfunction, hyperandrogenism, or hyperprolactinaemia should be simultaneously treated. Replacement therapy for catagen-promoting deficiencies such as iron, zinc, estradiol, or proteins can be also prescribed [2].

Today, no proven vitamins or supplements for any form of hair loss are commercially available. In case of a measurable deficiency such as iron-deficiency anemia, replacement therapy may be initiated. However, a balanced diet and stable body weight are the critical measures. In the literature, biotin supplementation has been shown no effect on TE [36]. Despite their claimed benefits, there are no controlled studies investigating the efficacy of iron or thyroxine replacement on TE [4]. In addition, maintaining serum ferritin above 40 ng/dL has been suggested to reverse hair loss [26]. In case of poor response, possible factors such as poor compliance, misdiagnosis, malabsorption, coexisting anemia, or persistent blood loss should be considered. Iron supplementation should be continued for 3–6 months, until the iron stores are replenished [37].

Of note, unnecessary long-term iron supplementation may result in iron overload [38]. On the other hand, there is no proven effect of antioxidants or other supplements on TE [39].

## 14. Conclusion

In conclusion, TE is a common disease that causes diffuse hair loss. The diagnosis of acute TE is based on patient's history and examination findings. Since acute TE is self-limiting, the clinician should monitor the patient, until spontaneous resolution. However, in case of severe or prolonged shedding, further investigations are warranted. On the other hand, chronic TE can be only diagnosed, after other causes of chronic diffuse telogen hair loss are ruled out. There is no specific treatment for TE. In the management of TE, the major aspect is to educate the patient relating to the natural history of the condition.

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