

# Self-Biting with Multiple Finger Amputations following Spinal Cord Injury

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## Summary

*We have observed mutilative self-biting leading to multiple finger amputations in two patients following C<sub>4</sub> complete spinal cord injury (SCI). Both men were of normal intelligence without psychosis and each had a neurotic personality and history of fingernail biting. They related the self-biting to anxiety and depression. We believe these to be the first English language reports of multiple finger amputations due to self-biting following SCI.*

**Key words:** Spinal cord injuries; Tetraplegia; Self mutilation; Fingers.

## Introduction

Within the general population, the most prevalent self-destructive oral habit is fingernail biting, a subject which has received a good deal of attention in the English language medical literature (Azrin, 1980; Ballinger, 1970; Barmann, 1979; Birch, 1955; Bornstein, 1980; Bucher, 1968; Clark, 1970; Davidson, 1980; Deardorff, 1974; Ellerbroek, 1978; Gruenewald, 1965; Hill, 1946; Horne, 1980; Klatte, 1981; Koupernik, 1964; Lowry, 1965; Malone, 1952; Massler, 1950; McKerracher, 1967; McNamara, 1972; Pennington, 1945; Wechsler, 1931). Such behaviour is usually subtle and often goes unnoticed. More vigorous self-biting resulting in trauma of the upper extremities has been seen with some regularity in psychiatric institutions (Ballinger, 1971). We have observed two unique cases of progressive self-biting of the fingers and hands, resulting in extensive mutilation including multiple finger amputations, in individuals of normal intelligence who displayed this behaviour following spinal cord injury (SCI). Both patients bit their fingernails prior to SCI.

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In seeking background information on possible causes of the biting behaviour we observed, we performed a computerised search of the literature utilising both the PsychINFO data base and the National Library of Medicine's MEDLINE data base. We were unable to identify reports of cases similar to ours and we believe these to be the first English language reports of multiple finger amputations due to self-biting following SCI.

## Case reports

### *Case 1*

While away without leave from the Army during the Viet Nam Conflict, a 21-year-old man, under the influence of multiple centrally-acting drugs, dived into shallow water and sustained a C<sub>4</sub> complete SCI with transitional levels at C<sub>5-6</sub>. He had a history of seizure disorder from childhood head trauma and history of heroin addiction. He underwent decompressive laminectomy in a non-government hospital and was placed in a nursing home with no rehabilitation training. He received an undesirable discharge from the Army. He continued to abuse ethanol and was discharged from numerous nursing homes over the next 8 years. A difficult relationship with his father precluded his living at home. After revised policy converted his military discharge status, he was admitted to our facility. On physical examination, the right index finger had been amputated to the middle of the proximal phalanx and the distal phalanx of the right ring finger had been partially amputated. Most of the nails of the left hand were gone. He explained that he had chewed the fingers and nails off. Serum chemistry and haematology, chest roentgenogram, and electrocardiogram were within normal limits. He was taking no prescription medication. Phenytoin had been discontinued years earlier. Psychological: Past history was significant for childhood hyperactivity and nailbiting. Nailbiting recurred after SCI, when he was drug-free. The patient admitted to anxiety and depression during the 8 post-SCI years, when he began nibbling his fingers. He viewed his self-biting as an extension of nailbiting and had not been inclined to stop. There were indications of restlessness, impulsivity, and low frustration tolerance. He was alert, cooperative, oriented in all spheres, and exhibited no bizarre symptomatology. He was of average intelligence with good abstract reasoning ability and adequate potential to complete the college programme already begun. There was no evidence of psychosis. Although projective personality test data identified problems of anxiety management, identity, and sexuality, the patient did not manifest symptoms. Clinical observations and test results reflected a neurotic individual.

After rehabilitation, the patient was discharged to an independent living centre. Three months later, he had a major motor seizure during a glucose tolerance test. Phenytoin was initiated. Seven months later, the finger stumps had healed. After 4 more months, he was readmitted with pressure sores and open ulcers on the stumps. Serum phenytoin concentration was negligible, suggesting non-compliance. Therapy was reestablished and phenytoin levels were therapeutic. There were no signs of self-biting for the next 2 years, after which it recurred intermittently.

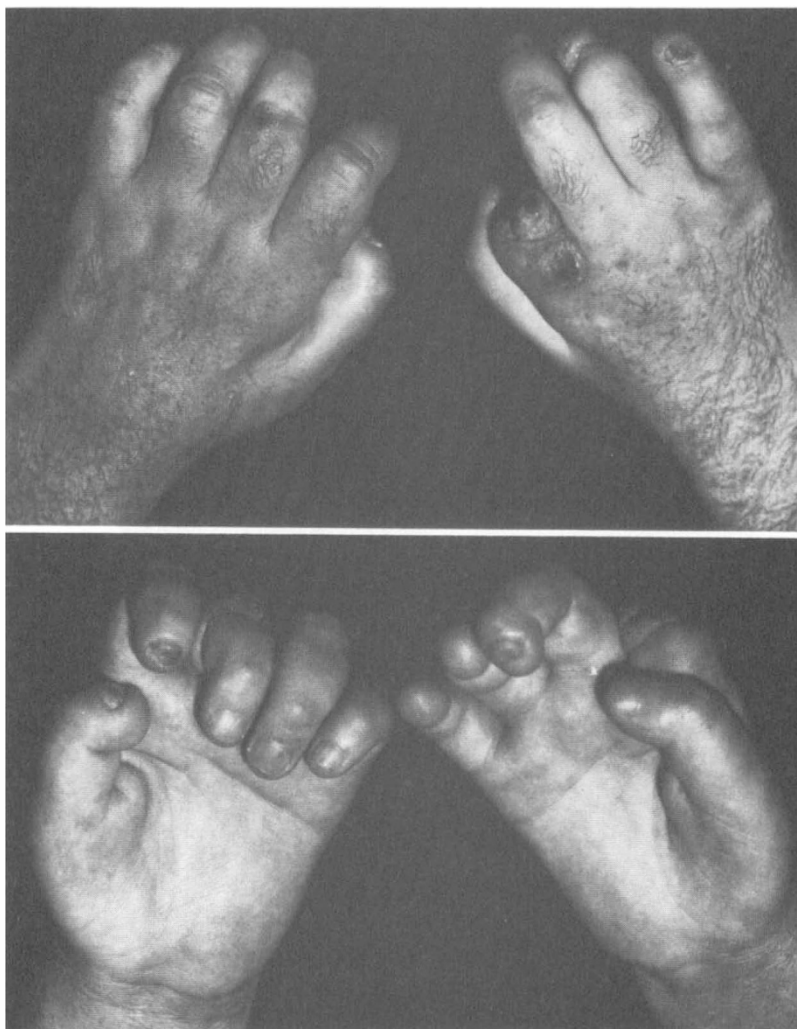
*Case 2*

While on combat duty in Viet Nam, a 24-year-old man sustained a C<sub>4</sub> complete SCI with transitional levels at C<sub>5-6</sub> from bullet fire. Following this, he separated from his wife and son. He was hospitalised several times over the next few years for bilateral hamstring tenotomies, lumbar sympathectomy, management of multiple pressure sores, and circumcision and implantation of penile prostheses for a condom catheter. Medication included diazepam and propantheline bromide. Six years post-SCI, when admitted with ischial osteomyelitis, the patient chewed his nails and fingers leading to bleeding and displayed increasing frustration. He requested that his fingers be covered with tape, to thwart the biting. When discharged, the fingers were healed. When seen as an outpatient afterwards, he had eaten away flesh from 2 fingers, exposing bone. One distal phalanx had broken off. Another was completely denuded. Five years later, he was admitted with multiple infected, necrotic, communicating Grade IV (Enis, 1973; Shea, 1975) pressure sores and osteomyelitis of the hip. He reported that the sores had been present since his last hospitalisation but that his dislike for hospitals had kept him home. On physical examination, the two distal phalanges of the right middle, ring, and small fingers had been amputated. The right index finger had been amputated to the middle of the proximal phalanx. Significant scarring and clubbing were present. On the left, the index, ring, and small fingers had been amputated just distal to the metacarpal-phalangeal joints and bleeding ulcerations were present on the stumps. The patient explained that this was all due to persistent finger-nibbling. One penile implant was gone and the other was eroding though the penile skin. Laboratory values were within normal limits except for hypochromic, microcytic anemia and hypoalbuminemia (2.8 g/dL) which required correction. Psychological: Past history was negative for emotional difficulty. Upon graduating from high school, the patient had been employed as a telephone company lineman. He attended college for a short time on a football scholarship but did poorly due to socialising. He left college to be married and was later drafted into the Army. He reported pre-SCI fingernail biting. He displayed severe self neglect. He was found to have bright normal intelligence and to be immature. He had been an underachiever and tended to exaggerate past deeds and accomplishments. He was overly self-confident regarding his abilities and future plans. Resumption of self-biting while hospitalised was related to his anxiety response to hospitals. He spoke freely of his self-mutilation but denied it was a problem. He tended to act out anger and depression. He was demanding and self-centered. The psychiatric diagnosis was of depressive neurosis.

During later hospitalisations, the patient stopped self-biting when staff focussed attention to it but when visited at home, he was found repeating the behaviour on several occasions.

**Discussion**

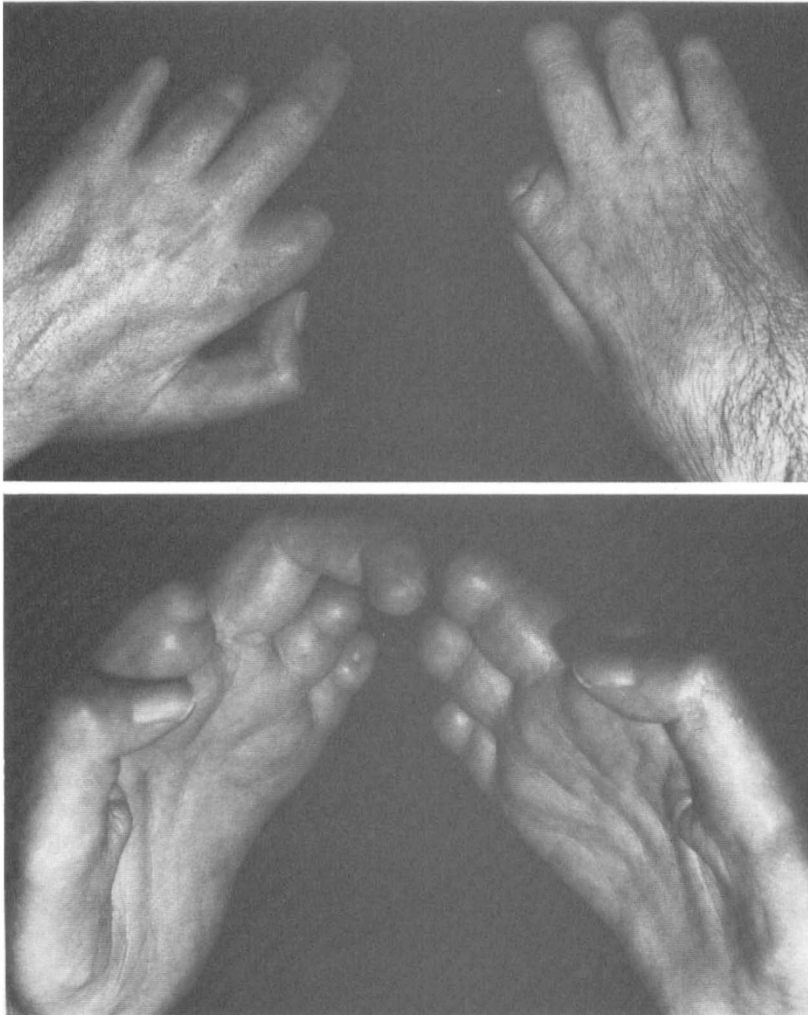
The term self-injurious behaviour (SIB) describes various repetitious acts directed by individuals toward themselves which impart physical harm or tissue damage (Dorsey, 1980; Schroeder, 1978; Tate, 1966); this includes self-biting.



**Figure 1.** Photographs of hands (Case 1), demonstrating mutilation by self-biting. (A) (Dorsal View) Note amputations of right index finger and tip of right ring finger, trauma over knuckles, and damaged nail. (B) (Palmar View) Note damaged nails.

Self-biting of the upper extremities has been associated with a number of diagnoses but SCI has never been implicated prior to this report, as far as we can ascertain. SCI shares certain characteristics with some, but not all, of the conditions previously associated with this form of SIB.

Ballinger observed that 14 of 93 hospitalised persons with below normal intelligence quotients who engaged in SIB did so by biting, which was usually confined to the upper extremities (Ballinger, 1971). Self-biting has been noted in cases of microcephaly (Singh, 1980), following neonatal brain damage (Gross, 1980), and in mental retardation (Dorsey, 1982; Gaylord-Ross, 1980; Lockwood, 1982). Lesch-Nyhan Syndrome (LNS), an X-linked inborn error of metabolism, is characterised by mental retardation, spastic cerebral palsy, choreoathetosis,



**Figure 2.** Photographs of hands (Case 2), demonstrating mutilation by self-biting. (A) (Dorsal View) Note bilateral multiple finger amputations. (B) (Palmar View) Note absence of trauma to thumbs bilaterally.

and compulsive self-biting of the lips, fingers, and hands (Anderson, 1978; Christie, 1982; Cudzinowski, 1979; Duker, 1975; Lesch, 1964; Nyhan, 1972; Nyhan, 1980). In one series, 3 of 15 hand/finger biters with LNS self-amputated portions of digits (Christie, 1982). Unlike these patient groups, our SCI patients had normal intellectual function. Unlike LNS patients, in whom sensory pathways are intact (Anderson, 1978; Cudzinowski, 1979; Seegmiller, 1980), our patients did have sensory deficits.

The most effective deterrent against the chewing away of viable tissue is probably pain sensation. Social mores also serve to dissuade self-biting. Persons with congenital abnormalities of pain perception have inflicted extreme self-mutilation upon themselves by biting, as in reversible somatotropin deficiency, which may be associated with pain agnosia (Money, 1972). Patients with



**Figure 3.** Roentgenograms of hands of Case 1 (3A) and Case 2 (3B), showing phalangeal loss and destruction due to self-biting.

congenital sensory neuropathy with anhidrosis begin self-chewing with the eruption of primary incisors (Lee, 1976; Mazar, 1976; Thompson, 1980). They are generally of low intelligence, with emotional disorders including autistic behaviour. They may present with numerous scars, calluses, abrasions, and nail deformity or loss (Pinsky, 1966; Thompson, 1980). Familial dysautonomia (Riley, 1957) has also been associated with SIB (Thompson, 1980).

Congenital sensory neuropathy without severe mental retardation leading to compulsive self-biting has been described (Haddow, 1970). Self-biting has been observed with a range of intellectual function, from borderline to

superior, in cases of congenital indifference-to-pain (Appenzeller, 1972; Gwinn, 1966; Ingwersen, 1967; Jewesbury, 1951; MacEwen, 1970; McMurray, 1950; Saldanha, 1964; Thrush, 1973). One would expect that an individual with impaired sensory function, but normal comprehensive ability, would curtail self-biting upon visual recognition of bleeding, as described by Appenzeller (Appenzeller, 1972). Recognition of trauma, bone denudation, and amputations did not effectively attenuate self-biting in our SCI patients.

Boredom-induced self-biting has been described in the absence of sensory deficit (Fahmy, 1981). Certainly, boredom may have contributed to the behaviour of our patients, who spent much of their day bedridden and alone. Parenthetically, we have observed self-biting of the digital pulp in a third C<sub>4</sub> complete SCI patient with normal intelligence who states that he bites his fingers out of boredom. Covert maternal deprivation has resulted in mutilative self-biting (Slaughter, 1977). Similarly, peer-infant separation syndrome has included handchewing in Macaque infants (Maxim, 1980). Both of our patients experienced relative social isolation following SCI. Carroll *et al.* state that non-suicidal self-mutilation is typically followed by a sense of calm, relief, and decreased tension. They related self-inflicted injury in adults to excessive violence in the home during childhood and to physical abuse by parents (Carroll, 1980). We are unaware of such childhood experiences in our patients.

Wart-like hyperkeratotic lesions due to gnawing of the dorsal aspects of the hands has occurred in response to stress (MacWilliams, 1974). Spontaneous self-aggression including extensive biting of the upper limbs has also developed in housed Macaques experiencing stress (Pond, 1983). Both of our patients related fingernail biting, which progressed to severe mutilation, to anxiety and depression. It has been found that disfigurement, embarrassment, and comments from peers do not necessarily diminish self-mutilative behaviours (Azrin, 1982). Our patients were both very self-conscious of the appearance of their hands after the finger amputations and yet both were observed repeating the self-biting behaviour.

Two cases from the literature resemble ours in terms of central nervous system involvement. Altman *et al.* described SIB in a one-year-old child with spina bifida and normal intellectual function. Pin-prick and temperature sensitivities were normal on the right upper extremity but not on the left. Normal withdrawal response was elicited by testing all areas except the left forearm and hand. Frequent episodes of finger-biting led to severe trauma of the left index finger (Altman, 1983). Arons *et al.* recently reported self-mutilation of the hands by chewing in a female born with a large cervical meningocele, which was surgically repaired after birth. She began chewing her hands at age 3 months and was able to describe pain and itching of the fingers at age 2 years. By age 6, tissue destruction and osteomyelitis had resulted. The thumbs were eaten away. She had normal mentation. Iron deficiency anemia was felt to be the result of a prolonged daily loss of blood due to biting (Arons, 1984). Anemia and hypoalbuminemia in our second patient were believed to be due to poor nutritional intake and extensive pressure sores, to which bleeding finger ulcerations probably contributed.

Self-biting behaviours associated with neurologic deficits resemble the phenomenon of post-neurosurgical mutilative self-chewing in animals. Multiple

dorsal rhizotomy or spinal cord trauma induce predictable self-chewing with amputations (Basbaum, 1974; Lombard, 1979; Sweet, 1981; Wiesenfeld, 1980). Increased pain-related activity accelerates this type of biting (Dennis, 1979). In a unique study, Berman and Rodin investigated the combined effects of T<sub>13</sub>-L<sub>6</sub> unilateral dorsal rhizotomy and isolation. Rats chewed the ipsilateral hindlimb and contiguous area when isolated pre- and postoperatively. Rats isolated preoperatively but paired postoperatively displayed no chewing (Berman, 1982), implicating isolation as a factor in self-biting.

In the unilaterally deafferented cat, spontaneously hyperactive neurons have been found in the dorsal horns bilaterally, from one cord segment below to two segments above the rhizotomised region. Lumbar cord hemisection is followed by spontaneous hyperactivity on the side of the lesion (Loeser, 1967). These findings support a quasi-epileptic focal etiology of self-biting in animals, in which contralateral inhibitory pathways may play a suppressant role (Duckrow 1977). High frequency and burst firing patterns from T<sub>10</sub> to L<sub>1</sub> have also been seen in human L<sub>1</sub> complete SCI (Loeser, 1968).

Management of self-biting involves behaviour modification, which has often proven ineffective. Restraints have produced bone demineralisation, tendon shortening, and reduced motor function (Dorsey, 1982; Lovaas, 1969) and could lead to pressure sores. While the efficacy of drugs in reducing SIB has produced varying results (Ballinger, 1971; Schroeder, 1978), selective pharmacologic manipulation has provided clues to understanding the behaviour.

Neuroleptics, such as droperidol (Burns, 1980) and chlorpromazine (McKinney, 1973) have attenuated self-mutilation in humans and Macaques. Their antiserotonergic action suggests self-biting is mediated by serotonin (SER) but the effectiveness of lithium therapy (Cooper, 1972; Sovner, 1981) suggests that the behaviour is initiated by relative SER depletion. Animal studies have supported the theory that self-biting in LNS is due to central dopaminergic dysfunction (Mueller, 1982; Mueller, 1982; Mueller, 1983). Decreased striatal dopamine (DA) neuron terminal function in LNS (Lloyd, 1981) implicates DA receptor supersensitivity (Mueller, 1982). Clinically, striking reduction in self-biting in LNS using a SER precursor gives credence to the hypothesis that the behaviour in LNS is due to subnormal central SER levels (Muzino, 1974; Nyhan, 1980), although some researchers disagree (Anderson, 1976; Buzas, 1981; Frith, 1976).

The narcotic antagonist naloxone attenuates or eliminates self-abusive episodes in mentally retarded patients with extensive history of SIB, possibly due to pain threshold lowering. SIB may be related to blunted nociception maintained by endogenous beta endorphins (Sandman, 1983). SIB could be a type of reinforced addictive response to the release of endorphins by pain-inducing behaviour (Richardson, 1983).

In rats, after unilateral C<sub>5</sub>-T<sub>1</sub> dorsal rhizotomy, self-chewing progressing to amputations, often to the elbow, occurred in 89 per cent without treatment and in 45 per cent with phenytoin treatment (Duckrow, 1977). While this supports the quasi-epileptic focus theory of self-biting, phenytoin also accelerates SER synthesis in the brain (Green, 1978), again implicating SER depletion. Arons' meningomyelocele patient responded to phenytoin when the antiepileptic diminished her dysesthesias (Arons, 1984). It is interesting that our first patient



nibbled his fingers extensively during a phenytoin-free period, although these are unscientific observations made retrospectively.

As discussed, SIB has been linked to central neurochemical alterations. Since stress and SCI have also been associated with such imbalances, they may contribute to SIB through that mechanism. SER activity increases (Culman, 1984; Driscoll, 1983; Richardson, 1984; Yehuda, 1984) or decreases (Driscoll, 1983; Smythe, 1983) in areas of the brain after stress. In humans, central SER stores are depleted (Hoes, 1982).

Elevation of central DA activity follows stress (Herman, 1982; Jensen, 1982; Miller, 1984; Reinhard, 1982; Richardson, 1984; Saavedra, 1981). Stereotypic behaviour in stressed pigeons is reduced by the neuroleptic haloperidol, apparently via DA antagonism (Goodman, 1983). In rats, stress-induced acceleration of DA metabolism in the medial frontal cortex can be reversed by naloxone, implying endogenous opioid facilitation of the DA stress response (Miller, 1984). DA antagonism could have been responsible for the reduction by naloxone of self-abusive behaviour reported by Sandman *et al.* (Sandman, 1983).

SCI is followed by long periods of adjustment, often extending to years and often accompanied by emotional stress. Central neurochemical imbalances might occur, as a result. Research has defined catecholamine fluctuations around the site of experimental SCI (Alderman, 1980; Bingham, 1975; Brodner, 1977; Brodner, 1980; Hinwood, 1980; Naftchi, 1981; Nemecek, 1977; Rodriguez, 1977; Shigetomi, 1980; Zivin, 1976) but the focus has been on acute cord response to trauma, rather than chronic abnormalities. Questions remain concerning SER and DA changes which may accompany the chronic SCI state, which might be compounded by stress, and which could contribute to self-biting.

## Conclusion

Self-biting of the upper extremities has generally been associated with specific pathology or physiologic abnormality or with mental retardation but the actual underlying cause continues to elude researchers. Certain factors may predispose an individual to self-biting. We believe that the fingernail biting habits and neurotic personalities of our two patients provided a foundation for this behaviour. After they sustained C<sub>4</sub> complete SCI, the contributory effects of stress, isolation, and most importantly loss of sensation resulted in the development of self-biting and led to multiple finger amputations. Alterations in central neurotransmitters and neuronal hyperactivity may have also been factors.

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## Résumé

Nous avons observé deux patients traumatisés médullaires C<sub>4</sub> complets qui ont rongé leurs doigts de façon très mutilante. Les deux patients étaient de sexe masculin, d'intelligence normale, ne

présentant aucun signe de psychose. Les deux ont une personnalité neurotique et des antécédents d'avoir été des 'rongeurs d'ongle.' D'après eux, l'anxiété et la dépression en sont responsables. Nous pensons que ces deux cas sont les premiers à être présentés dans la presse médicale en langue anglaise.

### Zusammenfassung

Wir berichten über 2 Fälle von Selbstverstümmelung, wobei die Patienten ihren eigenen Finger abgenagt haben. Beide Patienten waren C<sub>4</sub> Rückenmarkverletzte mit normaler Intelligenz und neurotischer Persönlichkeit. Psychose war nicht diagnostiziert. Sie haben eine Vorgeschichte von Nagel beißen. Sie klagten, dass Angst und Niedergeschlagenheit dafür verantwortlich waren. Es ist uns nicht gelungen anderen Fälle von Rückenmarkverletzung mit Selbstverstümmelung der Finger und Händen in der englischen Litteratur zurückzufinden.

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