Original Article

The early electroclinical manifestations of infantile spasms: A video EEG study

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Abstract

Purpose: Infantile spasms are described as flexor extensor and mixed; but more features of their semiology and ictal electroencephalography (EEG) changes are sparse in the literature. The purpose of the study was to describe the clinical and ictal video-EEG characteristics of consecutive cases with infantile spasms and to try to find an association with the etiology. **Materials and Methods:** The clinical phenomenology and EEG characteristics on video-EEG were analyzed in 16 babies with infantile spasms. **Results:** A total of 869 spasms were reviewed. Nine (56.3%) showed focal seizures at least once during the recording and 1 (6.3%) had multifocal myoclonus in addition to the spasms. The duration of the cluster and interval between spasms was totally variable in all patients. Lateralizing phenomena were present in at least some of the spasms in all patients. Unilateral manual automatism in the form of holding the pinna was noted in three patients following the spasm. The ictal EEG activity in the majority (75%) was the slow wave. Four (25%) showed fast generalized spindle-like ictal discharges. Spikes, spike and wave activity, or electrodecremental pattern alone during the ictus was seen in none. On bivariate analysis, no factor noted on the video EEG had association with the etiology. **Conclusion:** Infantile spasms could be associated with focal and other seizures, has unique, non-uniform and variable semiology from patient to patient. The ictal EEG manifestation in the majority (75%) of our patients was the slow wave transient with 25% showing generalized fast spindle-like activity.

Key Words

Ictal EEG, infantile spasms, video EEG, west syndrome

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Introduction

Infantile spasms are an epilepsy syndrome characterized by flexor or extensor spasms that tend to cluster. The onset is usually less than 2 years of age and electrographically has hypsarrhythmia.[1] Studies on the electroclinical manifestations of infantile spasms using video EEG are rare in the literature.[2-5] There was much discussion on the motor manifestations that characterize the spasm. The semiology that was used to distinguish a spasm from a myoclonus or a tonic seizure included the muscle contraction that lasted from 1 to 2 seconds, with a peak attained more slowly than a myoclonus and more rapidly than a tonic seizure. Clinical observation has also shown that the spasm is preceded or associated with focal clinical signs like ocular movements and facial grimaces. Various authors have noted different ictal characteristics like the percentage with clustering, the number with other seizures associated and the number with features of lateralization.

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Hypsarrhythmia has been extensively researched by many, but the ictal EEG correlate has been less addressed. Kellaway *et al*., in 1979 noted that the ictal EEG pattern in infantile spasms varied from patient to patient and described 11 different ictal EEG patterns.[4] We wanted to find out the pattern of seizures and EEG correlate in children with infantile spasms in our region. The primary objective of this study was to describe the semiology and the ictal EEG characteristics in these children. Our secondary objective was to look for associations of the various electro-clinical characteristics with the etiology of the spasms.

Materials and Methods

Subjects

We prospectively recruited consecutive patients in this study done in a tertiary care pediatric neurology center in South

India, from August 2009 to February 2010. We included newly diagnosed children fulfilling the ILAE criteria for infantile spasms with an age of onset between 1 and 12 months and who had hypsarrhythmia or modified hypsarrhythmia on EEG before treatment.[6] The present study excluded non-consenting patients and those previously treated with adrenocorticotropic hormone (ACTH), steroids, or Vigabatrin. During analysis of the video EEG, ictal epochs that showed non-negligible artifacts were excluded.

The variables noted were sex, religion, urban or rural place of residence, birth order, etiology, developmental delay prior to onset of spasms, age at presentation, age at onset of spasms, latent period for presentation, presence of microcephaly, and flexor vs. extensor spasms as described by the parent.

The etiology was reclassified into three groups as cryptogenic, perinatal insults and other causes. The perinatal causes included hypoxic ischemic encephalopathy and hypoglycemic, post-meningitic and post-ventilation neonatal cerebral injury. The other causes included structural malformations, inborn errors of metabolism, tuberous sclerosis and intrauterine infection. The term cryptogenic was used in cases where an etiology could not be detected.

Tools and techniques

A semi-structured pretested questionnaire was used to collect demographic details, and history. Inborn errors of metabolism were screened with Tandem mass spectrometry in all cases with the slightest clinical suspicion. Chromosomal analysis was done in cases where it was clinically deemed necessary. MRI scan of the head using a 1.5 Tesla machine was performed.

Video EEG protocol

An awake and sleep video EEG record was taken for minimum three hours before the commencement of treatment. A second awake and sleep video EEG for >3 hours was performed at 28 days after commencement of ACTH treatment.

Technical considerations

Video-EEG monitoring (VEM) was performed by using 16-channel digital video-EEG systems (BIOLOGIC) with gold plated — silver disk electrodes placed according to the full 10-20 system of electrode placement and the restricted 10-20 system of electrode placement when the head size was too small.[7] Electrode and movement artifacts were most commonly encountered as there was massive muscle movement during the ictus. Electrodes were fixed using 10-20 conductive neurodiagnostic electrode paste marketed by Weaver and company, and this was found very useful to minimize electrode artifacts. The ictal EEG recordings were visually examined; using when required, an expanded time-scale and appropriate amplitude display in order to evaluate more precisely the EEG waveform preceding during and following the spasm and to exclude any superimposed artifacts. When myogenic artifacts were seen, we changed the high-frequency filter to exclude the artifact and evaluate the waveform better. We then excluded all spasms with artifacts that did not allow us to study the ictal pattern.

Patient surveillance during the EEG was done by the EEG technologist and a relative of the subject, who was familiar with the habitual seizures. At least 25 spasms were recorded with a minimum total duration of 180 minutes. Both awake and sleep EEG and EEG on awakening was obtained in all cases.

Events detection and analysis

The events were detected by patient's caregiver triggered event button. The ictal semiology and ictal EEG were analyzed to look for any specific ictal pattern. A slow wave or spike seen during a spasm was not considered an artifact when it recurred with the same morphology from spasm to spasm.

Tables 1 and 2, respectively show the semiological details examined in the video and the factors looked for during analysis of the EEG. The characteristics of the spasms of each patient were classified depending on the characteristics of more than 75% of the spasms of that patient.

The ictal EEG correlates of the spasms was observed and classified as:

- High-voltage, generalized slow wave transients.
- Low-amplitude fast activity.
- Marked diffuse attenuation of EEG electrical activity (electrodecremental ictal EEG pattern).
- High-voltage slow waves with positivity at the vertex and followed by attenuation.
- Low-voltage rapid discharges on an attenuated background (or fast EEG ictal activity).

Ethical considerations

After approval by the institutional ethics committee, informed verbal and written consent was obtained from each child's parent or guardian after explaining the protocol.

Statistical analysis

The data was coded, entered and analyzed using Windows SPSS version 11. Descriptive statistics was used to describe the demographic profile and the electro-clinical observations on the video EEG recorded. Bivariate analysis was used to assess the relation of the ictal EEG and semiology to the etiology.

Results

Baseline characteristics of the patients

Table 3 shows the baseline characteristics of the patients. The sex distribution was equal. The majority was firstborn, symptomatic cases, presented to the hospital within one month of symptom onset; had an age of onset less than six months, microcephaly, flexor spasms and hailed from a rural background. All patients had developmental delay prior to onset of spasms and half the cases presented before six months of age.

Analysis of the ictal video EEG record obtained in 16 patients

A total of 869 spasms were reviewed in 16 children with infantile spasms. Well-discerned spasms with no movement artifacts on the corresponding EEG record ranged from 27-118 per patient.

Table 1 shows the ictal and inter-ictal video semiology observed in the 16 children on analysis of 869 spasms.

Table 2 shows the inter-ictal and ictal EEG changes. It was noteworthy that though the ictal characteristics on the EEG varied from child to child, it was consistent in all the spasms recorded in a single child. The ictal EEG activity in the majority

Table 1: The seizure semiology observed on the recorded video

Characteristic	Number (%)
Spasm type – flexor	16 (100)
Spasm symmetry	
Symmetric	9 (56.25)
Asymmetric	5 (31.25)
Subtle	2 (12.5)
Non uniform spasms*	13 (81.25)
Number in whom other seizure types in addition to spasms were noted	10/16 (62.5)
Behavioural arrest as a separate ictal phenomenon†	11 (68.75)
Irregular interval between individual spasms	16 (100)
Frequency of spasms in a cluster-	
>10/minute	11 (68.75)
5-10 per minute	2 (12.5)
2-5 in a minute	1 (6.25)
Nearly 1/minute	2 (12.5)
Maximum spasms in a series	
50-100	5 (31.25)
20-50	7 (43.75)
10-20	1 (6.25)
1-10	3 (18.75)
Duration of a cluster	
5-10 minutes	5 (31.25)
1-4 minutes	8 (50)
<1 minute	2 (12.5)
<30seconds	1 (6.25)
Time for peak contraction <2 seconds	9 (56.25)
The time for complete relaxation <2 seconds	11 (68.75)
Lateralizing phenomena like eye deviation, posturing or asymmetric spasms	16 (100)
Spasms associated with lateralizing phenomena	
With all spasms in the same patient	1 (6.25)
>half the spasms in the same patient	12 (75)/
<half the spasms in the same patient	3 (18.75)
Behavior preceding the seizure	
Not remarkable	9 (56.25)
Cessation of respiration only	3 (18.75)
Cessation of all motor activity	2 (12.5)
Fixed gaze	2 (12.5)
Behavior at seizure onset	
Nothing remarkable	10 (62.5)
Semipurposeful picking	2 (12.5)
Version	4 (25)
At seizure termination	
Not remarkable	4 (25)
Chewing movements	6 (37.5)
Terminated with a high pitched cry	3 (18.75)
Unimanual ear clutching‡	3 (18.75)

*The spasms varied in the distribution of limb and neck contraction in at least some of the spasms, †With EEG correlate, ‡Seen to hold the ear at seizure termination with at least some of the spasms

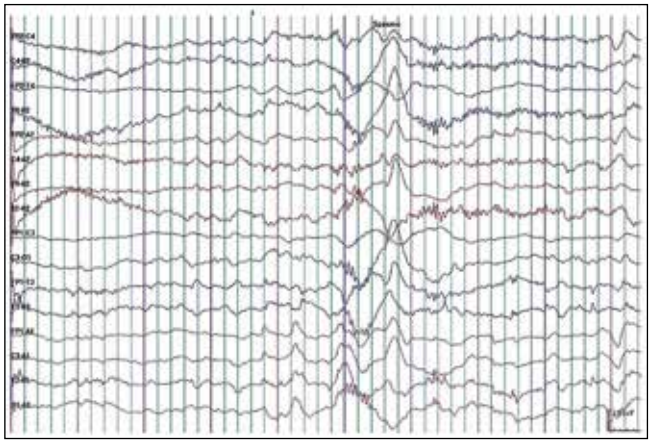

Figure 1: Generalized slow waves corresponding to the spasm and preceded by fast spindle-like activity. Ictal EEG record displayed at 30 mm/s paper speed and 15 microvolt/mm gain

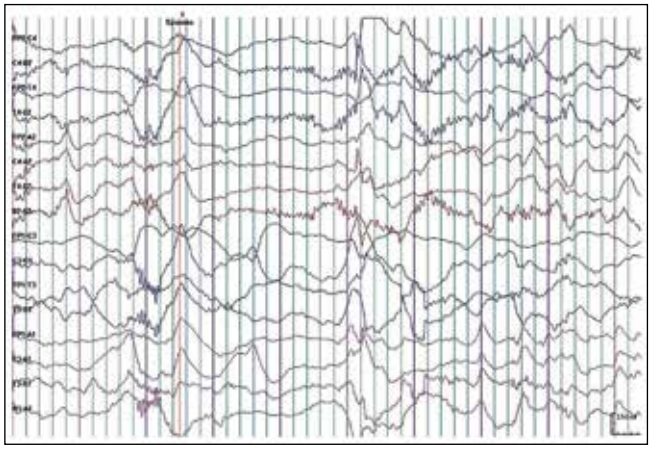

Figure 2: Generalized slow waves corresponding to the spasm, preceded by fast spindle-like activity and followed by voltage suppression. Ictal EEG record displayed at 30 mm/s paper speed and 15 microvolt/mm gain

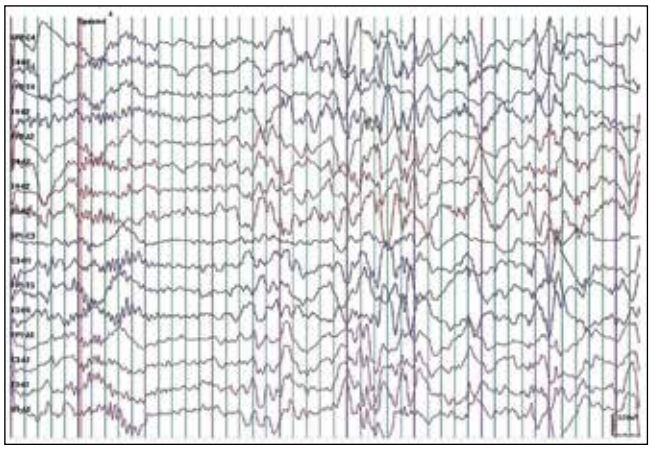

Figure 3: Fast spindle-like activity corresponding to the spasm. Ictal EEG record displayed at 30 mm/s paper speed and 10 microvolt/mm gain

Table 2: The EEG characteristics seen during video EEG recording of the 16 children

EEG characteristics	
The background hypsarrhthmia	
Typical with high amplitude polymorphic delta with multifocal spikes	10 (62.5)
With a consistent focus	5 (31.25)
With generalized spikes indicating interhemispheric synchrony	1 (6.25)
Attenuation of the background at any point of time	6 (37.5)
The voltage of spikes in the background	
>500 microvolt	6 (37.5)
300-500 microvolt	5 (31.25)
100-300 microvolt	4 (25)
<100 microvolt	1 (6.25)
Consistency of ictal EEG pattern from spasm to spasm in the same child	16 (100)
Ictal EEG	
High voltage slow waves or slow sharp wave complexes	9 (56.25)
A fast spindle like rhythm	4 (25)
High voltage slow wave followed by attenuation	3 (18.75)
Electrodecremental pattern only during the spasm	0/16
Assymmetry of the ictal activity	9/16
The EEG duration of the ictus	
<1 second	6 (37.5)
1-5 seconds	9 (56.25)
5-10 seconds	1 (6.25)
Interruption of hypsarrhythmia during ictus	16 (100)
Postictal slowing	
Generalized	10 (62.5)
Focal	6 (37.5)
Low amplitude fast activity leading to the ictal discharge	8 (50)

(75%) was the slow wave transient with or without voltage attenuation to follow [Figures 1 and 2]. Figure 3 shows the fast spindle-like activity seen in 25%. However spikes, spike and wave activity, or electrodecremental pattern alone during the ictus was seen in none. The ictal activity seen in our patients recurred from one spasm to the other in the same patient with a consistent pattern.

The follow-up video EEG at 28 days on ACTH showed continuing hypsarrhythmia with no change in 12 (75%) cases and inter-ictal spikes only in 4 (25%).

Table 4 shows the ictal EEG finding, the underlying etiology, the radiological changes, the side of brain affection judging by radiology and clinical features, presence of fast activity in 500 ms preceding the ictal discharge (its symmetry and side of occurrence), and the side of onset of the ictal discharge in each child. Nine of the 16 patients showed an onset of ictal activity on one hemisphere.

Among the 12 patients with clinical and radiological symmetry of affection, five showed a focal onset of ictal discharges. One with a right hemiplegia had ictal electrical discharges arising from the opposite side. The only three with a clearly lateralized affection on brain MRI showed ipsilateral onset of the ictal discharges. Fast activity preceding the ictus was noted in patients with and without a defined etiology.

Table 3: The baseline characteristics of the patients

Characteristic	Total no. = 16
Sex - Male	8
Rural	15
Birth order – first born	13
Etiology	
Probably symptomatic*	5
Perinatal insult†	5
Others‡	6
Developmental delay prior to onset of spasms	100%
Age at presentation < 6 months	8 (50%)
Age at onset less than 6 months	13
Latent period for presentation one month or less	13
Microcephaly	13
Spasm as reported by mother	
Flexor spasms	10
Extensor or mixed	6

*all patients had developmental delay though investigations failed to prove etiology, †Hypoxic ischemic encephalopathy, hypoglycemic, post meningitic and post ventilation neonatal cerebral injury, ‡Structural and metabolic causes, tuberous sclerosis and intrauterine infection

Among the four patients who had an ictal EEG consisting of fast spindle-like rhythm, three patients had symmetric brain affection on MRI. In three of these four children, fast spindle-like activity was seen in 500 ms preceding the ictus and leading to the ictus.

Association of the ictal EEG findings and ictal seizure semiology with the etiology of the spasms

On bivariate analysis, no electrographic or semiologic feature noted on the video EEG analysis of 16 children had association with the etiology of the spasms.

Discussion

In this study, we have made an attempt to describe the ictal electroclinical manifestations of infantile spasms in patients from South India. The predominantly rural population we encountered reflects the population here. We had an equal sex distribution, which is reported in some series but unlike the male preponderance noted by the majority.[8] The majority (68.75%) of our children were symptomatic as observed by others too.[9] Though 81.5% of our children were firstborn, this factor has not been noted in other studies and could reflect the antenatal and natal risks inherent to the primigravida mother. Microcephaly was seen in 72.7% in an Indian series (81.5% in our patients).[10]

All 16 patients (100%) studied had predominantly flexor spasms, though by history only 10/16 (62.5%) of the total had purely flexor spasms. This may be due to reporting bias. The majority in our study showed symmetric spasms that varied in semiology in 13/16 (81.25%) in the same patient. Similar to our observation of non-uniform spasms in 81.25%, Fusco and Vigevano noted that more than one child had spasms of various types.[2] The majority (62.5%) had

Table 4: The ictal EEG and clinical, electrical and radiological associations

Sex	ICTAL EEG	Etiology	Radiology†	Side affected‡	Fast activity§	Onset of ictal activity‖
F	HVS followed by attenuation	Epidermal nevus syndrome	Hemimegalencephaly right	Right	None	Right
F	Fast spindle like rhythm	Aicardi Goutierre syndrome	Bilaterally symmetrical calcification	Symm	Symm	None
M	Fast spindle like rhythm	Post meningitic sequelae	Symmetric frontal atrophy subdural fluid collection	Symm	Symm	None
F	HVS	HIE	Temporo-parieto occipital cystic changes-symmetric	Symm	Left	left
M	HVS	Idiopathic	Normal	Symm	None	None
F	HVS followed by attenuation	Idiopathic	Normal	Symm	Left	Left
M	HVS	Schizencephaly	Schizencephaly left	Left	None	Left
M	HVS	Idiopathic	Normal	Symm	None	None
M	HVS	Idiopathic	Normal	Symm	None	Right
F	HVS	Idiopathic	Normal	Symm	None	None
F	HVS	Cytomegalo virus infection	Bilateral thalamic hyperintensity	Symm	Right	Right
M	HVS	MCAD deficiency*	Normal	Symm	Symm	None
F	Fast spindle like rhythm	Gaucher disease	Normal	Symm	Left	Left
F	Fast spindle like rhythm	HIE with right hemiplegia	Normal	Left	None	Left
M	HVS	Hypoglycemic brain injury	Normal	Symm	Symm	None
M	HVS followed by attenuation	Neonatal stroke	MCA infarct right with atrophy	Right	None	Right

HVS – High voltage slow waves, Symm – Symmetric, *MCAD medium chain acyl triglyceride dehydrogenase, †By MRI brain and CT brain taken if warranted by the scenario, ‡By radiology and clinical findings, §In the 500 ms immediately preceding the ictus, ‖onset of ictal activity on one hemisphere in the form of slowing or fast activity preceding the spasm

other seizures like focal seizures and multifocal myoclonus independent of the spasms, as described in other studies (20% in Singhi *et al*.).[8,10] It is worth noting this phenomenon of spasms occurs with other seizure types, as this is a unique phenomenon reported in infants but not highlighted in the description of the semiology of infantile spasms. These episodes of "arrest" noted in the majority of our patients are well documented as early as 1979 by Kellaway *et al*.,[4] The variable duration and interval between spasms again emphasize the heterogeneity of this epileptic syndrome. The brief time taken for the peak of contraction and relaxation could lead to non-recognition of these seizures. Lateralizing phenomena seen in all our patients is also noteworthy and could highlight surgical candidature in intractable cases. Lateralizing signs in symmetrical spasms have been recorded by Fusco and Pachatz *et al*.[2,11]

Behavior preceding or at the onset of seizure was not remarkable in the majority. However at seizure termination, six (37.5%) showed chewing movements and three patients were seen grasping the pinna of one side following majority of spasms. Several automatisms have been described in infantile spasms but this unilateral manual automatism is not described in the literature. Unilateral manual automatisms accompanied by contralateral arm dystonia seen with temporal lobe epilepsy usually indicates seizure onset from the cerebral hemisphere ipsilateral to the manual automatisms.[12] This phenomenon needs further exploration.

There are varying patterns of ictal activity described in various studies.[2-5] Kellaway *et al*., reviewed 5,042 spasms in 24 infants and noted the generalized slow wave pattern followed or not by a period of voltage attenuation present in 48.8% of seizures and a generalized sharp and slow wave complex followed or not by voltage attenuation seen in 30.6% (total, 79.4%).[4] This is comparable to our 75% having the slow wave transient with or without attenuation of the background. The same authors had observed that the fast activity termed spindle-like was not related to a clinical spasm, but to a phenomenon called akinetic spasms. We had 4 (25%) children whose spasms (true spasms), on video EEG correlated with fast spindle-like activity on the EEG. Pachatz *et al*., have recorded diffuse slow waves in all 13 of their ictal records; fast activity associated in 9/13 and associated voltage attenuation in one.[11]

Fusco and Vigevano reviewed 955 spasms and slow waves, spindle-like activity and decrement of activity was described as ictal activity. They noted that every ictal discharge was associated with at least a single slow wave and that the onset of the spasm correlated with the onset of the slow wave. They also observed very low amplitude fast activity often riding on the slow wave. In their series, decrement of activity appeared in 69% of cryptogenic cases and 70% of symptomatic cases.[2] Haga *et al*., in 1995 noted high-voltage slow waves as the ictal EEG correlate in all patients. In their study, superimposition of fast activity on the slow waves was noted in a majority and spike and slow wave activity in a minority.[3] Menezes and Rho in 2002 recorded a slow wave transient followed by an

attenuation of the background amplitude as the most common ictal EEG pattern.[5]

On reviewing the literature, only Haga *et al*., and Curatolo *et al*., have observed spike as an ictal electrical phenomenon in at least a minority.[3,13] The slow wave transient with or without a decrement is the commonest observed electrical ictal phenomenon in infantile spasms in our study and the commonest observed in the literature available.[2,4,5,11] The observation of spasm to spasm consistency of the slow wave seen as the ictal electrical correlate in the same patient was made earlier by Gobbi *et al*., and later by Fusco and Vigevano.[2,14]

Presence of hypsarrhythmia in between spasms was found to be a good prognostic factor by Fusco and Dulac *et al*., independently in 1993; but all our patients had this pattern and therefore no comparison could be made.[2,15]

Kobayashi *et al*., in 2005 studied the slow wave associated with the spasm and showed that this could occur symmetrically in symptomatic infantile spasms with focal lesions.[16] The fast activity seen in 50% of our patients preceding the spasms has been interpreted by Kobayashi *et al*., in 2004 as an evidence for neocortical involvement.[17]

In the series by Fusco and Vigevano (1993) and in the 42 patients by Haga *et al*., (1995), the ictal pattern did not correlate with the etiology as is our observation.[2,3] In 2003, Pachatz *et al*., concluded that occurrence of focal seizures with infantile spasms can be related to various etiologies and could occur in the absence of localized lesions in the brain. They could not correlate the different spasm semiologies they recorded with the etiology.[11]

The persistence of hypsarrhythmia at 28 days after initiation of treatment in 75% of patients possibly reflected the lack of effect of the low dose of ACTH used in our study.

Study limitations

Though we did record video EEG with every child asleep over hours, we could not study the difference in the ictal pattern in the awake and sleep state as we could record only very few episodes in the sleep state; majority of the spasms were recorded on awakening the child from sleep.

Conclusion

The ictal EEG pattern was consistent in the same patient; though the semiology varied from spasm to spasm. High-voltage widespread slow/slow sharp wave transient with or without decrement was the ictal correlate in the majority of our patients (75%). Seizures other than infantile spasms were noted in 62.5% of our patients; majority showing focal seizures and all the patients had lateralizing phenomena.

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