A Developmental Study of the Relationship Between ERP P3a Latencies and Eyelid Reflexes Elicited by Startle Acoustic Stimuli in Normal Children

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Abstract

A late positive component (P3) in event-related potentials (ERPs) is elicited by attended or task-relevant stimuli with the peak latency of about 300 msec at the parietal and vertex scalp electrode locations. The P3 latency in response to an infrequent target tone occurring in a sequence of standard tones decreases progressively as a function of age, whereas the older subjects have a remarkably a longer P3 latency than do the younger adolescents. These latency changes of P3 may parallel the development of information processing speed and memory span with reagard to reaction times (RTs). However, the P3 is generated in a number of mammalian species when attention is elicited from significant environmental events. Startle or unexpected information should evoke large amplitudes in several ERP components in contrast to amplitudes evoked by expected, highly probable background stimuli. The P3 is mainly composed of two (or more) different components, that is, the P3a and P3b.

The majority of developmental research on ERPs has focused on the changes of the P3b component with respect to aging, not on the P3a at Cz. The P3a has a central scalp distribution under an unpredicted, unassigned task or ignored condition, while the P3b has a parietal location under an attended, assigned condition. This research investigated the developmental changes of the P3a component, which might overlap with the latency of P3b, to unattended, startle acoustic stimuli in normal children (of ages 3-8 years) as compared to young adults.

The result showed that the P3a latencies decreased progressively as a function of age, as well as the eyelid reflex latencies, but the P3a amplitudes changed as an inverse U-shape function of age.

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Key Words: development, children, auditory evoked potentials (AEPs), P3a,
           maturation (aging)

Introduction

It has been shown that exogenously evoked potentials (EPs) reflect the in-
tegrity and organization of the sensory pathway, whereas the characteristics of
endogenous event-related potentials (ERPs) are determined by cognitive processes
and psychological factors rather than by the physical properties of the stumulus
events (Picton & Hillyard, 1988). Auditory evoked potentials (AEPs) are classi-
fied globally into the three information processing levels: the shortest latency
brain-stem potentials (waves I- VII) which appear in the first 10 msec after
stimulus onset, the middle latency components (No, Po, Na, Pa, Nb, and Pb) in
the second 10-50 msec, and the late components (P1, N1, P2, Nd, N2a, N2b, PN,
MMN, Nc, Pc, P3, and SW) in the third 50-1000 msec duration. The late AEPs have
been also classified into sensory exogenous and cognitive endogenous components
(Hillyard & Kutas, 1983; Hillyard & Picton, 1987). The P3 of endogenous brain
potentials is the most widely studied event-related component in tasks requiring
subjects' attentional and categorizing skills (the oddball task is probably one
of the most widely used paradigms in all ERP investigations). It has been asso-
ciated with psychological constructs including orientation, attention, stimulus
evaluation and memory, and seems to reflect the selective detection and classi-
fication of unexpected, significant information in any modality (Dustman & Beck,
1967; Hrbek et al., 1973; Hansen & Hillyard, 1983; Karis et al., 1984; Mullis et
al., 1985). The proposal of oddball experimental paradigms suggests that the P3
wave is a reflection of the revision or updating of memory brought about by the
unexpected event (Donchin, 1981; Donchin et al., 1984).

The latency of AEP P3 component decreases progressively as a function of age
to reach adult values in about 20 years (Goodin et al., 1978; Ford et al.,1979a,
1979b, 1982; Pfefferbaum et al., 1979, 1980). These changes may show that the
speed (Howard & Polich, 1985; Bashore, 1990b) of information processing increas-
es with age with regard to reaction times (ETs) (Marsch, 1975; Courchesne, 1978;
Ford et al., 1982; Howard & Polish, 1985; Bashore, 1990a). Goodin et al. (1978)
found that the P3 latency in response to an infrequent target tone (a detected
oddball tone) occurring in a sequence of standard tones increased as a function
of advancing age at a rate of 1.8 msec/year, ranging from 15 to 76 year-old
adults (Fig. 1). This finding has been replicated, and it has demonstrated the
general trend toward increased latency and decreased amplitude with increasing
age (Beck at al.,1980; Syndulko et al., 1982; Brown et al.,1983; Pfefferbaum et

al., 1984; Picton et al., 1984, 1986). Some studies reported that in the re-
search of subjects between adolescence and 90 years of age, P3 latencies gradu-
ally increased for auditory and visual stimuli, respectively, and they have also
reported a slight difference between male and female subjects, with female sub-
jects having earlier peak latencies than male subjects, although others have not
found such a difference. Moreover, other investigators have extended their re-
search modalities to the visual and somatosensory, and found modality-specifica-
tions of maturation for P3 latency (Pfefferbaum et al., 1984; Picton et al.,
1984).

In summarizing the results from a variety of the developmental ERP literature,
Courchesne (1990) reviewed the developmental differences in children's auditory
and visual P3s, so that the AEP P3 latencies and amplitudes appeared to increase
or decrease abruptly from pre- to post adolescence, whereas the latency and am-
plitude of visual P3s seemed to decrease abruptly from childhood to adulthood.
He also observed that meaningless, novel pattern stimuli and meaningful visual
stimuli both evoke a frontally distributed negative late component (Nc) and pos-
itive component (Pc) that are apparently specific to infants and children
(Courchesne, 1978, 1983). The novel auditory events and sudden visual stimuli
elicit an anterior Nc-Pc complex in infants which occurs at progressively short-
er latencies and smaller amplitudes with increasing age, and it is afterwards
replaced by a centro-frontally distributed P3 wave in adults. The Pc is elicited
by meaningful visual stimuli, whereas the Nc provoked by novel, uninterpretable
events was considered an activity of attention-getting events for which there is
no immediately available interpretation (Courchesne, 1977, Courchesne et al.,
1981). However, further developmental research should be continued, since very
little data and information exists on the Nc and Pc waves.

The changes in P3 component as a function of age have been documented in stud-
ies of normal adult aging, whereas if compared to those seen in adult subjects,
maturational or developmental investigations of the P3 changes are minor in
studies of infants (e.g. Hofmann & Salapatek, 1978) and children (e.g. Fig. 1),
because developmental EP research is often hampered by muscle artifacts and lim-
ited in available control repertoires.

However, the large ERP P3 is elicited not only by an attended or task-relevant
(oddball condition) stimuli, but also by a non-assigned task, startle acoustic
stimuli (Roth et al., 1984; Putnam & Roth, 1990) with the peak latency of about
300 msec at the parieto-central and fronto-central scalp electrode distribution.
Moreover, the P3 is mainly composed of two (or more) different waves (Squires et
al., 1975), that is, the P3a and P3b. The P3b is a large, positive component
which is usually maximum at electrode sites over the parietal scalp with a la-
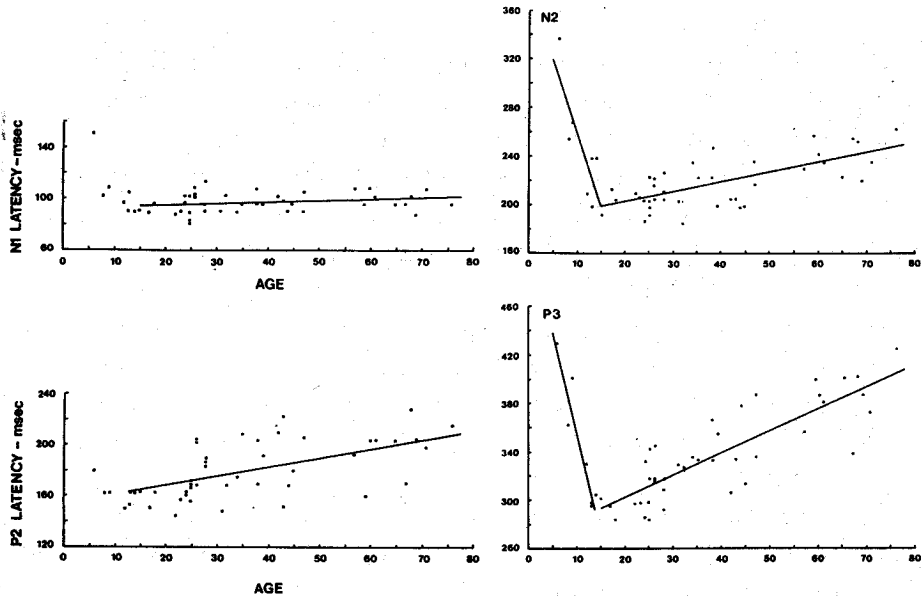tency between 310 and 400 msec, and is thought to reflect cognitive processes

Fig. 1

The developmental changes of AEP latencies of late components (reproduced from Goodin et al., 1978). They presented the aging ERP data from 6 to 76 year-old subjects. The adult P2 latency increased significantly at the rate of 0.7 msec/ year. The difference in slopes between the age-latency regression lines for N1 and P2 was significant. The N1-P2 amplitude decreased with age at a rate of 0.2 μV/year. The shortest N2-P3 latencies were from subjects in their late teens and early twenties. Latencies were longer for both the older and the younger subjects. A similarly significant increase in latency with age was found for N2, although the rate of change, 0.8 msec/year, was less than that for P3 (1.8 msec/ year, ranging from 15 to 76 year-old adults). The adult N2-P3 amplitudes also decreased with age at the rate of 0.2 μV/year. However, further analysis of the child population would be needed because there were only 8 children in the study.

involving revision or updating of the schema (Donchin et al., 1984). On the oth- er hand, the P3a is thought to reflect an automatic process (probably brainstem activity) that is elicited by acoustical deviations occurring infrequently in a sequence of identical sounds under involuntary control with a latency between 250 and 330 msec.

   The purpose of the present research is to provide data on the developmental and maturational changes of AEP P3a latencies and amplitudes and eyelid reflexes * elicited by startle acoustic stimuli in normal children (of ages 3-8 years) as compared to young adults. It is not yet known whether the P3a develops at the

same rate as the developmental course of P3b activity changes, because there was
no the systematic developmental investigation of the P3a components.

 * [Eye movements and eyelid reflexes provide much important information (Volk-
mann et al., 1980) on human perceptual and cognitive information processing.
However, when we investigate simultaneously the ERP and startle reflex, ocular
activities usually contamimate the EEG response recording (Corby & Kopell, 1972;
Girton & Kamiya, 1973; Verleger et al., 1982; Fortgens & De Bruin, 1983; Gasser
et al., 1985). Recently, it has been possibe to record and analyze simultaneous-
ly the ERP and eyelid response by some new method of off-line or on-line removal
of ocular artifact (Woestenburg et al., 1983; Gratton et al., 1983; Elbert et
al., 1985; Lutzenberger, 1985; Berg, 1986)] .

Method

Subjects

   Seventy-seven normal children (40 males and 37 females) whose ages ranged from
3 to 8 years, and 16 normal adults (7 males and 9 females) of ages 18-24 years
were screened for good health, normal development, and absence of any symptoms
suggesting neurological pathology (Table 1). None had any history of neurologi-
cal or middle, inner ear pathology.

Table 1. Numbers of subjects in each age group

	males	females	total
3 years	8	4	12
4 years	7	10	17
5 years	16	10	26
8 years	9	13	22
Adults	7	9	16
	47	46	93

Stimuli

   White noise burst tones (50 msec duration, 104 dB, SPL, zero rise-fall time)
were presented binaurally to the right and left ear through TDH-49 circumaural
earphones applied to the subject's head with a random schedule of inter-trial
interval (ITI) every 25-45 seconds, during which the subjects watched silent
color TV cartoons (for the children) or movies (for the adults), sitting in a
comfortable chair.

Procedure and Recording

Before beginning the experimental session, practical trials were carried out
to familiarize the subjects with the experimental situation, and were made as
enjoyable as possible. One week after the preliminary training session, each
subject returned for the second 20-min laboratory session. The subjects were
seated comfortably in a reclining chair in a sound-attenuated, electrically
shielded room, where they were instructed to watch the TV and to remain quiet
throughout the period of novel, unexpected sound stimulus presentation.

Electroencephalographic (EEG) potentials recorded monopolarly from midline
placements over central (Cz) and parietal (Pz) scalp electrode locations in the
international 10-20 system with linked mastoids (A1-A2) as reference using stan-
dard chlorided silver disc electrodes. Electrodes were also placed on the later-
al canthus and above the supercilium of the left eye in order to measure the am-
plitude and latency of the eyelid reflex. The Fpz was the earth electrode. Elec-
trode impedances were less than 5 kOM. The amplifiers were set to a high fre-
quency cut off of 100 Hz and a lowcut of 0.1 Hz. All mean peak latencies and am-
plitude measurements of AEP components were quantified with a baseline-to-peak
measurement by subtracting the average activity in the 100 msec prestimulus
baseline from the amplitude of the peak in the search epoch with an analysis
time of 1.2 sec (giving a resolution of 1 msec/point). EEG responses and AEP P3
waves contaminated by large electrooculographic (EOG) and electromyographic
(EMG) responses were rejected from the analyzed data. All single-trial and aver-
age data were recorded on a floppy disk for further analysis. The statistical
significance of the AEP differences of the amplitude and latency values among
different age groups was evaluated by the repeated measurement analysis of vari-
ance (ANOVA).

Results

In this study, AEPs to 104 dB (SPL) clicks were compared among 77 normal chil-
dren (40 males, 37 females) and 16 normal adult (7 males, 9 females) subjects in
five groups. Amplitudes and latencies of AEPs for binaurally delivered stimula-
tion were calculated in each subject group. Three prominant components were
recorded at vertex (Cz) and parietal (Pz) in the areas over the auditory cortex.
The vertex components were a 110 msec negative peak (N1), a 180 msec positive
peak (P2), a 240 msec negative peak (N2), and a 300 msec positive peak, usually
called P3a. The P3a waves at Cz, which were identifiable in all the subjects,
were treated here as a major ERP component, because the P3a waves were designat-

ed as the vertex potentials of maximum positive peak measured from base-line to
peak at Cz after the acoustic stimulus presentation.

The typical AEP P3a wave-forms of a younger child and an adult subject elicit-
ed by the infrequent-startle auditory stimuli are shown in Fig. 2. The means and
standard deviations of the amplitude and latency measurements for P3a are pre-
sented for the subjects at ages 3, 4, 5, 8 and 18-24 in Table 2 and Fig. 3. As
can be seen in Table 2 and Fig. 3, the younger children displayed significantly
longer latencies of the P3a to novel-startle stimuli, as compared to the elder
subjects (3 year-old subjects: n=12, 327.8±29.7msec; adults: n=16, 278.1±29.9
msec).

It was not shown that P3a latency variability was greatest in the youngest
subjects and steadily decreased with age. On the other hand, there were apparent
amplitude differences of the P3a components elicited by the click stimuli among
each developmental group. The clearest evidence was the longest latencies of P3a
components in the 3 year-old group, when compared to the P3a latencies of the 8
year-old and adult subjects. The adult subjects displayed amplitudes of the P3a
components which were lower more than the children's amplitudes, although the
amplitude differences among each children's group were relatively very few and
did not reach statistical significance for large deviations. The AEP data were
statistically analyzed using the ANOVA among each group. The result showed that
the P3a latencies decreased progressively as a function of age, but the P3a am-
plitudes changed as an inverse U-shape function of age.

The developmental eyelid reflexes showed clear evidence that the EOG peak la-
tencies and amplitudes decreased progressively as a function of age (Table 3.
and Fig. 4). The developmental EOGs and EMGs elicited by startle acoustic stim-
uli did not show the same developmental courses. It suggests that these activi-
ties are organized by apparetly different neural systems (auther's colleagues
will present and discuss in detail the developmental changes of EOG and EMG ac-
tivities else where, and is in preparation).

Table 2

The means and standard deviations of P3a latencies and amplitudes for binaural
acoustic stimulation in different-aged children and adult subjects.

	Latencies (msec)*	Amplitudes (μV)*
3 years	327.8(±29.7)	51.2(±39.3)
4 years	310.6(±27.8)	72.7(±31.9)
5 years	303.1(±19.9)	73.9(±60.7)

| 8 years | 291.9($\pm$20.2) | | 47.0($\pm$36.1) |
| 18-24 adults | 278.1($\pm$29.9) | | 23.4($\pm$24.7) |

* : significant P< .01
ANOVA (analysis of variance)

Table 3

The means and standard deviations of the latencies and amplitudes of eyelid reflex (EOG) for binaural acoustic stimulation in different-aged children and adult subjects.

	Latencies (msec)*	Amplitudes (μV)**
3 years	128.0($\pm$12.8)	
4 years	121.9($\pm$ 5.8)	89.8($\pm$34.0)
5 years	119.5($\pm$10.2)	89.2($\pm$42.3)
8 years	112.5($\pm$ 5.3)	73.0($\pm$33.3)
18-24 adults	108.1($\pm$30.8)	62.4($\pm$23.2)

* : significant P< .01   ** : significant P< .05
ANOVA (analysis of variance)
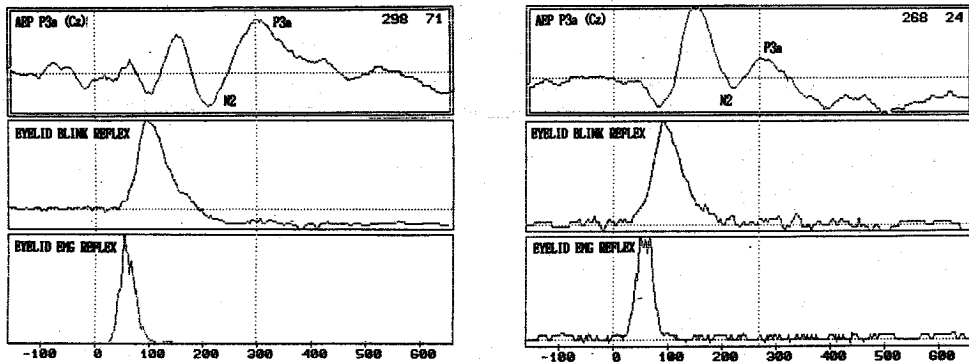
Fig. 2
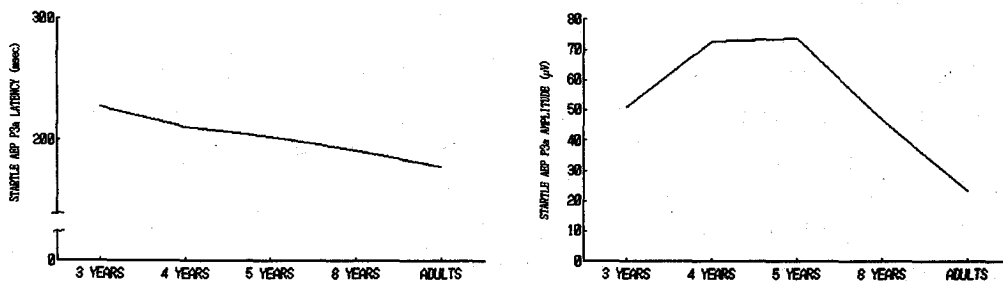AEP wave forms at Cz and eyelid reflexes elicited by startle acoustic stimuli.

Fig. 3

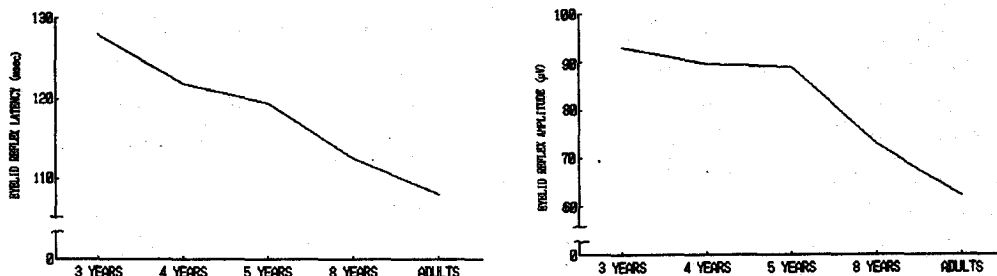The mean P3a latencies and amplitudes for each age group.

Fig. 4

The mean latencies and amplitudes of eye blink reflexes for binaural acoustic
stimulation in children and adult subjects of different ages.

Discussion

The latency of the AEP P3 wave in young children is longer than in adults, and
decreases progressively as a function of age, reaching adult values by late ado-
lescence or the early twenties. On the other hand, some researchers have report-
ed a decrease in amplitude of the P3 wave with increasing age, but others have
found that the amplitude increases or remains stable (Beck et al., 1980; Pfef-
ferbaum et al., 1979, 1980, 1984; Ford et al., 1979a, 1979b, 1982). The fronto-
central P3a and parieto-central P3b not only have different scalp latency and
amplitude distributions, but also might reflect different developmental courses
and modes of information processing within each auditory, visual and somatosen-
sory modality. Recent studies have shown clear evidence that P3bs are indeed

modality-specific, having different scalp distributions among auditory, visual and somatosensory modalities, such as the P3b of VEP, which shows a more central -frontal distribution than AEP P3b.

Unfortunately, the variability (Ellingson et al., 1974; Polish, 1986) unrelated to age is generally greater than the variability related to age within any given age range (especially in amplitudes; see Table 2), although the P3 latency consistently decreases with increasing age. We do not yet have a clear understanding of the neural generator of the P3 wave, and the persuasive reason for the P3 latency change with age remains unknown.

However, this effect of prolonged latency and small amplitude in infantile subjects might be explained by assuming that the P3b is only generated in older subjects after a revision or updating of information provided by the sensory evaluation. The distribution change of the P3 wave with age becomes mostly frontal in scalp distribution, which may be related more to sensory processing than to cognitive processing in younger children. Another possibility is that some smaller amplitudes in computer-averaged waveforms may have been caused by an increased variability in the P3 latency with increasing age. If the peak latency variability is corrected before averaging, the P3 amplitudes might be enhanced more than at the averaging point of 100 msec prestimulus onset. However, Table 2 shows the variabilities of P3a for each age group, and it did not support the possibility that the small amplitudes were caused by an increased variability of peak latency. The P3a is elicited more by acoustical deviations occurring infrequently in a sequence of identical sounds, than by target or counting task stimulations. It has the largest amplitude at sites with a latency range of 250-330 msec, which partially overlaps with the P3b latency. The P3 component of ERPs is generated in humans and other mammalian species when attention is drawn to infrequent stimuli. Auditory P3s are elicited by target (P3b) and nontask, unexpected novel stimuli (P3a) without instruction to subjects in monaural and dichotic signal detection experiments. In younger subjects, the parietal P3b elicited by the targets would overlap with the vertex-dominant P3a wave, and the P3 would come to indicate the increased temporal separation of the P3a and P3b subcomponents at the vertex and parietal lobe with increasing age (Smith et al., 1980; Picton et at., 1984, 1986). The decrease of parieto-central P3 amplitude might have resulted from the increased temporal separation of the fronto-central P3a and parieto-central P3b subcomponents due to aging, and this latency would reachthe adult latency level by adolescence. This decrease in latency may parallel the development of information processing speed and memory span.

Courchesne (1977, 1978) and Friedman et al. (1984) reported the absence of any age-related changes in either latency or amplitude for the N1 or P2 in children.

The N2 does not generally affect on task, and is predominantly larger over the
right hemisphere. It would be expected that the P3a component has different de-
velopmental time tables as compared with the P3b elicited by an oddball paradi-
gm. It reflects the activity of an intracranial source, and overlaps with the
P3b which appears to depend on the stimulus modalities. It is a question whether
the AEP potentials being studied, which were elicited by startle acoustic stim-
uli in these children and adults, are the same as those that have been observed
in classical P3s. The answer to this question is apparently clear; that is, the
previously reported P3s or P3bs were mixed with the P3a. The P3 may be an index
of context-updating operations in the working memory, but it also indicates that
affective-motivational factors affected by ascending reticular activation from
the brain-stem may contribute to the ERP P3 amplitude changes.

Recently, there is some evidence in the literature that the hippocampal forma-
tion and amigdala in medial temporal lobes or the auditory association cortex in
the human temporal-parietal junction may be involved in the P3 generation (Hal-
gren et al., 1980; Okada et al., 1983; Wood et al., 1984). But other regions of
the brain may also contribute to the scalp recording. In paticular, the P3a,
most of which overlaps with the P3b, may be generated or affected by the brain-
stem activity of neural "bottom-up" circuits, because the developmental P3a
changes in parallel with the developmental startle response (see Table 3, Fig.
3) which is generated in the brainstem. It would be expected that in infantile
subjects, startle modulation mediated by polysynaptic brainstem mechanisms is
not yet matured in a lower level. The results in infantile individuals show that
the increased latencies of P3a might suggest that they may have a limited capac-
ity not only to process novel information which takes place at higher levels,
but also in orienting responses in the central nervous system.

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