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Enhanced mismatch negativity in adolescents with posttraumatic stress disorder (PTSD)

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ABSTRACT

The mismatch negativity (MMN) is observed following rare or unique sensory events, and reflects pre-attentional sensory processing of unexpected stimuli. The MMN is altered in several mental illnesses, including post-traumatic stress disorder (PTSD), but did not yield consistent results. We measured MMN in 27 survivors of the Wenchuan earthquake, including 13 who were diagnosed with PTSD, to determine if pre-attentive processing in the auditory cortex was altered by this disease. The amplitude of MMN was significantly greater in the PTSD group compared to the control group. In contrast, no significant group difference was found in the N1 potential, an event-related potential that reflects cortical transmission of sensory information. These results demonstrated an increased sensitivity to deviant stimuli in PTSD that may reflect a chronic state of hyperarousal and hypervigilance in trauma victims.

1. Introduction

Mismatch negativity (MMN), a component of the event-related potential (ERP) evoked by rare changes or "deviants" aimed repetitive sensory stimuli, was first described over 30 years ago (Näätänen et al., 1978). The MMN is studied extensively in cognitive and clinical neuroscience because it is believed to reflect a pre-attentive cortical processing that is largely independent of conscious awareness or rehearsal. It can even be recorded in the absence of focus attention or specific task instruction, which makes it particularly suitable for clinical populations (Näätänen, 2003). For example, MMN has been used to study the information processing in patients with dyslexia (Schulte-Körne et al., 2001), schizophrenia (Baldeweg et al., 2004; Michie, 2001; Shelley et al., 1991) and depression (Kähkönen et al., 2005; Ogura et al., 1993).

MMN has been also used to explore the pre-attentive processing in patients with posttraumatic stress disorder (PTSD). Most individuals with PTSD are characterized by persistent reexperiencing of the traumatic event, persistent avoidance of stimuli associated with the trauma, and chronic hyperarousal (DSM-IV, American Psychiatric Association, 1994). Previous studies provided evidence for alterations in information processing in individuals with PTSD, but some results and conclusions have been contradictory. Morgan and Grillon (1999) found that the amplitude of the MMN, evoked in an auditory frequency discrimination task, was significantly greater in a PTSD group of sexual assault survivors than in a control group. It was concluded that there were abnormalities in the preconscious auditory sensory memory in PTSD subjects that manifested as increased sensitivity to stimulus changes. In contrast, a reduced MMN was demonstrated in PTSD patients when the discrimination involved sound duration or sound gaps, but not when the deviant stimuli differed in frequency (Menning et al., 2008). A reduction in pre-attentive auditory sensory memory in PTSD was proposed, possibly due to hyperarousal and impaired concentration.

The main difference between these two studies was the paradigm employed. Morgan and Grillon (1999) used the traditional paradigm (Näätänen et al., 1982) that employs just one kind of deviant stimulus imbedded in one kind of repeating standard stimulus. Menning et al. (2008) used an optimal paradigm, in which five kinds of deviants, varying in frequency, intensity, direction, gab and duration, could be manipulated within the same experiment (Näätänen et al., 2004). Both paradigms have limitations. The more complex optimal paradigm may engage additional or entirely different processing mechanisms. The utility of the traditional MMN paradigm has also been challenged because that refractoriness of organized cortical neurons was not excluded in this paradigm (Jacobsen and Schröger, 2001). Moreover, the effect of deviance on ERP characteristics in the traditional paradigm was mainly due to an effect on the N1 amplitude (Horvath et al., 2008). These conflicting data suggest two alternative explanations for MMN. The sensorial view states that MMN is caused by refractoriness of frequency-specific afferent cortical neurons, while the cognitive view implicates a mismatch between the sensory input of the deviant tone and a sensory memory trace for the frequently presented standard tone (Maess et al., 2007).

In order to exclude the effect of refractoriness and to measure the genuine memory comparison-based MMN, a controlled MMN paradigm

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was developed (Schröger and Wolff, 1996). In this paradigm, in addition to the oddball block, a control block with 10 tones, including standards and deviants, was presented additionally. There was a deviant-related negativity in the deviant-control comparison, which eliminated the influence of different probability. It was proposed that these results were best explained by the memory-comparison hypothesis. In later studies, the controlled MMN paradigm was used to explore this pre-attentive change-detection system widely (Jacobsen and Schröger, 2003; Maess et al., 2007; Horvath et al., 2008).

The goal of the current study was to explore how PTSD symptoms influence change detection processing in the auditory cortex as measured by MMN. The controlled MMN paradigm was used to investigate the pre-attentive processing of earthquake survivors. With this version of the task, we could compare the difference in the sensorial, non-comparator mechanism and the cognitive comparator mechanism between two groups. We predicted that brain potentials indicative of pre-attentive auditory processing differ in PTSD subjects.

2. Method

2.1. Participants

Volunteers were recruited from the Beichuan Vocational High School in Sichuan Province. They all experienced the tremendous earthquake in Wenchuan County of Sichuan Province, China on May 12, 2008, that measured 8.0 on the Richter scale and left about 69,000 people dead. These subjects were not injured and did not receive any psychiatric treatment or medication before this study. They completed the PTSD Checklist-Civilian Version (PCL-C) for PTSD screening (Andrykowski et al., 1998; Blanchard et al., 1996), and a PTSD group and a control group were selected using the results. Thirteen participants who had scores above 44 on the PCL-C and met the ICD-10 diagnostic criteria (International Classification of Diseases, World Health Organization, 1992) were designated as the PTSD group. For the control group, we chose a more conservative cutoff score, 14 participants who scored below 30 on the PCL-C were designated as control group. There were no significant differences in demographic features between groups (see Table 1). All subjects were right handed, had normal auditory acuity and normal or corrected-to-normal visual acuity. None of the subjects reported a history of neurological disease, psychiatric disorders, or substance use and none were taking medication or alcohol. They gave informed consent and were paid for their participation. Diagnostic interviews and ERP recordings were performed in June, 2009, about 13 months after the earthquake.

2.2. Clinical assessment

The diagnosis of PTSD for the earthquake survivors was determined by a clinical psychologist using the Chinese version of PCL-C (Weathers et al., 1993; Yang et al., 2007; Wu et al., 2008) and the ICD-10. The PCL-C is a self-report inventory for assessing PTSD, and it has also been used to gauge the 3 PTSD symptom clusters of re-experiencing, avoidance/numbing, and hyperarousal as defined in the Diagnostic and Statistical Manual of Mental Disorders, 4th Edition (DSM-IV). In addition, all participants completed the Chinese version of the Spielberger State/Trait Anxiety Inventory (STAI), a self-report assessment that evaluates state and trait anxiety (Spielberger et al., 1983; Zheng et al., 1993), and the Hopkins symptom checklist-25 (HSCL-25), a symptom inventory which measures symptoms of anxiety and depression (Derogatis et al., 1974).

Table 1
Subject characteristics in the PTSD and control groups.

	PTSD (n = 13)		Control (n = 14)		Group comparison	
	Mean	S.D.	Mean	S.D.		
Age (years)	16.15	0.98	15.93	0.83	$t = 0.64$	$p = 0.53$
Gender(male/female)	8/5	–	12/2	–	$\chi^2 = 1.78$	$p = 0.204$
Education (years)	8.23	0.44	8.35	0.49	$t = 0.69$	$p = 0.492$
STAT (state)	48.15	2.86	38.79	1.71	$t = 2.86$	$p = 0.008$
STAT (trait)	51.54	2.05	41.07	1.30	$t = 4.38$	$p = 0.00l$
PCL-C	52.62	2.11	24.50	0.95	$t = 12.43$	$p = 0.00l$
HSCL-25	61.46	2.85	35.00	1.38	$t = 8.55$	$p = 0.00l$

2.3. Stimuli

Ten sinusoidal tones of 600, 700, 800, 900, 1000, 1100, 1200, 1300, 1400, and 1500 Hz were generated by Cool Editor Pro V.2.0 (Syntrillium Software Corporation, USA). Tones were 75 ms long including 5 ms rise and 5 ms fade-out times and were adjusted to a comfortable listening level of ~70 dB SPL. In addition, a silent movie of nature scene was used.

2.4. Apparatus

Acoustical stimuli were administered by a SoundMAX Integrated Digital Audio board in an IBM R52 computer and delivered binaurally through air-headphones in Stim interface system (Neuroscan Labs, Sterling, VA). The movie was played on a 14.1 in. computer screen with a visual angle of approximately 8 degrees vertical and 13 degrees horizontal that was 60 cm from the viewer. The electroencephalogram (EEG) was recorded from 64 scalp sites using Ag/AgCl electrodes mounted in an elastic cap (NeuroScan Inc., USA).

2.5. Procedures

Participants were seated comfortably and instructed to watch a silent movie on the computer screen and to ignore the auditory stimuli. Tones were sequentially presented with a stimulus onset asynchrony of 500 ms. There were two experimental conditions. In the oddball condition, frequent "standards" (1000 Hz tones) were occasionally interrupted by infrequent "deviants" (1100 Hz tones). Deviants were presented among standards with a relative frequency of 0.1 in a pseudorandomized fashion with the constraint that two deviants could not be presented in direct succession. In the control condition, all of the 10 tones (600–1500 Hz) were presented with equal probability (relative frequency = 0.1) in a pseudorandomized sequence. Each condition was presented in two blocks; each block consisted of 600 trails. Block sequence was ABAB or BABA, which was counterbalanced across participants. An experimental session lasted approximately 20 min for data acquisition.

2.6. EEG recording

The EEG was continuously recorded from 64 scalp sites, with an electrode placed on the tip of nose as a common reference. The vertical and horizontal electrooculograms (VEOG and HEOG) were recorded from two pairs of electrodes, one pair placed above and below the left eye, and another pair placed at 10 mm from the outer canthi of each eye. All interelectrode impedance was maintained $<5\ k\Omega$. Signals were amplified with a 0.05–100 Hz bandpass filter and digitized at 500 Hz.

2.7. Data analysis

The EEG data were digitally filtered with a 30 Hz lowpass filter and were epoched into periods of 550 ms (including a 100 ms prestimulus baseline) time-locked to the onset of the presented sound. Ocular artifacts were removed from the EEG signal using a regression procedure implemented in the Neuroscan software (Semlitsch et al., 1986). Trials with various artifacts were rejected, with a criterion of $\pm 70\ \mu V$. The ERPs were then averaged separately for each experimental condition.

ERPs were derived for deviants, standards and controls. Deviants (D) were 1100 Hz tones (relative frequency of 0.1) in the oddball condition, which were embedded in a series of 1000 Hz tones. Standards (S) were 1000 Hz tones in the oddball condition (relative frequency of 0.9). Finally, controls (C) were 1100 Hz tones in the control condition in which tone from 600 to 1500 Hz was presented with equal probability. Grand-average ERPs were subsequently computed from the individual-subject average. Grand-average difference waves were computed by subtracting ERPs to control point by point from ERPs to deviants (DC) and standards from deviants (DS) respectively. For ERP quantification, individual N1 and MMN amplitudes were computed as the mean amplitudes in a uniform 20-ms window around the respective grand-average peak amplitudes. The ERPs and difference waves were measured in each condition at the following five sites: Fz, Cz, Pz, M1 and M2. MMN were measured and analyzed according to Jacobsen and Schröger (2003); Schröger (1998).

The ERP data were analyzed with SPSS 13.0 using analyses of variance (ANOVAs) with repeated measures. The between subject factor "Group" had two instances: "PTSD" and "control group". The two within subject factors were "location" (Fz, Cz, and Pz) and "condition" (D, S, and C). The MMN polarity inversion was analyzed in similar ANOVAs, except the "location" factor (M1, M2). The MMN of difference waveforms was assessed in an analysis of variance involving "group" (PTSD, control), "location" (Fz, Cz, and Pz) and "condition" (deviant-control, DC; deviant-standard, DS). Only significant results are reported. The Greenhouse–Geisser correction was used to compensate for sphericity violations. Post-hoc analyses were conducted to explore interaction effects, and Spearman rho correlations were calculated between the magnitude of the MMN at Fz and the STAI and PTSD-scores, where the MMN was the largest.

3. Results

3.1. Psychometric data

The Spielberger anxiety, PCL-C, and HSCL-25 scale scores were shown in Table 1. All scores were significantly higher in the PTSD group than in the control group, but there was no significant correlation between psychometric data and ERP data in the PTSD or control groups.

3.2. ERP data

ERPs and difference waves were presented in Fig. 1. The ANOVA (group × location × condition) was tested for N1 amplitude. No group effect was detected on N1 amplitude. The main effect of location on N1 mean amplitude was significant, $F(2, 50) = 10.384$, $p < 0.001$, however, as was the location × condition interaction, $F(2, 50) = 2.798$, $p < 0.05$. Accordingly, the condition effect was assessed at Fz, $F(2, 50) = 4.239$, $p < 0.05$. Planned paired comparisons showed that N1 to deviants was larger than N1 to standards, $F(1, 25) = 10.589$, $p < 0.01$. The peak latencies for N1, as defined by the time after stimulus onset, were 110 (standard, S), 112 (deviant, D), and 102 ms (control, C) for control group and 110 (S), 110 (D), and 102 ms (C) for PTSD group. No group difference was detected in latency.

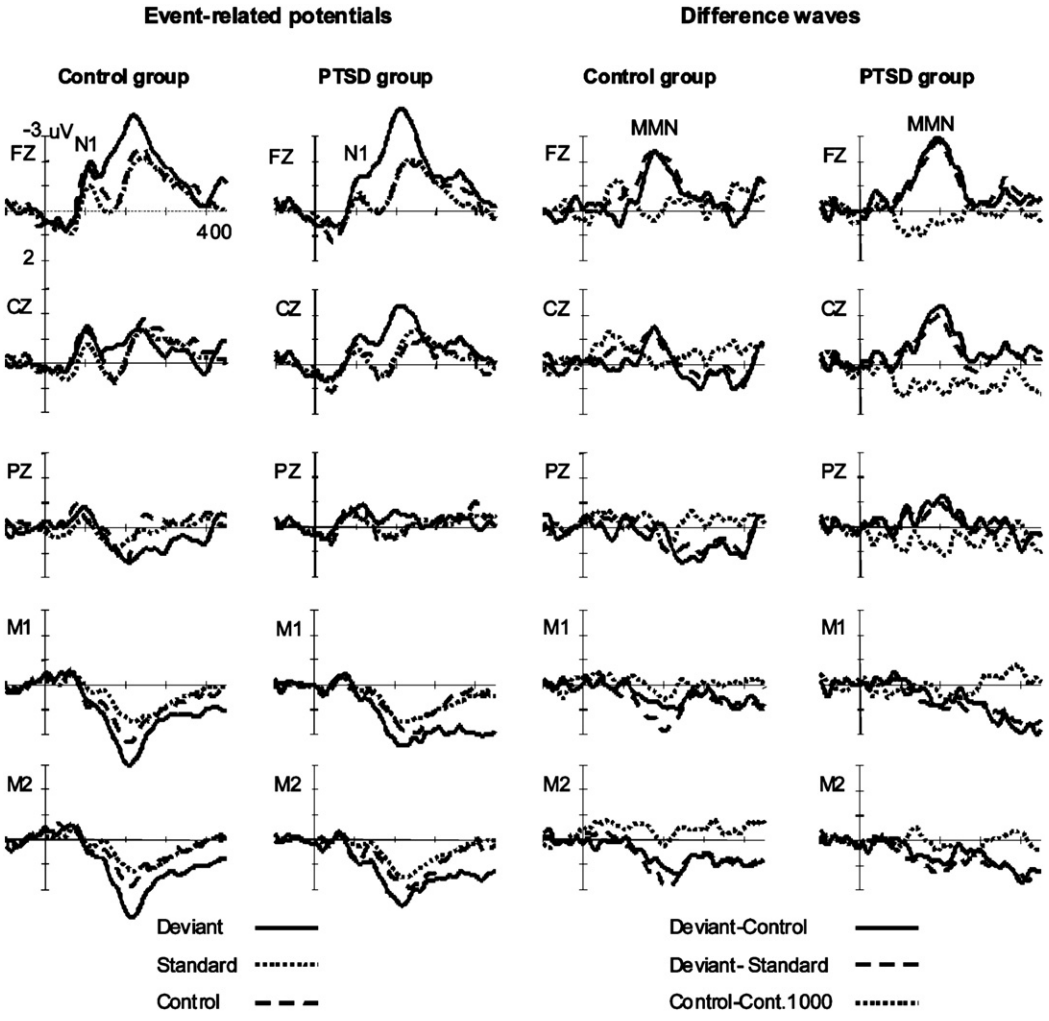

Fig. 1. ERPs and difference waves for two groups.

The ANOVA of MMN amplitudes yielded a main effect of group, $F(1, 25) = 4.862$, $p < 0.05$, with a larger average MMN amplitude in the PTSD group than in the control group. The main effect of location was also significant, $F(2, 50) = 24.188$, $p < 0.001$. Peak latencies for MMN were 178 ms (DC) and 180 ms (DS) after stimulus onset in the control group and 192 ms (DC) and 194 ms (DS) in the PTSD group. Additionally, the polarity of MMN inverted at the mastoids, $F(2, 50) = 10.038$, $p < 0.001$.

ANOVA with repeated measures was used to measure the interaction effect between group (PTSD and control) and ERP (ERP to deviant tone and ERP to control tone in the MMN time window at Fz). The main difference of ERP was significant, $F = 47.875$, $p < 0.001$. The amplitude of ERP to deviants was much greater than that to control. No significant interaction effect was detected. The amplitude of ERP to deviants in PTSD group was higher than that of control group, although it was not statistically significant. The N1 and MMN amplitude measures also were tested in a 2×2 ANOVA design (group by ERP component) to deviant tones at Fz. The main difference between N1 and MMN was significant, $F = 12.842$, $p < 0.001$. The MMN amplitude was much higher than that of N1, especially in PTSD group. No significant interaction effect was found.

4. Discussion

Patients with PTSD exhibited significantly increased MMN amplitudes, compared to control group, in both traditional MMN (DS) and controlled MMN (DC). In contrast, there were no significant group differences in the amplitude of N1, which was thought to reflect the transmission of sensory information to the cortex.

The present paradigm, which we believe to be more rigorous because it employs an additional control block (Jacobsen and Schröger, 2001; Horvath et al., 2008), was designed to disentangle non-comparator-based versus comparator-based contributions to MMN. These results confirmed that the enhanced MMN in the PTSD subjects was specific to the neural mechanisms sensitive to stimulus changes, because it was consistent with a previous study (Morgan and Grillon, 1999). It reflected alterations in cortical mechanisms that detect stimulus changes, rather than changes in sensory processing due to accommodation.

The controlled protocol for the MMN to frequency deviations usually yields a biphasic ERP pattern when difference waves are analyzed (N1 refractory effects as a relative positivity followed by the genuine MMN; Jacobsen and Schröger, 2001; Jacobsen et al., 2003; Maess et al., 2007; Horvath et al., 2008; etc.). The present data showed the monophasic data pattern of the traditional MMN method. Base on previous studies, the N1 refractory effect could be demonstrated by the ERP to the standard is different with the ERP to same stimulus from the control block. Unfortunately, in this study, no significant difference was detected between ERP to the standard and ERP to same stimulus from the control block, ERP to the deviant and ERP to the control. So no biphasic ERP pattern was showed when difference waves are analyzed. One possible reason for this result is the order of deviant in tonal succession set. In previous studies, the deviants came from the endpoints of the tonal succession, the standards being their closest neighbors in their studies. But in our study, the deviant came from the middle of the tonal succession. The different order of the deviant may cause different processing during the experiment. The underlying mechanism of it is similar to the serial order effect in memory, which was demonstrated in neuroscience by the amplitudes to probe items presented in the last position in the memory set was larger than that to probes presented in the middle position (Patterson et al., 1991). The other reason is maybe the difference came from participants. All participants in this study experienced the Wenchuan earthquake. The control group was also influenced by this disaster in some way. A comparable group who did not experience the earthquake is needed for further study and analysis.

Based on our results, the MMN amplitude was much higher than N1 amplitude, it implied a genuine MMN. The amplitude of ERP to deviant of PTSD was higher than that of control group. Although these results did not reach statistically significant, these trends also implied some mechanisms. Fortunately, the difference between two groups in grand-average ERP to control tone in the MMN time window was also not significant. We could infer that processing of control tone was not affected by PTSD symptom. The increased MMN was due to increased sensitivity to deviant tone in PTSD group. Deservedly, our inference should be proved more effectually in the future study.

The MMN is believed to reflect a pre-attentive change detection mechanism (Näätänen, 1992) that responds to the violation of a rule, established by a sequence of sensory stimuli. Indeed, the ability to detect unusual (and possibly dangerous) events amid monotony in the environment is a fundamental to survival. This cognitive comparator could be described as a cortical circuit that evaluates each successive stimulus against a memory trace encoding the repetitive stimulus. The enhanced MMN suggests that the sensitivity of this comparator is enhanced in PTSD patients. Post-traumatic stress disorder is a constellation of debilitation behavioral and emotional abnormalities, including the reexperiencing of the traumatic events (flash backs), the avoidance of stimuli associated with the trauma, persistent hyperarousal, inappropriately directed hypervigilance, insomnia, and an exaggerated startle response (DSM-IV; American Psychiatric Association, 1994). One explanation advanced for the increased MMN in PTSD group is that it reflects generalized chronic hyperarousal that sensitizes PTSD victims to any change in the environment (stimulus deviance). Significantly, this trait extends to automatic (preconscious) reactions as well as sensory events that are consciously attended to. Furthermore, MMN can be enhanced in healthy subjects under stressful conditions. Cornwell et al. (2007) found that the right auditory cortical regions of healthy subjects under threat of shock showed greater activity in response to stimulus deviance than did control subjects. These findings illustrated that sensitization to deviant stimuli can be influenced by anticipatory anxiety.

Our results add to a growing body of work showing changes in various ERP signal components in PTSD (Karl et al., 2006). For example, a greater P2–N2 ERP gradient has been reported in PTSD subjects (McPherson et al., 1997). The P300 response in a PTSD group was either reduced or augmented, depending on the pattern of symptom presentation and the relative involvement of different neurotransmitter systems (McFarlane and Weber, 1993). Still others have shown that P300 amplitudes, elicited by distracters in oddball tasks, were increased in PTSD patients but not in healthy controls (Kimble et al., 2000). In addition to effects on early sensory processing functions, PTSD subjects also exhibited deficits in attention and working memory, as well as difficulty encoding information and inhibiting distracting stimuli (Vasterling et al., 2002). An increased MMN, like increased P300 elicited by distracters in oddball tasks, reflects an enhanced response to unattended distracters.

Our results differ, however, from those previously reported by Menning et al. (2008). While impaired processing of "time-related" information in PTSD subjects was observed in their study, MMN amplitudes for frequency mismatch were not significantly different. The mechanisms for processing of temporal characteristics (duration, gaps, etc.) are different from those that evaluate and compare tone (frequency) information. For example, evidence from fMRI studies demonstrated that the neural generators underlying the auditory pre-attentive change-detection system varied as a function of the eliciting stimuli (Molholm et al., 2005), with frequency deviation resulting in a different pattern of MMN-related activation in auditory cortex that duration deviation. Furthermore, attentional effects on the MMN amplitude depended on the type of stimulus deviation (Muller-Gass et al., 2005; Näätänen, 1991; Näätänen et al., 1993), with the intensity-dependent MMN much more sensitive than the frequency-dependent MMN to the withdrawal of focused attention (Woldorff et al., 1991). Clearly, a systematic analysis of how MMN is altered by dimension, disease etiology, distracter type, and subtle changes to the stimulation paradigm is needed to resolve these issues.

Certain limitations in our study should be noted. Firstly, the control group was selected based on the score of a report scale (the PCL-C), not by professional diagnostic interviews. Secondly, just a single feature deviant was used in our study, and others have shown that MMN is highly dependent on both the sensory dimension, type of change, and level of distraction. Finally, the age of all subjects was under 18 and all subjects were earthquake survivors. Further studies are needed to determine whether these findings can be generalized to adults and to other groups that experience different sources of trauma.

In conclusion, PTSD subjects showed an increased sensitivity to unattended changes (deviants) in pre-attentive auditory processing compared to trauma-exposed, non-PTSD subjects, although a biphasic ERP pattern was not found in our study. The augmented amplitude of MMN suggests that PTSD patients are hypersensitive to sensory changes, possibility due to chronic cortical hyperarousal. These results, however, should be replicated using other auditory deviant, with a larger sample and across varied trauma populations before reaching definite conclusions.

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