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Effects of Superior Colliculus Inhibition on Three-Dimensional Visual Motion Processing in the Lateral Suprasylvian Visual Area of the Cat

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Purpose: To determine whether visual inputs from the tectothalamocortical pathway influence three-dimensional motion processing within the lateral suprasylvian (LS) area of the cat.

Methods: Tungsten microelectrodes were used for recording visual-evoked potentials (VEPs) from the LS area of 4 cats. Random dot stereograms were used as visual stimuli. Three-dimensional, motion-triggered VEPs were recorded from the LS area. Each motion sequence consisted of an abrupt onset of motion disparity with a 2° amplitude followed by an abrupt offset and a stationary phase of 900 ms. The velocity of the motion disparity was varied in eight steps from 10° to 400° per second. The onset of motion disparity was used as the trigger for recording the VEPs. Single or multiple injections (two to three) of muscimol were made mainly into the rostral superior colliculus (SC). The amplitudes of the VEPs before and after the muscimol injection were compared.

Results: A large negative wave (N1) with an implicit time of 92.7 ± 13.5 ms (mean ± SD, n = 98) was recorded consistently. The amplitude of N1 was significantly larger on stereovision of motion disparity than on either binocular vision of two-dimensional lateral motion or monocular vision, indicating that N1 contains neurons sensitive to motion disparity. The amplitude of N1 was not altered by muscimol injection into the SC at velocities ≤50°/s. On the other hand, the amplitude of N1 was reduced to 66–71% of that observed before muscimol injection at velocities ≥75°/s.

Conclusions: These findings suggest that the LS area processes three-dimensional motion inputs via two parallel pathways, the geniculostriate pathway and the tectothalamocortical pathway, at velocities of motion disparity ≥75°/s, while the three-dimensional motion inputs project to the LS area only via the geniculostriate pathway at velocities of motion disparity ≤ 50°/s. **Jpn J Ophthalmol 2001;45:475–481**

Key Words: Lateral suprasylvian area, motion disparity, superior colliculus, visual-evoked potential.

Introduction

Recent neurophysiological studies in monkeys and humans suggest that two general information-processing streams exist in the visual cortex.[1–5] The first stream is considered to subserve form and color vision, to lie ventrally and to terminate in the temporal lobe (temporal stream). The other stream is considered to be specialized for visual motion, to lie dorsally and to terminate in the parietal cortex (parietal stream). The middle temporal (MT) area in the monkey is assumed to be an important neural substrate for visual motion perception in the cerebral cortex.[1–6] In the cat, the lateral suprasylvian (LS) visual area is the region suggested to be functionally analogous to the MT area of the monkey.[1,7–10]

The LS area receives visual inputs from the geniculostriate pathway and from the extrageniculate system via the tectothalamocortical pathway.[11–18] The tectothalamocortical pathway consists of projections from the superior colliculus (SC) to the LS area through the pulvinar and the lateral posterior nucleus of the thalamus.[11–20] Recently, we reported that the LS area processes two-dimensional (2D) motion

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inputs via the tectothalamocortical pathway and the geniculostriate pathway at velocities >75°/s.[21] The LS area, especially the postero-medial lateral suprasylvian cortex (PMLS), is also involved in three-dimensional (3D) motion processing, and it is probable that the LS area receives 3D motion signals via the geniculostriate pathway.[22] However, whether the tectothalamocortical pathway conveys 3D motion signals to the LS area has not been determined.

In this study, using muscimol injections, we investigated the effects of SC inhibition on the amplitudes of the 3D motion-triggered visual evoked potentials (3Dm-VEPs) within the LS area. We attempted to clarify whether the visual inputs from the tectothalamocortical pathway to the LS area influence 3D motion processing in the LS area by investigating the effects SC inhibition on the amplitude of 3Dm-VEPs.

Materials and Methods

Surgical Preparations

This study was conducted on 4 cats weighing 2.5–3.5 kg. Each cat was deeply anesthetized with 2–4% halothane. After the trachea and saphenous veins were cannulated, halothane anesthesia was replaced by ketamine hydrochloride (initial dose, 25 mg/kg, intramuscular [IM]) and α-chloralose (25 mg/kg, intravenous [IV]). The animal was immobilized with pancuronium bromide (initial dose, 0.1 mg/kg, IV) and artificially ventilated. Thereafter, pancuronium bromide (0.05 mg/kg, IV) was administered every 60 minutes.

The head of the animal was placed in a stereotaxic head-holder frame. Two small holes were drilled in the parietal skull for the later insertion of microelectrodes and glass micropipettes into the LS area and the SC. All incisions and pressure points were infiltrated with 2% lidocaine hydrochloride. Rectal temperature was maintained at 37.5°C using a feedback-controlled heating pad. During the experiment, supplemental doses of ketamine hydrochloride (15 mg/kg, IM) and α-chloralose (10 mg/kg, IV) were administered every 30 minutes. All experimental protocols were approved by the Sapporo Medical University Animal Care and Use Committee and complied with the ARVO Statement for the Use of Animals in Ophthalmic and Vision Research.

Visual Stimuli

The pupils were dilated with topical phenylephrine hydrochloride. In order to determine the relative position of the area centrales, retinal landmarks, such as the optic discs and some major blood vessels, were projected onto a tangent screen placed 57 cm in front of the animal using the tapetal reflection method.[23] The visual axes of the two eyes were optically adjusted to converge at the center of the tangent screen. The adjustment of the visual axes was performed at least several hours after the pancuronium bromide application. Contact lenses were placed on both eyes so that the eyes were focused on the screen.

Random dot stereograms (0.5° × 0.5× square, 20 cd/m^2, 99.8% contrast, and stimulus size, 15° × 15°) and a background random dot pattern (40° × 40°) were projected onto the tangent screen (Figure 1). The background random dot pattern and the random dot stereograms for the right and left eyes were projected using three different slide projectors. The stereograms for the right and left eyes were moved rightward and leftward using two mirrors attached to two galvanometers whose movements were controlled by a microcomputer. To induce stereovision, each eye was stimulated independently using polarizing filters. Each motion sequence consisted of an abrupt onset of approaching/receding motion from 0° disparity with 2° amplitude followed by an abrupt offset and a stationary phase of 900 milliseconds (Figure 1). The velocity of the motion disparity was varied in eight steps (10, 20, 50, 75, 100, 150, 200, and 400°/s).

Recording and Muscimol Injection

Tungsten microelectrodes, insulated with Isonel 31 (Nisshoku, Osaka), were used for recording the visual evoked potentials. The electrodes were introduced into the medial bank of the LS area at an angle of 30° to 35° from the vertical axis in the coronal plane and were positioned at stereotaxic coordinates A0 to A1, which is the area corresponding to the PMLS.[24] Activities from single neurons were initially recorded, and the receptive field and directional selectivity of each neuron were determined. In each cat, a neuron that was selective for motion disparity was found, and the tungsten electrode for recording the VEPs was positioned at this point. VEPs were then recorded. The onset of motion was used as the trigger for recording 3Dm-VEPs. Potentials evoked by either approaching or receding motion were averaged depending on the motion selectivity of the neuron in each cat. After amplification and with a band-pass filter of 0.5 to 100 Hz, 128 epochs of 1-second duration were averaged and digitized with a computer at a sampling rate of 10 kHz.

The concentration of γ-aminobutyric acid (GABA) is high in the SC, and neurons are inhibited by muscimol (GABA agonist).[25,26] In the previous study, we demonstrated that the tectothalamocortical pathway was blocked by muscimol injections into the SC.[21]

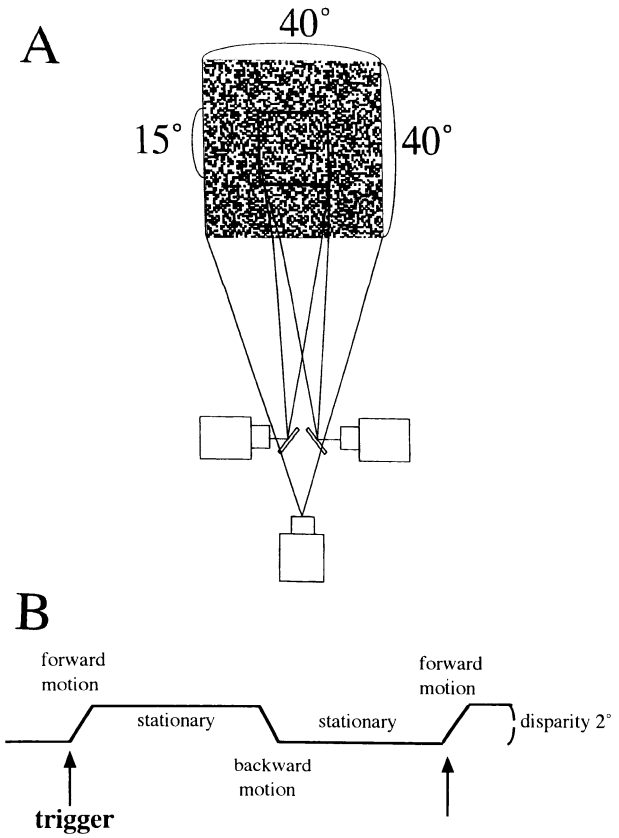

Figure 1. (**A**) Schematic diagram of system for recording three-dimensional (3D) motion-triggered visual-evoked potentials (VEPs). (**B**) Random dot stereograms (0.5° × 0.5° square, 20 cd/m^2, 99.8% contrast, 15° × 15° stimulus size) and background random dot pattern (40° × 40°) were projected onto tangent screen positioned 57 cm in front of animal. Electrodes were introduced into lateral suprasylvian area at angle of 30° to 35° from vertical axis in coronal plane, and positioned at stereotaxic coordinates of A0 to A1. Saline solution of muscimol was injected stereotaxically into the superior colliculus along the vertical axis on same side as recording side of VEPs. (**B**) Visual stimulus paradigm. Each motion sequence consisted of abrupt onset of motion in depth whose amplitude was 2° followed by abrupt offset and stationary phase of 900 milliseconds. Velocity of motion disparity was varied in eight steps (10, 20, 50, 75, 100, 150, 200, and 400°/s). Duty cycle (motion phase/stationary phase) was 10%, irrespective of velocity of motion disparity. The onset of motion was used as trigger for recording 3D motion-triggered visual-evoked potentials.

Glass micropipettes, filled with 1 μg/μL of muscimol (Sigma, St. Louis, MO, USA) in saline, were introduced stereotaxically into the SC along the vertical axis. The projection from the SC to the LS area is exclusively ipsilateral.[27–29] However, the LS area also receives projections from the contralateral LS area.[18] Therefore, the SC on both sides were inhibited by muscimol. The injection sites were stained with fast green for later identification. Before muscimol injections, the receptive field was recorded at the injection site using glass micropipettes. Single or multiple injections (2–3) of muscimol, spaced 0.25–0.5 mm apart, were performed within the region of the SC that corresponds to the area of the representation of the visual field compatible with the receptive field location at the recording site. The total amount of muscimol injected ranged from 0.3 to 0.6 μL. In the controls, 0.3–0.6 μL of saline was injected into the SC to demonstrate the lack of an effect.

Data Analysis and Histological Processing

We evaluated the amplitudes and implicit times of the positive and negative peaks of the VEPs for each stimulus velocity before and after muscimol injection into the SC. Evoked potentials were recorded 6–8 times for each stimulus velocity in each animal. The amplitudes of the VEPs before and after muscimol injection were compared by two-way repeated-measures analysis of variance.

After the experiments, the animals were deeply anesthetized with pentobarbital sodium and perfused transcardially. Two liters of physiological saline were infused, followed by 2 L of fixative solution containing 10% paraformaldehyde and 1% glutaraldehyde in 0.1 M phosphate buffer (pH 7.4). Following perfusion, the brains were exposed, blocked in the stereotaxic plane, placed in 0.1 M phosphate buffer containing 30% sucrose, and kept in a refrigerator overnight. The brains were then sectioned into 100-μm serial coronal sections using a freezing microtome and collected in compartmentalized trays. The sections were then mounted on gelatin-coated slides and stained with neutral red.

Each section was examined by light microscopy using both low- and high-magnification under bright-field illumination. The distribution of the injection sites of muscimol was plotted on sheets of paper with the aid of a drawing tube attached to the microscope.

Results

Single neuronal activities were initially recorded, and activity from a single neuron that was associated with motion disparity was identified in each cat. The receptive fields of four neurons in the 4 cats were located in the lower quadrant of the visual field on the side contralateral to the recording side with sizes ranging from 15° to 30° in width (Figure 2). Visual-evoked potentials were recorded from the point

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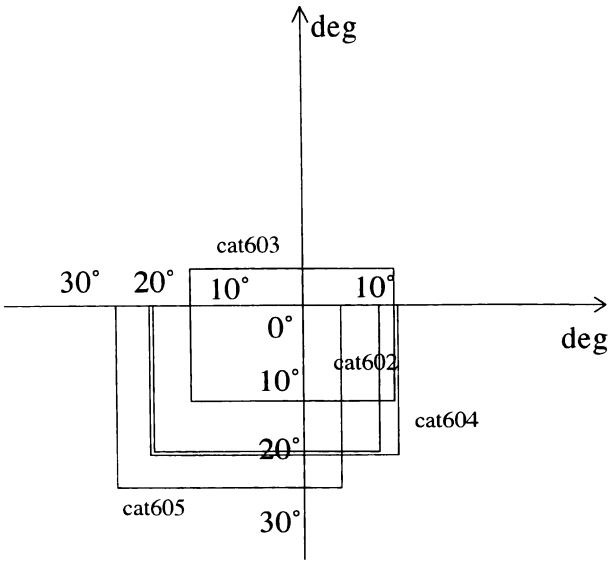

Figure 2. Receptive fields of four neurons at recording sites in 4 cats. Receptive fields were located on lower quadrant of visual field on side contralateral to recording side, with sizes ranging from 15° to 30° in width.

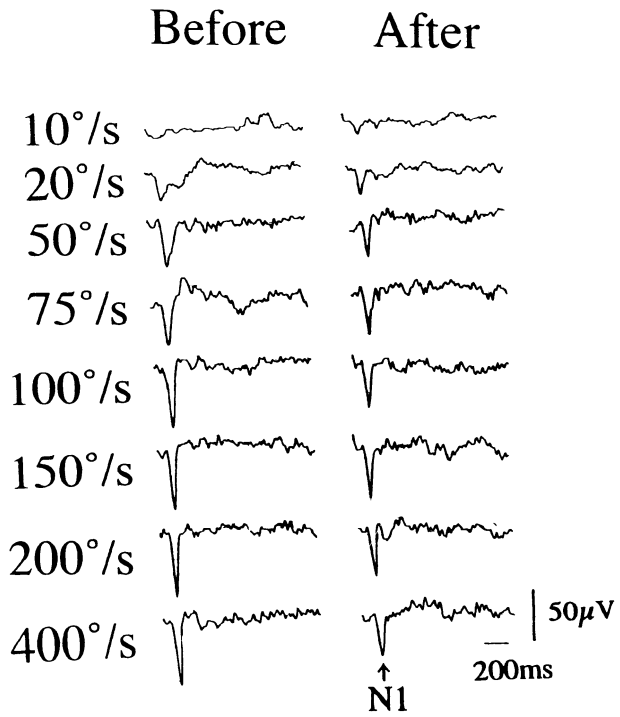

Figure 3. Examples of motion-triggered visual-evoked potentials for various stimulus velocities in 1 cat (cat 203) before and after muscimol injection. Large negative wave was recorded consistently and was designated as N1 (arrow).

where the single neuronal activity was recorded in each cat. The pattern of the VEPs recorded was essentially the same for the 4 cats and was dependent on the velocity of motion disparity. Figure 3 shows examples of 3Dm-VEPs for various stimulus velocities in 1 cat. A large negative wave was recorded consistently for stimuli of approaching motion but not for that of receding motion. This wave was designated as N1.

In the following experiments, the parameters of N1 evoked by approaching motion were analyzed. The mean implicit time of N1 was 92.7 ± 13.5 ms (mean ± SD, n = 98).

Figure 4 shows the relationship between the velocity of motion disparity and the mean amplitude of N1 in the 4 cats for each stimulus velocity under three visual conditions: stereovision, binocular vision of 2D lateral motion, and monocular vision. The amplitude of N1 changed with the velocity of motion disparity; it increased as the velocity of motion disparity increased up to velocities of 200°/s, but was fairly constant at velocities >200°/s. The amplitude of N1 was significantly larger under conditions of stereoscopic vision than under either binocular vision of 2D lateral motion or monocular vision. These characteristics of N1 were consistent for all the trials for the four cats. The amplitude under conditions of binocular vision of 2D lateral motion decreased to 65.9–83.2% of that under conditions of stereovision.

Muscimol (1 μg/μL saline solution) was injected stereotaxically into the SC, and the injection sites in the 4 cats are shown in Figure 5. For all the cats, muscimol was injected into the SC at stereotaxic coordinates Al–A3, and the effects of muscimol on the amplitude of N1 was essentially the same. Figure 3B shows examples of VEPs after muscimol injection. The mean implicit time of N1 after muscimol injection was 89.15 ± 23.26 ms (mean ± SD, n = 183) which was not significantly different from the value before the muscimol injection. The waveforms were also similar before and after the injection. The mean amplitudes of N1 in the 4 cats for each stimulus velocity before and after muscimol injection are shown in Figure 6. The amplitude was significantly reduced following the muscimol injection to 66–71% of that before the injection for velocities ≥75°/s. On the other hand, the amplitude of N1 remained unchanged following muscimol injection at velocities ≤50°/s.

Discussion

The amplitude of N1 was significantly larger under conditions of stereovision than under those of binocular vision of 2D lateral motion or monocular vision

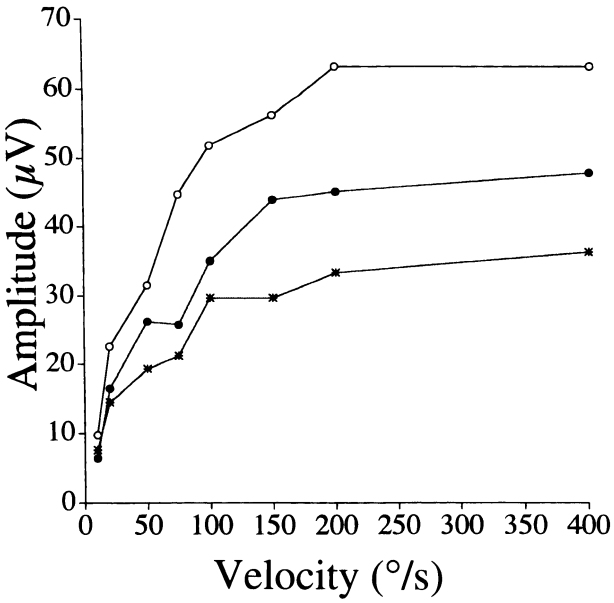

Figure 4. Relationship between velocity of motion disparity and mean amplitude of large negative wave in 4 cats under conditions of stereovision (○), binocular vision of lateral motion (•) and monocular vision (*) for each stimulus velocity. Error bars represent standard deviations.

(Figure 4). These findings indicate that N1 represents neuronal activities related to 3D motion processing in the LS area, and that 3D motion processing in the LS area is independent of visual inputs from the tectothalamocortical pathway at relatively slow velocities <50°/s (Figure 6). On the other hand, the visual inputs from the tectothalamocortical pathway to the LS area contribute partially to 3D-motion processing at velocities ≥75°/s (Figure 6). In our study, N1 was observed consistently for stimuli of approaching movement but not for that of receding movements. Neurons in the PMLS have also been reported to respond preferentially to approaching motion,[22] and our results are compatible with the neuronal responses in the PMLS.

Previous studies have shown that disparity-sensitive cells are found in the superficial layers of the SC.[30,31] Among the binocular cells in the SC, 65% were found to be sensitive to spatial disparities. Disparity-sensitive cells were prominent in the SC as well as in the PMLS. These cells may project visual inputs to the LS area via the tectothalamocortical pathway.[11–18] Our previous study also demonstrated that visual inputs via the tectothalamocortical pathway to the LS area contribute partially to 2D motion processing at velocities ≥75°/s. Toyama et al[22] reported that many neurons in the LS area, particularly those in the PMLS, respond preferentially to 2D motion, 3D motion, or both, and that neurons in the PMLS had broad velocity tuning. The tectothalamocortical pathway contributes to the broad velocity tuning of PMLS neurons by conveying high-velocity motion signals to the PMLS for both 2D and 3D motion processing, although the geniculostriate pathway is the main pathway for conveying visual motion signals.

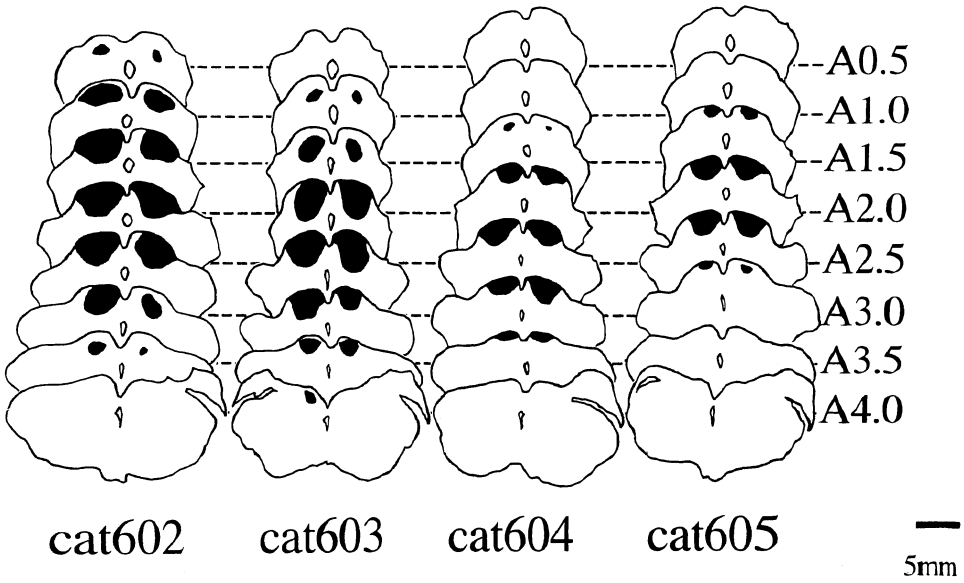

Figure 5. Drawings of serial coronal sections through superior colliculus in stereotaxic plane showing locations of muscimol injections in 4 cats. Solid black region represents extent of fast green staining. Bar = 5 mm.

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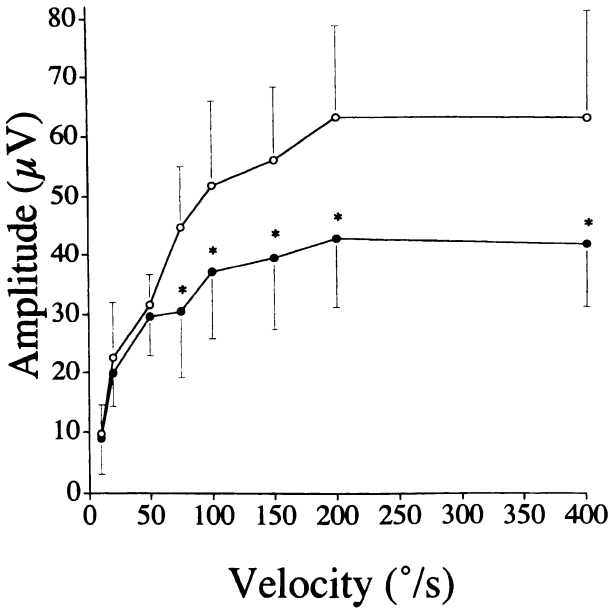

Figure 6. Relationship between velocity of motion disparity and mean amplitude of large negative wave in 4 cats before (○) and after muscimol injection (•) for each stimulus velocity. Error bars represent standard deviations. *$P < .01$.

The results of previous studies have suggested that the PMLS is also associated with the control of accommodation and vergence eye movements.[32–35] Visual motion signals may be involved in providing motor commands of accommodation and vergence in the PMLS. The accommodation and vergence areas in the PMLS send heavy projections to the SC.[18,36] Recent studies suggest that the SC plays an important role in the control of accommodation and vergence in the brainstem.[36–40] Therefore, it is probable that the functional linkage between the PMLS and SC is involved not only in visual motion processing but also in the control of accommodation and vergence.

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