Semicircular Canal Function Before and After Surgery for Superior Canal Dehiscence

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Objective: To characterize semicircular canal function before and after surgery for superior semicircular canal dehiscence (SCD) syndrome.

Study Design: Prospective unblinded study of physiologic effect of intervention.

Setting: Tertiary referral center.

Patients: Patients with SCD syndrome documented by history, sound- or pressure-evoked eye movements, vestibular-evoked myogenic potential testing, and high-resolution multiplanar computed tomographic scans.

Intervention: Nineteen subjects with SCD had quantitative measurements of their angular vestibulo-ocular reflexes (AVOR) in response to rapid rotary head thrusts measured by magnetic search coil technique before and after middle fossa approach and repair of the dehiscence. In 18 subjects, the dehiscence was plugged; and in 1, it was resurfaced.

Main Outcome Measures: The AVOR gains (eye velocity/head velocity) for excitation of each of the semicircular canals.

Results: Vertigo resulting from pressure or loud sounds resolved in each case. Before surgery, mean AVOR gains were normal for the ipsilateral horizontal (0.94 ± 0.07) and posterior (0.84 ± 0.09) canals. For the superior canal to be operated on, AVOR gain was 0.75 ± 0.13; but this was not significantly lower than the gain for the contralateral superior canal (0.82 ± 0.11, p = 0.08). Mean AVOR gain decreased by 44% for the operated superior canals (to 0.42 ± 0.11, p < 0.0001). There was a 13% decrease in gain for the ipsilateral posterior canal (p = 0.02), perhaps because plugging affected the common crus in some cases. There was a 10% decrease in gain for excitation of the contralateral posterior canal (p < 0.0001), which likely reflects the loss of the inhibitory contribution of the plugged superior canal during head thrusts exciting the contralateral posterior canal. Mean AVOR gain did not change for any of the other canals, but two subjects did develop hypofunction of all three ipsilateral canals postoperatively.

Conclusion: Middle fossa craniotomy and repair of SCD reduce the function of the operated superior canal but typically preserve the function of the other ipsilateral semicircular canals.

Key Words: Semicircular canal—Tullio phenomenon—Vertigo—Vestibulo-ocular reflex.

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through a middle cranial fossa approach. As previously reported, our experience with resurfacing was disappointing because resurfacing of the superior canal led to complete resolution of symptoms in only 7 of the 11 patients who underwent this procedure. Four of the patients who underwent canal resurfacing had initial resolution of symptoms but then developed recurrent vestibular symptoms and signs at 3 to 6 months after the surgical procedure. These recurrences led us to use the plugging procedure in subsequent cases. In contrast, complete resolution of symptoms and signs has been achieved in eight of nine patients who underwent canal plugging surgery, with the remaining patient achieving partial resolution of symptoms (2).

We hypothesize that the resolution of the vestibular symptoms and signs brought about by surgery to plug the superior canal is due to the correction of the abnormal flow of endolymph in the canal under the provocative stimuli. However, a less ideal mechanism would be the loss of neurosensory function in the entire labyrinth. Although this result would alleviate vertigo due to pathologic endolymph flow, it would also lead to the typical signs and symptoms of unilateral vestibular hypofunction, which can themselves be debilitating for some patients.

The goal of this study was to characterize the function of all three semicircular canals before and after surgery for superior canal dehiscence and to determine whether the function changes only in the operated canal or in all of the canals. Traditional caloric and slow harmonic rotational tests of canal function are not suited to the investigation of superior and posterior canal function. Instead, the function of all semicircular canals can be determined in the physiologic range of head rotational frequencies and accelerations by using rapid rotary head thrusts of canal function. The auditory brainstem response, facial nerve, and somatosensory evoked potentials were monitored in all of the surgical procedures. The auditory brainstem response was carefully monitored during the brief periods of manipulation of the membranous labyrinth, and the response was maintained in each case. All patients received 6 to 8 mg of dexamethasone three times daily, which was tapered gradually, typically during the initial 2 weeks after the procedure.

Magnetic Search Coil Testing

The 3-D angular vestibulo-ocular reflexes (AVOR) for passive head thrusts were recorded in all subjects using magnetic search coils before and after surgery. Horizontal, vertical, and torsional components of the movement of one or both eyes were recorded using a dual search coil embedded in a silicone annulus that was placed around the cornea (Skalar, Delft, The Netherlands). The eyes were anesthetized with two drops of topical proparacaine (0.5%) before placement of the search coil. Another pair of search coils firmly attached to a bite bar sensed head movements. The bar consisted of a Plexiglas plate coated with hardened dental impression compound molded to the subject’s dental occlusion. The magnetic fields generating the voltages in the search coils were produced by three orthogonal pairs of coils, with a diameter and a distance of 1.02 m. The techniques for search coil calibration and recording have been described previously (14).

The subject, seated with the head centered in the magnetic field, was instructed to gaze at a light-emitting diode located 124 cm directly forward at eye level. The examiner stood behind the subject and grasped the head over the temporoparietal area. The head was kept stationary in a comfortable, upright position before each head thrust. This position placed the Reid stereotactic line (inferior orbital rim to superior external auditory canal) within 5 degrees of the earth-horizontal plane. From this position, the examiner rapidly rotated the head by 10 to 20 degrees in one of three planes: the earth-horizontal plane, the plane containing the left anterior (superior) and right posterior canals (LARP head thrusts), or the plane containing the right anterior and left posterior canals (RALP head thrusts). For horizontal head thrusts, the head was turned to the left to excite the left horizontal canal (HC) or to the right to excite the right HC. For LARP head thrusts, the head was turned down

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and counterclockwise (with respect to the subject) in the LARP plane to excite the left anterior canal or up and clockwise to excite the right posterior canal. For RALP head thrusts, the head was turned down and clockwise to excite the right anterior canal or up and counterclockwise to excite the left posterior canal. The subject knew which plane would be stimulated in each trial, but could not predict the direction of the rotation.

**Data Analysis**

Eye and head positions in three dimensions were expressed as rotation vectors and used to derive the angular velocities of the eye and head. We discarded data from head thrusts during which the eye responses included blinks, saccades, or eye movements that commenced before the onset of head movement. The starting point of the head thrust was taken when the measured head velocity exceeded the mean velocity at rest, which was close to 0 degree/s, by 8 * standard deviation (SD) + 2 degrees/s (15).

Eye and head velocities were derived from the coil signals and initially expressed in a Cartesian coordinate system obeying the right-hand rule with the x axis aligned with the naso-occipital axis, the y axis aligned with the interaural axis, and the z axis aligned with the earth-vertical axis. The positive ends of these axes pointed out the nose, left ear, and top of the head, respectively. We then rotated the coordinate system + 45 degrees around the z axis so that the new coordinate axes roughly aligned with the axes of head rotation, axes that lay perpendicular to the canal planes (13). The 3-D eye and head velocity vectors then had components corresponding to each of the three canal planes.

For each head thrust, we calculated AVOR gain from the components of eye and head velocity along the axis of rotation. For example, gain for the HC pair was calculated from horizontal head thrusts using the component of eye velocity along the z axis divided by the component of head velocity along the z axis. Similarly, the gain for the LARP pair was calculated from LARP head thrusts using the eye and head velocity components along the axis perpendicular to the LARP plane.

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**FIG. 1.** Angular vestibulo-ocular reflex gains for head thrusts exciting each of the semicircular canals in Subject 6 before surgery to plug the dehiscent left superior canal. Gain was measured in the 30 ms before peak head velocity for each trial. Mean ± SD values are shown in the upper right corner of each panel. Head and eye velocities are expressed as positive in all cases for purposes of comparison.

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Finally, the gain for the RALP pair was calculated from RALP head thrusts using the eye and head velocity components along the axis perpendicular to that plane. As a representative “best value” of gain for each head thrust, we took the highest gain value that occurred in a 30-ms period before peak head velocity. Later times are avoided because of the possible intrusion of nonvestibular eye movements, particularly the refixation saccades that may compensate for a deficient AVOR in the early portion of the head thrust (16). The rotational data were analyzed using a program that we developed for the Labview software platform (National Instruments Corporation, Austin, TX).

Means and SDs were calculated for 5 to 20 trials for each of the 6 canals in each subject. Means and SDs over all subjects were computed for each canal before and after surgery. Paired t tests were used to compare these mean canal gains before and after surgery. A Bonferroni correction for multiple t tests was performed. Statistical calculations were done with Microsoft Excel 2003 (Microsoft Corporation, Redmond, WA).

RESULTS

Between June 1, 2001, and May 1, 2006, all of 32 subjects who were about to undergo surgery participated in preoperative quantitative testing of the AVOR with magnetic search coil testing. As of May 1, 2006, 19 (59%) of these subjects had also completed postoperative testing. Results from these 19 subjects are reported here. Four subjects (13%) are still healing from recent surgery and are awaiting postoperative testing. Five (16%) declined postoperative testing because of inconvenience, principally because they did not live locally; but each of these subjects had complete relief of their symptoms. One (3%) did participate in postoperative testing, but was not able to complete all of the testing paradigms to measure AVOR gains. Only three subjects (9%) did not complete postoperative testing because of recurrence of symptoms due to superior canal dehiscence. Each of these three subjects had an attempted resurfacing procedure before we abandoned that in favor of canal plugging.

Surgery relieved symptoms of vertigo and/or oscillopsia resulting from sound or pressure stimulation of the affected ear in each of the 19 subjects whose preoperative and postoperative AVOR results are reported here. Surgery consisted of plugging the dehiscence in 18 of the 19 subjects (95%) and of resurfacing the dehiscence in 1 (5%) subject. One case was complicated by an immediate postoperative epidural hematoma. This was evacuated, and the patient made a full recovery. One patient developed a wound cellulitis that resolved with antibiotic treatment, and one developed transient diabetes insipidus. Results of the surgery on hearing status of the inner ear is provided in a separate report.

The AVOR responses before surgery for Subject 6 are shown in Figure 1. This subject was a 40-year-old woman who developed vertigo when she went to a loud concert 9 years previously. Since then, vertigo could be provoked by any loud noises, by Valsalva maneuvers, and by bending. Over time, she developed a constant sense of disequilibrium. She also felt a sensation of fullness in the left ear, and she noted autophony and hyperacusis in that ear. As revealed by examination, the AVOR elicited by rapid head thrusts in the planes of each of her six semicircular canals seemed normal. As revealed by examination under Frenzel lenses, the administration of tones at frequencies of 250 and 1,000 Hz at brief intensities of 110 dB normal hearing level to the left ear evoked eye movements that were characteristic of left superior canal excitation: The slow phase of the nystagmus moved the eyes upward and rotated their superior poles toward the subject’s right side. Valsalva against pinched nostrils...
elicited a similar eye movement. Valsalva against a closed glottis evoked an eye movement in the opposite direction, characteristic of superior canal inhibition; but on sudden release, it reverted to an eye movement characteristic of superior canal excitation. The Weber tuning fork test (512 Hz) lateralized to the left ear. Rinne tuning fork testing demonstrated that she heard air conduction greater than bone conduction bilaterally. Her audiogram showed a 10- to 20-dB air-bone gap for low frequencies in the left ear. The air-conduction thresholds were normal across all frequencies for the left and for the right ear. Vestibular evoked myogenic potentials were elicited at a low threshold of 75 dB in the left ear (10). There was no VEMP response evoked from the right ear. Her temporal bone CT scan showed dehiscence of bone overlying the left superior semicircular canal (Fig. 2).

The AVOR responses in Figure 1 are similar to those we have observed in normal subjects. The right and left eye-in-head velocities closely follow the head velocity throughout the brief head impulse. All values here are expressed as positive to facilitate comparisons, although in reality the eye moved opposite in direction to the head. The nearly equal velocities meant that the eyes in space remained relatively stable, which is the goal of the AVOR.

Figure 3 shows the AVOR responses after surgery in the same subject. The AVOR response for excitation of the plugged ipsilateral superior canal is diminished. The mean gain is reduced from the preoperative value of 0.76 ± 0.08 (mean ± SD) to a postoperative value of 0.53 ± 0.05, a 30% reduction. Responses for excitation of the other canals are not affected.

Compensatory AVOR were measured in all subjects before surgery. The AVOR gains approximated 1 (perfectly compensatory) for the horizontal canal (Table 1, Fig. 4). For the superior and posterior canals, AVOR gain values were typically not as large as for the horizontal canal, as has been previously observed in normal

FIG. 3. Angular vestibulo-ocular reflex gains for head thrusts exciting each of the semicircular canals in Subject 6 after surgery to plug the dehiscent left superior canal. Format is the same as for Figure 1.
subjects (12). The values observed here are within the range we have reported using the same measurement techniques in normal subjects and subjects with Ménière disease before intratympanic gentamicin treatment (17,18).

For the group as whole, before surgery the AVOR gain values for head thrusts exciting the superior canal to be repaired tended to be lower than gain values for head thrusts exciting the contralateral superior canal. For the ipsilateral superior canal, AVOR gain was 0.75 ± 0.13. For the contralateral superior canal, gain was 0.82 ± 0.11. The difference did not reach significance (p = 0.08 for paired t test). It is notable, however, that 11 of the 19 subjects had bilateral dehiscences; and 7 of these individuals had some symptoms from the contralateral superior canal dehiscence. Thus, contralateral superior canal gains may have been lowered in some cases as well, making the differences between the sides less significant.

We examined the relationship between the length of the dehiscence measured at the time of surgery and the preoperative gain of the affected superior canal. Figure 5 demonstrates that there was a weak negative linear correlation (r² = 0.3, 95% confidence interval [CI] −0.71 to −0.27) between dehiscence size and preoperative AVOR gain for the affected superior canal. The two subjects with the lowest gains in Figure 4, with gain values of 0.42 (Subject 3) and 0.45 (Subject 5), had dehiscences measuring 6 and 5 mm, respectively. Yet three dehiscences of equal or greater size were associated with higher gains.

There were no significant differences in the gain values between the ipsilateral and contralateral sides for the horizontal canals (95% CI for difference: −0.05 to 0.00, p = 0.09) and posterior canals (95% CI: −0.04 to 0.02, p = 0.5) before surgery.

Postoperative testing of the AVOR was conducted between 46 and 206 days after surgery (mean ± SD: 109 ± 55 days), except in one recent subject in whom testing was completed 6 days after surgery. Plugging the dehiscent superior canal (18 cases) or resurfacing it (1 case, Subject 5) resulted in significant reduction in gain for that canal (Table 1, Fig. 4). Postoperatively, the mean gain value for plugged superior canals was 0.42 ± 0.11, a 44% reduction compared with preoperative values for the same canal in the same subjects (p < 0.0001, paired t test).

The mean changes in the ipsilateral horizontal canal gain (−5%) and contralateral horizontal canal gain (−4%) were not significant (Table 1).

### TABLE 1. Angular vestibulo-ocular reflex gains (mean ± SD) for head thrusts exciting the ipsilateral and contralateral horizontal, superior, and posterior canals before and after surgery for superior canal dehiscence. The changes in gain after surgery are given in percentages, and p values are for paired t tests.

<table>
<thead>
<tr>
<th></th>
<th>Ipsilateral</th>
<th></th>
<th>Contralateral</th>
<th></th>
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<tbody>
<tr>
<td></td>
<td>HC</td>
<td>SC</td>
<td>PC</td>
<td>HC</td>
</tr>
<tr>
<td>Preop</td>
<td>0.94 ± 0.07</td>
<td>0.75 ± 0.13</td>
<td>0.84 ± 0.09</td>
<td>0.97 ± 0.08</td>
</tr>
<tr>
<td>Postop</td>
<td>0.90 ± 0.24</td>
<td>0.42 ± 0.11</td>
<td>0.73 ± 0.20</td>
<td>0.93 ± 0.09</td>
</tr>
<tr>
<td>% change</td>
<td>−5%</td>
<td>−44%</td>
<td>−13%</td>
<td>−4%</td>
</tr>
<tr>
<td>p</td>
<td>0.33</td>
<td>&lt;0.0001</td>
<td>0.018</td>
<td>0.09</td>
</tr>
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*HC, horizontal canal; SC, superior canal; PC, posterior canal; Preop, before surgery; Postop, after surgery.*

FIG. 4. Mean AVOR gains before and after surgery for all subjects. Preoperative and postoperative gain values for the same subject are connected by a line segment.

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For the group mean values, there were also mild but significant reductions in the gains of the contralateral posterior canal (10%, $p < 0.0001$) and ipsilateral posterior canal (13%, $p = 0.018$). Only the contralateral posterior canal reduction remained significant after applying a Bonferroni correction for the fact that six paired $t$ tests were performed (requiring alpha $= 0.05 / 6 = 0.008$ for significance.)

Subjects 3 and 10 were distinct outliers who lost 59 and 82% of their preoperative gain values for the ipsilateral horizontal canals (postoperative gains of 0.36 and 0.16, respectively, in Fig. 4). Subjects 3 and 10 also lost 57 and 30% of their preoperative gain values for the ipsilateral posterior canals (gains of 0.32 and 0.44, respectively). These subjects did not experience any associated hearing loss. Subject 9 had a reduction in posterior canal gain to 0.39 but no reduction in horizontal canal gain (0.94). He had a transient high-frequency sensorineural hearing after surgery that completely resolved within 3 weeks; his AVOR testing was conducted 7 weeks after surgery. His dehiscence, measuring 2 mm, was the smallest that we have observed intraoperatively.

The single subject in this report who underwent a procedure with an intent to resurface the dehiscent superior canal had diminished gain for the affected superior canal (0.45 ± 0.13) preoperatively. Postoperatively, this further decreased to 0.35 ± 0.08. His ipsilateral horizontal canal gain remained robust (0.83 ± 0.04), but his ipsilateral posterior canal gain dropped (from 0.92 ± 0.07 to 0.59 ± 0.05).

**DISCUSSION**

Surgical plugging of a semicircular canal has long been known to decrease its rotational sensitivity. Manipulation of individual semicircular canals was, in fact, essential to Ewald’s demonstration of the physiologic mechanism of the canal (19). Canal plugging has a younger history in clinical use. Parnes and McClure (20) introduced the procedure of posterior canal plugging for intractable vertigo due to benign paroxysmal positioning vertigo. Shaia et al. (21) have recently confirmed previous reports of the success of this surgery at relieving positional vertigo while preserving hearing in most cases. Extended approaches to the petroclival region have been devised in which both the superior and posterior canals are partially removed and the lumina of the residual canals are plugged. These approaches can be done with preservation of hearing (22). Several centers have reported success in plugging or resurfacing dehiscent canals (5,23–26). However, little is known regarding the effect of surgical plugging of a single canal on the function of the remaining canals. Cremer et al. (13) demonstrated in three subjects after unilateral posterior canal occlusion for benign paroxysmal positioning vertigo that the AVOR gain for the plugged canal was reduced, whereas that of the remaining canals remained intact.

In the present study, we measured the function of each semicircular canal in 19 subjects before and after plugging (18 cases) or resurfacing (1 case) of the dehiscent superior canal. The data indicate that the surgery reliably reduces the rotational sensitivity of the affected canal. However, the function of the ipsilateral horizontal and posterior canals remained unaffected for most subjects. The results indicate that the middle fossa approach and plugging technique used in this group of patients can preserve function in the rest of the labyrinth in most cases, and that vertigo relief is obtained by virtue of the plug, not global labyrinthine hypofunction.

Plugging the superior canal should eliminate the flow of endolymph in that canal. However, it is also possible that abnormal pressure effects related to dilation of the canal that occur as a consequence of the dehiscence are eliminated by the plug. Hair cells transduce shearing motion of their stereocilia. This shearing motion has traditionally been thought to occur because of cupular displacement by endolymph flow. However, Yamauchi et al. (27) have demonstrated that cupular stretching/compression as a result of dilational pressure operating on the ampulla can also activate the vestibular afferent nerves. It is possible that plugging the dehiscent canal has its beneficial effect by eliminating transmission of pressure that could dilate and contract the ampulla of the superior canal.

Plugging the dehiscent superior semicircular canal effectively relieved symptoms of superior canal dehiscence in 18 of the 19 subjects in this series. A resurfacing procedure worked for 1 of these 19 subjects, but we have since abandoned that procedure in favor of plugging because of the higher rate of symptom recurrence that we experienced with resurfacing in other patients (2). This may be because the bone graft and other materials used for resurfacing can shift or be reabsorbed postoperatively (28). The data for the patient who underwent the resurfacing procedure in
this study do not suggest that it preserved function of the resurfaced canal. In fact, not only did his superior canal gain decrease after surgery, but so did his posterior canal gain.

Subject 9 also had a reduction in posterior canal gain after plugging surgery, but no reduction in horizontal canal gain. This raises the possibility that superior canal plugging can affect the posterior canal without causing global labyrinthine hypofunction. We hypothesize that this may be due to threading the plug far enough into the nonampullated limb of the dehiscent canal that it impinges on the membranous common crus. In this case, the dehiscence was only 2 mm in length. However, there was a much longer segment of the canal with thin, transparent, “blue-lined” bone on either side of the dehiscence. We threaded fascia strips and bone chips into the canal over sufficient distances to completely underlay these regions of thin bone. This was done as a prophylactic measure: Should the thin bone later break down, the patient would not be subject to recurrent symptoms because the more distant canal would remain plugged. This more aggressive approach to packing may, however, place the common crus at risk of being plugged as well. This subject did note that he still had mild oscillopsia with vertical head movements 6 weeks after surgery. The additional burden of losing posterior canal function may contribute to such symptoms. Attention to the distance through which the plug is threaded in the nonampullated limb of the canal may help avoid this situation.

Two of the 19 subjects (10%) did develop global ipsilateral vestibular hypofunction as the result of the surgery. Subject 3 was 68 years old at the time of her surgery. She required vestibular physical therapy to compensate for the unilateral vestibular loss, but she has resumed all of her previous activities and has relief of vertigo previously caused by Valsalva maneuvers. Subject 10 was 41 years old at the time of surgery. He also required vestibular physical therapy but has resumed all of his normal activities and has had relief of vertigo that had been induced by loud sounds or pressure changes in the right ear. Interestingly, neither of these subjects developed sensorineural hearing loss in association with this labyrinthine function loss. This suggests that the damage, possibly a chemical labyrinthitis, did not extend to the cochlea. These two subjects had dehiscences that measured 5 and 6 mm, in the midrange of those encountered (Fig. 5). This does not suggest that the damage can be attributed to a larger dehiscence size and more aggressive packing. Neither of these subjects had previous inner ear surgery, and there were no deviations from the described surgical technique in their cases. These cases were done after our experience with 30 previous ones, so that inexperience would not seem to be the reason. We conclude that there is some inherent risk of global loss of ipsilateral vestibular function with this approach, but that risk seems to be acceptable in that it is approximately 10% and has not been debilitating. However, in patients who may already have hypofunction in the contralateral labyrinth, the potential disability from such loss may be greater.

We have previously reported decreased gain for the affected superior canal in subjects with superior canal dehiscence in which the dehiscence length measured at least 5 mm on CT scans (7). In the present study, intraoperative measurements of dehiscence length were made; and we found a weak negative correlation between dehiscence size and gain of the affected superior canal (Fig. 5). We hypothesize that this relationship is due to the prolapse of dura into the dehiscent canal. When the bony dehiscence is of sufficient length, prolapsing dura may compress the membranous semicircular canal. This is, in effect, a natural equivalent of canal plugging. However, in cases in which sound or pressure changes still evoke vertigo from such canals, the dural plug must be incomplete or intermittent, such that these stimuli can overcome the effect of plugging and produce the abnormal stimulation of the canal.

In summary, results of this study suggest that in most cases, plugging the superior canal via the middle fossa approach using the technique that we describe is associated only with a decrease in function of the plugged canal, not the entire labyrinth. This is highly effective in relieving symptoms of vertigo due to sound or pressure changes. However, there is an approximately 10% risk of loss of function of all of the ipsilateral semicircular canals associated with this surgery. Hearing loss does not necessarily accompany this labyrinthine loss. Aggressive packing of the superior canal lumen toward its nonampullated end may occlude the common crus as well, leading to diminished posterior canal function and the risk of oscillopsia with vertical head movements.

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