Supplementary Materials and Methods

Subjects

Four adult subjects (2 men, 2 women) with BVL participated in this study. Inclusion criteria included age 22-90 years, summed responses to warm/cool caloric vestibular stimulation below 10°/s per ear, MRI and CT imaging confirming normal ear and vestibular nerve anatomy, and sufficient hearing in the contralateral ear to support communication. All subjects must be ≥12 months post-onset and must have completed ≥6 months of vestibular rehabilitation therapy exercises while off vestibular-suppressant medications. Exclusion criteria included vestibular areflexia etiologies outside of the labyrinth, medical conditions that could impede a subject's ability to complete testing, or any medical contraindication to the planned surgery.

Prior to enrollment, subject MVI001 (male, 61 years old on entry into the study) received intravenous gentamicin for 14 days in 2012 for a leg injury. Post-aminoglycoside treatment, the subject reported symptoms of profound BVL which persisted for over 4 years following a plateau of compensation from rehabilitation therapy. The subject was implanted with the MVI system in the left ear in August 2016. Subject MVI002 (male, 57 years old) suffered from vertigo, imbalance, and oscillopsia in 2006 following spinal surgery. The subject was further treated with bilateral intratympanic streptomycin in 2007 and experienced symptoms consistent with BVL thereafter. On presentation to Johns Hopkins in 2016, the subject reported an incomplete recovery from vestibular rehabilitation exercises started in 2010. He underwent implantation of an MVI stimulator in his left ear in November 2016. Subject MVI003 (female, 63 years old) suffered from symptoms of BVL after 7 days of intravenous gentamicin treatment for a kidney stone urosepsis in 2015. The subject performed vestibular physical therapy for over a year without sufficient

recovery and was implanted in her left ear with the MVI in February of 2017. Subject MVI004 (female, 62 years old) was treated for 14 days with gentamicin after an operation to treat a pelvic abscess in 2015. After the onset of BVL symptoms, she participated in vestibular therapy, which incompletely alleviated her symptoms. This subject's left ear was implanted with an MVI stimulator in December 2017.

Surgery

Each subject was implanted with the receiver/stimulator component of a Labyrinth Devices MVI system in the left ear via a post-auricular incision and transmastoid approach similar to that typically used for cochlear implantation or labyrinthectomy, except that no entry was made into the cochlea and instead four ~0.6-0.75 mm diameter openings were made into the labyrinth (in the anterior and horizontal semicircular canal ampullae and in the thin segment of the posterior canal near the ampulla and near the common crus), via which electrodes were inserted. Bone pate was applied around the points where the electrode arrays entered the labyrinth. The receiver/stimulator (Figure 2) was secured with suture in a bone well and sub-periosteal pocket in the post-auricular region. All surgeries were performed on an outpatient basis at the Johns Hopkins Hospital or Johns Hopkins Outpatient Center by the same surgeon (CCDS). Two subjects (MVI002 and MVI003) were discharged directly from the post-anesthesia recovery unit within 2 hours after surgery; the other two stayed for extended observation until the next morning and were then discharged to a nearby hotel.

3D eye movement recording

Eye movements were recorded using 3DBinoc™ video-oculography (VOG) goggles (Labyrinth Devices, LLC), which use a single camera to track binocular 3D eye position while illuminating the subject's eyes using infrared LEDs (allowing ocular tracking without visible light). The goggles assay horizontal and vertical components of eye position via pupil tracking and measure torsional angular position using iris pattern template matching (44). Custom VOG software acquired 3D angular eye position data at 100-180 frames-per-second with a peak-to-peak noise floor of 0.15°, 0.07°, and 0.06° for the torsional, vertical, and horizontal 3D components, respectively. The goggles use a pair of IR-pass optical filter insets to block the subject's view of visible light during data acquisition. 3DBinoc™ goggles interface with a host PC through a galvanically isolated USB connection and directly connect to each subject's PCU for stimulus trigger synchronization.

Data analysis

The 3DBinoc™ system reports 3D angular position data as gaze direction (i.e., horizontal and vertical position of the pupil) and torsion around the eye's line-of-sight. We processed raw angular position data traces with third-order median filters to recover VOG tracking dropouts. 3D angular position data were converted to rotation vectors (47) and filtered with second or third order Savitsky-Golay filters (76) for high frequency noise rejection. We first computed 3D angular velocity in X ('roll'), Y ('pitch'), and Z ('yaw') head-fixed coordinates then transformed angular velocity data into anatomic canal coordinates by applying a -45° passive reorientation of the head-fixed coordinate system about the yaw axis (45, 77, 78).

Nystagmus quick phases were detected and excised using an eye acceleration threshold. Spline interpolation across excised segments produced smooth slow phase eye velocity traces. We

computed cycle averages after removing trails corrupted by blinks or drops in pupil/iris tracking. Eye position data collected during adaptation to MVI activation were processed with custom software that fit a least-squares linear model to slow phase responses between auto-detected quick phases. The slope of the fitted line for each 3D component and corresponding angular position values was used to compute 3D angular velocity using standard rotational kinematic techniques (78).

Electrode characterization and activation

For pre-activation electrical stimulation characterization measurements, a 200pps pulse train was delivered to each electrode contact (with pulse train duration 200ms and inter-train-interval 300ms) to assay responses at each tested current level. Pulse train stimuli were first used to characterize perceptual thresholds, VOR thresholds, and maximum current levels for each tested electrode/phase duration combination. These data were used to generate 10 (MVI001 and MVI002) or 7 (MVI003 and MVI004) current amplitudes spanning the VOR threshold and maximum current level to assay acute 3D VOR responses during MVI stimulation (Table S1).

Following electrode characterization, a stimulation parameter set (including electrode contact, current amplitude, and phase duration) was programmed into the subject's PCU for each canal. Stimuli were chosen to optimize evoked VOR magnitude and response alignment with target canal anatomic axes (Table S2). Device activation (including adaptation to baseline tonic electrical stimulation) was performed with the subject on a bite-block in total darkness. Each active electrode was programmed to provide biphasic, charge balanced current pulses at 100pps on all three active canal channels.

The nystagmus evoked by the onset of stimulation was measured in darkness for 1 minute, after which the lights in the experimental room were turned on and the IR-pass filters removed from the 3DBinoc™ goggles for 4 minutes. When in light the subject was instructed to focus on an Earth-fixed target to promote adaptation to prosthetic baseline stimulation via retinal slip error signals during the evoked nystagmus. This procedure (1 minute in darkness to assay vestibular nystagmus responses and 4 minutes to promote adaptation) was repeated until all components of the spontaneous nystagmus in darkness were <5°/s. Subjects MVI002, MVI003, and MVI004 were later adapted to a 150pps baseline pulse rate on the same day.

Virtual head rotation (prosthesis-only) stimulation

For experiments examining responses solely driven by prosthetic input, the PCU was programmed to bypass gyroscopic input from the HWU and provide 'virtual' head motion signals processed by the subject's PCU. The device communicated with the implanted stimulator to provide a sequence of pulsatile stimuli encoding head rotations according to each canal's stimulation parameters describing relationships between head velocity and both pulse rate and pulse amplitude for each active electrode (Table S3). These stimuli were delivered with the subject in darkness and their head stationary on a custom bite-block to prevent visual, latent vestibular, or cervico-ocular reflex responses from influencing recorded eye movements.

Rotary chair testing

An Earth-vertical rotary chair (NeuroKinetics, Inc., Pittsburgh, PA) was used to provide whole body, *en bloc* sinusoidal rotations in darkness using frequencies between 0.1–2Hz at 100°/s peak velocity. Rotatory testing was performed both pre-operatively and post-operatively before any

electrical stimulation to assay changes to mechanical vestibular function due to surgical procedures. After activation of the MVI system, testing was completed in the following two conditions: a) with the device set to provide pulsatile stimulation that modulated according to parameters programmed into the subject's PCU ("Modulation ON") and b) with the PCU set to provide non-modulating, constant-rate and -amplitude tonic stimulation on each active canal electrode ("Modulation OFF").

Subject stimulus parameter mappings

The MVI encodes 3D head velocity via modulation of pulse rate and/or pulse amplitude of the adapted electrical stimulus delivered to each active canal electrode. For each active contact within a canal ampulla, a head velocity-to-pulse rate and head velocity-to-pulse amplitude mapping was programmed into the subject's PCU to update pulsatile stimulation parameters and encode 3D head rotation components about each anatomic canal axis. Mappings were programmed as either:

1) Flat

   Where pulse rate or amplitude is set to a single value that does not change with head velocity.

2) Piecewise-Linear

   A two-segment, piecewise-linear map constructed by mapping the head velocity input range from [-400°/s, 0°/s, +400°/s] to the minimum, baseline, and maximum pulse rate or amplitude determined during electrode characterization.

3) Sigmoidal

A non-linear mapping created using a hyperbolic tangent function to mimic the response dynamics of primary vestibular afferents in non-human primates (30, 31, 79) defined by:

$$Pulse(t) = 0.5 * (P_{MAX} - P_{MIN}) * \left(1 + tanh\left(X + C_{RATE} * \left(\frac{v(t)}{v_{MAX}}\right)\right)\right) + P_{MIN}$$

and

$$X = atanh\left(2 * \frac{P_{BASELINE} - P_{MIN}}{P_{MAX} - P_{MIN}} - 1\right)$$

Where:

Pulse(t) = Mapping describing either pulse-amplitude- or pulse-rate-modulation of prosthetic stimulation as a function of head velocity.

v(t) = Input head velocity waveform. This can be generated either via measurements made using the 3-axis gyroscope sensors in the subject's HWU or generated by a galvanically isolated computer with the subject's head stationary.

v_{Max} = Maximum input head velocity magnitude. Set to 400°/s for all subjects. The head velocity input range is set to [$-v_{Max}$, v_{Max}].

$P_{Baseline}$ = Pulse rate or amplitude used when the subject's head is stationary (i.e., 0°/s)

P_{Max} = Maximum pulse rate or amplitude used to encode the maximum input head velocity (v_{Max}).

P_{Min} = Minimum pulse rate or amplitude used to encode the minimum input head velocity ($-v_{Max}$).

C = Compression factor defining the slope of the head velocity-to-pulse rate or -pulse amplitude curve.

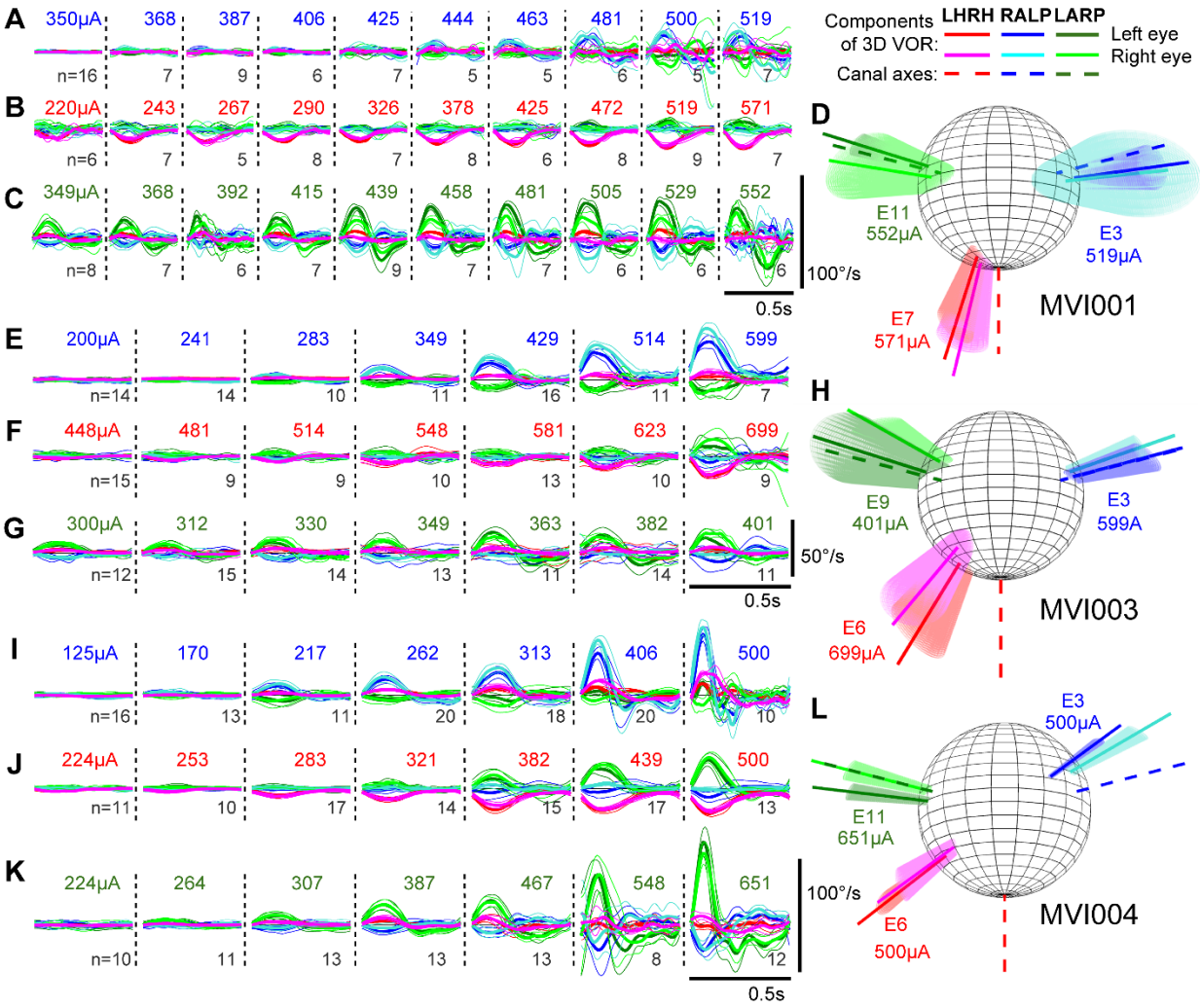
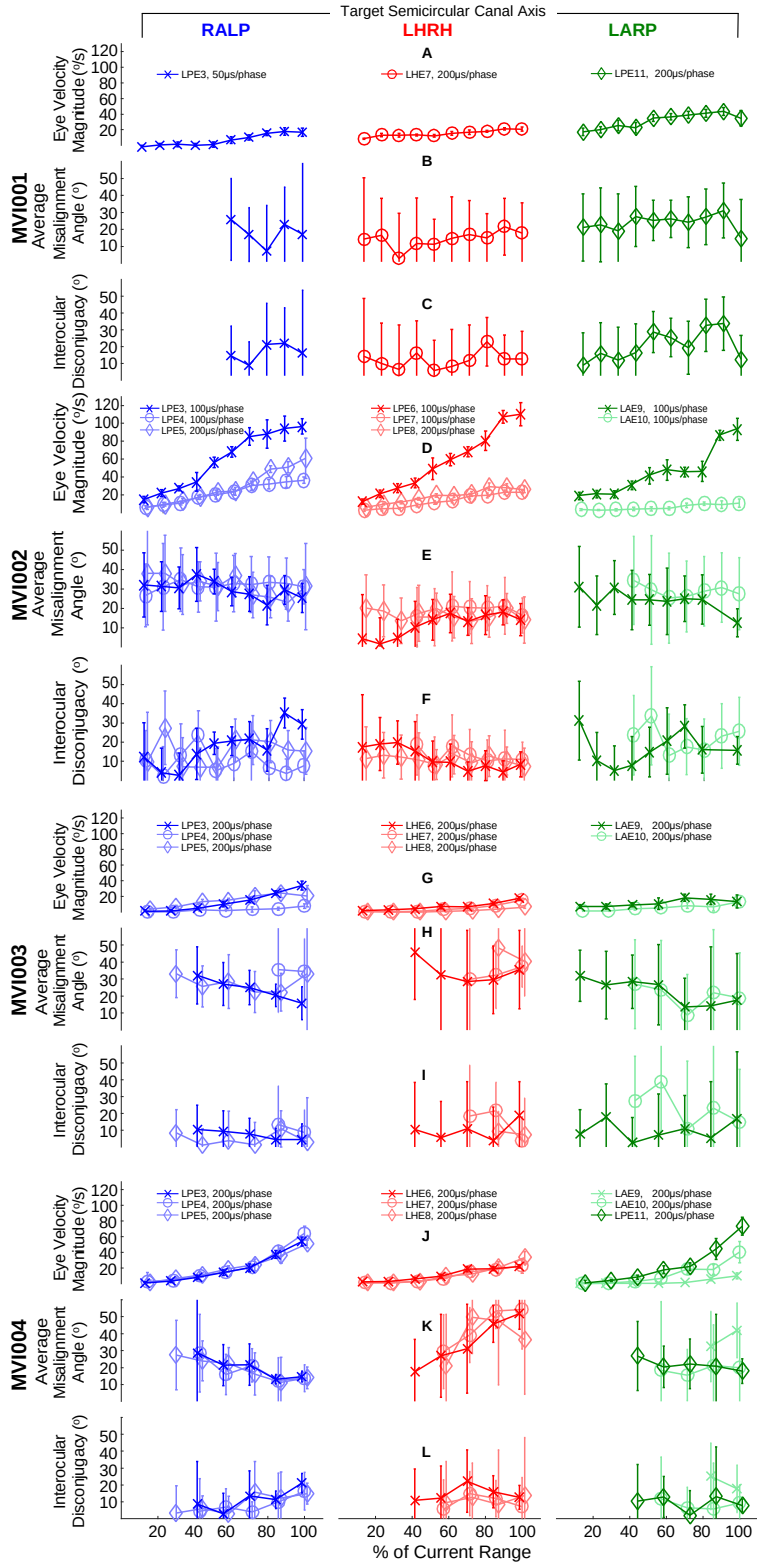


Figure S1. Binocular 3D Responses from subjects MVI001, MVI003, and MVI004.
Binocular cycle averaged 3D slow phase eye velocity traces in response to 2Hz, 40% duty cycle pulse trains oscillating between 200pps (200ms) and 0pps (300ms). Data are shown as mean±SD for n cycles. Pulse trains where delivered to isolated electrode contacts on subjects MVI001 (A-D), MVI003 (E-H), and MVI004 (I-L). For subject MVI001, pulse trains delivered to (**A**) electrode E3 using 50μs/phase biphasic current pulses, (**B**) electrode E7 using 200μs/phase, and (**C**) electrode E11 using 200μs/phase pulses produced canal specific responses. The axis of rotation of the eye movement evoked by the maximum current level for each of the three electrodes is plotted in (**D**). When tested on electrodes (**E**) E3, (**F**) E6, and (**G**) E9 using 200μs/phase current pulses, subject MVI003 produced eye movements well aligned with the LP and LA canals. Stimuli delivered to the LH canal produced a response consistent with activation of the LH ampullary nerve and spurious stimulation of the LA canal, depicted graphically in (**H**). Lastly, subject MVI004 produced similar responses when tested on electrodes (**I**) E3, (**J**) E6, and (**K**) E11 using 200μs/phase current pulses, where stimulation of electrode contacts in the LA and LP ampullae produced well aligned responses, while stimulation in the horizontal canal produced a mixed LHRH/LARP response.

Figure S2. Current fitting summaries for subjects MVI001, MVI002, MVI003, and MVI004.

Vestibulo-ocular reflex eye movement velocity magnitude, misalignment angle and interocular disconjugacy for responses to electrical stimulation delivered individually via each stimulating electrode in each subject's implanted (left) labyrinth. Data are shown as mean±SD for n=5 to 20 (median 12) cycles. Electrode contacts chosen for continuous motion-modulated stimulation for device activation are bolded while data collected from the remaining contacts are faded. The misalignment angle plotted as a function of current amplitude intensity, where misalignment is computed as the angle between the mean binocular axis of rotation and the intended canal axis of rotation. Binocular disconjugacy is plotted as the angle between the mean VOR rotation axes of the left and right eye as a function of current intensity. (**A-C**) MVI001 was only thoroughly tested on electrodes E3, E7, and E11 in the LP, LH, and LA canals respectively due to restricted time. (**D-F**) Subject MVI002 produced robust responses when assayed using the electrodes on the tip of each electrode shank (i.e., electrode E3 in the LP canal, E6 in the LH canal and E9 in the LA canal. (**G-I**) Subject MVI003 produced eye movements that approximated the target canal axis of rotation, with overall smaller amplitude eye velocities compared to MVI002. (**J-L**) MVI004 produced strong eye movements during stimulation of electrodes in the LP and LA canals (**G**, left and right) that were consistent with selective stimulation of their respective ampullary nerve branches. Stimulation of electrodes in the LH canal (**H**, middle) produced eye movements with a large LARP component (as seen in Figure S1J). All stimulus parameters are listed in Table S1. Responses with peak eye velocities <5°/s were too small to provide accurate estimates of misalignment or interocular disconjugacy and are not displayed in the figure.



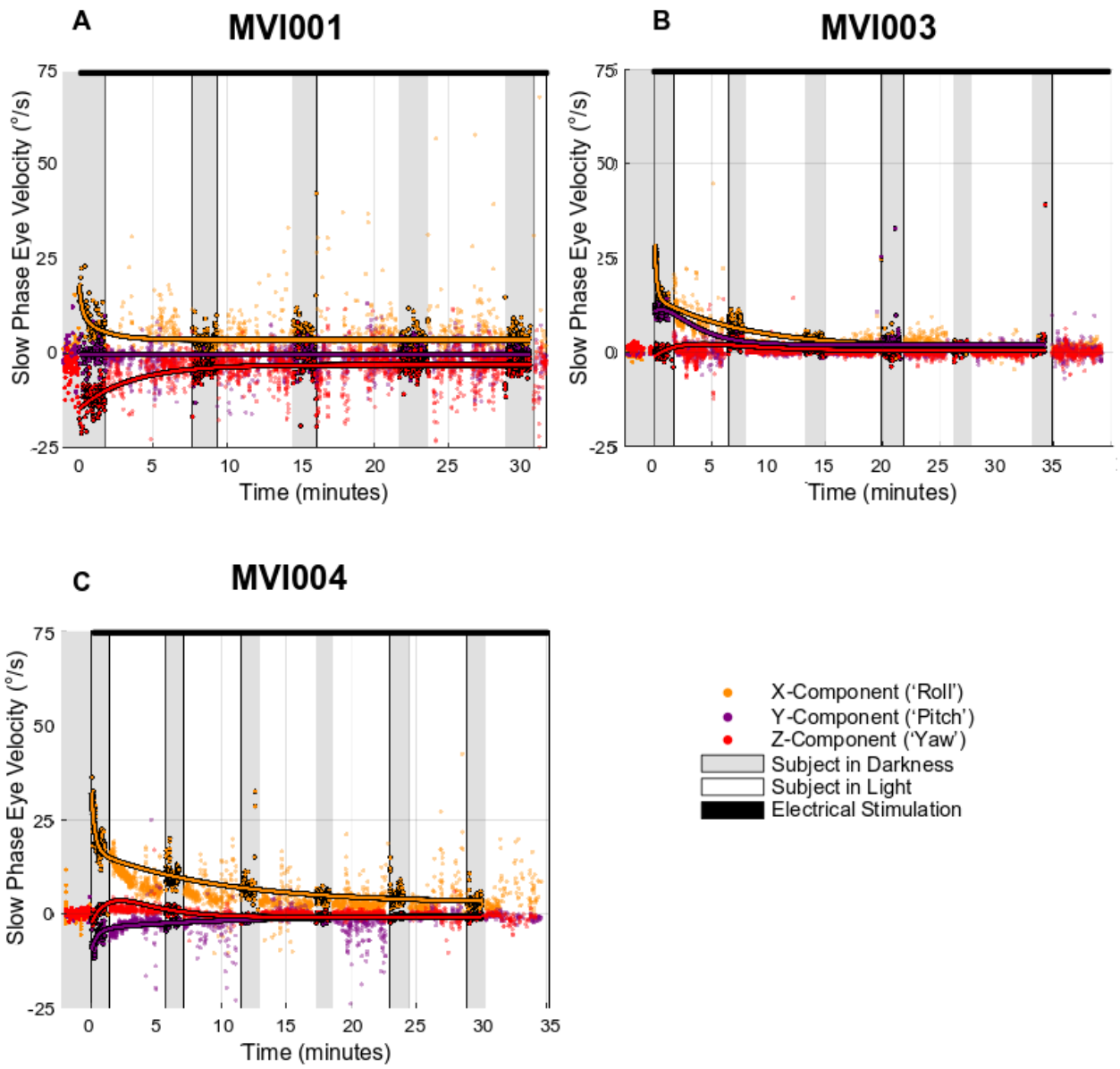
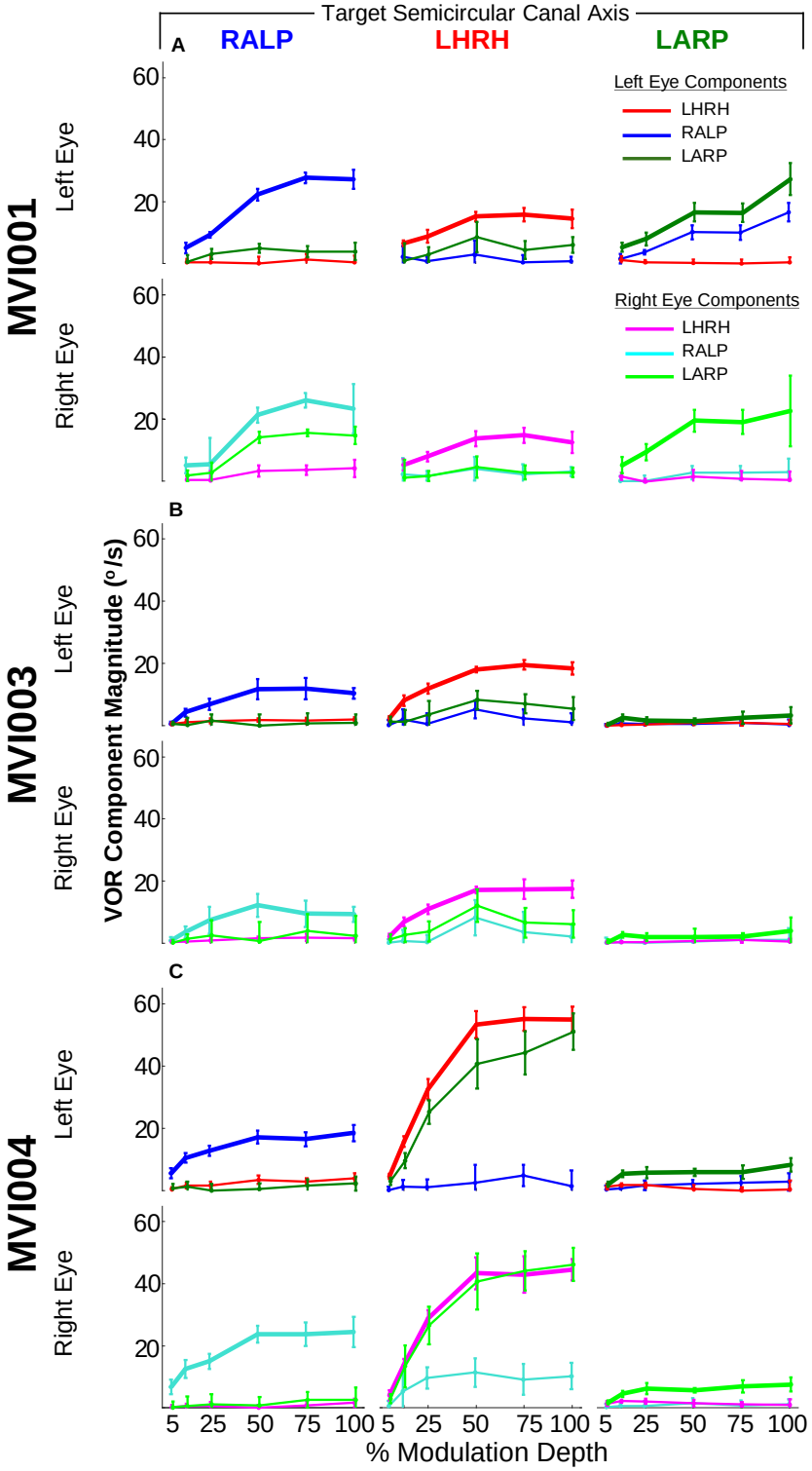


Figure S3. MVI001, MVI003, and MVI004 adaptation to constant rate and current electrical stimulation. Subjects (**A**) MVI001, (**B**) MVI003, and (**C**) MVI004 all produced nystagmus responses to the onset of prosthetic vestibular stimulation delivered to electrodes in each canal that decayed to imperceptible levels within ~30 minutes. (**A**) Subject MVI001 produced a brisk nystagmus that decayed within the first minute in darkness. (**B**) Subject MVI003 produced a positive roll component that decreased rapidly within the first minute in darkness. The subject also produced a positive pitch component in addition to the positive roll component, consistent with a higher level of evoked activity in the LP ampullary nerve branch relative to the LA nerve group. Also, this subject and subject (**C**) MVI004 produced a nystagmus with a small negative (~-3°/s) yaw component that reversed direction within the first minute in darkness. This finding is consistent with reversal phases of evoked nystagmus during sustained vestibular stimulation. Each point represents one slow phase nystagmus segment. Second order exponential fits of slow phase velocity data acquired in darkness revealed dominant roll time constants of 1.5, 7.8, and 9.6 minutes (RMSE = 3.1, 1.7, and 2.8°/s) for MVI001, MVI003, and MVI004, respectively.

Figure S4. Eye movement responses to 2Hz synthetic sinusoidal head velocity waveforms (MVI001, MVI003, and MVI004). Excitatory half-cycles of eye movement responses to "virtual head rotation" prosthesis-only stimulation using 2Hz sinusoidal modulation targeting one electrode in each canal reveal ability to drive canal-specific VOR responses after 8 wk of motion-modulated prosthetic stimulation. All data were collected with the subject's head stationary on a bite-block and the test environment darkened by occluding the subject's vision with visible-light blocking filters and turning off the experimental room lights. Target canal components are bolded in each figure. Data are shown as mean±SD for n=7 to 20 (median 16) cycles. (A) Subject MVI001 produced stable VOR responses that grew in magnitude with modulation intensity. VOR responses tended to rotate about an axis that closely approximated the target anatomic canal axis, except during RALP stimulation in the right eye and LARP stimulation in the left eye. (B) Testing subject MVI003 with virtual head velocity sinusoids produced eye movements resembling the intended canal axes of rotation, though with lower amplitude peak eye velocities compared to subject's MVI001 and MVI002. Modulation of the current stimulus on electrode E9 in the LA canal (B, right) produced small amplitude eye movements when tested at 100% modulation depth (Left eye: 3.4±2.6°/s, Right eye: 4.0±4.2°/s). (C) Subject MVI004 produced 3D responses that approximated the intended anatomic canal axis of rotation during modulation of the electrical stimulus delivered to electrodes E3 in the LP canal (C, left) and E11 in the LA canal (C, right). During modulation of electrode E6 in the LH canal (C, middle), the intended 3D component aligned with the subject's LHRH axis (red and pink traces) grew with modulation depth with an unintended LARP component (dark and light green) that was likely due to co-activation of the LA nerve branch.



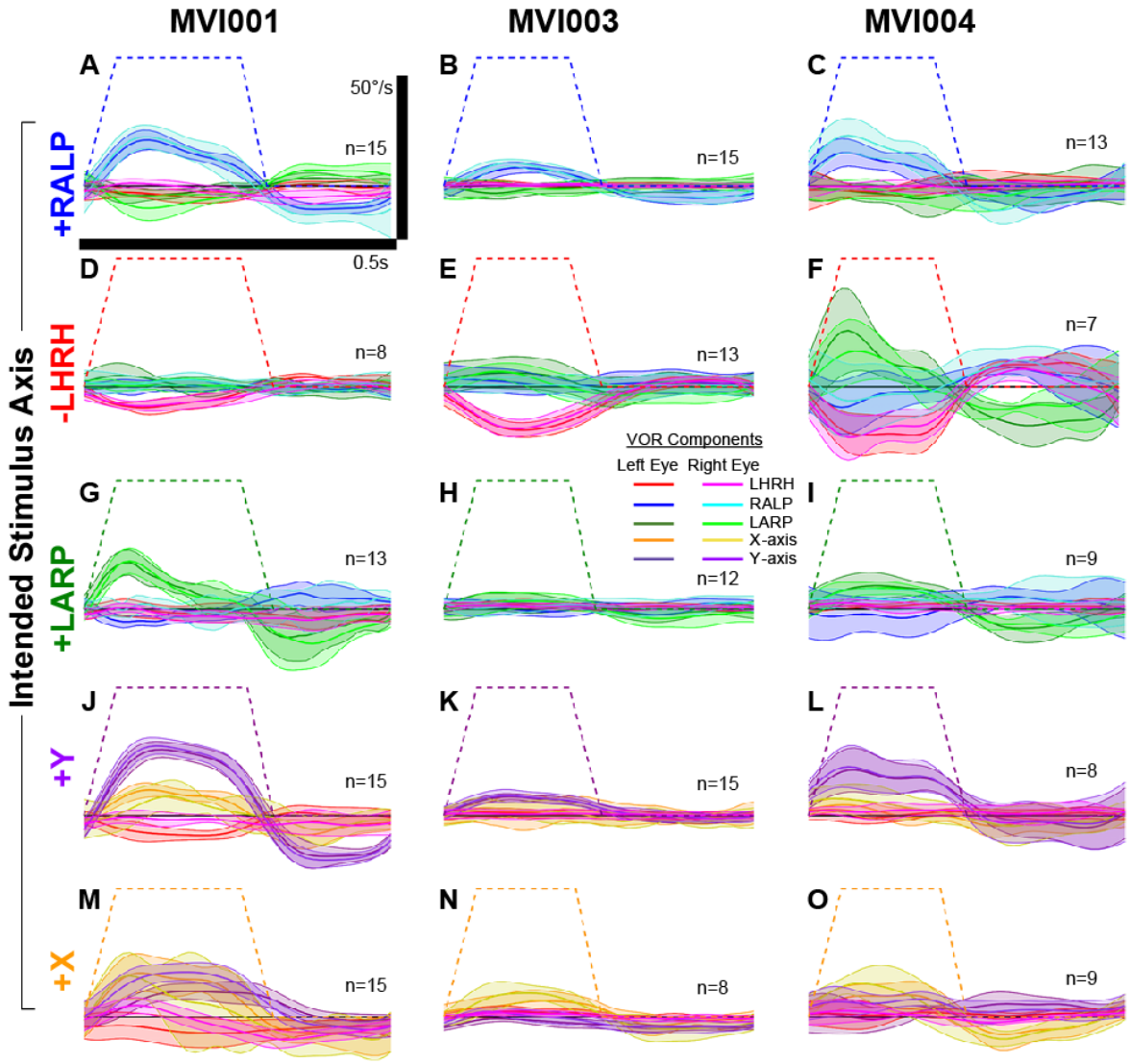


Figure S5. Responses to combinations of canal electrode approximate non-canal axes of rotation (MVI001, MVI003, and MVI004).
Mean±SD 3-dimensional vestibulo-ocular reflex velocity for n cycles of responses to coordinated stimulation via multiple electrodes can approximately encode arbitrary head rotation axes (MVI001, MVI003, and MVI004). (**A,D,G,J,M**) For subject MVI001 combining modulation of electrodes E3 and E11 out of phase produced a predominantly vertical eye movement (**J**), while modulating E3 and E11 in-phase (**M**) evoked a binocular eye movement with comparable positive vertical and torsional eye velocities (which is a principally RALP eye movement when converted into anatomic canal coordinates). (**B,E,H,K,N**) MVI003 produced eye velocities well aligned with the intended head motion vectors, though with smaller eye velocities compared to the other subjects. (**C,F,I,L,O**) Subject MVI004 produced selective eye movements for all tested vectors, save for stimuli targeting the LHRH axis (**F**) where an unintended LARP component grew in amplitude with the targeted horizontal eye movement.

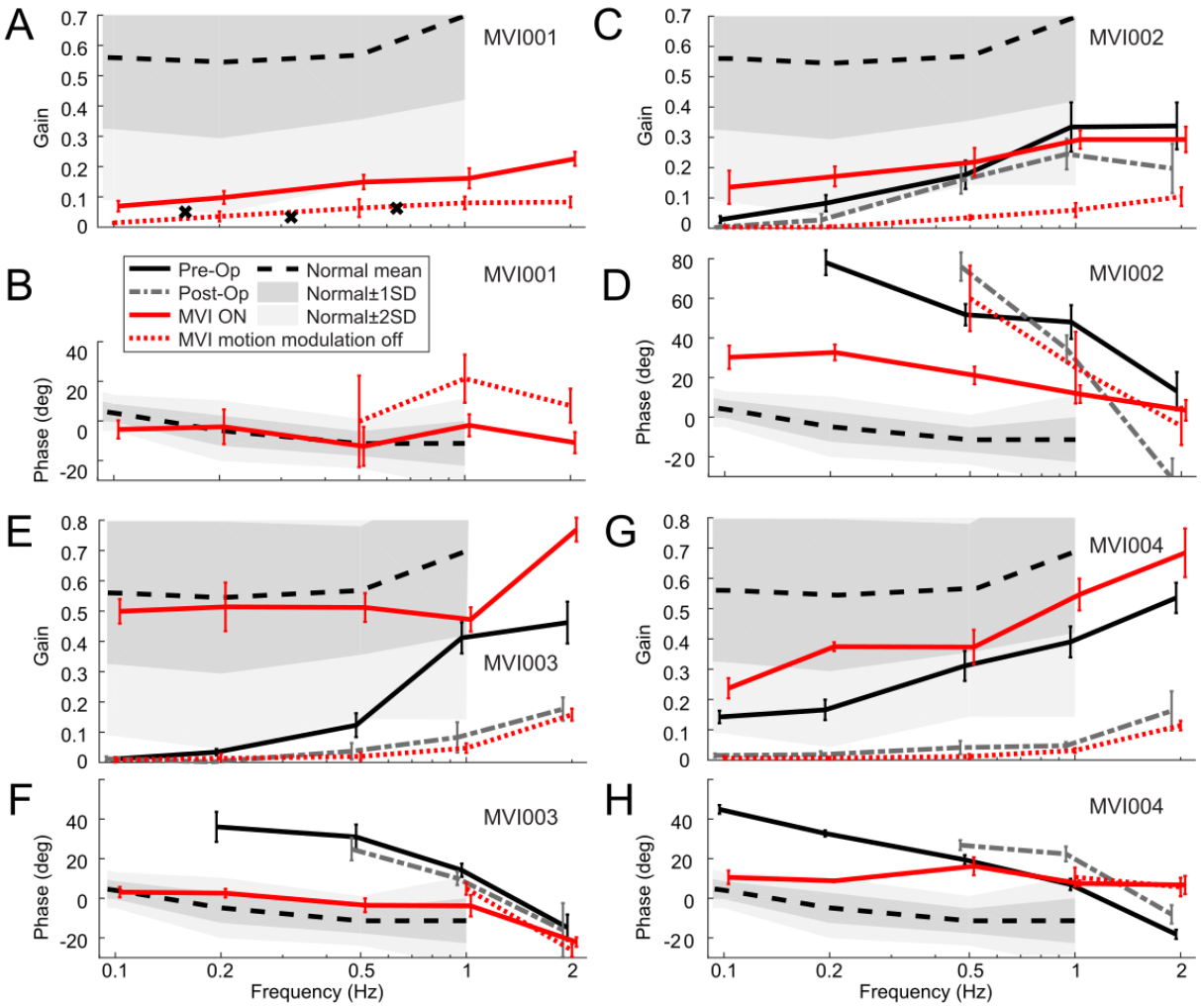


Figure S6. Vestibulo-ocular reflex gain and phase during whole-body rotation in darkness. Mean±SD horizontal vestibulo-ocular reflex (VOR) gain and phase lead for 0.1/0.2/0.5/1/2 Hz 100°/s peak rotary chair testing of subjects MVI001 (**A,B**), MVI002 (**C,D**), MVI003 (**E,F**) and MVI004 (**G,H**), performed before implantation (*Pre-Op*, for all subjects except MVI001); 3 wk post-implantation, just before initial MVI activation (*Post-Op*); and at the most recent study visit (after 812, 738, 782 and 354 days of continuous stimulation, respectively) with MVI motion-modulation on ("MVI ON") or with a placebo constant-rate stimulus ("modulation OFF"). Each data point is the cycle-averaged mean for n=2 to 32 (median 13) cycles. Phase was only computed for VOR responses >1.5°/s. Normative data range shown is from Wall et al. for 25 normal subjects age 50-69 years.(49) (**A**) For subject MVI001, who was not tested at 0.1/0.2/0.5/1/2 Hz pre-operatively, black x's show pre-op data from a test at another institution prior to study enrollment. "MVI ON" VOR gain significantly exceeds "Modulation OFF" gain at every tested frequency (Wilcoxon rank sum test at each frequency: $p<0.01$) but is still below normal. (**C**) Subject MVI002's MVI ON gain significantly increased vs. pre-op at 0.1-0.5Hz ($p<0.05$) but decreased at 1-2Hz ($p<0.05$). MVI ON gain exceeds modulation OFF gain at every frequency ($p<0.001$). (**E**) Subject MVI003's MVI ON gain significantly increased vs. pre-op and vs. modulation OFF at every frequency ($p<0.05$). (**B,D,F,H**) Phase responses improved toward normal over 0.1-1Hz.

Implanted Canal			Left Posterior			Left Horizontal			Left Anterior		
Electrode			E3	E4	E5	E6	E7	E8	E9	E10	E11
MVI001R019	Phase Duration (µs)		50	-	-	-	200	-	-	-	200
	Current intensity level (%)	10%	350	-	-	-	220	-	-	-	349
		20%	368	-	-	-	243	-	-	-	368
		30%	387	-	-	-	267	-	-	-	392
		40%	406	-	-	-	290	-	-	-	415
		50%	425	-	-	-	326	-	-	-	439
		60%	444	-	-	-	378	-	-	-	458
		70%	463	-	-	-	425	-	-	-	481
		80%	481	-	-	-	472	-	-	-	505
		90%	500	-	-	-	519	-	-	-	529
		100%	519	-	-	-	571	-	-	-	552
MVI002R004	Phase Duration (µs)		100	100	200	100	100	200	100	200	-
	Current intensity level (%)	10%	300	349	300	50	250	300	201	250	-
		20%	330	373	330	71	269	330	227	260	-
		30%	363	401	363	93	288	363	255	269	-
		40%	397	430	397	116	316	397	283	278	-
		50%	430	456	430	151	354	430	321	288	-
		60%	463	486	463	196	392	463	373	297	-
		70%	496	515	496	241	434	496	430	312	-
		80%	529	543	529	289	472	529	486	330	-
		90%	562	571	562	359	510	562	543	349	-
		100%	599	599	599	448	552	599	599	373	-
MVI003R140	Phase Duration (µs)		200	200	200	200	200	200	200	200	-
	Current intensity level (%)	14%	200	201	300	448	349	300	201	151	-
		29%	241	219	345	481	396	345	208	168	-
		43%	283	241	396	514	448	396	217	184	-
		57%	349	262	448	548	500	448	224	200	-
		71%	429	283	495	581	548	496	235	217	-
		86%	514	307	548	623	599	548	241	234	-
		100%	599	349	599	699	699	599	250	250	-
MVI004R201	Phase Duration (µs)		200	200	200	200	200	200	200	200	200
	Current intensity level (%)	14%	125	125	175	224	175	224	25	125	224
		29%	170	168	212	253	219	264	58	179	264
		43%	217	212	250	283	267	307	91	234	307
		57%	262	257	288	321	321	387	125	288	387
		71%	316	300	349	382	415	467	191	382	467
		86%	406	387	425	439	505	548	257	491	548
		100%	500	477	500	500	599	651	349	599	651

Table S1. Pulse parameters used for current fitting experiments.
Phase durations (µs) and current amplitudes (µA) used in fitting experiments (Figures 3, 4, S1 and S2) for all tested electrodes. Current intensity levels are expressed as fractions of the range from the current at which a subject first reported illusion of head movement to the maximum tolerated current. Each level was tested using 15-20 repetitions of 200 pulse/s pulse trains lasting 200 ms each and repeated every 500 ms. Subjects MVI003 and MVI004 were tested with only seven levels each due to time constraints.

	Subject ID	MVI001	MVI002	MVI003	MVI004
	Date Implanted	9/7/2016	11/30/2016	2/23/2017	1/5/2018
	Ear implanted	Left	Left	Left	Left
RALP	Electrode	E3	E3	E3	E3
	Phase duration μs	50	100	200	200
	Pulse current μA	390	600	600	406
	Pulse Rate pulses/s [pps]	100	100	100	100
LHRH	Electrode	E7	E6	E6	E6
	Phase duration μs	200	100	200	200
	Pulse current μA	570	160	620	320
	Pulse Rate pps	100	100	100	100
LARP	Electrode	E11	E9	E9	E11
	Phase duration μs	200	100	100	200
	Pulse current μA	480	600	400	550
	Pulse Rate pps	100	100	100	100

Table S2. Electrical stimulation parameters used during device activation.
Stimulation parameters used during initial device activation (onset of continuous stimulation) in all three canals for all subjects, corresponding to data in Figures 5 and S3. The table lists the active electrode contact number, phase duration, pulse amplitude, and stimulus pulse rate for each channel. At the time of initial activation, subjects were not yet acclimated to non-zero pulse rates. Continuous pulse rates representing zero head motion were subsequently increased to 150 pulse/s for subjects MVI002, MVI003 and MVI004 on all channels.

	Modulated Parameter	Subject ID	MVI001	MVI002	MVI003	MVI004
		Date of Testing	11/02/2016	1/24/2017	04/18/2017	02/27/2018
		Days Since Activation	**56**	**55**	**54**	**53**
RALP		Electrode ID	E3	E3	E3	E3
		Phase duration µs	200	100	200	200
	Current	Map Type	Flat	Linear	Flat	Linear
		Minimum current µA	N/A	500	N/A	125
		Zero-motion current µA	450	600	600	406
		Maximum current µA	N/A	700	N/A	500
		Compression Factor	N/A	N/A	N/A	N/A
	Rate	Map Type	Sigmoidal	Sigmoidal	Sigmoidal	Sigmoidal
		Minimum pulses/s [pps]	0	0	0	0
		Zero-motion rate pps	100	150	150	150
		Maximum rate pps	400	450	450	450
		Compression Factor	2	5	2	5
LHRH		Electrode ID	E7	E6	E6	E6
		Phase duration µs	200	100	200	200
	Current	Map Type	Flat	Linear	Sigmoidal	Sigmoidal
		Minimum current µA	N/A	150	610	220
		Zero-motion current µA	600	350	620	320
		Maximum current µA	N/A	550	700	470
		Compression Factor	N/A	N/A	20	3
	Rate	Map Type	Sigmoidal	Sigmoidal	Sigmoidal	Sigmoidal
		Minimum pulses/s	0	0	0	0
		Zero-motion rate pps	100	150	150	150
		Maximum rate pps	400	450	450	450
		Compression Factor	2	5	2	5
LARP		Electrode ID	E11	E9	E9	E11
		Phase duration µs	200	100	100	200
	Current	Map Type	Flat	Linear	Sigmoidal	Linear
		Minimum current µA	N/A	500	400	220
		Zero-motion current µA	700	600	400	550
		Maximum current µA	N/A	700	420	600
		Compression Factor	N/A	N/A	20	N/A
	Rate	Map Type	Sigmoidal	Sigmoidal	Sigmoidal	Sigmoidal
		Minimum pulses/s [pps]	0	0	0	0
		Zero-motion rate pps	100	150	150	150
		Maximum rate pps	400	450	450	450
		Compression Factor	2	5	2	5

Table S3. MVI stimulation parameters for each subject during testing after 8-weeks of continuous, motion-modulated electrical stimulation. Stimulation parameters used by each subject's MVI for pulse-rate- and pulse-amplitude-modulation at test session8 weeks after activation (yielding data in Figures 6 and S4).

	Modulated Parameter	Subject ID	MVI001	MVI002	MVI003	MVI004
		Date of Testing	10/29/2018	11/12/2018	03/27/2019	12/03/2018
		Days Since Activation	**812**	**738**	**782**	**354**
RALP		Electrode ID	E3	E3	E3	E3
		Phase duration µs	200	100	200	200
	Current	Map Type	Flat	Linear	Flat	Linear
		Minimum current µA	N/A	500	N/A	125
		Zero-motion current µA	450	600	600	406
		Maximum current µA	N/A	700	N/A	500
		Compression Factor	N/A	N/A	N/A	N/A
	Rate	Map Type	Sigmoidal	Sigmoidal	Sigmoidal	Sigmoidal
		Minimum pulses/s [pps]	0	0	0	0
		Zero-motion rate pps	100	150	150	150
		Maximum rate pps	400	450	450	450
		Compression Factor	5	5	5	5
LHRH		Electrode ID	E7	E6	E7	E6
		Phase duration µs	200	100	200	200
	Current	Map Type	Flat	Linear	Sigmoidal	Sigmoidal
		Minimum current µA	N/A	150	350	220
		Zero-motion current µA	600	350	400	320
		Maximum current µA	N/A	550	620	470
		Compression Factor	N/A	N/A	10	3
	Rate	Map Type	Sigmoidal	Sigmoidal	Sigmoidal	Sigmoidal
		Minimum pulses/s [pps]	0	0	0	0
		Zero-motion rate pps	100	150	150	150
		Maximum rate pps	400	450	450	450
		Compression Factor	5	5	5	5
LARP		Electrode ID	E11	E9	E9	E11
		Phase duration µs	200	100	200	200
	Current	Map Type	Flat	Linear	Sigmoidal	Linear
		Minimum current µA	N/A	500	180	220
		Zero-motion current µA	700	600	200	550
		Maximum current µA	N/A	700	300	600
		Compression Factor	N/A	N/A	10	N/A
	Rate	Map Type	Sigmoidal	Sigmoidal	Sigmoidal	Sigmoidal
		Minimum pulses/s [pps]	0	0	0	0
		Zero-motion rate pps	100	150	150	150
		Maximum rate pps	400	450	450	450
		Compression Factor	5	5	5	5

Table S4. MVI stimulation parameters for each subject during most recent test session

Electrical stimulation parameters used by each subject's MVI for pulse-rate- and pulse-amplitude-modulation at the time of the most recent test session (yielding data in Figures 8 and S6).

Video S1. Evoked eye movements during manipulation of head worn unit on MVI004.

After 5 days of continuous, motion-modulation prosthetic electrical stimulation via electrodes in the three semicircular canal of the left ear, pitch rotation of subject MVI004's head worn unit (HWU) about the subject's +Y axis evokes compensatory vertical eye movements.