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Lateral semi-circular canal pressures during cochlear implant electrode insertion: A possible mechanism for postoperative vestibular loss

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Abstract

Hypothesis—Insertion of cochlear implant electrodes generates transient pressure spikes within the vestibular labyrinth equivalent to high intensity acoustic stimuli.

Background—Though cochlear implant (CI) surgery is regarded as having low-risk of impacting the vestibular system, several studies have documented changes in vestibular function after implantation. The mechanism of these changes is not understood. We have previously established that large, potentially-damaging pressure transients can be generated in the cochlea during electrode insertion, but whether pressure transients occur within the vestibular labyrinth has yet to be determined. Here, we quantify the exposure of the vestibular system to potentially-damaging pressure transients during CI surgery.

Methods—Five human cadaveric heads were prepared with an extended facial recess and implanted sequentially with eight different CI electrode styles via a round window approach. Fiber-optic sensors measured intralabyrinthine pressures in scala vestibuli (SV), scala tympani (ST), and the lateral semicircular canal (LSCC) during insertions.

Results—Electrode insertion produced a range of high-intensity pressure spikes simultaneously in the cochlea and LSCC with all electrodes tested. Pressure transients recorded were found to be significantly higher in the vestibular labyrinth than the cochlea and occurred at peak levels known to cause acoustic trauma.

Conclusion—Insertion of CI electrodes can produce transients in intralabyrinthine fluid pressure levels equivalent to high-intensity, impulsive acoustic stimuli. Results from this investigation affirm the importance of atraumatic surgical techniques and suggest that in addition to the cochlea, the vestibular system is potentially exposed to damaging fluid pressure waves during cochlear implantation.

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Conflict of Interest Statement:

SPC is a consultant for Cochlear Corporation.

Keywords

Cochlear implant; intracochlear pressures; vestibular loss; semicircular canals

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Introduction

Use of cochlear implants (CI) to treat severe to profound sensorineural hearing loss has increased as excellent communication outcomes have made it the modality of choice. Though cochlear implantation is generally considered as a low-complication procedure¹, recent reports have described a wide range of incidence of adverse outcomes affecting balance, with estimates as low as 0.3%¹⁻⁴ and as high as 75% of patients^{5,6}.

Changes in vestibular function following cochlear implantation can be evaluated with both subjective and objective measures that do not always correlate with vestibular symptoms^{5,7-11}. The clinical implications of changes can be difficult to assess and the mechanism of implantation-related loss of vestibular function remains poorly understood.

A series of recent investigations¹²⁻¹⁶ reported pressure changes within the cochlea during the placement of CI electrodes. These studies hypothesized that insertion-induced pressure changes can stimulate the cochlea in an “acoustic” manner, with levels at times equivalent to those known to cause acoustic trauma. Given the interconnected nature of the fluid spaces in the inner ear, it would follow that these same high level pressure changes may also propagate through the vestibular labyrinth. Though we have previously demonstrated that high-level acoustic stimuli can cause pressure changes in the vestibular system in the absence of pathologic canal dehiscence in cadaveric specimen¹⁷, it is unknown if CI insertion-related pressure changes are transmitted to the vestibular end organs. To the best of our knowledge, no intralabyrinthine measurements specifically examining electrode insertion-related pressure changes within the vestibular system have been reported previously. Here, we aim to overcome this knowledge gap and characterize intralabyrinthine pressure changes associated with CI insertions in both the cochlear and vestibular systems of cadaveric specimen.

Methods

Data was collected and analyzed from five ears in fresh-frozen whole cadaveric heads. All specimens were confirmed to have intact temporal bones and verified to have no history of middle ear disease (MD Global, Aurora, CO, USA). The use of cadaveric human tissue was in compliance with the University of Colorado Anschutz Medical Campus Institutional Biosafety Committee and Review Board (COMIRB EXEMPT #14-1464).

Temporal Bone Preparation

Temporal bone specimens were prepared as previously described by our laboratory^{12,17-24}. After overnight thawing in warm water, intact whole cadaveric heads were inspected to rule out injury or disease. A canal-wall-up mastoidectomy with an extended facial recess approach was performed. The cochlear promontory was blue-lined near the oval and round windows, as was the bone overlying the lateral semicircular canal. The specimens were

suspended with a Mayfield Clamp (Integra Lifesciences Corp., Plainsboro, NJ, U.S.A.) attached to a stainless-steel baseplate. Cochleostomies into the scala tympani (ST), scala vestibuli (SV), and the lateral semicircular canal (LSCC) were created using a fine pick under a droplet of water, and fiber-optic pressure sensors (FOP-M260-ENCAP, FISO Inc., Quebec, QC, Canada) were inserted (see Fig. 1) using micromanipulators (David Kopf Instruments, Trujunga, CA, U.S.A.) rigidly mounted onto the head clamp. Probes were sealed in place with alginate dental impression material (Jeltrate; Dentsply International Inc., York, PA).

Baseline measurements of stapes velocity (V_{Stap}) and intralabyrinthine pressures (P_{IL}) were made to acoustic stimuli. Out-of-plane velocities were measured with a single-axis laser Doppler vibrometer (LDV) (OFV-534 & OFV-5000; Polytec Inc., Irvine, CA) mounted to a dissecting microscope (M400; Leica Microsystems, Buffalo Grove, IL, United States). Microscopic retro-reflective glass beads (P-RETRO 45–63 μm dia., Polytec Inc., Irvine, CA) were placed on the stapes to enhance measurement of the LDV signal [41, 42].

Electrode insertions were performed primarily by resident physicians under the guidance of Neurotology faculty. Implant electrodes were inserted into the cochlea as described in previous experiments¹². Cochleostomies for CI electrode insertion were made in the round window with a fine pick. Electrodes were inserted by hand underwater using an insertion tool, if provided by the manufacturer, and with a pair of fine forceps otherwise. CI electrodes used in these experiments were: Nucleus CI24RE Contour Advance (NCA; Cochlear Ltd, Sydney, Australia), Nucleus CI422 Slim Straight inserted to 20 mm (CI422; Cochlear Ltd, Sydney, Australia), Nucleus Hybrid L24 (HL24; Cochlear Ltd, Sydney, Australia), Nucleus CI532 Slim Modiolar (CI532; Cochlear Ltd, Sydney, Australia), HiFocus Mid-Scala (MS; Advanced Bionics AG, Stäfa, Switzerland), FLEX 24 (F24; Med-El, Innsbruck, Austria), FLEX 28 (F28; Med-El, Innsbruck, Austria), and Digisonic SP EVO (EVO; Oticon Medical, Vallauris, France). Electrodes were inserted one after another, in random order.

Sound Presentation, Data Acquisition, and Data Analysis

All experiments were performed in a double-walled, sound-attenuating chamber (IAC Inc., Bronx, NY). Acoustic stimuli were presented as described previously^{12,17–24} in order to generate baseline transfer functions used to calculate estimated equivalent ear canal pressures for each insertion. Briefly, digitally generated stimuli were presented via a closed-field magnetic speaker (MF1; Tucker-Davis Technologies Inc., Alachua, FL) coupled directly to the ear with a foam insert altered to accommodate flexible speaker tubing. The speaker was driven by an external sound card (Hammerfall Multiface II, RME, Haimhausen, Germany) amplified with one channel of a stereo amplifier (TDT SA1). Sound intensity in the ear canal was measured with a probe-tube microphone (type 4182; Bruel & Kjaer, Nærum, Denmark), also placed through the modified foam earplug. Baseline acoustic transfer functions were generated from presentation of short (1 s duration) tone pips between 100 and 12000 Hz ramped on and off with one half (5 ms) of a Hanning window. Input from the microphone, LDV, and pressure sensors were simultaneously captured via the sound card analog inputs. The fiber-optic sensors were factory calibrated and sensitivity was verified by normalizing the sensor response to LDV displacement measurements made while generating

a known motion in a cup of fluid controlled by a B&K mini-shaker (Brüel & Kjær Type 4810, Nærum, Denmark). The magnitude of the LDV signal was adjusted using a cosine correction ($1/\cos(\theta)$) based on an estimate of the difference in angle between the primary axis of the stapes and the orientation of the LDV laser ($\sim 45^\circ$). Signals acquired were band-pass filtered between 15–15000 Hz with a second order Butterworth filter.

Baseline temporal bone measurements are shown as acoustic transfer functions, which are generated by measuring the response of a system to a known stimulus. In this case, we use acoustic stimuli and characterize the response of the external, middle, and inner ears utilizing intralabyrinthine pressures. We are then able to use the resulting transfer function to generate a filter and calculate an estimate of the intensity in decibels sound pressure level (dB SPL) of acoustic stimulus in the external ear canal (EAC) that would be required to generate a given pressure measurement¹². These equivalent sound pressure levels in the ear canal (dB SPL Eq) are provided as peak pressure (in Pa) observed, and in dB SPL Peak ($20 * \log_{10}(\text{peak pressure}/20\mu\text{Pa})$). Differential pressure (P_{Diff}), which is thought to be the input to the cochlear partition and the driving force for auditory transduction^{25–29}, is calculated as the difference in pressures between scala vestibuli and scala tympani. Responses were only analyzed for recordings with a signal to noise ratio greater than 6 dB, and SNR was higher than 10 dB for the majority of the recordings.

Intralabyrinthine pressure changes with CI electrode insertion were determined according to methods previously detailed^{12,19}. Briefly, to investigate maximum pressure exposure, the absolute peak pressures were found in each recording using the *findpeaks* function in Matlab (R2014b; The Mathworks, Inc., Natick, MA, U.S.A.). Filter criteria required peaks to a minimum peak amplitude of the greater of either four times the standard deviation within each recording or 100 Pa. Peaks were only assessed if they occurred within 10 ms of one another across recording locations to avoid inclusion of artifacts.

Results

Acoustic closed-field transfer functions

Baseline responses of V_{Stap} and P_{IL} were assessed after placement of intralabyrinthine pressure probes and prior to making the RW cochleostomy for CI insertion in order to verify the condition of each temporal bone. Mean ($\pm$ SEM) closed-field acoustic transfer function magnitudes for specimens are shown in Figure 2. Responses are overlain onto the 95% confidence interval for stapes velocities³⁰, and the mean and the range of responses observed for intracochlear pressures reported previously by Nakajima et al²⁶. Responses collected were consistent with previous reports^{12,18,20–24}.

Closed-field acoustic transfer functions were also determined for the lateral semicircular canal. Given the relatively smaller number of specimens in this study ($n=5$), we elected to include lateral canal measurements from previous investigation of our lab¹⁷ in the calculation of the mean lateral canal transfer function. Figure 3 shows both the individual lateral canal pressure responses for all specimens as well the mean $\pm$ standard error of the mean.

Peak Intralabyrinthine Pressures During CI Electrode Insertion

Figure 4 shows a summary of the absolute maximum peak pressures observed in all recordings in all specimens for all electrodes tested. Figure 4A shows the unfiltered recorded raw pressures while the maximum peak pressures in estimated EAC SPL are illustrated in Figure 4B. Estimated EAC pressures recorded during electrode insertion ranged up to 169 dB SPL Peak; these levels are consistent with data previously observed in our lab¹² and comparable in magnitude to high intensity acoustic stimuli.

Results of a one-way ANOVA, with peak pressures serving as the dependent variable and recording location as the independent variable, revealed a significant effect of electrode location on pressure for both raw pressures and estimated EAC SPL ($F=8.18$, $p<0.0001$ and $F=9.29$, $p<0.0001$, respectively). Post hoc Tukey honest significant difference (hsd) pairwise comparisons showed significantly higher pressure in the lateral canal than scala tympani and differential pressure, as well as significantly higher pressure in scala vestibuli than tympani in unfiltered data (see Table 1). Estimated equivalent EAC pressures were significantly higher in the lateral semicircular canal than the three other recording locations (see Table 2).

A breakdown of the absolute maximum peak pressures by electrode type is shown in Figure 5. Figure 5A shows the unfiltered recorded raw pressures while the maximum peak pressures in estimated EAC SPL are illustrated in Figure 5B. In general, the presence of high peak pressure transients was reasonably consistent across electrode styles (peak EAC SPL Eq exceeded 164 dB SPL Peak in all cases); however, differences were noted in the prevalence of large transients.

Peak pressures served as the dependent variable and electrode style acted as the independent variables for a one-way ANOVA for both unfiltered and estimated EAC SPL conditions. Significant differences were noted in both raw pressures and estimated EAC pressures for electrode type ($F=8.31$, $p<0.0001$ and $F=13.81$, $p<0.0001$, respectively). Post hoc testing also defined the effect of electrode type, with significant differences between electrode types illustrated in Figure 5 and Tables 3 and 4.

Discussion

Our results suggest that placement of CI electrodes into scala tympani via a round window approach can generate intralabyrinthine pressures in both the cochlea and vestibular organs comparable to high-intensity acoustic stimulation. Here, as in our previous study investigating pressures in the semicircular canals¹⁷, we confirm that pressure changes do indeed propagate from the cochlea to the vestibular labyrinth in normal temporal bones that are free of pathology, such as canal dehiscence. Estimated peak equivalent ear canal pressure was as high as 169 dB SPL, with significantly higher exposures associated with measurements made from the lateral semicircular canal. Additionally, two perimodiolar electrode styles (the NCA and the CI532) showed significantly higher pressures when compared with the remaining electrodes in both unfiltered pressures and the estimated equivalent SPL measurements. This trend for higher pressures with perimodiolar electrodes was present in our previous study¹², but the difference was not significant for all

perimodiolar electrodes as it is in results presented here. As previously noted, not every insertion generated a pressure spike above the equipment noise floor.

Vestibular Loss After Cochlear Implantation

Transient postoperative dizziness or imbalance is a commonly reported side effect of cochlear implant surgery¹, while prolonged or persistent symptoms are more rarely described. The reported rates of postoperative changes in vestibular function varies widely, with some studies reporting little to no changes^{1–3} and others seeing effects in the majority of patients^{5,6}. Methods for evaluation of postoperative dizziness generally fall into one of two categories: objective assessments of vestibular function, such as the videonystagmography (VNG), head impulse test (HIT), vestibular evoked myogenic potentials (VEMP), or caloric testing, and subjective testing, which includes both informal questionnaires and validated balance surveys, such as the Dizziness Handicap Index (DHI).

Postoperative loss of vestibular function as measured by postoperative changes in HIT ranges from 3.6–30%^{10,31}, though one study showed no significant change in postoperative HIT testing³. The range of postoperative change in caloric weakness was even greater, from estimates as low as 6.3% ranging upward to 60%^{5,7,8,10,32–35}. Systematic review of caloric changes found 34% of patients demonstrated new-onset unilateral canal weakness³⁶. Changes in VEMP, which include reduction in response amplitudes, development of unilateral weakness, or loss of response postoperatively was found to be most common, occurring in 31–60% of patients^{5,9,10,37}. Age of patient at implantation may be an important factor as one review saw significant changes in objective testing only in patients over 60 years of age³⁸. Another study saw no change between pre- and post-operative VEMP, caloric, or HIT, though the studied patients were all under the age of 5 at the time of implantation².

Subjective assessment of patient dizziness with the validated DHI can confirm worsening dizziness and resultant quality of life³¹; however, degree of handicap is frequently low^{4,11} and in some cases demonstrates no postoperative change³⁵. Direct questioning regarding the presence of postoperative dizziness and vertigo or the use of specifically-designed questionnaires places estimates of new symptoms between 24–53%^{5,7–9,32,39–43}, though some estimates are as low as 3%⁴⁴ and as high as 75%⁶.

In addition to examining objective and subjective vestibular changes independently, several studies have attempted to correlate changes on balance function tests with patient-reported symptoms. Mismatch between reported symptoms of balance changes and objective testing has been seen in several investigations, where patients complaining of new dizziness show no new abnormalities on vestibular function testing and those with objective findings do not necessarily report vertigo or imbalance^{5,7–11,44}. One study demonstrated a significant change in DHI associated with changes in vestibular function testing, though this was for a relatively small number of patients (n=10)³¹.

Possible Mechanisms for Vestibular Loss

Though many clinical studies have reviewed both subjective and objective changes in balance and vestibular function following cochlear implantation, the exact mechanism

underlying new onset vestibular remains unclear. Here, we propose that pressure changes generated from implant insertion can cause a stimulus similar to acoustic trauma, which may potentially cause disruption of the delicate sensory epithelium of the inner ear. Results from this study, as well as previous investigation¹⁷, have confirmed that pressure energy is propagated from the cochlear to the vestibular labyrinths in the absence of a third window. Others have suggested that direct trauma to the epithelium from contact with the electrode or translocation through the basilar membrane and subsequent mixing of endolymph and perilymph may explain changes in vestibular neurophysiology^{36,45}. Though not a direct measure of trauma, no association between insertion depth and vestibular function has been found⁴⁶.

Several studies have examined histopathological changes of the cochlea following cochlear implantation, but few have looked at the vestibular system. When the vestibular end organs of implanted patients are examined post-mortem, patterns that can be attributed to implantation include saccular membrane distortion^{47,48}, as well as fibrosis, calcification, and ossification⁴⁸. These changes are most often seen in the saccule, which is not surprising given the anatomic proximity to the cochlear implant, but have also been noted with less frequency in the utricle and semicircular canal⁴⁸. No deafferentation in the peripheral vestibular system has been seen on histologic examination⁴⁷.

Multiple theoretical mechanisms have been proposed to explain immediate or acute-onset vestibular symptoms after cochlear implantation. Benign positional vertigo has also been reported in the postoperative period in a small number of patients^{42,49}, and has been proposed to be due either to inadvertent entry of bone dust into the labyrinth or dislodging of otoconia⁵⁰. Iatrogenic perilymphatic fistula has also been proposed as a potential mechanism for postoperative vertigo^{5,43,51}, and a single case study has demonstrated this etiology⁵². Spreading of electric current from the implant to stimulate the otolithic organs has been described to cause dizziness in some patients who did not report preoperative sound-induced vertigo^{32,33,39,53}.

In addition to immediate onset changes in vestibular function, a delayed-onset vertigo has been reported in a subset of patients. These individuals have vertigo spells starting more than one month postoperatively, associated with Meniere's-like symptoms of fluctuating hearing changes and tinnitus^{42,51}. The presence of cochlear hydrops presenting with saccular membrane abnormalities on histology has led some to hypothesize that obstruction of endolymphatic flow can cause dizziness in implanted patients⁴⁷.

Source for Pressure Transients

We have previously theorized multiple sources for pressure transient generation during electrode insertion¹² and multiple potential mechanical mechanisms have been previously discussed for cochlear trauma during CI electrode insertion. Basilar membrane translocation of the electrode from scala tympani to scala vestibuli would likely generate pressure changes⁵⁴, though this seems unlikely to be the sole mechanism as it would likely generate only a single transient and we frequently see multiple trains of spikes within each recording. Contact with the crista fenestra or lateral wall may also be a source for implantation trauma⁵⁵, but this seems unlikely to be the only mechanism given the presence of spikes

with both lateral wall and perimodiolar style electrodes. The size and shape of the round window cochleostomy may also contribute to fluid build-up and release as the non-compressible cochlear fluid may face increased impedance releasing through a smaller round window cochleostomy⁵⁶. While data presented here is not able to determine to what degree these mechanisms may play a role, it is likely that some combination of the above-mentioned theories represent the true source for the pressure transients and additional study is needed in this area to determine the relevance of each proposed mechanism.

Limitations

Although this investigation represents the first description of pressure measurements made in both the cochlea and vestibular end organs resulting from insertion of CI electrodes, several limitations in our methodology limit the ability to generalize results from this study. Though full cephalic represent a more complete model than isolated temporal bones, cadaveric specimens may be subject to post-mortem degradation. Regulation of intracochlear pressure via the cochlear aqueduct in living humans is not present in cadaveric specimens and pressure changes from physiologic baseline cannot be verified. Additionally, while it is possible that the very nature of creating openings into the otic capsule may alter pressure dynamics within the labyrinth, it does not appear to be affected by a greater number of probe insertions (up to 4 in previous investigation)¹⁷. Two cochlear probes not only provide important acoustic baseline measurements and are necessary for the exclusion of artifacts, we think that the scala vestibuli probe may roughly estimate exposures of the otolithic organs and may provide insight into the relative potential trauma to each subsite of the vestibular system.

Additional testing on the specimens after insertions, such as imaging or histological studies, may provide additional information regarding the implant trajectory and damage to anatomical structures leading to further insight into the mechanism of this potential source of trauma, but these tools are not available for this study. The mechanism proposed here is speculative, as directly linking observed pressure transients to cellular level changes remains elusive.

Our objective in this study was to investigate a possible mechanism for vestibular trauma and not evaluate the effect of surgeon experience; however, one important consideration is that we may be overestimating potential trauma exposure given that electrode insertions were performed primarily by resident physicians. Additionally, it is possible given the repeated nature of insertions within a single specimen, anatomical damage occurred to specimen during initial insertions that affected subsequent measurements. Though this is not possible to confirm without additional anatomical dissection, we have attempted to mitigate any effect by randomizing the order of electrodes inserted. And finally, while we propose a single mechanism for postoperative changes in vestibular function, we also appreciate that studies performed to date have shown that these changes are likely to be multifactorial.

Conclusions

Patients undergoing cochlear implantation for the treatment of severe to profound hearing loss risk the development of postoperative dizziness and vertigo. Data presented here have

demonstrated that pressure changes in the cochlea and lateral semicircular canal resulting implant electrode insertion may be of sufficiently high amplitude to cause injury. Our data also suggest that these pressures may be higher in the vestibular system than the cochlea, though additional data is needed to further investigate this association. This study underscores the importance of atraumatic surgical techniques to minimize likelihood of generation of transient intralabyrinthine pressure spikes and confirms that the vestibular end organs, as well as the cochlea, are at risk for pressure-related trauma from cochlear implant insertion.

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Figure 1.

Photomicrograph of the right ear in a single specimen during data collection. Through the extended facial recess, the anatomical landmarks (stapes and round window niche) can be visualized. Pressure probes (P_{SV} , P_{ST} , P_{LSCC}) were placed in cochleostomies in scala vestibuli, scala tympani, and the lateral semicircular canal. All cochleostomies were sealed with dental impression material. Implant electrode (CI) was inserted into the round window.

Figure 2.

Mean baseline stapes velocity (V_{Stap}), scala vestibuli pressure (P_{SV}), scala tympani pressure (P_{ST}), and differential pressure (P_{Diff} ; $P_{\text{SV}} - P_{\text{ST}}$) transfer function magnitudes to air-conducted stimuli. Responses recorded in specimens are shown normalized to the SPL recorded in the ear canal (P_{EAC}) and are superimposed onto the 95% CI and range of responses (gray bands) observed previously^{26,30}. Colored bands indicate $\pm$ standard error of the mean.

Figure 3.

Individual and mean baseline and lateral semicircular canal pressure (P_{LSCC}) transfer function magnitude to air-conducted stimuli. Responses recorded in specimens are shown normalized to the SPL recorded in the ear canal (P_{EAC}). Data collected from individual specimens are shown as colored lines, the black line represents the mean, and gray bands indicate $\pm$ standard error of the mean.

Figure 4.

Summary of peak sound pressure levels observed in all specimens during all electrode insertions. Unfiltered peak intracochlear pressure measurements (A) and estimated EAC pressures (B) are shown for each pressure recording as a function of recording location. Box plots represent the median $\pm$ 25% of the range of pressures observed, whiskers show the full range of the estimated distribution, and +'s mark outliers. Significant differences between groups are indicated with asterisks (* $p < 0.05$).

Figure 5.

Peak sound pressure levels observed in all specimens during all electrode insertions as a function of recording location and electrode type. Unfiltered peak intracochlear pressure measurements (A) and estimated EAC pressures (B) are shown in the upper and lower panels, respectively. Box plots represent the median $\pm$ 25% of the range of pressures observed, whiskers show the full range of the estimated distribution, and '+'s mark outliers. Significant differences between groups are indicated with asterisks (* $p < 0.05$).

Posthoc Tukey honest-significant difference (hsd) pairwise comparisons for raw pressure by location

Table 1

Electrode	Electrode	Lower limit 95% CI for true mean difference	Difference between estimated group means	Upper limit 95% CI for true mean difference	p-value
1 (Diff)	2 (LSCC)	-1415.16	-773.52	-131.89	0.0105*
1 (Diff)	3 (ST)	-119.13	497.41	1113.95	0.1621
1 (Diff)	4 (SV)	-815.40	-198.86	417.68	0.8409
2 (LSCC)	3 (ST)	602.28	1270.93	1939.58	<0.0001**
2 (LSCC)	4 (SV)	-93.99	574.66	1243.32	0.1210
3 (ST)	4 (SV)	-1340.88	-696.27	-51.65	0.0283*

Table 2
Posthoc Tukey honest-significant difference (hsd) pairwise comparisons for SPL by location

Electrode	Electrode	Lower limit 95% CI for true mean difference	Difference between estimated group means	Upper limit 95% CI for true mean difference	p-value
1 (Diff)	2 (LSCC)	-1041.61	-684.10	-326.60	<0.0001**
1 (Diff)	3 (ST)	-581.22	-254.98	71.27	0.1850
1 (Diff)	4 (SV)	-380.59	-54.34	271.91	0.9737
2 (LSCC)	3 (ST)	61.61	429.13	796.64	0.0144*
2 (LSCC)	4 (SV)	262.25	629.76	997.28	0.0001**
3 (ST)	4 (SV)	-136.55	200.64	537.82	0.4202

Posthoc Tukey honest-significant difference (hsd) pairwise comparisons for unfiltered pressure by electrode pairs

Table 3

Electrode	Electrode	Lower limit 95% CI for true mean difference	Difference between estimated group means	Upper limit 95% CI for true mean difference	p-value
1 (NCA)	2 (CI532)	-599.74	654.89	1909.51	0.7613
1 (NCA)	3 (EVO)	825.19	1847.57	2869.94	<0.0001**
1 (NCA)	4 (F24)	672.48	1705.24	2738.00	<0.0001**
1 (NCA)	5 (F28)	462.66	1690.46	2918.25	0.0008**
1 (NCA)	6 (HL24)	874.26	2195.24	3516.22	<0.0001**
1 (NCA)	7 (MS)	202.05	1487.66	2773.27	0.0107*
1 (NCA)	8 (CI422)	1165.32	2230.98	3296.64	<0.0001**
2 (CI532)	3 (EVO)	237.57	1192.68	2147.79	0.0038**
2 (CI532)	4 (F24)	84.13	1050.35	2016.57	0.0221*
2 (CI532)	5 (F28)	-136.80	1035.57	2207.95	0.1295
2 (CI532)	6 (HL24)	270.72	1540.36	2809.99	0.0058**
2 (CI532)	7 (MS)	-400.02	832.77	2065.57	0.4496
2 (CI532)	8 (CI422)	574.78	1576.09	2577.40	0.0001**
3 (EVO)	4 (F24)	-778.53	-142.33	493.87	0.9976
3 (EVO)	5 (F28)	-1076.69	-157.11	762.47	0.9996
3 (EVO)	6 (HL24)	-693.06	347.67	1388.41	0.9727
3 (EVO)	7 (MS)	-1355.37	-359.91	635.55	0.9579
3 (EVO)	8 (CI422)	-304.91	383.41	1071.72	0.6950
4 (F24)	5 (F28)	-945.89	-14.78	916.34	1.0000
4 (F24)	6 (HL24)	-560.94	490.00	1540.94	0.8516
4 (F24)	7 (MS)	-1223.71	-217.58	788.55	0.9980
4 (F24)	8 (CI422)	-177.92	525.74	1229.39	0.3134
5 (F28)	6 (HL24)	-738.34	504.78	1747.91	0.9230
5 (F28)	7 (MS)	-1408.28	-202.80	1002.68	0.9996
5 (F28)	8 (CI422)	-426.96	540.52	1507.99	0.6917
6 (HL24)	7 (MS)	-2007.85	-707.58	592.68	0.7200

Table 4

Posthoc Tukey honest-significant difference (hsd) pairwise comparisons for SPL by electrode pairs

Electrode	Electrode	Lower limit 95% CI for true mean difference	Difference between estimated group means	Upper limit 95% CI for true mean difference	p-value
1 (NCA)	2 (CI532)	-583.16	21.02	625.19	1.0000
1 (NCA)	3 (EVO)	506.23	1015.30	1524.37	<0.0001**
1 (NCA)	4 (F24)	417.56	923.97	1430.37	<0.0001**
1 (NCA)	5 (F28)	-55.62	599.79	1255.19	0.1016
1 (NCA)	6 (HL24)	344.28	1078.28	1812.28	0.0002**
1 (NCA)	7 (MS)	12.63	678.23	1343.84	0.0422*
1 (NCA)	8 (CI422)	661.53	1185.78	1710.03	<0.0001**
2 (CI532)	3 (EVO)	528.71	994.29	1459.87	<0.0001**
2 (CI532)	4 (F24)	440.28	902.95	1365.62	<0.0001**
2 (CI532)	5 (F28)	-43.46	578.77	1201.00	0.0902
2 (CI532)	6 (HL24)	352.72	1057.26	1761.80	0.0001**
2 (CI532)	7 (MS)	24.25	657.22	1290.18	0.0353*
2 (CI532)	8 (CI422)	682.63	1164.77	1646.90	<0.0001**
3 (EVO)	4 (F24)	-420.25	-91.34	237.58	0.9907
3 (EVO)	5 (F28)	-945.89	-415.52	114.85	0.2539
3 (EVO)	6 (HL24)	-561.92	62.98	687.87	1.0000
3 (EVO)	7 (MS)	-880.00	-337.07	205.86	0.5636
3 (EVO)	8 (CI422)	-185.29	170.48	526.25	0.8326
4 (F24)	5 (F28)	-852.00	-324.18	203.64	0.5776
4 (F24)	6 (HL24)	-468.42	154.31	777.04	0.9954
4 (F24)	7 (MS)	-786.16	-245.73	294.70	0.8675
4 (F24)	8 (CI422)	-90.14	261.81	613.77	0.3191
5 (F28)	6 (HL24)	-270.44	478.49	1227.43	0.5256
5 (F28)	7 (MS)	-603.59	78.45	760.49	1.0000
5 (F28)	8 (CI422)	41.04	586.00	1130.96	0.0248*
6 (HL24)	7 (MS)	-1157.92	-400.05	357.83	0.7506