Newbold, C., S. Mergen, R. Richardson, P. Seligman, R. Millard, R. Cowan, and R. Shepherd. 2014. Impedance changes in chronically implanted and stimulated cochlear implant electrodes. *Cochlear Implants International*. **15**(4): 191-9.

Impedance changes in chronically implanted and stimulated cochlear implant electrodes

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Objectives: Electrode impedance increases following implantation and undergoes transitory reduction with onset of electrical stimulation. The studies in this paper measured the changes in access resistance and polarization impedance in vivo before and following electrical stimulation, and recorded the time course of these changes.

Design: Impedance measures recorded in (a) four cats following 6 months of cochlear implant use, and (b) three cochlear implant recipients with 1.5–5 years cochlear implant experience.

Results: Both the experimental and clinical data exhibited a reduction in electrode impedance, 20 and 5% respectively, within 15–30 minutes of stimulation onset. The majority of these changes occurred through reduction in polarization impedance. Cessation of stimulation was followed by an equivalent rise in impedance measures within 6–12 hours.

Conclusions: Stimulus-induced reductions in impedance exhibit a rapid onset and are evident in both chronic in vivo models tested, even several years after implantation. Given the impedance changes were dominated by the polarization component, these findings suggest that the electrical stimulation altered the electrode surface rather than the bulk tissue and fluid in the cochlea.

Keywords: Electrode impedance, Access resistance, Polarization impedance, Cochlear implant, Electrical stimulation

Introduction

Cochlear implants electrically stimulate residual spiral ganglion cells in the cochleae of severe to profoundly deaf patients, evoking auditory cues for speech comprehension. The power requirements of this system depend in part on the electrical impedance of the pathway between the electrode pair. Electrode impedance can be measured by recording the voltage developed across a pair of electrodes in response to a biphasic current pulse. Impedance provides information on the status of the electrode–tissue interface including whether or not the stimulator is operating within its voltage compliance. It can be broken down into access resistance (Ra) and polarization impedance (Zp). Ra represents the resistance of the electrolyte and its contents around the electrode; and Zp includes the resistance and capacitance of the double layer at the electrode surface and the corresponding Faradaic impedance across this layer (Dymond, 1976; Agnew et al., 1981).

It is widely accepted that electrode impedance increases following the implantation of an intracochlear electrode array (Saunders et al., 2002; Busby et al., 2002; Shepherd et al., 1990; Shepherd et al., 1994; Ni et al., 1992; Charlet de Sauvage et al., 1997; Xu et al., 1997). As such, the voltage, and therefore power, required to stimulate the auditory nerve at pre-set current levels increases. This increase in impedance is a result of tissue growth around the electrode array as the foreign body immune response encapsulates the electrode array in fibrous tissue within first few weeks of implantation (Kawano et al., 1998; Xu et al., 1997; Shepherd et al., 1990; Ni et al., 1992). Further changes in electrode impedance occur with the onset of electrical stimulation for tissue-covered electrodes. In chronically implanted experimental animals, the impedance of intracochlear electrodes was reported to be lower after stimulation compared with pre-stimulus levels (Charlet de Sauvage et al., 1997; Shepherd et al., 2001). Charlet de Sauvage et al. (1997) recorded up to a 17% drop in impedance following stimulation, from 17 to 14 kΩ after 40 days of stimulation. Shepherd et al. (2001) described smaller yet statistically significant drops in impedance (around 5–8%) following electrical stimulation. Impedance of thin-film iridium oxide electrodes in the cortex was also found to decrease after stimulation (Weiland and Anderson, 2000). These reductions in impedance were all temporary, with impedance returning to pre-stimulation levels after cessation of stimulation. It was suggested that these stimulation induced impedance changes were also related to the tissue growth around the electrode array (Charlet de Sauvage et al., 1997). Changes in electrode impedance with stimulation have also been described clinically. Two studies have reported a stimulus-induced reduction in impedance of 10–17% during the first stimulation session (Busby et al., 2002; Saunders et al., 2002). A later study measured the change in impedance before and after up to 4 hours of stimulation for the first 2 weeks of device usage (Paasche et al., 2009). They showed a 13–20% reduction in impedance with stimulation by the second week. Greater change was recorded for the first week (data not provided). Immediate impedance changes have also been modelled in vitro using cell-covered electrodes (Newbold et al., 2011). A rapid reduction in electrode impedance was measured within the first 10 minutes of stimulation, returning to pre-stimulation levels several hours after cessation of stimulation. Electrodes without cell cover showed only a fraction of change in impedance associated with stimulation.

Most of the in vivo studies referenced above only measured changes to the total impedance (Zt) of the electrode. However, changes in Ra and Zp should also be considered. Zt depends on a number of factors such as current density, current amplitude, and pulse duration, as well as any deposits on the electrode and the tissue between the stimulating and the return electrode. Changes in these factors give rise to changes of either Ra or Zp alone, or in combination. For example, increasing the current amplitude will increase Zt, mainly due to increases in Ra. Similarly, increasing the pulse width will increase Zt due to increases in Zp (Newbold, 2006). The biphasic pulse measurement used in this paper is a time-domain test. Its results have no direct correlation between frequency-domain tests such as electrode impedance spectroscopy. However, the biphasic pulse is used routinely in clinical cochlear implant stimulation and testing, and as such provides information relevant to clinicians and researchers.

Several studies describe changes in Ra and Zp with tissue growth (Xu et al., 1997; Shepherd et al., 2001; Tykocinski et al., 2001; Huang et al., 2007), however, only one publication was found that analysed Ra and Zp against both tissue growth and electrical stimulation (Tykocinski et al., 2005). Tykocinski et al. (2005) recorded the impedances of 21 cochlear implant recipients 1, 2, and 10 weeks after surgery, using a clinical impedance measurement technique similar to the one used in this paper,

which allowed calculation of both Ra and Zp. They showed an increase in Zp between the first and second week of implantation. By week 10, Zp had reduced below
week 1 levels, presumably due to the onset of electrical stimulation after the second week test. Ra was shown
to increase slowly over the 10 weeks assessed. The authors suggested that the changes in Ra were due to the development of tissue growth around the electrode, while Zp was affected by both protein adsorption (initial increase) and electrical stimulation (dispersion of this surface layer). The above-mentioned studies addressed some of the processes occurring at the electrode within the first few weeks of implantation (Tykocinski et al., 2005; Busby et al., 2002; Saunders et al., 2002). However, there is a lack of data on the dynamic changes in impedance during stimulation of long-term implanted electrode arrays, the timing of these changes, and the proportion of change of Ra versus Zp. In this paper, results from two in vivo studies examining the time course of stimulation- induced changes in electrode impedance are presented. The first study involved chronically implanted cats, tested 6 months after implantation. These animals were implanted with a percutaneous lead, allowing direct impedance recordings of the electrode. The second study was a clinical trial, conducted at least 1.5 years after cochlear implantation.

Methods

Animal experiments were performed in accordance with the Australian National Health and Medical Research Council guidelines for animal experimentation and approved by the Royal Victorian Eye and Ear Hospital (RVEEH) Animal Research and Ethics Committee (Project # 04/108A). The clinical data were obtained with approval and under guidelines set by the RVEEH Human Research and Ethics Committee (Project # 96/307H).
Electrode impedances are calculated from the voltage waveform developed across the electrodes in response to a charge-balanced biphasic current pulse (Fig. 1A). Ohm's law (voltage = current × impedance) is used to calculate the various components of the electrode impedance. The initial rise in voltage seen at the onset of the pulse (access voltage, Va, Fig. 1A) is used to derive Ra, the resistive component of the electrode–tissue interface. Owing to the highly capacitive nature of the electrolyte and its contents around the electrode, the voltage continues to rise for the duration of the pulse, in this paper termed polarization voltage (Vp,Fig. 1A). This rise is presumed to be dependent on the reactive components of the interface and is used to derive Zp. Total voltage (Vt, Fig. 1A) and hence Zt are measured at the end of the first pulse, thus, Zt is the summation of Ra and Zp.

Cat study

The animals in this study were part of a large study on the effect of electrical stimulation on cochlear health
and cortex reorganization (National Institute of Health NIDCD contract number NIH-N01-DC-3- 1005). The lead researchers in this study choose the stimulation mode, timing and levels, as was relevant to their work.
Four neonatally deafened cats were implanted and electrically stimulated via an intracochlear electrode array, designed in-house, consisting of eight platinum electrodes on a silicone rubber carrier (Fallon et al., 2009). Electrodes were located 0.4 mm apart and each were 0.45 mm in diameter and 0.3 mm wide. This created electrodes with surface areas of approximately 0.42 mm2. The arrays were inserted

3–4 mm into the lower basal turn of the scala tympani. Lead wires and connectors were manufactured in-house. The surgical procedure used was published previously (Xu et al., 1997). Chronic electrical stimulation commenced 2 weeks after surgery using Esprit 3G speech processors in conjunction with the Nucleus CI24 receiver-stimulator (Cochlear Ltd, Sydney, Australia). The receiver-stimulators were not implanted as would occur clinically, but were directly connected to the electrodes via a lead wire system that exited the skin (Fallon et al., 2009). The stimulation paradigm involved delivering charge-balanced biphasic current pulses to electrodes using a common ground configuration at levels determined by the electrically evoked auditory brainstem response thresholds for each electrode and confirmed as being supra-threshold by behavioural observation. Each pulse was 100 µs in duration, with amplitudes from 0.1 to 0.84 mA, thus the charge densities ranged from 2.4 to 20 µC/cm2 per phase. Each processor included a microphone, so that the stimulation level was modulated by environmental sound levels, as would be the case in the clinical setting. Animals received constant stimulation via the normal acoustic environment of their animal house, which included a radio left on at all times. Two electrodes of each animal were stimulated in common ground mode (all remaining electrodes used as the return electrode) for approximately 6 days per week, 24 hours a day. The break in stimulus on the seventh day each week was used to determine whether any change in impedance was seen with and without stimulation. The repeated measurements of electrode impedance involved in this study began after approximately 6 months of stimulation. Electrode impedance was measured just prior to commencement of the 6-day stimulation cycle, and then several times over the next 4 hours during the chronic stimulation programme. A biphasic current pulse of amplitude 76 µA and 50 µs pulse width was used in common ground mode. Measurements were conducted over 4 weeks. In-house software was used to calculate the electrode impedance values of the first phase (Fig. 1A). Changes in electrode impedance values following stimulation were analysed using paired t-tests. Each time point was compared with each other, creating a matrix of correlation. A regression analysis was also conducted on the percentage change in impedance following the first 15 minutes of stimulation. It was compared against the pre-stimulation impedance. Significance was determined using P-values of less than 0.05.

Clinical study

Three adult recipients were implanted by surgeons at the RVEEH and received clinical support through the RVEEH's Cochlear Implant Clinic. All had severe-to-total hearing loss in the implanted ear and had been implanted for between 1.5 and 5.5 years at the time of the study. The aetiologies for their hearing loss were described as either unknown, familial, or due to a childhood case of meningitis. Recipients received either a Nucleus 24 Contour or Contour Advance array (Cochlear Ltd). Both arrays have 22 half-banded platinum intracochlear electrodes moulded in a pre-curved silicone rubber carrier, and 2 extracochlear return electrodes. The intracochlear electrode surface areas range from 0.31 mm2 at the base to 0.28 mm2 at the tip. The clinical study involved the use of implanted receiver-stimulators and external speech processors (Cochlear Ltd). Each recipient had 20 of the 22 intracochlear electrodes programmed with the processing strategy Advanced Combined Encoder (ACE) at 900 Hz (Vandali et al., 2000) as part of the standard clinical procedure, using monopolar 1+2 mode of stimulation (i.e. using both extracochlear electrodes as return electrodes). This stimulation strategy also involved the delivery of charge-balanced biphasic current pulses at levels determined by threshold and comfortable levels particular to each recipient. The minimum and maximum current levels ranged from 50 to 690 μA. With an average electrode surface area of 0.295 mm2 and a pulse width of 25 μs/phase, the charge density ranged from 0.4 to 5.8 μC/cm2 per phase. Electrode impedance for all electrodes for each recipient was measured three times a day over three consecutive days: prior to switch-on (first thing in the morning), half an hour after switch-on, and just before switch-off (end of day). Standard clinical impedance measures provide only one value of impedance taken at the end of the measuring pulse (i.e. Zt). Unlike animal studies, which use electrode assemblies with direct percutaneous connections, clinical data relies on a transcutaneous connection via a radiofrequency link (Seligman and Shepherd, 2004). While percutaneous connections allow a view of the complete pulse, there exists a limitation in the data transfer for these transcutaneous links, with only a single measure of impedance available for each current pulse. Thus direct measures of the output voltage and therefore the impedance were not available in the clinical setting. To overcome this, in-house software was used to approximate the voltage pulse shape. Charge-balanced biphasic current pulses of fixed current (100 CL; 76 μA) and varying pulse width (7–100 μs) were applied to each electrode and a single impedance measure was taken at the end of each pulse (measured using monopolar +1, where the ball electrode is used for the return path). Taken together, this allowed an estimate of the shape of the first phase of the pulse to be made (Seligman, 2004). Fig. 2 shows schematically how this was achieved. Impedance values over the 20 stimulated electrodes of each subject were analysed with respect to the time and day of measure, electrode number, and the subject using a general linear model. Changes in electrode impedance values following stimulation were then analysed using paired t-tests. Each time point was compared with all others, creating a matrix of correlation. The percentage change in impedance following the first half an hour of stimulation was also compared against the pre-stimulation impedance using regression analysis. Significance was determined using P-values of less than 0.05. The amount of variance explained by the variables was evaluated using R2 adj, and tests of normality were conducted on each analysis' residuals.

Results

Cat study

Electrode voltage waveforms recorded from one animal are shown in Fig. 1B. A large drop in the amplitude of the electrode voltage waveform (i.e. impedance) was seen 15 minutes following the onset of stimulation, while only a small further reduction in impedance (if any) occurred at later time points. Fig. 1C shows impedance plots for one animal (average of both stimulated electrodes) over the four stimulus cycles tested. A rapid drop in impedance following onset of stimulation was observed in three of the four animals. Little to no change in impedance with stimulation was seen in one animal. The lowest electrode impedance was also recorded for this animal. The mean and standard deviation of all impedance values are plotted in Fig. 3. These values show the same pattern as the individual data: (1) a stimulus induced reduction in impedance, (2) the decrease in impedance was rapid and generally occurred within the first 15 minutes following stimulation, (3) both Ra and Zp decreased rapidly after onset of stimulation, and (4) both Ra and Zp increased slightly with ongoing stimulation. Paired t-tests indicated a significant difference in all impedance values between the first measure taken prior to stimulation and all other time points. There were no significant differences between the impedance measured 30 minutes following the onset of stimulation, and the later time points during the course of a day's stimulation. While both Ra and Zp showed significant differences between the initial and subsequent times, a greater drop in Zp was recorded in most cases. We also analysed the effect of the pre-stimulation impedance on the extent of the stimulus-induced reduction in impedance. A regression analysis compared the percentage of impedance change following the first 15 minutes of stimulation and the pre-stimulation impedance (Fig. 4). This analysis showed a strong relationship between Zt and Zp, such that the reduction in impedance following stimulation was larger when the pre-stimulation impedance levels were high. No significant relationship was determined for Ra.

Clinical study

The impedance measuring techniques used in the clinical study are illustrated in Fig. 2A and B. Fig. 2C illustrates how this technique resulted in electrode impedance values comparable with the chronic animal data presented above. The impedance curves shown are from one subject at three times: prior to stimulation (0 minutes), and at 30 minutes and 16 hours after stimulation onset. We plotted the averaged impedances of the subject's 20 stimulated electrodes with the error bars showing 1 SD. There was a small but measurable drop in impedance following stimulation at the longer pulse widths (time >25 μs). While the range of impedance values across electrodes was large, a small drop in Zt and Zp was seen within the first 30 minutes of stimulation for most days in each subject (Fig. 5). There was no observable difference in Zt and Zp between 30 minutes and 16 hours post-stimulation onset. Changes in Ra with the onset of stimulation were less consistent across subjects. The average electrode impedance values for all subjects (all stimulated electrodes over all days), are shown in Fig. 6, and grouped into the electrodes' cochlear position (basal electrodes 3–8; middle electrodes 9–15; apical electrodes 16–22). These graphs show the basal electrodes had higher impedances than those placed more apically. This is in contrast to the impedance difference the electrode surface area would create (basal electrodes have larger surface areas than apical electrode, leading to lower electrode impedances). Paired t-tests of the impedance values for each measurement point were conducted to assess if, and when, a significant change in impedance occurred. This analysis showed that Zt and Zp prior to stimulation were significantly higher than 30 minutes and 16 hours after stimulation onset ($P < 0.001$). No difference in Zt and Zp was seen between 30 minutes and 16 hours post-stimulation onset ($P = 0.536$ and $P = 0.055$, respectively). Taken together, the majority of stimulation-induced reductions in impedance occurred in the first 30 minutes of electrical stimulation. Access resistance (Ra) showed opposite results. There were significant differences in Ra between 16 hours post-stimulation and both pre-stimulation and 30 minutes post-stimulation. The t-values of these conditions were negative, signifying Ra was increasing rather than reducing with stimulation. The drop in Zp and rise in Ra after stimulation were such that the overall change in Zt was small. The effect of pre-stimulation impedance on the strength of the stimulation-induced impedance change was also analysed. Regression analysis for all stimulated electrodes showed no significant relationship between the percentage change in impedance following stimulation and the pre-stimulation impedance prior to stimulation. The first two basal electrodes of each array (electrodes 1 and 2) remained unstimulated and as such could be used as controls. These electrodes showed no significant change in impedance (Zt, Ra, or Zp) over the same measurement period.

Discussion

These results demonstrated that transitory stimulusinduced reductions in electrode impedance are present even after long periods of chronic implantation. The stimulation-induced impedance changes recorded in the first weeks of stimulation (Busby et al., 2002; Saunders et al., 2002; Tykocinski et al., 2005) is still evident after long-term implantation. There was a noticeable difference in the extent of impedance reduction measured between the studies, however. The cat study, recording changes after 6 months in vivo, showed about 20% reduction in Zt after stimulation. The clinical study, recording changes after 18–60 months in vivo, showed less than 5% Zt reductions. These differences may be related to the differing modes of stimulation and impedance recording used for the cat and clinical studies, common ground and monopolar +1, respectively. Common ground stimulation, particularly with small numbers of electrodes as was the case in the cat study, can create only a small surface area difference between the working and return electrodes. This would increase the contribution of the return electrode impedance to the recorded impedance values, otherwise deemed to be informative of the working electrode. In comparison, monopolar +1 uses a large ball electrode for the return path, creating a more accurate assumption that the recorded impedance was indeed the impedance of the working electrode. When stimulation was provided to the intracochlear electrodes of the cat study, their impedances would have individually dropped, creating the likely scenario that a greater change in impedance was recorded than that seen in the clinical study where the stable impedance of the ball electrode was used.

The stimulation-induced impedance reduction was also affected by the pre-stimulation impedance levels. In the cat study, when the pre-stimulation impedance value was large, stimulation induced a greater impedance reduction. We saw a similar trend in the clinical trial subjects, but this was not significant when considered as a group. Presumably, this base impedance level was affected by the volume of fibrous tissue within the scala and on the electrode pad. Histology data from these animals had not been analysed at the time of publication. It should be noted, however, that histology from other animals that underwent the same electrical stimulation showed no sign of stimulus-induced auditory nerve or general cochlear damage (Coco et al., 2007). The largest drop in clinical impedance measures was recorded for the basal electrodes, where tissue growth is known to be greatest (Xu et al., 1997), particularly in cases with cochleostomy insertions. The stimulation-induced reductions in impedance were rapid – the maximum reduction occurred within the first 15 minutes (cats) and 30 minutes (clinical) of stimulation onset. These impedance changes could have occurred earlier than the time points elected in these studies. In vitro studies of cell-covered electrodes showed impedance dropped markedly within the first 150 seconds of stimulation onset (Newbold et al., 2011). It is important to note that little to no change in impedance was recorded with unstimulated electrodes. All three cochlear implant recipients in this study had no stimulation applied to their two most basal electrodes. These unstimulated electrodes always had the largest impedance values, in turn suggesting the greatest tissue growth was present at the basal end of the cochleae. Similarly, the stimulated electrodes mostly returned to their pre-stimulation impedance values after several hours of no stimulation. The present studies examined the contribution of both Ra and Zp to the stimulus-induced changes in Zt. Both datasets showed the majority of change in impedance after stimulation was due to a drop in Zp. This matched with what was observed by Tykocinski et al. (2005), who showed after the first 8 weeks of stimulation that Zp was significantly reduced, whereas Ra showed a slight rise during the same period. Interestingly, our results show these changes are still occurring with daily stimulation at least up to 5 years after implantation. These results suggest that changes are occurring at or very near the electrode surface.

Conversely, stimulation did not seem to affect access resistance, Ra. A small, yet significant, reduction in Ra was observed with cats over the course of electrical stimulation, while in contrast a small rise in Ra was recorded in the clinical data, suggesting that stimulation did not directly alter the conductance of any fibrous tissue covering the electrodes. This result is not immediately intuitive, particularly when considering that greater reductions in impedance were recorded when pre-stimulation impedances were high, presumably due to a more extensive tissue response. This lack of Ra change was also seen, however, over the longer time period by Tykocinski et al. (2005). Thus, while a fibrous tissue matrix covering may lend itself to larger Ra and Zp values, the stimulation- induced changes are likely confined to the electrode interface. Possible changes that could be occurring are protein adsorption and desorption, and/or a reduction in adhesion of the fibrous tissue matrix to the surface of the electrode. Earlier work with stimulation of cell-covered electrodes created a different picture (Newbold et al., 2011). Sputter-coated gold electrodes were plated with a monolayer of highly adherent cells and with the onset of electrical stimulation, a reduction in Ra and an increase in Zp were recorded. Subsequent in vitro work using the same physical set-up with changes to the stimulation parameters applied (used anodic-first single pulse compared with cathodic-first pulse train, respectively), showed stimulation created a reduction in both Ra and Zp (data not published). In both cases, however, stimulation caused a major change in Ra, which contrasts with the in vivo data. In the in vitro situation, the cell monolayer would have adhered firmly to and completely over the electrode such that any charge passage would need to pass through the cell layer. Electrical stimulation in this condition may have created breaches in the cellular membrane, reducing the resistive pathway between the working and return electrodes, i.e. reducing Ra. Considering the lack of change in Ra seen in our in vivo data, similar changes to the cellular material over the electrodes in vivo was not occurring, rather just changes to the electrode surface.

Conclusions

This study has described changes in electrode impedance values for chronically implanted and stimulated cochlear implant electrodes. Electrical stimulation caused a transitory reduction in impedance values, seen several years after implantation, using charge densities well within safe electrochemical limits (Shannon, 1992). We showed this decline to be rapid (within 15 minutes of stimulation onset) and impedance remained low until stimulation stopped. Pre-stimulation impedance levels recovered following cessation of stimulation. The majority of impedance reduction occurred via a reduction in polarization impedance, Zp. These results suggest that stimulation affected the electrode surface, rather than creating change in the fluid and tissue of the scala tympani. It is important to note that while there were stimulation-induced changes in impedance, there has been no evidence of stimulation-induced damage to the cat cochleae. Greater understanding of the impedance changes that occur with electrical stimulation of implanted electrodes provides opportunity for improved device programming and efficient power usage.

Acknowledgements

This work was funded by the Cooperative Research Centre (CRC) for Cochlear Implant and Hearing Aid Innovation and the HEARing CRC, established and supported under the Australian Government's Cooperative Research Centres Program. The electrodes were made in-house by Ms Helen Feng. The data for the cat study were collected in conjunction with the Bionics Institute's study, supported by the National Institute of Health (NIDCD contract number NIH-N01-DC-3-1005). We gratefully acknowledge the time and effort taken by our three cochlear implant recipients. Dr Jin Xu, Ms Anne Coco, Ms Stephanie Epp, Ms Lauren Donley, Mr David Perry, and Dr James Fallon were responsible for the surgery, some impedance measures, and the day-to-day maintenance of the cats. The Bionics Institute acknowledges funding from the Victorian State Government's infrastructure grant scheme.

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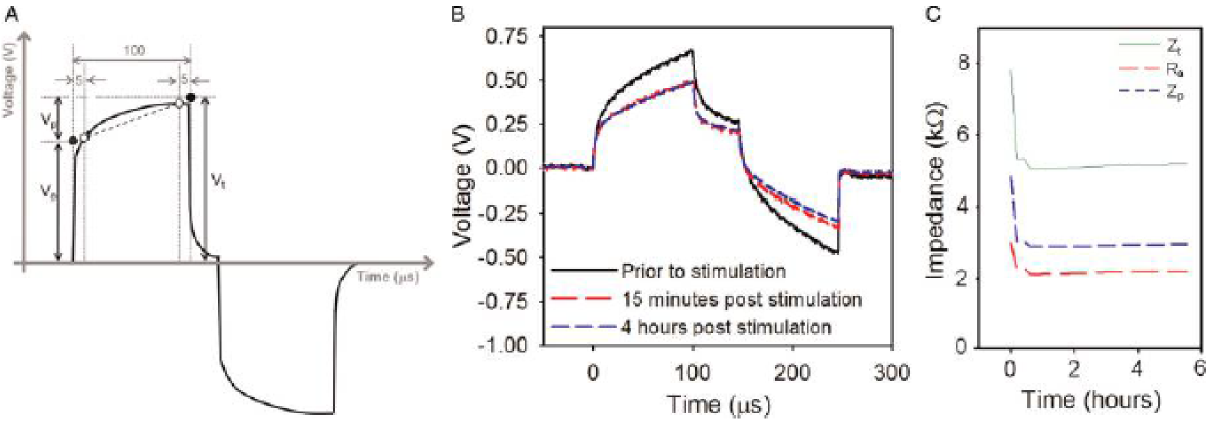

Figure 1 (A) In the cat study, an electrode voltage waveform was developed across an electrode pair in response to a biphasic current pulse. Impedance was calculated by measuring two points on the waveform (white circles), one at 5 μs from the start of the pulse, and one 5 μs from the end of the first phase of the pulse. A line between these points was extended to 0 and 100 μs (black circles), i.e. the start and end of the pulse, to determine the access voltage (Va) and total voltage (Vt). (B) Representative electrode voltage waveforms from the cat study. These waveforms were taken prior to stimulation, 15 minutes following, and 4 hours after the onset of stimulation. (C) Average electrode impedance values for one animal for 1 day. Total impedance, Zt, equates to Vt, access resistance, Ra, equates to Va, and polarization impedance, Zp, equates to Vp. This plot shows the timing of impedance change due to the onset of stimulation over the first 6 hours of four stimulation cycles.

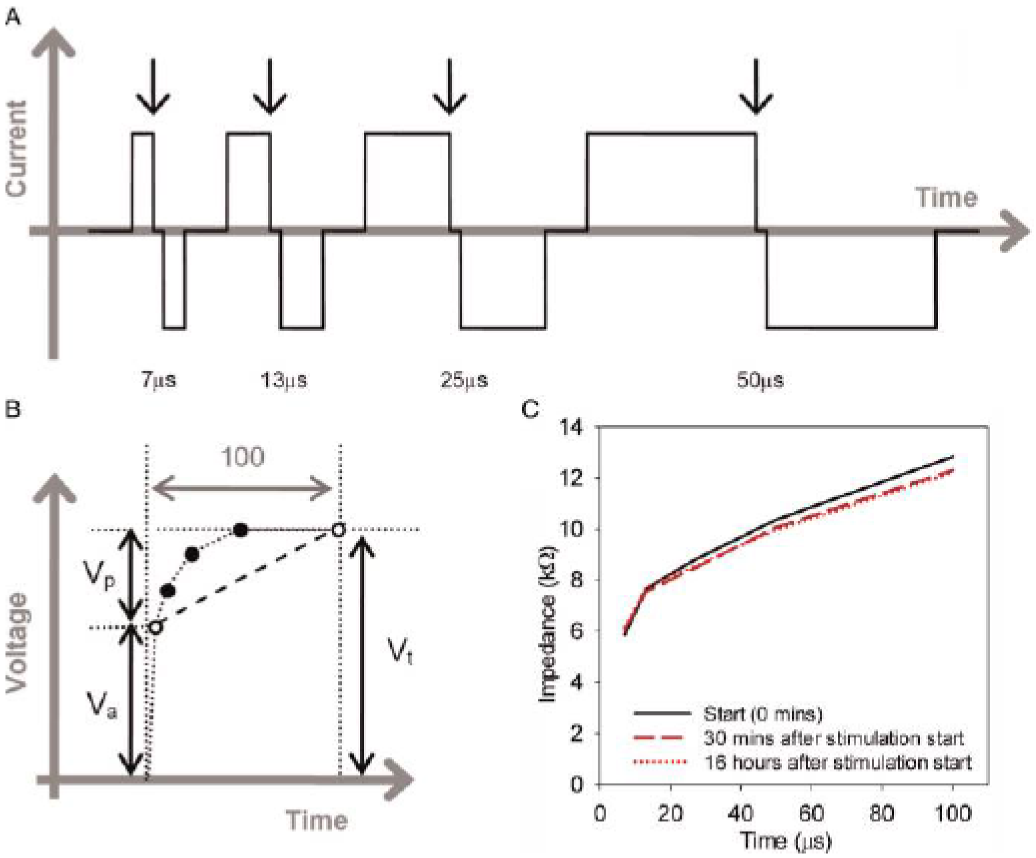

Figure 2 (A) Electrode impedancewas measured in the clinical study by applying current pulses of varying pulse widths to each electrode and impedance was measured at the end of the first pulse (arrow). (B) Impedance calculation from the

voltage ‘waveform’ for the clinical study. A line was taken between the voltages at 7 μs and the end of the ‘pulse’. A line between these points was projected to 0 μs, i.e. the start of the ‘pulse’, and this voltage was used to calculate the access impedance (Ra). Total impedance (Zt) was defined as the impedance at end of the ‘pulse’, while polarization impedance (Zp) was found using the difference between Zt and Ra. (C) Representative impedance ‘waveform’ obtained from one subject in the clinical study. The data shown are for electrode 3 on day 1 of the test, measured prior to stimulation onset, after 30 minutes of stimulation, and after 16 hours of stimulation.

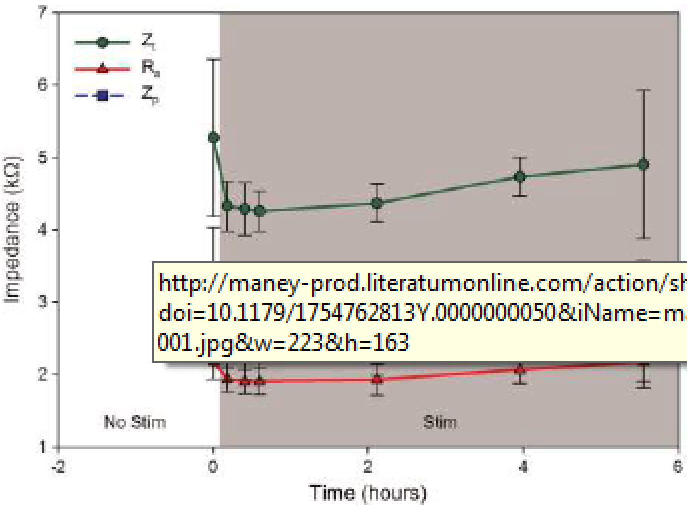

Figure 3 Average electrode impedance values for all animals in the cat study. These were measured after 166–199 days of implantation (N = 4). The error bars signify ±1 SD. Shaded block indicates the period of stimulation.

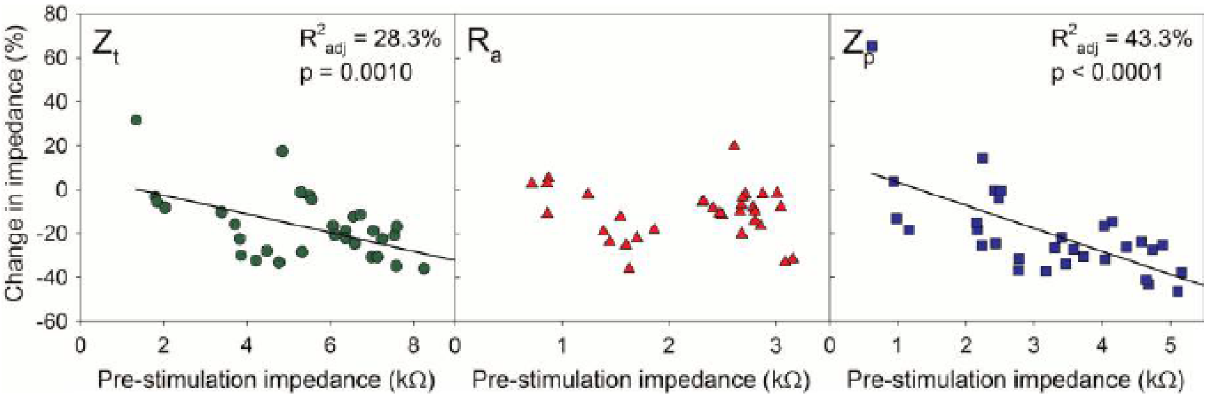

Figure 4 Stimulus-induced impedance changes against the pre-stimulation impedance for the cat study. Pre-stimulation impedance (i.e. before stimulation onset) significantly affected the percentage of impedance change measured following 15 minutes of stimulation for both Zt and Zp.

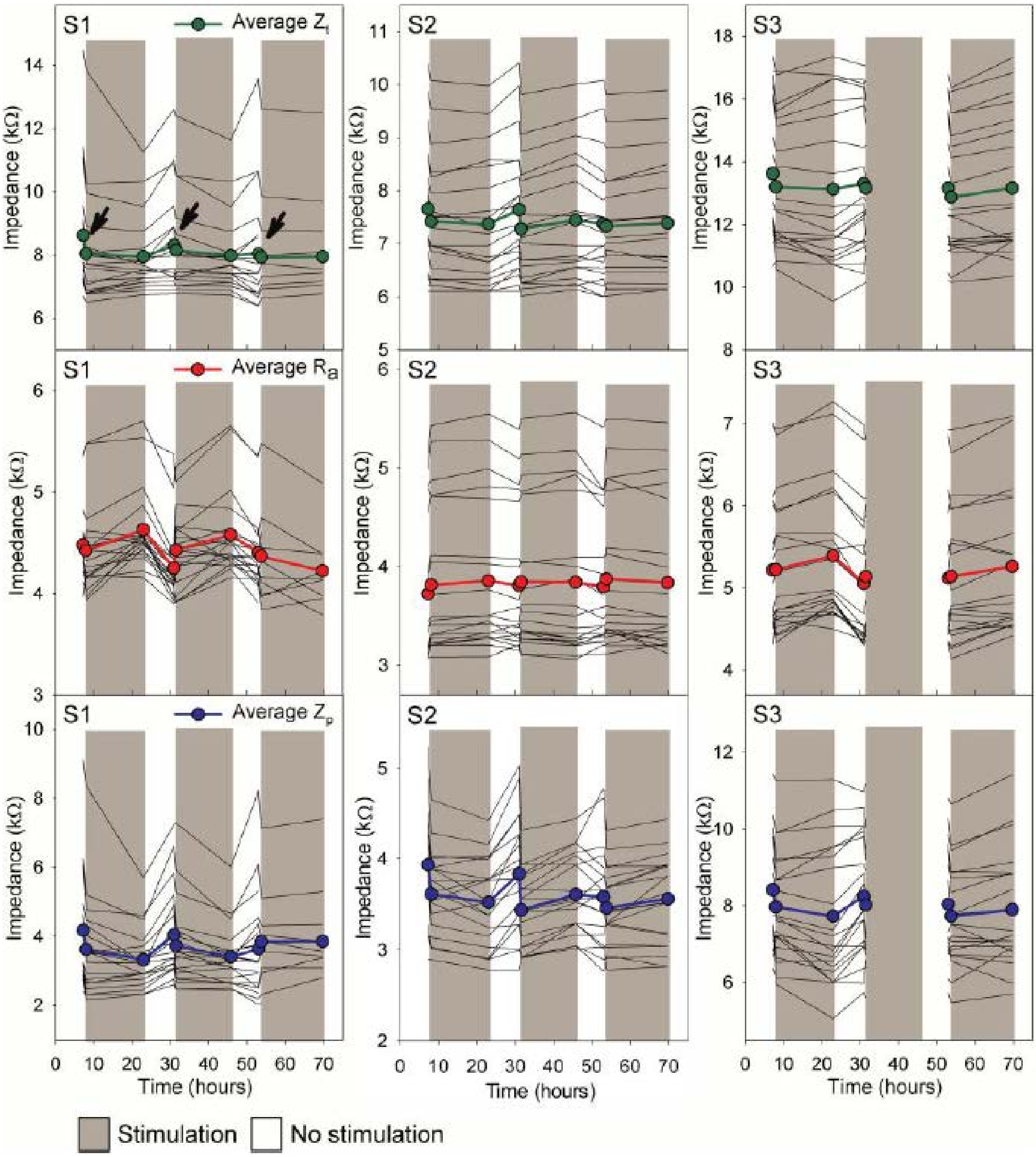

Figure 5 Electrode impedance values for the three cochlear implant subjects in the clinical trial. Top row: Zt, middle row: Ra, and bottom row: Zp. The shaded bars indicate times of stimulation. The 20 stimulated electrodes are shown for each subject, with the average shown in bold colour. The arrows in the first plot signify the reduction in impedance recorded 30 minutes following the onset of stimulation. Typically, the electrode impedance was greatest for the basal electrodes.

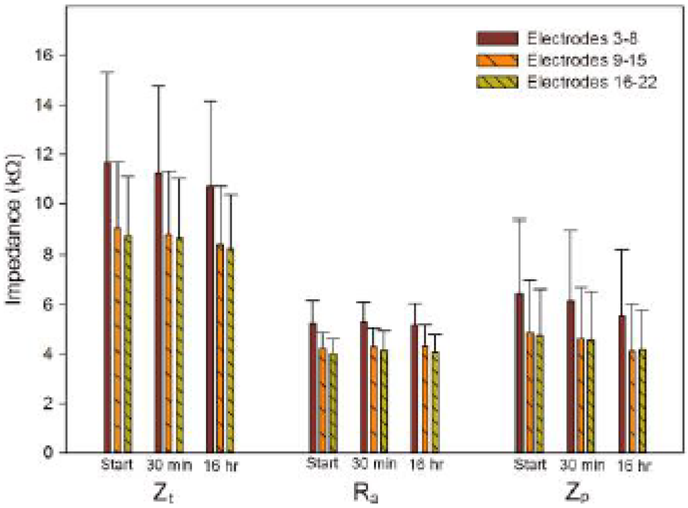

Figure 6 Average electrode impedance values for the clinical trial split for electrode position: 3–8 were the most basal electrodes; 16–22 were the most apical electrodes. This graph shows all stimulated electrodes of all subjects over all days in the trial. The error bars signify ±1 SD.