Direct Current Measurements in Cochlear Implants: An *in vivo* and *in vitro* Study

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ABSTRACT: Direct current (DC) was measured both *in vivo* and *in vitro* in cochlear implant electrodes with stimulation at moderate to high pulse rates in monopolar and bipolar modes. *In vivo* DC was approximately 2-3 times higher than that measured *in vitro*. *In vivo* DC levels were <100 nA even at very high rates, although DC levels increased as a function of stimulus rate and charge intensity. DC levels were lower in the monopolar than in the bipolar stimulation condition. Stimulation with a monopolar capacitively coupled extracochlear electrode showed even lower DC levels in the intracochlear electrodes. Our results indicated that the Nucleus electrode shorting system is able to maintain a low level of DC during very high rate stimulation for both monopolar and bipolar modes.

1. INTRODUCTION

Electrical stimulation of neural tissue involves the transfer of charge to tissue via electrodes. The conversion from electron to ionic current takes place at the electrode-tissue interface by electrochemical processes. With a charge-balanced biphasic current pulse, there is always a small amount of charge remaining on the electrodes immediately following the second phase as a result of the slow kinetics of the oxidative-reductive reactions that store charge on the electrode [1]. There are two methods of charge recovery typically used in neural prostheses: i) capacitive coupling; ii) electrode shorting [2]. Capacitive coupling between the stimulator and electrodes prevents any long-term residual charge. However, capacitors occupy a significant volume of the implant package in a neural prosthesis with a large number of electrodes. The Nucleus multichannel cochlear implant shorts all electrodes between current pulses. Any remaining charge flows through a low resistance path that brings all electrodes to the same electrode potential and effectively minimizes direct current (DC) [2].

While there is no clearly defined maximum safe limit for DC, chronic stimulation studies have consistently shown DC levels of less than 100 nA do not result in damage to the auditory nerve or the cochlea in general [3,4,5,6,7]. However, there is good evidence to suggest that DC levels in excess of 1-2 μ A can result in significant neural damage [8].

While electrode shorting has been shown to be a safe and efficacious technique for minimizing DC at low stimulus rates (and therefore relatively long

shorting periods), at higher rates (>500 pulses per second; pps) there is evidence of increased DC levels, particularly associated with high stimulus intensities [9,10].

The purpose of the present study was to examine DC levels *in vivo* using high stimulus rates at stimulus intensities both within and outside normal clinical levels with monopolar and bipolar stimulation.

2. MATERIALS AND METHODS

Six healthy guinea pigs were anaesthetised with ketamine (40 mg/kg) and xylazine (4 mg/kg) and were maintained at surgical levels of anaesthesia during the experiment. The four most apical platinum (Pt) electrodes of a standard Nucleus electrode array were carefully inserted into the scala tympani. An extracochlear Pt electrode was placed in the musculature outside the cochlea.

Biphasic constant current pulses were generated from a modified, box-mounted version of the Nucleus CI24M receiver/stimulator. The Nucleus CI24M device was powered and controlled from a purpose built interface board connected in to an IBM PC. This system allowed an arbitrary sequence of stimulation pulses to be defined and repeatedly presented to the electrodes with parameters such as pulse width, current amplitude and stimulus rate all independently variable. Current pulses at total rates of 1,200 – 14,500 pps were delivered to the four electrodes. The current amplitude was set at 0.2, 0.875 or 1.75 mA and pulse widths of 26, 50, 100 or 200 μ s/phase were used in monopolar and bipolar stimulation modes. Each stimulus condition in the monopolar mode was performed with and without a capacitor in series with the extracochlear electrode.

A parallel combination of a 47 μ F capacitor and a 10 k Ω resistor was placed in series with each intracochlear electrode. The voltage across each RC circuit was measured using battery operated digital voltmeters (10 M Ω input impedance; Fluke 8060A) providing a resolution of 1 nanoampere (nA).

The care and use of animals involved in this study was approved by the Animal Experimentation Ethics Committee of the Royal Victorian Eye and Ear Hospital ('Cochlear Implants: Neural damage mechanisms in the auditory nerve' # 92-016A).

3. RESULTS:

In a preliminary study using high intensity bipolar stimulation the DC level measured *in vivo* was 3.2

and 2.8 times that of the values measured in human serum albumen and saline respectively. Because of the large discrepancy noted between the *in vivo* and *in vitro* values the decision was made to use an *in vivo* rather than an *in vitro* model for this study.

Both monopolar and bipolar stimulation showed DC levels increased as a function of stimulus rate, pulse width and stimulus intensity. In general, DC levels were higher in bipolar stimulation than in monopolar stimulation. Monopolar stimulation with a capacitively coupled extracochlear electrode showed even lower DC levels in the intracochlear electrodes.

For monopolar stimulation, using a capacitor in series with the extracochlear electrode, the DC levels were almost zero at pulse widths of 25 and 50 μ s/phase even at the highest rate (8500 - 14,500 pps/4 channels). With pulse widths of up to 200 μ s/phase or stimulus intensities up to 1.75 mA, which are outside the normal clinical level, some DC levels (less than 30 nA) were observed. It should be noted that even in the case of a capacitively coupled extracochlear electrode it is still possible for small DC to be present in the intracochlear electrodes. This is because it is only necessary for the sum of DC in the intracochlear electrodes to be zero, not the individual electrode current. Stimulation without a coupling capacitor showed slightly higher DC level but still less than 100 nA for the same stimulus conditions.

Using a stimulus intensity of 0.875 mA, the mean DC levels for bipolar stimulation (16 stimulating electrodes from 4 animals) were 80 nA for 26 μ s/phase; 105 nA for 50 μ s/phase; and 138nA for 100 μ s/phase at the stimulus rates of 14500, 8500, and 4600 pps/4 channels respectively, corresponding to a shorting period as low as 7 μ s.

4. DISCUSSION:

There has been increasing interest in speech processing strategies using high pulse rate stimulation (>500-1000 pulses/s per channel) to provide fine temporal cues for cochlear implant patients. The present study has demonstrated that the Nucleus electrode shorting system can effectively minimize DC at stimulus rates of up to 14500 pps/4 channels with pulse widths of 26-50 μ s/phase for both monopolar and bipolar stimulation. Monopolar stimulation showed even greater reduction in DC level in the intracochlear electrode, especially with a capacitively coupled extracochlear electrode, with DC levels of less than 30 nA using stimulus intensities outside the normal clinical range. The results of this study showed DC increased as a function of stimulus rate, pulse width and stimulus intensity for both monopolar and bipolar stimulation. Our results were obtained from an animal model using a modified clinical device, indicating the data is clinically relevant. It has important practical implications for selection of safe stimulus parameters and stimulation configurations for cochlear implants. The mechanism(s) responsible for the significantly

elevated DC observed *in vivo* compared with *in vitro* are currently under investigation.

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