The Multiple-Channel Cochlear Implant: Interfacing Electronic Technology to Human Consciousness

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10.21437/Interspeech.2005-1

Abstract

Fundamental research on electrical stimulation of the auditory pathways resulted in the Multiple Channel Cochlear Implant, a device which provides understanding of speech to severely-to-profoundly deaf people. The device, a miniaturized receiver-stimulator with multiple electrodes fed with power and speech data through two separate aerials was first implanted in a patient in 1978 as a prototype, and since 1982, was commercially produced by Cochlear Limited, Australia. Speech processing is based on the discovery that the sensation at each electrode is "vowel-like". Initially, the second formant was coded as a place of stimulation, the sound pressure was coded as a current level, and the voicing frequency as a pulse rate. Further research showed that there were progressively better open-set word and sentence scores for the extraction of the first formant in addition to the second formant (the F0/F1/F2 processor), the addition of high fixed filter outputs (MULTIPEAK) and then finally 6 to 8 maximal filter outputs at low rates (SPEAK) and high rates (ACE). All the frequencies were coded on a place basis. World trials completed for the US FDA on late-deafened adults in 1985 and in 1990 on children from two years to 17 years proved that a 22-channel cochlear implant was safe and effective in enabling them to understand speech both with and without lip-reading.

Summary of the Keynote

The restoration of speech understanding in severely-to-profoundly deaf people was not thought possible in the 1960s. The criticisms were reasonable. One was that the innervation of the cochlea was too complex and the 10,000 nerves for speech frequencies too numerous, for a small number of electrodes to reproduce the coding of sound. This criticism had to be answered in the experimental animal before operating on people.

The coding of sound frequencies by the brain can occur through two mechanisms - a temporal and/or place code. With the temporal code there is a relation between the time intervals between nerve action potentials and the frequency of the sine wave. With the place code frequency is coded through site of stimulation in the brain. The inner ear filters sound frequencies, and the different regions excited are connected to all the brain centres spatially so that a frequency scale is preserved. In other words, the pitch of a sound is recognized according to the site of stimulation in the brain.

Personal studies on experimental animals from 1967 to 1969 demonstrated that it was not possible to use temporal coding to reproduce frequencies above about 500 Hz, through rate of stimulation. This is much less than the 4,000 Hz needed for speech. The limitation in the temporal coding of frequency with electrical stimulation was confirmed with behavioural studies on the experimental animal. The results of these and other studies showed that single-channel stimulation, using rate alone would not be able to reproduce speech frequencies from 500 Hz to 4,000 Hz. Thus important mid-to-high frequencies would need to be reproduced by place coding through multiple-channel stimulation.

At first localizing current to separate groups of nerve fibres in the cochlea for place coding of frequency appeared to be a major problem, as electrical current could short circuit through the fluid in the cochlea, preventing it being localized to nerve fibres. However, mathematical modelling of electrical resistances in the cochlea, and physiological studies demonstrated that monopolar or bipolar stimulation could both partially localize the current. The electrodes also had to be placed in the scala tympani close to the nerve fibres or spiral ganglion cells for optimal results.

These data showed that even with multiple-channel stimulation the "electro-neural" interface with the central auditory nervous system only allowed limited transmission of rate and place information to the brain, and was thus an electro-neural "bottle-neck". The modelling and animal electrophysiological studies also demonstrated that stimulation with electrodes inside the cochlea provided better localization of current for place coding of frequency than electrodes outside the cochlea. However, before passing a bundle of electrodes around the cochlea and stimulating the auditory nerves, it was necessary to answer the second question: would this be safe?

The key safety questions to be answered in the experimental animal were on: 1) surgical trauma; 2) electrical stimulus parameters; and 3) middle ear infection spreading to the cochlea and leading to meningitis.

Firstly, could electrodes be placed in the scala tympani of the cochlea close to the nerves for speech frequencies without injury and the loss of the auditory nerves it was hoped to stimulate? Studies on the experimental animal showed that if an implant tore the basilar membrane or fractured the spiral lamina there was localized loss of nerve and spiral ganglion cells. On the other hand, if the electrode was smooth and free-fitting it did not damage the cochlea, especially if inserted gently.

Furthermore, it was also necessary to know whether the electrode bundle could be passed some distance around the tightening spiral of the cochlea to reach the nerves transmitting speech frequencies without trauma? Initial studies, showed the upward progress of the array was impeded, and mechanical studies demonstrated this was due to friction between the electrode bundle and the outside wall.

The solution came serendipitously on the beach by inserting dune grasses into turban shells, that were a large-

scale model of the human cochlea. If the grass were flexible at the tip with increasing stiffness towards its basal end it would pass around the first turn. The same principle applied to an electrode carrier passed into the human cochlea. The graded stiffness was achieved through the incremental addition of wires from apex to base. To minimize friction and the resulting trauma the electrode pads were designed as bands that lay flush with the surface of the carrier. The flush circumferential bands also allowed the array to be easily removed from its fibrous tissue sheath and another re-implanted.

To ensure that these conclusions were correct in the human the electrode arrays were inserted into fresh human temporal bones. The damage was restricted to a small area of the basal turn and could be avoided if the insertion was stopped when resistance was first met.

The next safety question was: would the electrical currents used to stimulate the nerve fibres also damage them? What were the safe electrical charge densities and current levels?

To keep the charge densities to low safe levels the bands needed to be wide. But on the other hand, they had to be narrow so that current could to be localized to separate groups of nerve fibres for the place coding of speech frequencies. The bands were optimized to a width of 0.3 mm, and with an OD of 0.6 mm the charge density for a maximum current was approximately 20 $\mu C/cm^2$/phase. This was below the safe levels determined for the brain. The bands also had an inter-electrode spacing of 0.45 mm that allowed 20 electrodes to be sited opposite the nerves for the speech frequencies. In addition, the biphasic pulses were charge-balanced so there would be no build up in DC above 0.1 μA as studies have shown this does not damage the nervous system.

With the banded electrodes the maximum currents for stimulating patients were tested on the experimental animal. In an experimental study cat spiral ganglion cells remained normal after 2,000 hours of continuous stimulation with a charge density of 18 $\mu C/cm^2$ / phase. No damaging effect was seen in this or other animals up to 32 μC, and this was thus a conservative electrically safe upper limit. Later it was also found that stimulus rates up to 2,000 pulses/s for more advanced strategies did not lead to loss of ganglion cells.

The other very important safety concern was that the implant could lead to a dangerous spread of infection from the middle ear to the inner ear with the risk of meningitis. This concern was due to the fact a few patients, with a strut to replace the stapes in otosclerosis, had developed fatal meningitis following a middle ear infection. A series of 10 studies over 11 years was undertaken on experimental animals and it was found that the formation of a fibrous tissue sheath around the electrode reduced the entry of a later infection. The formation of the fibrous tissue sheath was facilitated by a fascial graft around the electrode at the time of surgery. It was also found that healing around the electrode at its entry point took one month, and this was a vulnerable period for the entry of infection. Care was thus required in the prevention and management of operative and postoperative infection.

The sheath for the electrode prevented the entry of infection through three defence mechanisms involving: 1) mucous and ciliated cells ; 2) phagocytic white cells; 3) lymphocytic white cells. Mucus is bacteriostatic, and the phagocytic and lymphocytic cells are brought to the site by blood vessels in the sheath.

These defence mechanisms are the reason that it was later shown by the US Centre for Disease Control that the risk of meningitis was similar to that of the community as a whole, providing care was taken to prevent early post-operative infection, a one -component array was used, and there were no risk factors such as a congenital ear.

After completing the efficacy and safety studies on the experimental animal the next major task was to discover whether speech could be coded with electrical stimuli of the auditory nerve in deaf people so that it could be understood.

Speech is a complex signal and it was not clear what information could or should be transmitted through the electro-neural “bottle-neck”. To do research on patients meant developing an implantable electronic unit, as the use of a plug and socket to transmit the stimuli through the skin, was found in the experimental animal to become infected.

Developing an implantable receiver-stimulator was going to be very expensive and as no peer group funding could be obtained the costs came from an appeal to the public which included standing on the city streets of Melbourne at lunch time shaking a tin and asking for donations.

The circuit for the receiver-stimulator was completed in 1976 after three years work, with its implementation as a bench top model. The bench-top circuit was miniaturized through silicon chips bonded to gold-plated tracks printed on silica wafers.

The University of Melbourne’s prototype receiver-stimulator ready for implantation was completed in 1978. It was hermetically sealed and the power to operate it and the data stream were transmitted by radio waves through two separate aerials.

The first patient was implanted with the prototype receiver-stimulator in Melbourne on the 1st August 1978. The first important question after surgery was: what did the patient hear for rate as well as place of stimulation? Firstly, for rate of stimulation on each electrode the patient could distinguish pitch, but only up to 300 Hz. This was predicted by the previous experimental animal findings in cats.

For localized stimulation of each electrode, “pseudo-bipolar” simulation was used with current flowing from each electrode to all the others connected together as a common ground. An interesting discovery was made. With place of electrical stimulation the sensation was perceived as timbre rather than pitch (timbre is the quality of the sound that distinguishes two musical instruments playing the same note). The timbre varied from sharp at the high frequency end of the cochlea to dull at the low frequency end. The sensations of timbre at the different electrodes could be well discriminated. It was therefore hoped that the perception of timbre through place of stimulation could be used to present the higher frequencies of great importance for intelligibility. In addition, increasing the strength of the current increased loudness.

A fixed-filter channel vocoder, speech processing strategy was initially used by Simmons in 1965 to process speech but without speech understanding. Then in 1980 it was reported to provide some speech understanding by Eddington at al. In 1978 in Melbourne a physiologically-based method of processing speech was evaluated. It separated speech sounds into different frequency bands like the normal cochlea, introduced frequency time delays appropriate for the basilar membrane travelling wave, and jittered the stimuli to copy

nerve responses. Speech understanding was very limited because the electrical currents on each electrode overlapped in space and time, and this produced unpredictable variations in loudness. This discovery, led to the important principle that only non-simultaneous stimulation should be used.

The clue to developing an effective speech processing strategy came in 1978 when the patient described the sensations at each electrode as "vowel-like". The vowels were similar to the one with a single formant frequency exciting the same area of the cochlea for example /ʌ/, and /ɪ/ and long vowel equivalents /a/ and /i/. The exception was /ɒ/ and /ɔ/ for the electrode outside the cochlea which stimulated low frequency fibres around the nerve. It was also noted that the vowel heard, could be changed by increasing or shortening the duration of the stimulus, as seen for vowels presented to normal hearing subjects.

Because of the importance of formant frequencies for speech understanding and in particular the second formant, the inaugural strategy selected this as place of stimulation, the sound pressure was coded as current level, and the voicing frequency which is low in frequency was coded as pulse rate.

This strategy was first implemented on the laboratory computer and provided a 300% improvement in lipreading and some speech understanding using electrical stimulation alone.

The strategy was then realized as a portable unit that the patient could wear.

Having shown proof that multiple-channel electrical stimulation of the auditory nerves could provide understanding of running speech, it was developed industrially by the pacemaker firm Telectronics/Nucleus and later its subsidiary Cochlear, with financial support from the Australian government. In 1985 after an international trial the Cochlear Limited implant became the first multiple-channel device to be approved by the US FDA as safe and effective in providing speech understanding both with and without lipreading for adults who had hearing before going deaf.

The research to understand why the electrical coding strategy was effective showed firstly that pitch was perceived for spatial as well as coarse temporal information, but not for fine temporo-spatial information, where the neural firing of neighbouring nerve fibres is correlated.

Additional research showed that there were progressively better open-set word and sentence scores for the extraction of the first formant in addition to the second formant (the F0/F1/F2 processor), the addition of high fixed filter outputs (MUPLTIPEAK) and then finally 6 to 8 maximal filter outputs at low rates (SPEAK) and high rates (ACE). All the frequencies were coded on a place basis. There was a steady improvement in results, but they are now reaching a plateau and need strategies to provide a fine temporo-spatial pattern of stimuli.

The last key question was would children born deaf be able to develop the right neural connections for understanding speech with electrical stimulation. To test children the implant was modified to have a magnet in the centre of the transmitting and receiving aerial so that it could be easily aligned with the external transmitting coil. The first three children to receive the multiple-channel cochlear implant, deafened early in life were operated on in 1985 and 1986.

Following the successful results on the first three children in Melbourne, a world trial was undertaken for the US FDA on children from two years to 17 years. The results for the F0/F1/F2 and Multipeak strategies for 91 children born deaf and 51 deafened after birth showed there were greater numbers of children able to reach open and closed-set word levels after the implant than before.

In 1990 the FDA announced that the 22-channel cochlear implant was safe and effective in enabling deaf children from ages two through 17 years to understand speech both with and without lipreading. It was the first cochlear implant to be approved by any world regulatory body for deaf children.

It was also important to determine whether electrical stimulation of the auditory nerve during the early phase of the child's brain development led to better speech perception. An analysis of results showed a trend for better speech perception the younger the child, and suggested they would be best under two years of age. There was also considerable variability in results.

In view of the improved results for young children, and a trend showing they could be even better under two years of age, further biological studies were undertaken before operating on these young children, as there were special safety issues to be addressed. These issues were the effect of head growth on the implant and visa versa, and preventing the spread of a Pneumococcal middle ear infection, to the cochlea with the risk of meningitis. It was only after the safety studies were complete, and showed minimal risk that operations were carried out on young children.

With all children the ultimate aim has been for them to develop normal spoken language for their day-to-day life. This goal is being achieved by early diagnosis, early intervention to develop listening skills and integration into mainstream primary schools for children with normal hearing. For example, in 18 children at a primary school which provides "Auditory-verbal" education, when their receptive language measured as PPVT was plotted versus chronological age, 61% had a slope>1) i.e. they were developing language faster than children with normal hearing. In addition, 40% of the 18 children showed normal or above normal receptive language. The remaining children had language levels comparable to that expected for children with hearing aids and a severe hearing loss.

This research is the result of a team effort at The Bionic Ear Institute and the University of Melbourne. The research is described in more detail in *Cochlear Implants: Fundamentals & Applications* by G.M. Clark (Springer-Verlag, 2003) in the AIP series in Modern Acoustics and Signal Processing (Editor-in-Chief Robert T. Beyer), and in "Cochlear Implants" by G.M. Clark in *Speech Processing in the Auditory System*, edited by Steven Greenberg, William A. Ainsworth, Arthur N. Popper, Richard R. Fay, volume 18 of the Springer Handbook of Auditory Research (Springer, 2004).