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COMPUTER SIMULATION OF AUDITORY EVOKED RESPONSES

Michael J. Pont and Kien Seng Wong

Bioengineering, Transducers and Signal Processing Research Group, Department of Engineering, University of Leicester, Leicester, UK.

INTRODUCTION

An auditory evoked response (AER) is a change in the on-going electrical activity of the auditory system that is stimulated by sounds. The AER, which may be measured by means of electrodes placed (non-invasively) on the scalp and near the ears, is of small amplitude (typically 0.5 - 2μv), and must be amplified and processed to separate it from general background brain activity and other noise. AERs recorded in this way consist of a series of low-amplitude positive and negative potentials ("waves").

Since the development of small flexible computers and powerful amplifying equipment in the mid-1960s, AERs have become central to the practices of audiology, neurology, otology, and related disciplines (Glattke, 1983): for example, the auditory brainstem response (ABR) finds clinical application in the diagnosis of multiple sclerosis, acoustic neuromas ("tumours" in the auditory nerve) and other nervous system diseases (Sehmi, 1988).

Origins of AERs

AERs originate from a polarisation of charge across the membranes of cells in the auditory nervous system. The ABR is most effectively obtained from a high intensity click stimulus, and can contain up to seven waves, numbered I to VII (Jewett *et al.*, 1970). These seven components of the ABR are assumed to have been generated by the auditory nerve and subsequent structures of the ascending brainstem auditory pathway. For instance, Wave I of the ABR is a negative potential recorded at the ipsilateral ear, and is thought to be a manifestation of the VIII nerve action potential generated in response to the click stimulus: similarly, Waves II and III are thought to originate from the cochlear nucleus and superior olivary complex respectively (Moore, 1987).

Applications

While it is accepted that the various waves of the ABR arise in large part through the activity of the brainstem nuclei, the precise correlations of scalp-recorded ABR components with particular nuclei is unknown, but certainly complex. As Sehmi (1988) has noted, the ABR generators within the brain might be serially or non-serially linked, simultaneously active, or have sustained activity. This situation restricts the value of ABR measurement as a clinical tool: for instance, in response to observed changes in Wave III, it is not possible to infer simply that some change or damage has occurred within the superior olivary complex. One aim of the research described in this paper is to develop a simulation of ABRs that can be used to assist clinical personnel in the interpretation of such waveforms.

From the point of view of non-clinical researchers involved in the study of the neurobiology of language, we believe that the simulation and recording of AERs has the potential to provide an important adjunct to more frequently used techniques. The equipment necessary for recording AERs is not expensive, and can be used by non-clinical personnel to make non-invasive recordings. We believe that measurements of the AER from volunteer subjects, used in conjunction with detailed anatomically- and physiologically-motivated simulations of the AER, have an important contribution to make to the investigation of the neural mechanisms underlying human speech and language processing. Specifically, we have begun a programme of work aimed at simulating the ABR of normal-hearing and hearing-impaired listeners. Inevitably, when developing such models, assumptions must be made due to a lack of appropriate data. By adjusting our models, as necessary, to improve the correlation between observed and simulated AERs, we hope to improve the quality of our simulations. Such studies will contribute to a general understanding of the mechanisms of hearing.

The particular aim of the studies described in this paper was to explore the possibility of using an existing computer simulation of processing within the auditory nerve and cochlear nucleus (Pont and Damper, 1991) to simulate aspects of the AER.

Simulation of APs

The whole nerve action-potential (AP) is arguably the simplest of the AER measures, and was therefore chosen as the topic of the first experiments described in this paper. The clinical measurement of the AP is typically made by placing a large electrode (non-invasively) near the cochlea and averaging the potential resulting from multiple applications of a stimulus with a rapid rise time (such as a click) that will generate simultaneous activity in a sufficiently large number of auditory (VIII) nerve fibres so as to allow a measurement to be made. A typical AP recorded in this way is shown in Figure 1.

Clearly, under normal conditions, it is impossible to record the AP without simultaneously recording responses from brainstem nuclei, particularly the nearby cochlear nucleus. We therefore felt that our simulation of processing of afferent neural activity within the AN and DCN was appropriate for use in the present study.

Simulation of ABRs

In the second half of this paper, we describe a prototype system capable of simulating the more general ABR response. This prototype is capable of simulating the evoked response to click and tone burst stimuli, at any required signal level. In addition, it allows the user to simulate responses from subjects with normal hearing, and with a range of auditory and general nervous system disorders.

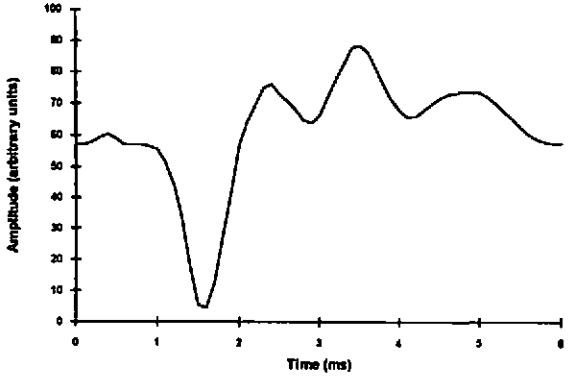

Figure 1: The whole nerve AP response recorded from a normal listener (Re-drawn from data in Glattke, 1983: Fig. 3.7).

THE COMPUTATIONAL MODEL

The Original Model

The computational model at the heart of the present study is essentially that described by Pont and Damper (1991). Briefly, the model simulates afferent neural processing up to the level of dorsal acoustic stria. The model consists of two scissile stages simulating (1) the cochlea and AN and (2) the DCN. The model derives its input from a 120-channel cochlear filterbank. Cochlear transduction, rectification, logarithmic compression, and two-tone suppression functions are performed at the first stage of the simulation. The 480 artificial neurons employed here model the cell at the level of transmembrane potential and have interconnections that follow closely those reported in recent anatomical and physiological studies.

Simulation of ABRs

Usually, the above model is used to simulate action potentials at the level of the auditory nerve, and from various units within the cochlear nucleus (e.g. Pont and Mashari, 1993). It was therefore necessary to make some modifications to the program source code for the present study.

Moore (1987) has argued that the best fit of known (human) brainstem anatomy to the ABR waveform is obtained by assuming that somatodendritic potentials in cell groups are reflected as negative deflections in the evoked response, and massed axonal activity as positive waves. For the first study described here, we made some minor modifications to our model to allow the simultaneous measurement and summation of such activity from the units in model. Further details of the simulation technique used can be found in Pont (1993) and will not be repeated here.

Stimuli

The stimuli used in the simulations described here were all ideal impulse stimuli, approximating the click stimuli commonly used in clinical studies. The stimuli were applied to the model at various signal levels.

SIMULATION OF THE WHOLE NERVE AP

Experiment One

This experiment involved the application of a click stimulus to an early version of our model at a level of approximately "70 dB SL" (measured as detailed in Pont and Damper, 1991). The stimulus was applied 100 times, and the responses were smoothed and averaged. The result is shown in Figure 2.

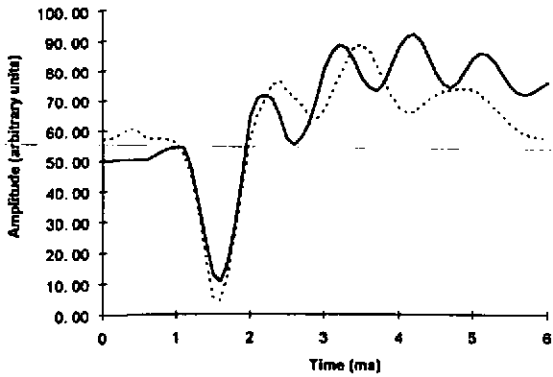

Figure 2: A simulated whole nerve AP response (solid line) compared with the typical response for a listener with normal hearing (dotted line).

Clearly, while the match between simulated and recorded AP is less than perfect, the responses are qualitatively similar. In particular, both responses show a pronounced negative peak, followed by a number of smaller positive deflections, on a similar time scale.

Experiment Two

One of the most important measures in clinical studies involving AERs is the latency of the N_1 wave following the application of the click. Typically, in a normal hearing listener, we expect a quasi-linear relationship between this latency measure and the level of the applied click stimulus (in dB), with a low latency (about 1.5 ms) at a signal level of some 80 dB SL, increasing to around twice this value at about 40 dB SL (see Figure 3a).

Figure 3a also shows the change in latency response expected for listeners with impaired hearing. Again, the relationship of latency to signal level is almost linear, but this time the rate of latency decline is greater.

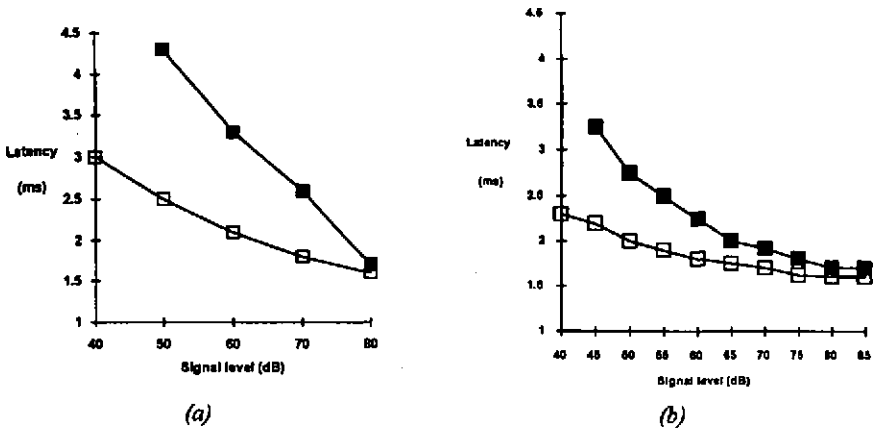

Figure 3: (a) A comparison of the latency of the N_1 response at different stimulus levels for listeners with normal (□) and impaired (■) hearing. (Data adapted from Glattke, 1983, Figure 6.4). (b) A comparison of the latency of the N_1 response at different stimulus levels for the simulation of listeners with normal (□) and impaired (■) hearing described in the text.

Simulation of listeners with normal hearing

In Figure 3b, the latency measures calculated for click stimuli at a range of levels are shown using a model described in detail in Pont (1993).

While the model response is an imperfect match to the original, and in particular the overall latency range is smaller (0.7 ms in the simulation cf. 1.4 ms from the listeners) the simulation response does show the same "linear" trend found in normal-hearing listeners.

Listeners with impaired hearing

For this experiment, we made some further modifications to the models to allow the simulation of responses from listeners with impaired hearing.

Following Moore (1991), we simulated a listener with noise-induced hearing damage in the high-frequency region by (1) increasing the bandwidth of the filters used in the filterbank by a factor of two, (2) raising the filter thresholds by 20 dB, and (3) reducing the range of frequencies spanned by the filterbank from 100 - 5000 Hz to 100 - 3000 Hz.

The impact of these changes is shown in Figure 3b. As might be expected in the light of the results for the "normal hearing" simulation above, the latency change obtained in the "impaired hearing" simulation is smaller than that measured experimentally. However, comparing the simulation results with those in Figure 3a, it is clear that the responses obtained are again qualitatively similar, and in particular that the rate of latency decline is greater in the impaired hearing simulation.

SIMULATION OF THE ABR

Encouraged by the simulation results obtained for the simulation of the AP response described above, we have begun a programme of work aimed at the development of a simulation of the ABR at a finer level of detail. We have at present developed a simple prototype system (described below) which will form a framework for our future work in this area. We describe this prototype and some of our preliminary results below.

The Prototype

A screen from the prototype simulation is shown in Figure 4.

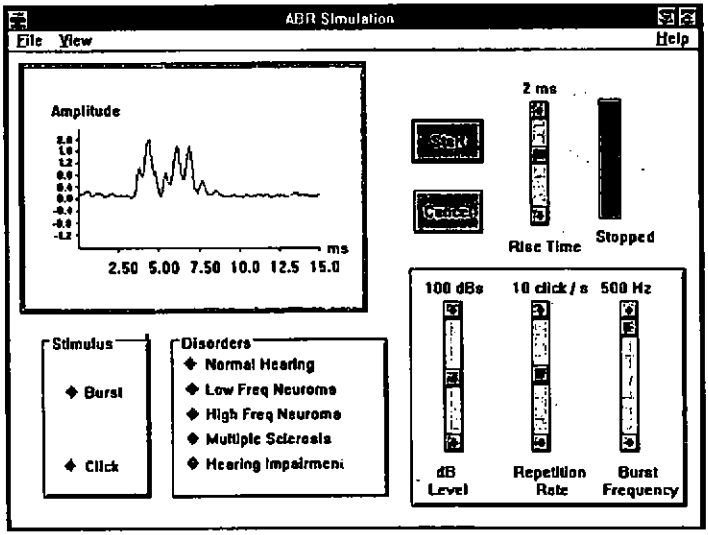

Figure 4: The main screen from the ABR simulator. This version of the program has been developed as a prototype system for decision support in the analysis of ABR waveforms.

The present prototype has a "user-friendly" graphical interface, and allows the user to simulate responses (which are displayed on the main screen at the top left of Figure 4) from normal hearing listeners, as well as those suffering from noise-induced hearing impairment, acoustic neuromas, and multiple sclerosis.

As is apparent from the figure, the timescale of the ABR response is roughly twice that for the lower-level AP described earlier. To generate the responses, the original computational model (Pont and Damper, 1991) has been extended to include aspects of processing within the ventral cochlear nucleus, inferior colliculus and medial geniculate body. These simulations are, at the present time, at a very low level of detail: each nucleus is modelled as an amplifier (plus delay) of the incoming signal, and the connections between nuclei are modelled as attenuators (plus delays). A major focus of our current work is to improve the resolution of all stages the present simulation.

Given the simple nature of this prototype simulation, it would be surprising if the match between observed and simulated responses was perfect. In fact, the responses obtained are broadly similar. One example is given in Figure 5. The figure shows an ABR from a normal-hearing subject, along with a simulation produced by the model. While the present model lacks the resolution required to reproduce the slow underlying alpha wave evident in the original, the multi-peak nature of the ABR is clearly evident.

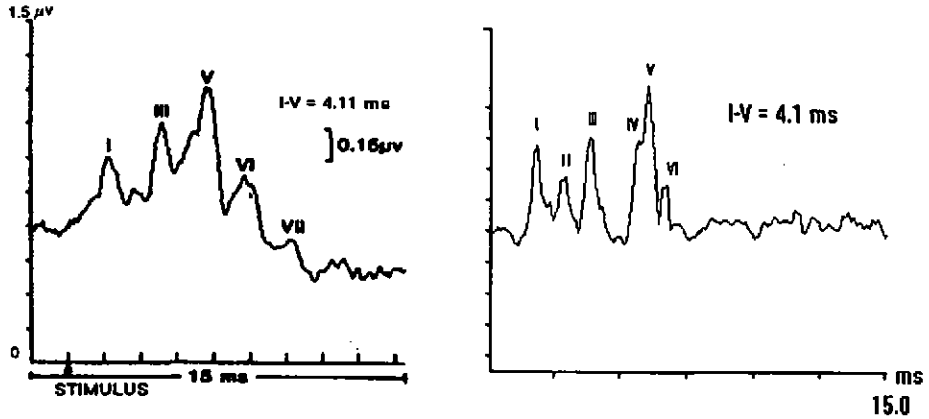

Figure 5: A comparison of the ABR response from a normal-hearing subject (left), with the simulated response from the model. Left hand figure adapted from Hall, J.W. (1992) "Handbook of Auditory Evoked Responses", Allyn and Bacon, MA, USA. Reproduced with permission of the publisher.

DISCUSSION AND CONCLUSIONS

In this paper, we have reported the results of a pilot study which attempted to simulate a simple ABR by means of a previously-developed computer model of the mammalian auditory nervous system.

The results demonstrate that, with our existing detailed model of auditory processing at up to the (dorsal) cochlear nucleus, we are able to reproduce the AP response, including the changes in latency with signal level, at a reasonable level of accuracy. Currently, using a simple model of auditory

processing within the remainder of the auditory brainstem, we are able to reproduce the general form of the generic ABR response.

The results are encouraging, and we feel that the area merits further study. By extending our existing computer model, in terms of both the areas of the nervous system modelled and the detail of the existing programs, we believe we can improve the correlation between the recorded and simulated ABRs. We then hope to be in a position to relate fine details of the simulated responses to particular structures and unit types within our model, and to reproduce the range of ABR responses seen from subjects of differing in age and gender.

ACKNOWLEDGEMENTS

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