COCHLEAR IMPLANTS IN THE SECOND AND THIRD MILLENNIA

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ABSTRACT

Much has been achieved in the Second Millennium in the development of cochlear implants for profoundly deaf people, but further advances in the Third Millennium should result in most severely to profoundly deaf people being able to communicate effectively in a hearing community.

1. THE COCHLEAR IMPLANT

The cochlear implant or Bionic Ear as illustrated in Fig. 1 is an electronic device that replaces the inner ear when it does not function in severely-to-profoundly deaf people.

It consists of a directional microphone which sends information to a speech processor which is worn on the belt or behind the ear. The speech processor extracts information of importance to speech understanding, produces a code for the signal and transmits it by radiowaves to the receiver-stimulator implanted in the mastoid bone. The receiver-stimulator decodes the signal and produces a pattern of electrical stimulus currents in an array of electrodes inserted around the basal turn of the inner ear.

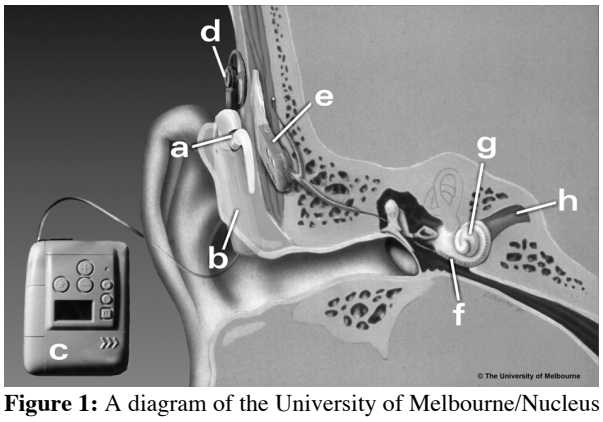

Figure 1: A diagram of the University of Melbourne/Nucleus (Cochlear Limited) multiple-channel cochlear prosthesis. a - microphone; b - cable; c - speech processor; d - transmitter coil; e - receiver stimulator; f - electrode array; g - cochlea; h - auditory nerve (1).

2. DEVELOPMENTS IN THE SECOND MILLENNIUM

In is now hard to realise that only in the 1960's and 1970's many scientists and clinicians said successful cochlear implants were not possible in the foreseeable future.

The fundamental objections, which were reasonable, were as follows:

- **The first objection was that the cochlear hair cells and their nerve connections were too complex and numerous to be replaced by a small number of electrodes.**

This objection was partly answered by research to see how well electrical stimulation could reproduce the temporal and place coding of frequency in normal and deafened ears. It was found that rate of electrical stimuli could not reproduce discriminable pitch above about 300 Hz which is much less than the 3000 Hz needed for speech understanding. However, the research showed that for certain modes of stimulation electrical current could be localized to discrete groups of nerve fibres for the place coding of frequency.

- **The second objection was that there would not be enough residual hearing nerves in the cochlea after die back due to deafness for speech information to be transmitted.**

This objection was partly overcome through studies on the experimental animal where it was found that varying populations of spiral ganglion cells had no significant effect on the discrimination of rate of stimulation. Furthermore, studies on patients comparing various aetiologies, which have different mean ganglion cell numbers, have not shown a significant relationship between cell numbers and speech results.

- **The third objection was that implanting electrodes in the cochlea would destroy the very nerves it was hoped to stimulate. Studies had shown that damage of the basilar membrane could result in nearly total loss of the spiral ganglion cells in the vicinity of the injury.**

This objection was overcome by showing that the gentle insertion of electrodes with the right bio-mechanical properties did not lead to loss of ganglion cells nor did charge-balanced electrical stimulation.

- **The fourth objection was that speech was too complex to be presented to the nervous system for speech understanding to occur.**

This objection was resolved when it was appreciated that due to the limitations of reproducing the temporal and place coding of frequency because of an electroneural "bottle-neck", some form of pre-processing of speech was required. It was also established that speech information was best transmitted through the "bottle-neck" if the electrical currents were not presented simultaneously to electrodes. Simultaneous stimulation led to an interaction of electrical fields which produced unpredictable variations in loudness.

While attempting to determine the pre-processing of speech required it was noted that the patient reported vowel sounds rather than simple sounds when each electrode was stimulated, and the vowels varied according to the electrode stimulated. It was also realised that single formant vowels were perceived when similar locations in normal hearing people, were excited.

As the second formant carries the most intelligibility a speech processing strategy was developed which extracted this second formant, and used the current level to stimulate the electrode closest to that frequency area in the cochlea. Voicing was presented as rate of stimulation, as it is low in frequencies that could be discriminated (Fig. 2)

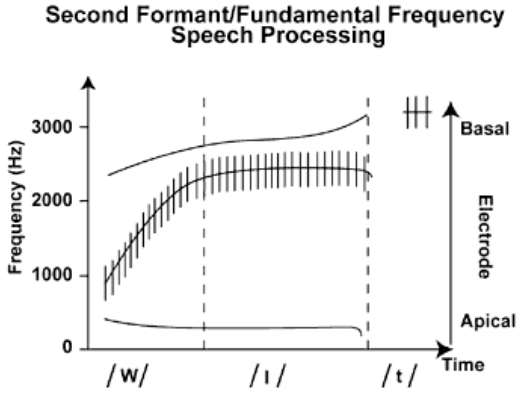

Figure 2: A diagram of the Second formant (F2)/ Fundamental frequency (F0) speech processing strategy for the word /wit/.

This strategy enabled the first patient, who was postlinguistically deaf, to understand running speech when combined with lip-reading, and some speech using electrical stimulation alone.

Further research has also shown that the extraction of additional formants or the outputs from band pass filters, and their presentation on a place coding basis, resulted in improved speech perception scores as shown in Fig. 3.

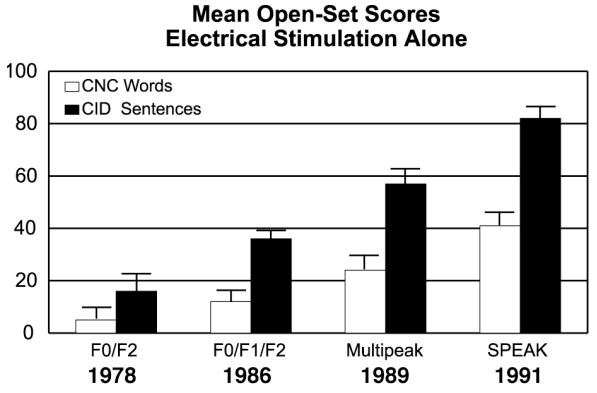

Figure 3: CID open-set sentence scores for electric stimulation alone for the F0/F2, F0/F1/F2, Multipeak, SPEAK strategies on unselected patients at the Royal Victorian Eye and Ear Hospital Cochlear Implant Clinic three months postoperatively. The speech processor which presents the strategies is shown following the strategy (1).

The improved results obtained by progressively increasing the amount of information presented on a place coding basis, as well as the way the information is extracted from speech, have now produced results that enable a majority of profoundly and post-linguistically deaf adults to understand significant amounts of running speech using electrical stimulation alone. The results are also comparable to those from severely deaf people with some residual hearing and a powerful hearing aid.

- **the fifth objection was that children born deaf or deafened early in life would not have developed or be able develop the right neural connections for hearing to be reproduced.**

This objection was resolved in Melbourne after establishing the benefits of the cochlear implant in adults. We implanted our first three children in 1985 and 1986. This also led to an international trial to determine whether the multiple-electrode cochlear implant would benefit children who became deaf during their early life or were born deaf. The trial showed that 60% of children born deaf were able to understand some open-set speech, and the majority of the others had significant help in lipreading.

The FDA approved the device as safe and effective for children two years of age and above in 1990. It thus became the first major advance for deaf children unable to obtain assistance with a hearing aid since sign language of the deaf was discovered in Paris 200 years ago.

A subsequent analysis of data on children in Melbourne has shown there is considerable variability in results Fig. 4. The data also emphasises that the results are better the younger the child, and if extrapolated back may even be better if the child is younger than two years of age.

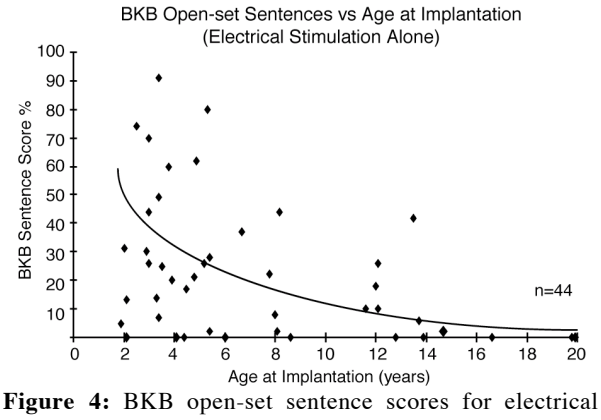

Figure 4: BKB open-set sentence scores for electrical stimulation alone versus age at implantation.

Before deciding to operate on children under two it was necessary, however, to undertake a series of biological studies to ensure that operating on a young child was safe. Young children of this age have special problems. These are firstly the effects of head growth, secondly frequent bouts of middle ear infection which could lead to inner ear complications, and thirdly the effects of electrical stimulation on the developing nervous system This research was part of a special five year contract to the US National Institutes of Health, and showed no cause for concern for operations on young children.

3. RESEARCH DIRECTIONS FOR THE THIRD MILLENNIUM

There are a number of research goals for the Third Millennium to further improve the cochlear implant performance and extend its benefits to as many hard of hearing people as possible. These have been reviewed by Clark (2).

- **the first research goal is to provide better reproduction of the way the brain codes sounds.**

Already research is contributing answers to these questions. Electrical stimulation has helped provide evidence that the temporal coding of frequency is not just through the timing of responses in single fibres, but an interaction between the timing and spatial distribution of the nerve connections to cells in the brain (Fig.5). When nerve fibres converge on a brain cell each fibre carries some but not all of the timing information in response to the sound waves. It is the role of the brain cells to correctly identify the frequency relayed by the group of fibres, and it may do so by using a time window so that the cells only fire if a certain number of appropriate time intervals of the arriving responses are within that time window.

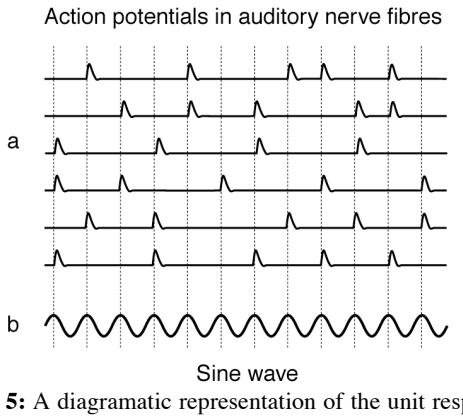

Figure 5: A diagramatic representation of the unit responses in an ensemble of fibres for a low to mid acoustic frequency. (a) Nerve action potentials in a population of neurons, (b) Pure tone acoustic stimulus (3).

Research in the Third Millennium should aim to provide patterns of electrical stimuli in small groups of nerve fibres that are similar to those of sound. To achieve this fine temporal and spatial pattern of responses a new electrode array will be required with many more electrodes to stimulate small groups of auditory nerve fibres (Fig.6).

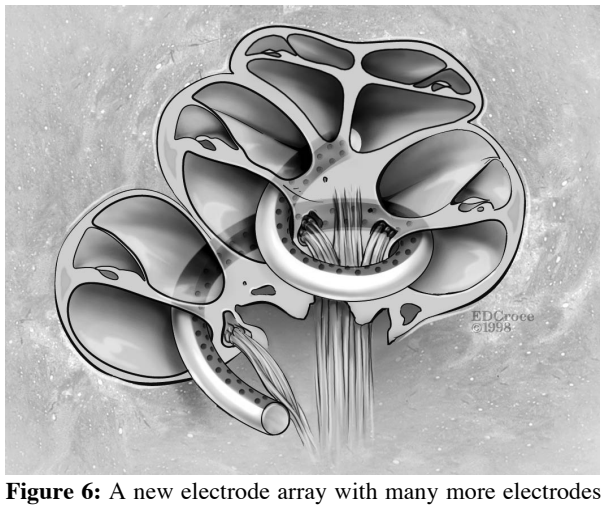

Figure 6: A new electrode array with many more electrodes to stimulate small groups of auditory nerve fibres.

This array should enable the responses to the rapid phase changes in basilar membrane vibration to be reproduced. These occur at the site of maximal vibration. The neural responses to these phase changes may be very important for frequency coding.

- **the second research goal is to further improve the perception of speech and other sounds in noise.**

Already improvements in speech processing in quiet have also resulted in better speech perception in noise. For example, in a group of 10 patients in Melbourne there was a mean CUNY sentence score of 90% for the Speak strategy in quiet, and 60% at a 10dB signal-to-noise ratio. However, a 100% score at a 0dB signal-to-noise ratio would be normal (Fig.7).

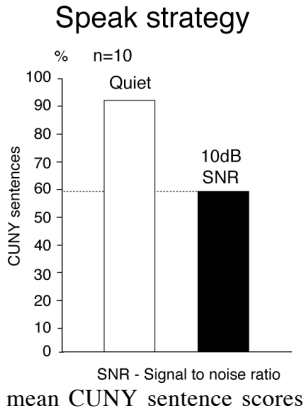

Figure 7: The mean CUNY sentence scores for the Speak strategy in quiet (90%), and at a 10dB signal-to-noise ratio (60%).

Further improvements in speech perception in noise have been achieved using the Griffiths/Jim adaptive beam forming technique and two microphones (4). The Griffiths/Jim beam former, uses an adjustable filter to annul the noise. It works well for a single noise source, but can break down in a reverberant field. The SIT sentence results for the Griffiths/Jim beam former with four cochlear implant patients using the Speak strategy were 82% for the beamformer and a control in quiet, and 44% for the beamformer and 9% for the control at a signal-to-noise ratio of 0dB (Fig. 8). However, the results for this and other beamformers are still not comparable to normal.

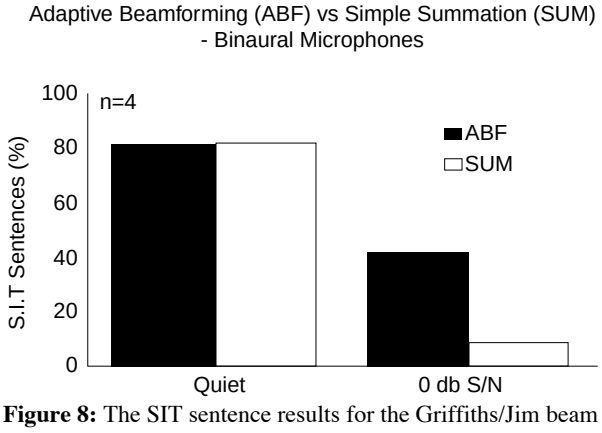

Figure 8: The SIT sentence results for the Griffiths/Jim beam former with 4 cochlear implant patients using the Speak strategy.

Improved perception in noise should be possible by better reproduction of the mechanisms used by the brain. Physiological research shows that binaural neural processing is very sensitive to phase differences between each ear, and this is critical for signal detection in noise. The mechanism for this is a series of delay lines and coincidence detectors. Replication of the parallel processing involved has been difficult electronically, but could provide better results. This should be possible in the future with the development of better electronic neural networks.

- **the third research goal is to develop a totally implantable cochlear implant**

Making the cochlear implant invisible could be important. Teenagers, for example, are very sensitive about their deafness, and may not use their device in company. It would mean implanting the speech processor as well as the microphone in the body.

Some have been approaching the problem by implanting an electret microphone under the skin behind the ear or more recently under the skin of the ear canal. A piezo-electric microphone has also been developed for implanting under the skin of the ear canal. As these approaches have inherent difficulties of frequency response and extrusion, sensors of tympanic membrane and ossicle vibrations are being developed. These are a piezo-electric bimorph cantilever, accelerometer, or fibre optic lever system being researched at the University of Melbourne (5).

The fibre optic lever system detects the ear drum vibrations through the modulation of light intensity. The device has a sensitive membrane coupled to the ear drum, and an optical fibre bundle directs a laser beam at this vibrating membrane. The vibrations cause the laser intensity to fluctuate, and these fluctuations are reflected and picked up by the implanted photo detector electronics, and then converted to a code for stimulating the auditory nerve. The sensor would be placed in the middle ear, and all the electronics and the rechargeable battery in the mastoid process as illustrated in Fig. 9.

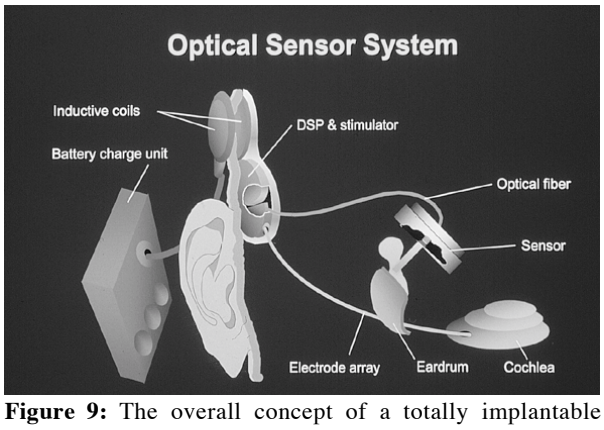

Figure 9: The overall concept of a totally implantable cochlear implant with the sensor placed in the middle ear, and all the electronics and the rechargeable battery in the mastoid process.

- **the fourth research goal is the use of nerve growth factors to protect the hearing nerve from die back after deafness.**

This research using nerve growth factors or neurotrophins may lead to better results with the cochlear implant with more nerves to stimulate, and ultimately the pharmacological cure of a sensori-neural hearing loss. Research in Melbourne (6) has commenced to determine the best combinations and doses of neurotrophins to use. It is being undertaken on cultures of rat spiral ganglion cells, and has shown considerable facilitation in survival when neurotrophin NT-3 was used in combination with neuronal cytokine transforming growth factor TGF-3 (Fig. 10).

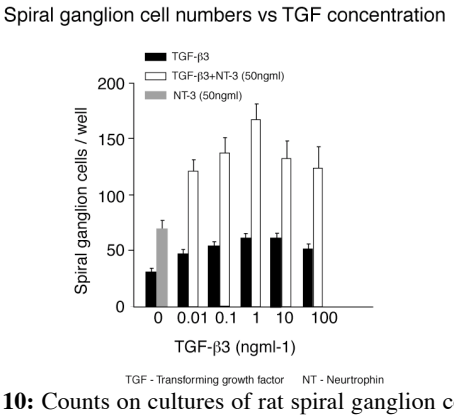

Figure 10: Counts on cultures of rat spiral ganglion cells for a constant dose of the Neurotrophin NT-3 with increasing concentrations of neuronal cytokine transforming growth factor TGF-3.

There are a number of possible ways of delivering the neurotrophin to the site of action and these include a micro-pump (Fig.11), slow release from polymers, and viral vectors. The use of different combinations of factors is being tested in vivo by injecting them into the inner ear with a micro pump.

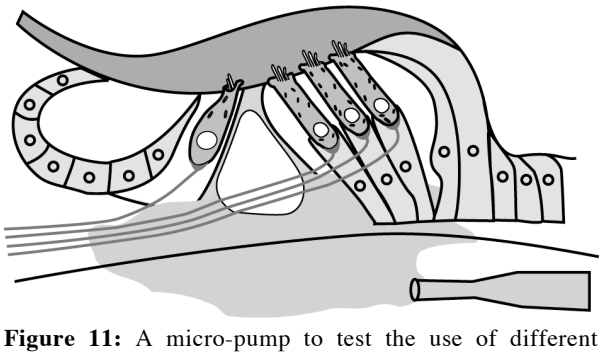

Figure 11: A micro-pump to test the use of different combinations of neurotrophins in vivo.

Not only could these nerve growth factors protect the auditory nerve from die back, but they could cause the auditory nerve and hair cells to regenerate, and ultimately lead to the pharmacological cure of sensori-neural deafness.

- **the fifth research goal is to use and re-establish auditory plasticity, to achieve optimal speech perception in children.**

This research will firstly aim at overcoming defects in temporal and place coding of frequency, and secondly defects in the perception of speech elements, by specific training at these levels. Research in Melbourne has found that some children with poor speech perception have good electrode place discrimination, but they cannot distinguish between the formants required for vowel recognition.

The first and second formant frequencies of vowels are represented with speech processing strategies by two places of stimulation within the cochlea. The spatial separation of electrodes representing the formants in vowel pairs has been calculated as the lengths of vectors in a Euclidean space (Fig.12).

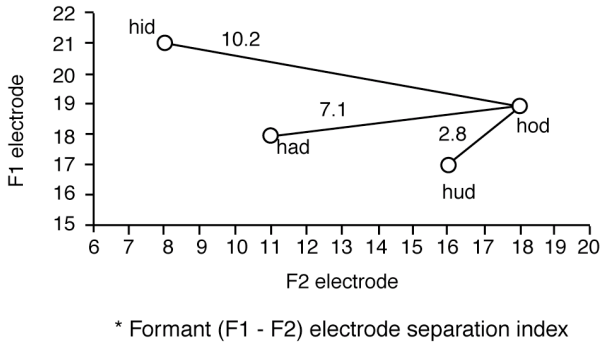

Figure 12: The formant separation indices for some selected vowel pairs.

A study has been undertaken on five children with poor speech perception, but good electrode place discrimination, to train them in formant discrimination when this was poor (7). As four out of five of the children evaluated could discriminate the percepts for different places of electrode stimulation, this should have provided the perceptual information required to distinguish the formant frequencies that are important for vowel discrimination. It was hoped this would also lead to better speech perception overall.

The training was carried out initially to achieve discrimination of the vowel pairs where the formants were most widely separated. It was argued this would result in the changes required for learning to discriminate more closely separated vowel pairs. It was found that two of the four children improved in their abilities to distinguish vowel pairs. In one child gains of minimal vowel pair recognition carried over to improved speech recognition. These results suggest that training to distinguish vowels with widely separated formant frequencies, and then more closely separated ones can be effective, and the benefits retained and carry over to the perception of speech (Fig. 13).

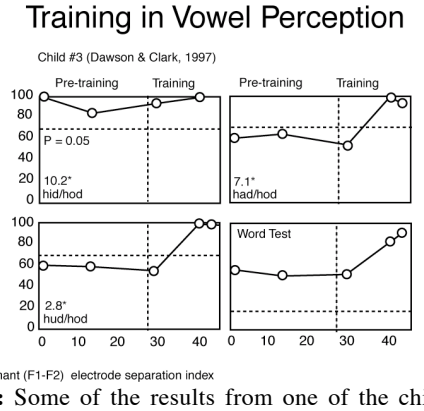

Figure 13: Some of the results from one of the children. This child had an electrode difference limen of 2. The formant electrode separation indices are also shown.

Secondly, in those children where the critical period for plasticity has passed it may be possible to use neurotrophins to facilitate a return of the plasticity required to develop the neural connections for the coding of the frequencies needed for speech perception.

In this situation the neurotrophin would be released from the cochlear implant electrode (Fig.14). The neurotrophin would pass to a receptor site on the synapse between the auditory nerve fiber and cochlear nucleus cell. This would facilitate the release of transmitters across the synaptic cleft. These

would activate a signal cascade of proteins in the cytoplasm that would re-activate the gene for the neurotrophin. Messenger RNA for the protein would be transcribed, and then translated into the neurotrophin in the cytoplasm. The release of the neurotrophin would not only facilitate further transmitter release, but cause the neural sprouting required for the coding of sound, and so re-establish auditory plasticity. The neurotrophin produced in the cochlear nucleus would also propagate to higher auditory centres, establishing appropriate neural connections along the auditory pathways.

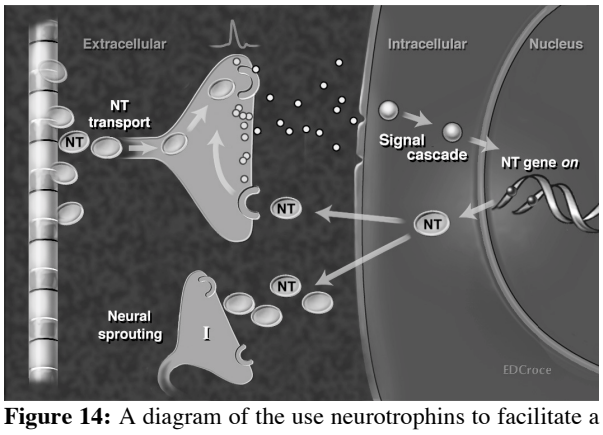

Figure 14: A diagram of the use neurotrophins to facilitate a return of the plasticity required to develop the neural connections for the coding of the frequencies needed for speech perception.

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