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Open Set Speech Perception with Auditory Brainstem Implant?

Vittorio Colletti; Robert V. Shannon

Objective: **Only a small percentage of auditory brainstem implant (ABI) recipients treated for neurofibromatosis type 2 (NF2) have proved capable of identifying words using only the sound from the ABI. Recently, the ABI was applied to a series of patients with no cochlear nerve or with cochlear disorders that could not benefit from a cochlear implant (i.e., cochlear nerve aplasia or posttraumatic avulsion) or whose benefit was or would be severely compromised. A significant number of these patients have proven capable of understanding speech, including effortless telephone use. In the present study, a series of psychophysical tests were administered to determine the cause of the difference in performance between tumor (T) and nontumor (NT) ABI patients.** ***Study Design:*** **Retrospective case review.** ***Setting:*** **Tertiary referral center.** ***Patients:*** **Twenty patients with ABIs participated in the investigation. Ten were NF2 patients and 10 NT subjects. Patient ages ranged from 24 to 61 years. Eleven were males and nine females.** ***Intervention:*** **Auditory rehabilitation in auditory disconnection caused by cochlea or cochlear nerve disorders.** ***Results:*** **There was a significant correlation between modulation detection thresholds and speech understanding and a significant difference in modulation detection between T and NT patients.** ***Conclusions:*** **The difference in modulation detection between the two groups suggests a difference in the survival of specific cells in the cochlear nucleus that support modulation. The pattern of results indicates a separate pathway of auditory processing that is specialized for modulated sounds, and that pathway is critical for speech understanding. In NF2 patients, the tumor and surgery may selectively damage this pathway, resulting in poor speech recognition with prosthetic stimulation.** ***Key Words:*** **Auditory brainstem implant, speech perception, amplitude modulation.**

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INTRODUCTION

Patients deafened by the bilateral loss of the primary auditory nerves have been treated with the auditory brainstem implant (ABI) since 1979 to restore auditory sensations.[1,2]

The ABI is placed in the lateral recess of the IV ventricle of the brainstem on the surface of the cochlear nucleus (CN). Most patients receiving the ABI cannot benefit from a cochlear implant (CI) because their VIII nerve is removed during removal of a vestibular schwannoma, a condition typically caused by neurofibromatosis type 2 (NF2).

To date, worldwide, approximately 500 NF2 patients have been implanted with ABIs, receiving a functionally beneficial level of auditory information to assist them in their communication abilities. ABI provides subjects with access to auditory information such as environmental sound awareness together with information on stress and rhythm in speech that will assist them in lip reading. Only a small percentage is able to identify words or sentences in the auditory-only mode, and the ability is usually minimal.

The overall performance is therefore considered no better than that achieved by single-channel CIs.[2,3] Multichannel CIs, in contrast, typically restore speech understanding in postlingually deafened adults to a level where most can converse on the telephone.

The cause of the large difference in performance of the CI and ABI has not been clear. The two devices use similar signal processing and a similar number of stimulating electrodes, but differ primarily in the location of stimulation: cochlea versus CN.

One hypothesis (the selectivity hypothesis) is that the ABI does not make selective contact with the tonotopic dimension of the CN, limiting the number of independent channels of spectral information. If each electrode activates a broad area of neurons, and adjacent electrodes activate largely overlapping populations, this overlap will have the effect of smearing the representation of spectral place.

An alternate hypothesis (the bypass hypothesis) is that the difference in speech understanding between CI and ABI is caused by the ABI bypassing or distorting activation of specialized neural circuitry occurring in the CN. The CN is a complex structure with many types of

From the Department of Otolaryngology (V.C.), University of Verona, Verona, Italy, and the House Ear Institute (R.V.S.), Los Angeles, California, U.S.A.

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Send Correspondence to Professor V. Colletti, Policlinico G.Rossi, P.le L.A. Scuro, 10, 37134 Verona, Italy. E-mail: vittoriocolletti@yahoo.com

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specialized neurons. Projections to higher auditory centers are both ipsilateral and contralateral, with significant projections between cell types within the CN. Stimulation of a CI activating the VIII nerve may still allow some of the intrinsic specialized CN circuitry to work in a relatively normal fashion, whereas direct activation at the surface of the CN may simultaneously activate competing pathways and so may not allow the specialized functions to work effectively.

Recently, the ABI was applied to a series of patients that presented disconnection of the central auditory system from the external sound environment because of a variety of cochlear or cochlear nerve malformation or disruption (e.g., aplasia or hypoplasia of the cochlear nerve, skull basal fracture, VIII nerve avulsion, cochlear ossification, etc.).[4,5]

A significant number of these patients showed surprising auditory performance, being able to understand speech at a level comparable with the most successful CI users, including conversational telephone use. No NF2 ABI patient has achieved this level of performance.[4,5]

These observations suggest that stimulation of the CN can produce functional hearing in the absence of tumors but that the tumors or tumor removal process may cause damage to the CN that significantly diminishes speech understanding. In addition, these outcomes eliminate both the selectivity and bypass hypotheses because they demonstrate that the existing surface electrode ABI can produce high levels of speech understanding.

The differences between these two patient groups appear to be subtle: both groups have no functioning auditory nerve, and both groups have the same ABI device with the same stimulation strategy. Yet, there is a large difference in performance that is related to the etiology. The existence of this patient dichotomy provides us with considerable leverage on a key question in auditory processing: is there a specialized physiologic pathway for speech recognition?

To determine the cause of the difference in performance between tumor (T) and nontumor (NT), 20 subjects (10 T and 10 NT) from a total population of 71 patients fitted with ABI were submitted to a series of psychophysical tests aimed to investigate the accuracy of electrical placement, stimulation selectivity, and physiopathologic properties of each group.

MATERIALS AND METHODS

From April 1997 to April 2005, a total of 71 patients (53 adults and 17 children; age range 14 months to 70 years) were fitted with ABIs in the ENT department of the University of Verona. All patients were suffering from a variety of T and NT diseases of the cochlear nerve or cochlea. The retrosigmoid-transmeatal approach was used in T patients and the retrosigmoid approach in NT patients.[6]

Twenty ABI patients (10 NT and 10 T [NF2]), consecutively operated on from May 1999 to December 2003 (T patients) and from August 2002 to December 2003 (NT patients), were selected to have a follow-up of at least 1 year. They were submitted to a series of psychophysical tests to determine the cause of the difference in auditory performance between the two groups. The auditory performance of both groups was comparable with that obtained in the remaining ABI population. In particular, open set sentence recognition score in the auditory only mode, obtained at 1 year after implantation, was 10% in the tested T group and 15% in the remaining T patients (P = ns). Performance was 65% in the tested NT group and 61% in remaining NT patients (P = ns).

Stimulation thresholds were measured as an indication of electrode proximity to stimulable neurons.[7,8] Electrode selectivity was evaluated with forward masking, a technique to quantify interaction between electrodes.[8] Modulation detection was investigated as an indication of temporal resolution, a measure that is strongly correlated with phoneme recognition in CIs.[9] Speech understanding was measured for phonemes (vowels) and simple sentences (Hearing in Noise Test).[10]

Electrical Threshold

This test furnished an indication of electrode proximity to stimulable neurons. A 500 millisecond burst was presented at a randomly selected level near threshold. The level was increased to the lowest level at which the listener reported hearing the burst on at least three of five presentations.

Electrode Selectivity

A forward masking technique was used. A 250 millisecond masker was placed on a middle electrode at a level that was comfortably loud. Ten milliseconds after the offset of the masker, a 25 millisecond signal was presented on another electrode in the same row of electrodes along the implant array. The listener heard two intervals; both contained the masker, but only one selected randomly contained the signal. The level of the signal was adjusted adaptively according to a three down, one up rule to converge on the signal level that would produce 79% correct detection. This level was used as the masked threshold. Masking was calculated as the elevation of the signal above quiet threshold for the same stimulus, measured in decibels. Masking typically diminished as the distance was increased between the masker and signal electrodes. The width of the interference was interpolated as the distance in millimeters where the masking dropped by 1 dB from the peak masking level.

Amplitude Modulation

Detection of amplitude modulation was measured in a two-alternative, forced-choice adaptive task. A 400 millisecond unmodulated biphasic pulse train (200 us/phase, 250 pps) was presented in one of the intervals selected at random. The other interval contained the same carrier stimulus sinusoidally amplitude modulated at either 10 or 20 Hz. The modulation depth was adjusted adaptively to obtain the level that produced 79% correct responses. Modulation detection threshold was computed from the final eight reversals in the adaptive procedure.

Speech Understanding

Vowel recognition was measured using 12 medial vowels in an h/V/day context. Tokens were spoken by five male and five female talkers selected from a speech database.[11] Vowels were presented in random order, and the listener selected the vowel heard from a 12 alternative matrix presented on a computer screen. Six Italian vowels were recorded by a single male talker in /V/, b/V/l, and n/V/t/V contexts. Listeners selected the vowel heard from alternatives presented on a computer screen.

Simple sentences[10] were presented in random order in a sound field at a comfortable listening level. Listeners were instructed to repeat whatever they heard, and sentences with each word correctly repeated was scored. The same sentences were translated into Italian and spoken by a single male native Italian talker. Approval of the Verona University Hospital Ethical Committee was obtained before initiating the study.

RESULTS

Electrical Threshold

T1 Auditory thresholds were as low as 2 nC (Table I), indicating a distance of less than 1 mm between electrodes and auditory brainstem neurons.[7,12] Similar thresholds were observed in NT ABI patients with excellent speech recognition and in NF2 ABI patients with poor speech recognition, indicating good electrode placement and good survival of stimulable neurons in both T and NT cases. There was no significant correlation between electrical thresholds and sentence recognition ($r = -0.21$) or vowel recognition ($r = -0.18$) and no significant difference in threshold levels between NF2 and NT patients ($P = .22$), suggesting that electrode proximity to the auditory brainstem was not different between NF2 and NT patients.

Electrode Selectivity

Electrode stimulation selectivity in NT listeners was measured by placing a masking stimulus on one electrode to produce a comfortably loud sound. Interference was measured as a function of distance from the masker electrode by forward masking. Selectivity was quantified as the distance in millimeters at which the interference dropped by 1 dB from the maximum masking. These selectivity measures were also not significantly correlated with vowel recognition ($r = 0.25$) or sentence recognition ($r = 0.45$), suggesting that the selectivity hypothesis was not correct. Some patients with excellent speech understanding had relatively poor selectivity, and some patients with poor speech understanding had excellent selectivity.

Amplitude Modulation

Amplitude modulation detection was measured at 10 or 20 Hz modulation frequency as a function of the loudness of the carrier signal.[9] Average modulation detection threshold was computed for at least four levels of loudness ranging from very soft to very loud. There was a significant correlation between average modulation detection threshold and sentence recognition ($r = -0.69$), a significant correlation between modulation detection and vowel recognition ($r = -0.59$), and a significant difference between T and NT patients in modulation detection ($P < .005$), vowel recognition ($P < .001$), and sentence recognition ($P < .005$) (Fig. 1). F1

DISCUSSION

ABI is indicated in a series of T and NT disorders characterized by a "disconnection" between environmental sounds and the central auditory system.[1,2,4,5] The degree of auditory benefit varies as a function of the underlying pathologic conditions, with NT subjects exhibiting significantly better outcomes than the T patients. In particular, Colletti et al.[4] obtained average open set sentence recognition score in the auditory only mode of 63% in the NT group and 12.2% in the T group. In view of the site and nature of the lesions, NT patients were considered ideal for ABI application because the brainstem was anatomically intact, unlike in NF2.[4] AQ: 2

In the present investigation, although ABI patients with tumors had excellent electrode placement and selectivity, they had significantly poorer modulation detection and speech understanding than NT patients. Both etiology groups had a wide range of pitch perception across electrodes and a full range of loudness percepts, suggesting that both groups had sufficient surviving neurons in the CN to support pitch and loudness. The difference in modulation detection between the two etiology groups suggests that there may be a difference in the survival of

TABLE I.
Psychophysical and Speech Results from NF2 and NT ABI Listeners.

Nontumor						NF2					
Subject	HINT, %C	Vowels, %C	Average Modulation, dB	Thresh (nC)	Tuning (mm)	Subject	HINT	Vowels	Modulation, dB	Threshold	Tuning (mm)
NT1	88.5	61.3	−29.3	3	0.98	NF2-1	0	28	−17.1	2.6	0.58
NT2	45.2	60	−20.2	3.7	1.75	NF2-2	0	17	−11.4	3.9	
NT3	97.5	94.7	−27.6	3	2.54	NF2-3	10	21	−18.3	7.6	
NT4	90.6	85	−37.6	12.3	3.14	NF2-4	0	25	−12.2	49.3	
NT5	42.5	44.6	−25.5	4	0.73	NF2-5	3	25	−5.4	7.7	
NT6	0	23	−20.9	22.1	1.6	NF2-6	8	6	−18.3	16.9	
NT7	0	17	−22.6	2.8		NF2-7	0	29	−10	4.8	
NT8	6.3	35.2	−22.3	3.6	3.2	NF2-8	0	8	−21.1	6.7	
NT9	49	65.3	−11.2	8.5		NF2-9	0	8	−10.9	9.9	
NT10	87	96	−28.2	11.1		NF2-10	6	21	−23.5	2.9	
AVG	50.66	58.21	−24.54	7.41	1.99	AVG	2.70	18.80	−14.82	11.23	
St dev	39.07	28.27	6.96	6.28	0.99	St DEV	3.89	8.66	5.68	14.02	
t test, T versus NT	0.0018	0.0008	0.0016	0.22							

NF2 = neurofibromatosis type 2; T = tumor; NT = nontumor; HINT = Hearing in Noise Test; %C.

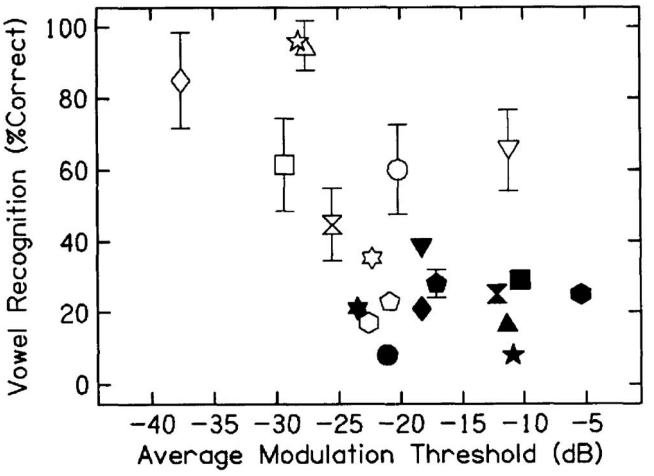

Fig. 1. Legend to come.

a specific neuronal pathway that is critical for modulation detection. The difference in speech recognition between NF2 and NT groups suggests that this putative pathway is also linked to speech recognition.

It is not clear why there might be a difference in the survival of specific cell types in the CN for NF2 and NT patients because all pathologies included in this population involve the VIII nerve. One hypothesis is that the growth and removal of NF2 tumors can compromise the vasculature of the CN. The tumor or its removal causes some type of damage to a system that is critical for speech recognition. If the tumor removal damages a specific cell type or region of the CN that is important for speech, it would be an important new advance in understanding the role of peripheral physiology in perception. NF2 tumors that are larger than 2 cm will contact the surface of the brainstem. Although benign, NF2 tumors produce an angiogenesis factor that attracts vascular blood supply from the surface of the brainstem in the cerebellopontine angle (i.e., the surface of the CN). Although the existence of the tumor and the shared vasculature may not impair the functioning of the CN (some patients with 4–5 cm tumors can still have normal hearing and speech understating before surgical removal), tumor removal and surgical cautery may damage CN cells that share blood supply with the tumor.

It is clear from the threshold measures and selectivity measures that not all CN cells are compromised in NF2 patients, implying that the modulation-specific cells are more labile or more vulnerable than other CN cell types to vascular damage or transient anoxic episodes, as can occur during surgical removal of tumors. Chopper cells are more susceptible to transitory anoxia and hypoxia than other CN cell types because of their large size and so large metabolic requirement. The small cell cap (SCC) of the CN (also called marginal cells) is also a candidate for the damaged pathway. The SCC also receives projections from small cells in the vestibular nerve root[13] and the VS of NF2 originates on the vestibular branch of VIII nerve. The SCC lies on the surface of the nucleus and would be in contact with large NF2 tumors. It is also known that cells in the SCC have larger dynamic ranges[13] and receive projections from high-threshold, low-spontaneous rate auditory neurons, which preserve spectral profiles across frequency in firing rate, even at high sound intensity levels.[14]

AQ: 3

Although the SCC is not well characterized, it is thought to project to medial olivary complex and to possibly have a role in intensity coding because of the wide dynamic range of its neurons.[15] The primary auditory neurons that project to the SCC also show little saturation with level and wide dynamic ranges. It has been shown that the high-threshold, low-spontaneous rate neurons could provide the basis for rate coding of spectral profiles because they preserve spectral profiles at moderate loudness levels without saturation. Central mechanisms that were selectively attentive to these neurons could provide a specialized pathway for coding complex pattern information. The SCC is the primary target for the initial synapses of this neural population and so could provide a physiologic subsystem specialized for spectral pattern processing. The loss of the SCC as a consequence of tumor removal could explain the difference between ABI results in NF2 and NT patients.

These unfavorable conditions are absent in NT patients implanted with ABI. The absence of distortion in the anatomy of the auditory nuclei made it possible to achieve an effective and fairly well-organized activation of the auditory pathways.

Overall, the pattern of results suggests a separate pathway of auditory processing that is specialized for modulated sounds and for speech recognition. NF2 tumors and their removal may selectively damage this pathway, resulting in limited speech recognition with prosthetic stimulation.

In conclusion, excellent speech recognition is possible with surface ABI (not bypassing critical neural processing) in NT patients with anatomic or functional disconnection between the cochlea and the central auditory system caused by ossification or fracture of the cochlea, cochlear nerve avulsion or aplasia, auditory neuropathy, etc. Modulation specialized cells in the CN, fundamental for speech perception, are preserved in such disorders, whereas they probably receive irreversible damage in the presence of a cerebellopontine angle tumor. It will be interesting to evaluate the psychoacoustic and auditory performance of ABI in patients with small acoustic tumors before irreversible vascular and neural damage occur.

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