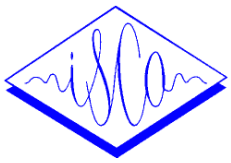

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CORTICAL ACTIVATION WITH SPEECH IN COCHLEAR IMPLANT USERS: A STUDY WITH POSITRON EMISSION TOMOGRAPHY

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ABSTRACT

The objective of this study is to investigate the pattern of cortical activation associated with speech sound stimulation in cochlear implant (CI) users. Six postlingually deaf patients using CIs were examined with positron emission tomography (PET) using intravenous injection of 15-O labeled water. PET activation studies were performed in two sets of three different conditions; (1) no sound stimulation as control, (2) hearing white noise and (3) hearing sequential words. Under sequential words' stimulation, regional cerebral blood flow (rCBF) in the primary auditory cortex, the auditory integration region and the auditory association area increased from the base line value. In contrast, white noise stimulation resulted in an increase of rCBF only in the primary auditory and auditory integration regions, and the increase was less than that observed in word stimulation. These results suggest that, in spite of non-physiological stimulation of the auditory primary afferents, speech being input through CIs are processed in the ordinary auditory cortices.

I. INTRODUCTION

Clinical application of cochlear nerve electrical stimulation in deaf patients started in 1970's by implanting an electrode in the cochlea [1]. The number of stimulating electrodes of a prosthesis was increased to improve its performance, and the design of cochlear implant has shifted from single channel to multi-channel system which can stimulate spiral ganglion cells in different locations independently [2].

Recognition of speech in cochlear implant users, however, is not yet complete probably due to unphysiological processes involved in speech sound coding in the cochlear nerve by electrical stimulation. In addition, little is known about the mechanisms how speech is recognized in the auditory cortex of patients with profound hearing loss. Recent investigations using Positron Emission Tomography (PET) have demonstrated decreased metabolism and rCBF in the auditory cortex in patients with profound hearing loss, and its increase by sound stimulation through cochlear implant [3],[4]. However, modes of cortical activation during sound stimulation in cochlear implant users have not yet been fully analyzed. The purpose of the present study is to investigate how cortices related to speech sound recognition in patients with profound hearing loss are activated with verbal and non verbal stimulation through cochlear implants, by measuring relative rCBF change using ^{15}O-labeled water PET.

II. SUBJECTS AND METHODS

Six postlingually deaf patients (age 10-54) using multi-channel cochlear implant system (Cochlear Implant Mini system 22, Cochlear Ltd., Melbourne, Australia) were included in the present study. Two of the patients were female and four were male, and all were right handed. None of the patients had a history of psychiatric and neurological disorders other than bilateral profound sensori-neural hearing loss. Informed written consent was obtained from all patients after full explanation of the study. This study was approved bye the Ethics Committee of Kyoto University Faculty of Medicine.

The subject lay supine on the PET scanner bed in a dimly lit room. The head was fixed using an individually molded helmet-shaped head holder. The right cubical vein was cannulated. The sound stimulation paradigms employed consisted of two sets of three different conditions; (1) no sound stimulation with the speech processor of the cochlear implant system switched off, (2) hearing white noise and (3) hearing sequential Japanese words. White noise in condition (2) was input directly into the speech processor at the loudness that the subject perceived noise distinctly but below uncomfortable level. In condition (3), the sensitivity of the processor was set at the level that the subject heard words distinctly but not uncomfortably loud. Words were read at the rate of approximately 40 words per minute without showing examiner's mouth. Each session continued for 2.5 minutes starting 30 seconds before the intravenous injection of the ^{15}O-labeled water, with an intermission of 15 minutes between each other.

For each stimulation condition, 30mCi of ^{15}O-labeled water was injected into the right cubital vein 30 seconds after the beginning of each session. The head was scanned for the radioactivity using a multi-slice PET scanner (PCT 3500W, Hitachi Medical Co., Tokyo, Japan) for 90 seconds from the beginning of the injection.

The original 15 slice data set was interpolated to an isotropic volume through a linear interpolation, giving a 50 slice data set with 2 x 2 x 2 mm voxel size. Using the midsagittal and two parasagittal MR images which had

been rotated into the Talairach proportional sterotaxic system [5], contours of the corpus callosum, brainstem, cerebellum, thalamus and visible cortical boundaries were identified. These outlines were transferred to the corresponding parasagittal slices of the resting PET data, which were then rotated and translated to fit the outlines. The necessary rotation was applied to all scans for each subject. These methods of the co-registration of MRI and PET data were the same as reported by Shibasaki et al [6], which based on the methods reported by Friston et al [7] and Evans et al [8].

All data were linearly scaled to adjust the inter-commisural distance of each subject to that of Talairach (25mm), of which scaling factor was applied to all directions. Two data of identical sound stimulation condition were averaged on a pixel-by-pixel basis. PET data of the silent condition (1) was subtracted from those of the noise stimulation (2) and sequential words stimulation (3) respectively. To search for changes of rCBF in cortices related to sound perception and speech recognition, two regions of interest (ROI) which included primary auditory cortex and auditory association area were selected (Fig.1) by comparing the images with an anatomical reference atlas of Talairach and Tournoux [5]. The ROI-A in Level 1 included the primary auditory cortex in the transverse temporal gyrus (Broadmann's area 41), the integration region (Broadmann's area 42) and a part of the auditory association area (Broadmann's area 22). The ROI-B in Level 2 was 12 mm below ROI-A, and included the auditory association area (Broadmann's area 22) almost exclusively. These regions were determined by three authors (Y.N., H.O. and Y.Y.) by taking into account the results of PET activation study on complex sound analysis and lexical processing [9]. The mean per cent increase of rCBF within each region of interest was measured for noise stimulation (white noise versus silence) and word stimulation (sequential words versus silence).

III. RESULTS

In subtracted PET images, rCBF increase was observed in the bilateral superior temporal lobe both with noise and word stimulation. Noise stimulation induced rCBF increase in the left primary auditory cortex in patients with an implant in the right ear (Fig.2-a), and in those who had an implant in the left ear had greater increase in the right side. Word stimulation induced rCBF increase both in the primary auditory cortex and the auditory association areas in all six patients, and the increase was more prominent in the left hemisphere than the right hemisphere (Fig.2-a). The auditory association areas inferior to the primary auditory cortex showed distinct rCBF increase with word stimulation, but much less increase with noise (Fig.2-b).

In the primary auditory cortex in ROI-A, the mean rCBF increase of the left and right hemisphere by noise stimulation was not significantly different (right: 7.5%, left: 11.3%). However, if we divided the subjects according to the side with an implant, rCBF increase by noise was significantly greater in the contralateral side of

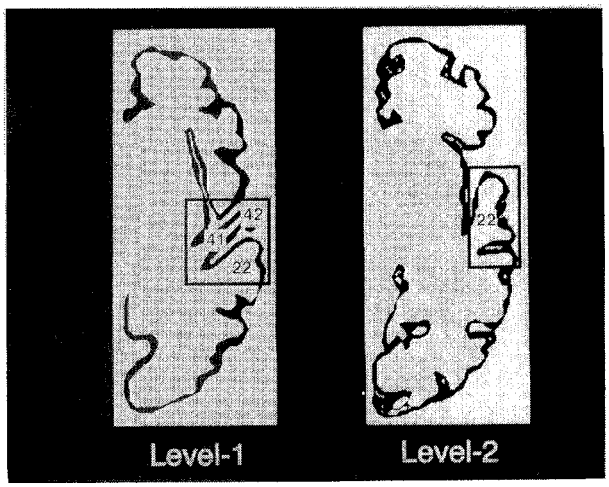

Figure 1. Regions of interest were selected to search for changes of rCBF in cortices related to sound perception and speech recognition. The ROI-A in Level 1 included the primary auditory cortex in the transverse temporal gyrus (Broadmann's area 41), the integration region (Broadmann's area 42) and a part of the auditory association area (Broadmann's area 22). The ROI-B in Level 2 was below ROI-A, and included the auditory association area (Broadmann's area 22) almost exclusively.

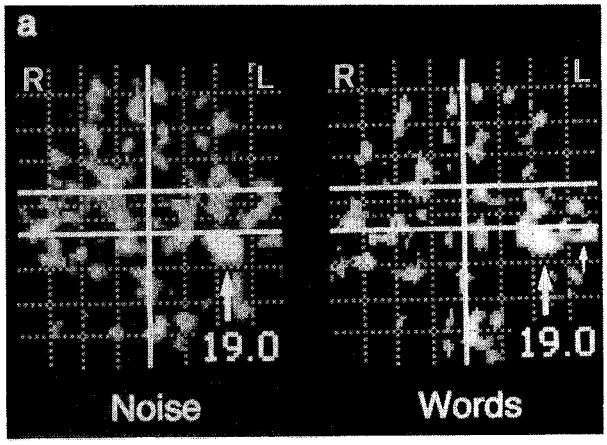

Figure 2. Subtracted PET images of a patient who had a cochlear implant in the right ear. Regions where rCBF increased are indicated in white.
a: In Level 1, noise stimulation induced more rCBF increase in the auditory cortices contralateral to the ear with an implant (arrow: Noise). Word stimulation induced rCBF increase both in the primary auditory cortex (large arrow: Words) and the auditory association areas (small arrow: Words), and the increase was more prominent in the left hemisphere.

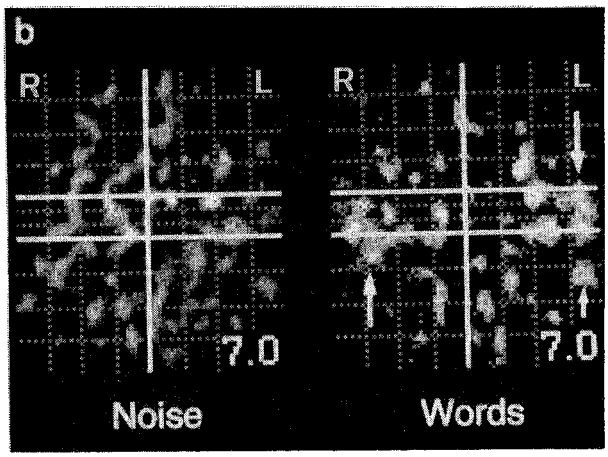

Figure 2. Subtracted PET images of a patient who had a cochlear implant in the right ear. Regions where rCBF increased are indicated in white.
b: In Level 2, the auditory association areas inferior and anterior to the primary auditory cortex showed distinct rCBF increase with word stimulation (arrows: Words), but much less increase with noise stimulation (Noise).

the implant than the ipsilateral side (contra: 13.2%, ipsi: 5.6%). Increases of rCBF in ROI-A were higher with word stimulation than with noise stimulation in both the left and right hemispheres, with greater rCBF increase in the left side.

In ROI-B, word stimulation caused high rCBF increase (right: 13.9%, left: 17.5%), and the increase with noise stimulation was very limited (right: 4.3%, left: 4.1%). rCBF increase in ROI-B caused by word stimulation was greater in the left hemisphere than in the right hemisphere.

IV. DISCUSSION

Complex auditory input of speech sound is thought to first undergo early acoustical analysis in the auditory cortex, and is passed to the auditory input lexicon, the site of encoded entries for familiar words [10]. Subsequent activation of the appropriate entry in the semantic system makes the meaning of spoken language apparent to the subject. In the PET study by Howard D et al. [9], processing of complex auditory stimuli was found to yield bilateral activation of extensive areas of both the primary auditory cortex (Heschl's gyrus) and the auditory association cortex. The ROI-A in the present study was determined to correspond to this area for complex sound processing. Access to the lexicon for heard words has been reported to be in the left superior and middle temporal gyri beneath the primary auditory cortex [9], which corresponded to the present left ROI-B.

Although the current cochlear implant system is designed after physiological sound coding in the cochlea, it still includes aspects that are different from physiological processes. For example, as cochlear electrode can be placed only in the basal half of the cochlear duct, the cochlear nerve fibers that can be stimulated by the implant are limited to those that have higher characteristic frequencies. Unlike extremely fine frequency tuning in the normal cochlea, the auditory nerve in cochlear implant users can be tuned only grossly because of limited number of stimulating electrodes. Although a cochlear nerve fiber can fire at various phase of complex speech sound, electrical stimulation in the present implant system is locked on the fundamental frequency of the speech sound. Consequently, neuronal activities in the primary auditory cortex derived from speech coding in the cochlear nerve by cochlear implant system may be different from those what the patients had before the they lost hearing. What is remarkable is that cochlear implant users with post-lingual deafness recognize speech considerably well, in spite of those fundamental differences described above. The question is whether processes similar to normal speech recognition work for unphysiological input coded by a cochlear implant system.

The result that white noise caused more activation in the primary auditory cortex in the contralateral hemisphere than in the ipsilateral side suggests that signals caused by non-verbal sound stimulation primarily reach and are processed in the contralateral hemisphere. Anatomically, fibers ascending from the cochlear nuclei and the superior olive project more to the contralateral lateral lemniscus, and the asymmetry occurred at this level is kept up to the auditory cortex [11], and sound stimulation in a ear is reported to cause increase in neural metabolism in the contralateral upper brain stem auditory tract neurons [12]. Penfield [13] reported that electrical stimulation of the temporal lobe induced sensation of buzzing sound in the opposite ear. These findings suggest stronger connection between a ear and the contralateral hemisphere. Activation of contralateral hemisphere with white noise by cochlear implant further support the idea of crossed connection between the ear and the cortex.

Cortical activation in ROI-A by word stimulation was higher than that caused by noise, which indicates that neuronal signals for speech sound coded by a cochlear implant system produces more intensive activation of neuron-networks in the primary and the surrounding auditory cortices than those for noise. Verbal stimulation activated the auditory areas more in the left hemisphere even in subjects whose implants are in the left ear. This indicates that speech sound signals that reached the right hemisphere might be transmitted to the opposite side and processed predominantly in the left hemisphere.

The difference in rCBF increase between noise and verbal stimulation was greater in the auditory association area (ROI-B) than in the area above this region (ROI-A). Although rCBF increase during verbal stimulation was observed in both hemispheres, activation in the left ROI-B was higher than in the right one. According to Howard et al [9], the region beneath the primary auditory cortex in the left hemisphere, which correspond to the left ROI-B in the present study, is

primarily involved in lexical processing. These results indicate that, in spite of unphysiological sound coding by the cochlear implant system at the entrance of the ascending auditory pathway, speech sound may be processed in the left auditory association areas similar to those used in normal subjects.

The present method provides an objective technique to assess cortical activities for speech recognition in patients with profound hearing loss. Further studies on post- and pre-lingually deaf patients using cochlear implant may give us a new perspective in the research on the development and plasticity of the human auditory cortex.

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