Case Report

Ear movement induced by electrical cortical stimulation

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A B S T R A C T

Cortical areas that control ear movement have not been reported in humans. We describe a rare case in which ear auricle movement was induced by extraoperative electrical cortical stimulation. A 21-year-old man with intractable localization-related epilepsy was admitted for presurgical evaluation. Subdural electrodes were implanted over the right temporal and frontal regions. Tonic upward contraction of the left ear auricle was elicited by stimulating the subdural electrode on the posterior portion of the right superior temporal gyrus close to the end of the Sylvian fissure. No other body movements or auditory symptoms were elicited. A possible mechanism underlying this rare phenomenon is discussed.

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1. Introduction

The somatotopical representation of the cortical areas was clarified by the pioneering work of Penfield and Jasper [1]. However, ear movement has never been evoked by electrical cortical stimulation in previous human studies. We experienced a rare case of localization-related epilepsy in which ear movement was induced by electrical stimulation of the brain through subdural electrodes. Although ear movement by cortical stimulation has been reported in monkeys [2–4], this is the first report of ear movement elicited by electrical cortical stimulation in humans.

2. Case report

2.1. Medical history and noninvasive presurgical evaluation

A 21-year-old right-handed man was admitted for presurgical evaluation for epilepsy surgery. The patient had no specific past medical or familial histories, and had experienced intractable seizures since 9 years of age. His seizures were characterized by motionless staring, followed by vocalization, stiffening of all limbs, and generalized violent movements, lasting less than 1 minute. Seizures tended to occur during sleep with a frequency of several times per day to once per month. An interictal scalp electroencephalogram (EEG) showed epileptiform discharges in the right anterior temporal (maximum in Sp2) and right frontal (F4) regions. The ictal EEG showed rhythmic slow waves in the right midposterior temporal region (T4 and T6). Brain MRI revealed no abnormality. Interictal single-photon-emission computed tomography (SPECT) with N-isopropyl-4-[123I]iodoamphetamine ([123I]IMP) showed hypoperfusion in the right posterior temporal region. Interictal SPECT with Technetium-99 m ethyl cysteinate dimer (99mTc-ECD) showed hypoperfusion in the right frontal pole and posterior temporal regions. Ictal SPECT with 99mTc-ECD revealed hyperperfusion in the right midtemporal and frontal regions. Neurological examination revealed no significant abnormality. The patient could not move his ears voluntarily.

2.2. Intracranial EEG and functional mapping

The patient underwent a right frontotemporal craniotomy, and subdural electrodes were implanted over the right temporal and frontal regions. In addition to the electrodes covering the basal frontal (OF), anterior temporal (AT), and basal temporal (TBA and TBP) regions, the right lateral temporal–frontal–parietal areas were also covered by a 4×6 subdural grid (Fig. 1). Each electrode was 2.3 mm in diameter, and the center-to-center interelectrode distance was 1 cm. The location of the grid was confirmed before and after functional mapping by X-ray. Electrical stimulation was performed via the implanted subdural electrodes for functional mapping. Repetitive square wave electric currents of alternating polarity with a pulse
The ear movement seen in the current patient was not accompanied by any other symptoms, such as somatosensory, auditory, autonomic, or other higher cortical functions. Therefore, it is assumed that this movement was caused by activation of the pure motor function of the ear, rather than by the activation of the auditory association cortex.

Most of the somatotopical representations have been identified in human brain [1]. However, a cortical area that controls ear movement has not been reported in humans, although the primary sensory area of the ear was identified around the primary sensory area of the neck and face [6,7]. In studies of macaque monkeys, Bon et al. [4] reported that electrical stimulation of area 8b, which is in the rostral area of the frontal cortex, evoked contralateral ear movements with or without conjugate movements of the eyes. Other investigators also evoked ear movements by stimulating the frontal eye field and the supplementary eye field associated with the eye movements [2,3]. Considering the location of the ear sensory area and these animal studies, it is likely that the “ear motor center” is in the frontal lobe, probably within or close to the eye fields, even in humans.

There are, however, no previous reports demonstrating ear movements on stimulation of such frontal areas in humans. This discrepancy may be explained by the evolution of the species. Ear movements are important in localizing the origin of sound in monkeys and other animals, but are not as useful in humans. Therefore, it is presumed that the human “ear motor center” has degenerated or even disappeared in the course of evolution.

In the current study, auricle movement was evoked by stimulating the posterior portion of the superior temporal gyrus. The location was within or close to the auditory association cortex, but far from motor areas. Therefore, it is very unlikely that this stimulated point itself has a motor function, and it is possible that the electrode position that induces ear movement may have some neural connections with an “ear motor center.” This kind of response was previously reported by Penfield and Jasper as a “distant response” [1].

Reasons why direct stimulation of the frontal areas, which may include an “ear motor center,” failed to show ear movements, not only in this study but also in previously reported patients, may be: (1) The cortical representation of the area responsible for ear movement may be very small in humans; (2) the electrical threshold to stimulate ear movement may be higher than that of other body parts; (3) not all humans may have an “ear motor center” as expected from clinical observations; and/or (4) physicians have not paid much attention to ear movements previously.

In addition to these reasons, distortion of normal cortical function by epileptogenicity may play some role in the ear movement elicited in the current patient. Repeated epileptic seizures could alter normal brain function, and nonphysiological ear motor function may appear within the temporal cortex. This phenomenon may also explain why a hand motor response could be recorded by stimulating B6, an area located lateral to the tongue motor area (A4 and B5). This phenomenon was called “epileptic sensitization” by Penfield and Jasper [1].

Although it is still uncertain if an “ear motor center” is present in all humans, if the center is in the frontal area or in the temporal area, and if a “distant response” or “epileptic sensitization” distorted the normal function in this patient, it is still interesting that the ear movement could be seen by stimulating the superior temporal gyrus. It might be clinically useful in functional mapping if ear movements could be elicited only when the superior temporal lobe is stimulated. In addition, because repeated epileptic seizures may alter normal brain function, if ear movement associated with the remote auditory cortex is found, a stricter and more careful presurgical evaluation for locating the epileptogenic zone and brain function areas could be performed by clinicians before epilepsy surgery. However, because this is only a single case report, additional research is required to clarify the clinical usefulness of this ear movement, as well as the underlying mechanism.
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Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at doi:10.1016/j.yebeh.2010.05.016.

References


