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Absence of Visually Influenced Cells in Auditory Cortex of Normal and Congenitally Deaf Cats

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Single cells recorded from primary auditory cortex (AI and AII) of unanesthetized paralyzed cats were tested for responses to visual stimulation. Forty-six cells recorded from normal animals responded to acoustic stimuli but showed no discrete response to light flashes or small spots and bars. Twenty-two cells recorded from a congenitally deaf cat were spontaneously active but showed no discrete response to either auditory or visual stimulation.

Introduction

There have been numerous reports of multisensory effects on single cells within the visual cortex of the cat (2–4, 6, 8). This experiement was an attempt to determine if multisensory processing is characteristic of other primary sensory areas. We examined the activity of single cells in primary auditory cortex (AI, II) of unanesthetized cats during visual stimulation.

Methods

One congenitally deaf [2] and three normal cats were used in the experiments. The former animal was judged as deaf by the absence of any overt behavioral response to loud environmental sounds and the failure of click stimuli to evoke potentials from auditory cortex.

Procedures for recording from the visual system of cats were similar to those reported previously from this laboratory (5). One week prior to the first experimental session the animals were anesthetized with sodium pentobarbital (35 mg/kg) and the temporalis muscle and outer bony table overlying the anteriomedial portions of the ectosylvian gyrus (AI and

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AII) removed bilaterally. Screws were mounted to the skull with dental acrylic to serve as a means for supporting the animal at the subsequent sessions.

On the day of a recording session, the animal was anesthetized for approximately 30 min with halothane (Fluothane, Ayerst) during which all surgical procedures were performed. An endotracheal tube was inserted and the head rigidly suspended from the previously attached dental acrylic platform. The remaining bone and dura overlying auditory cortex of one hemisphere was removed and the cavity sealed with agar. All incision points were coated with a long-acting local anesthetic (Ziljecton). The general anesthesia was then discontinued, the animal paralyzed by intravenous infusion of gallamine triethiodide (Flaxedil, Davis and Geck) (50 mg/hr), artificially ventilated, and body temperature maintained between 38 and 40 C. One hemisphere was explored each session, and each cat was used for two sessions separated by at least 1 week.

Contact lenses (1 diopter) were used to prevent corneal drying and to focus the retina on a translucent screen placed 57 cm in front of the animal.

Instrumentation was similar to that reported previously for work on the visual system of the cat (5). Tungsten probes with 1-μ diam. tip and 200-Mohm impedance (tested at 60 Hz) served as microelectrodes. Neural activity was amplified and displayed in the conventional manner, including oscilloscope traces and audiomonitoring. Each cell studied was isolated sufficiently to permit a Schmitt trigger to capture each action potential without intrusion of potentials from other units. Schmitt trigger pulses were led to a Computer of Average Transients.

Stimuli for computer averages were 2.8 log ft-L light flashes generated by a tungsten filament lamp placed 91 cm from the cats' eye and focused on the center of the retina. Light and dark stimuli were used in searching for receptive fields. Luminous spots and bars were 1 to 1.5 log units above a -2 log ft-L background, while shadows of cardboard cutouts were 1 log unit below a -1 log ft-L background.

Locations of penetrations are shown in Fig. 1. Biphasic action potentials of greater than 1-msec duration were considered to be generated by cell bodies. The potentials were usually initially positive and often showed an a-b break. When a cell had been identified, it was isolated and tested for auditory and visual responses.

Results

Sixty-eight cells were studied, forty-six of which were from normal cats. All of the cells from the normal animals responded to complex sounds (e.g., shuffling of feet, hand claps, whistles) and often to tones or clicks. The 22 cells studied in the congenitally deaf animal were spontaneously active but could not be influenced by acoustic stimuli.

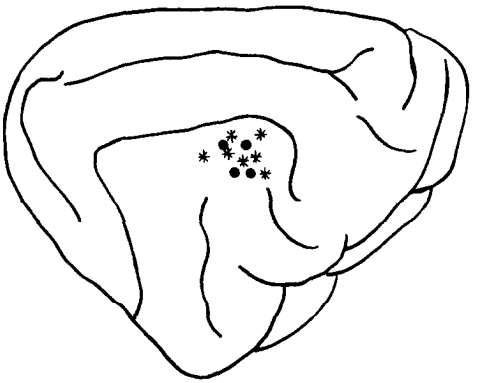

FIG. 1. Location of electrode penetrations in a congentially deaf (•) and three normal (*) cats.

Poststimulus time histograms of each unit's activity for 0.5 and 2.0 sec following a light flash were summed over at least 50 repetitions for each cell. In no instance was a discrete response observed, that is, a response time-locked to the stimulus and occurring with relatively short latency (less than 250 msec). On the other hand, many cells displayed a small diffuse shift (either increase or decrease) in level of activity beginning about 0.5 sec after stimulus presentation and sometimes lasting for a second or more. These diffuse shifts would usually be found on the first poststimulus time histogram set but in no instance were they reproducible.

The majority of cells (including all 22 from the deaf animal) were isolated for a sufficient length of time to permit a complete exploration of the entire visual field with small spots and bars. No responses were observed while monitoring activity on the oscilloscope and loud speaker.

Discussion

In the present experiment, cells in primary auditory cortex of unanesthetized cats did not show discrete responses to visual stimulation. Diffuse long-latency shifts in background activity frequently occurred, but their amplitude was small and their occurrence variable.

Visual influences in auditory cortex might be so slight as to be masked by response to background acoustic noise. This appears unlikely since visual responses were also not observed in the congenitally deaf cat. Results from this animal bear on the problem of cortical synaptic organization following the loss of primary afferent input. It is our opinion that the synaptic security of any nonacoustic input to auditory cortex should have been increased in the congenitally deaf animal. The presence of "spontaneous" activity suggests that these cells may have some function, but their activity is obviously not specifically related to vision.

Recent anatomical work (7) indicates sparse retrograde degeneration within the dorsal lateral geniculate nucleus after ablation of the anterior and medial ectosylvian gyrus. Our experiments suggests that this projection is not sufficient to produce discrete visual responses. It is possible that the diffuse shift in background activity may be related to a geniculate projection but both their long-latency and variability suggest influences from nonspecific systems. Similar long-latency changes have also been observed in visual cortex to acoustic stimulation (1).

Two recent reports have estimated that 28% (8) or 38% (6) of units in primary visual cortex show discrete responses to auditory stimulation. Our results suggest that there are far fewer multisensory cells in primary auditory cortex than in primary visual cortex of cat. The similarity in experimental procedures among the three studies (e.g., species, anesthesia, paralytic agent) might be expected to produce comparable results. The difference could be explained if many of the units reported both by Murata *et al.* (6) and Spinelli *et al.* (8) were from the visual association regions (area 18) instead of primary visual cortex (area 17). It is difficult to be certain of having obtained records from area 17 in cat without histological verification.

We suggest that the percentage of multimodal cells (i.e., neurons which can be independently activated to produce discrete responses by more than one stimulus modality) is very small in primary sensory cortex.

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