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Response Plasticity of Single Neurons in Rabbit Auditory Association Cortex During Tone-Signalled Learning

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Single unit activity was monitored in rabbit auditory association cortex (AC) throughout the acquisition of a classically conditioned, nictitating-membrane response The CS was a tone burst at the characteristic frequency of each neuron Rabbits which were pseudoconditioned or received conditioning trials but did not learn the response served as control groups Significant alterations in CS-evoked firing rate were termed 'response plasticity' Neurons in conditioned animals were more than twice as likely to show response plasticity during the 250 ms CS–US interval than neurons in control animals Such differences were evident both in the early (0–60 ms) and late (60–250 ms) portions of the CS–US interval Most early changes appeared at 21–40 ms after CS onset Response plasticity was most commonly manifested as an increase or decrease in CS-evoked firing rate with little change in the response pattern (PST histogram shape) In some neurons, subcomponents of response patterns (early or late portions of the CS–US interval) were observed to change independently of each other Spontaneous rate and UCS-evoked activity were not modified with learning Early in training (transition trials), neural activity evoked by the tone CS in conditioned animals was not different from that in controls Response plasticity was most pronounced after the CR was first learned (trained trials) and stabilized once the CR was well established (overtrained trials) Recording sites of neurons showing conditioning-related response plasticity were co-extensive with those of cells that did not

INTRODUCTION

Neural events in auditory cortex (AC) can be affected by non-acoustic factors 'Response plasticity' refers to the way that neurons may respond differently to invariant acoustic stimuli depending on whether or not the sound signals the occurrence of an environmental event such as reinforcement Previous work[11,15,16,24,49,50] showed that multi-unit activity in AC may be systematically modified during the acquisition of a tone-signalled task Observed changes consisted primarily of increases in stimulus-evoked spike discharge Behavioral contexts other than learning have also been shown to affect the activity of single AC neurons Alterations in neural activity in these experiments were not attributed to a learning process (since the animals had been previously trained), but rather to other variables surrounding the performance of a previously learned task Such variables included attention[26], task performance[2,4,31,40,53,62], reinforcement contingencies[2] and task complexity[4,5] These studies, employing single unit recording techniques, have shown that neural changes accompanying such behavioral manipulations can vary from cell to cell Investigations of single cell activity in conscious, untrained animals have also shown that neuronal activity in AC is heterogeneous, that is, neurons differ in response pattern, onset latency and sensitivity to acoustic stimuli[1,10,21,32,41] Thus one might hypothesize that response plasticity of different neurons during learning would also be heterogeneous

The present experiment was an attempt to bridge

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the gap between multi-unit learning experiments and single unit experiments in previously trained animals Single unit activity was monitored continuously throughout the course of training in order to determine whether learning-associated changes reflected the heterogeneity of neural response activity characteristic of single units or the more invariant trends found in multi-unit conditioning experiments The question whether response plasticity during learning is a property of all neurons or specific to a subset of cells was also addressed

MATERIALS AND METHODS

Surgery

Subjects were 63 New Zealand white rabbits, approximately 6 weeks old and weighing 0 8–1 2 kg Thorazine (8 mg/kg) and atropine sulfate (0 2 mg/kg) were administered 1 m , 0 5 h prior to surgery Sodium pentobarbitol (30 mg/kg) was given through the marginal ear vein and the wound area was infiltrated with Xylocaine

Restraining head-bolts were embedded in dental cement and fixed to the skull with stainless steel screws The head-bolts were attached with the animal's head at a constant angle, lambda 1 5 mm below bregma, the standard rabbit horizontal stereotaxic plane[19] Bregma was (0,0) A 2 mm diameter trephine hole exposing the dura was placed over auditory association cortex at (—1 5, 9), centered in the skull surface depression between the temporo-parietal suture dorsally and the zygomatic process of the temporal bone ventrally A Trent–Wells chamber was oriented perpendicular to the skull and cemented in place The inside of the chamber was rinsed with sterile saline and filled with chloramphenicol, a topical antibiotic which also served to keep the dura moist The chamber was sealed with a plastic screw cap Sutures were placed in the upper and lower eyelids of the left eye so that it could be kept open on the day of the experiment

Acoustic stimulation

The experiments were conducted in a sound attenuated, vibration isolated, single-walled chamber (Industrial Acoustic Corporation) Pure tones were generated by a General Radio 1900 wave analyzer and signal frequency was monitored with a Hewlett Packard 5521 electronic counter Stimulus output was fed into a Grason-Stadler 1287 electronic switch, Hewlett Packard 350 D attenuator and a Sansui BA 60 stereo amplifier

The amplified output was transduced by a Yamaha HP1A dynamic earphone A silastic earmold, fitted to the rabbit's external auditory meatus, positioned the earphone so that the sound was delivered close to the tympanic membrane A General Radio 0 5 in electret condensor microphone, operating in the measuring mode, was coupled to a 3/4 in long probe tube The probe tube was concentric within the sound delivery cavity and its tip extended to the tip of the earmold The output of the microphone, measuring sound near the eardrum, was monitored by a General Radio 1900A wave analyzer

Physiological recording system

Bak parylene-coated tungsten micro-electrodes were advanced into the cortex from outside the chamber with a Trent–Wells hydraulic microdrive A standard amplifier and spike amplitude–time waveform identifier[48] were used to monitor single cells and small cell clusters Cells with similar waveforms were sometimes combined by this technique and were not further discriminated

Behavioral preparation

The rabbit NM paradigm has been used extensively in behavioral studies[17,58,59] Two days after surgery, the animals were habituated to restraint in the stereotaxic device and given random puff stimuli at an average of 1/min for 2 h periods on two separate days The experiment was controlled by an IMSAI microprocessor computer system with an ICOM dual floppy disk drive Pure tones were 400 ms long, air puffs were 150 ms long During conditioning, tone onset preceded the puff by 250 ms and the two stimuli terminated simultaneously During pseudoconditioning, tones and puffs were presented separately in a random sequence The intertrial intervals ranged from 20 to 100 s and averaged 60 s during conditioning They averaged 30 s during pseudoconditioning Measurements of nictitating-membrane (NM) responses were done with a light reflection transducer which provided a sensitive record of NM activity without being directly

attached to the membrane[17] The animal's trial by trial performance was scored on line

Experimental procedure

The CS was chosen individually for each unit at the characteristic frequency (CF) of the neuron CF was defined as that frequency which elicited an excitatory response with minimal stimulus intensity[11] CS intensity was 80 dB re 20 $\mu N/m^2$ Typically, 100 conditioning trials were presented However, conditioning was continued for up to 200 trials if the CR had not been acquired Pseudoconditioning consisted of 100 tone and 100 puff trials At the completion of each conditioning experiment, 30 μA of current was passed through the electrode for 30 s

Histology

The animal was deeply anesthetized with sodium pentobarbital and perfused through the left ventricle with normal saline, followed by 10% formalin Middle ears were examined to insure that the animals were free from infection Brains were frozen-sectioned and alternate sections stained with cresyl violet and Weil stains Electrode locations from conditioned animals were pooled to form a composite map of recording sites on the lateral surface of the rabbit's right hemisphere The caudate nucleus and the floor of the 3rd ventricle were used as landmarks for this reconstruction

Data analysis

The behavioral data were analyzed to determine the trial of the 10th CR This trial has been shown to be a reliable index of the transition point to asymptotic behavioral performance[17,20], and it was used as a reference point for neural analysis

The neural data for each conditioning and pseudoconditioning experiment were stored on floppy discs as spike counts in 1 ms binwidths Each trial consisted of a 100 ms pre-stimulus period followed by 400 ms of stimulus-evoked activity The pre-stimulus period provided a measure of the cell's spontaneous rate PST histograms were compiled offline with a PDP 11-40 or 11-60 Experience with the data showed that 10 ms binwidth histograms were the most informative about response patterns with these auditory association cortex neurons

The trials comprising each set of PST histograms were *behaviorally* determined It was of greatest interest to compare the neural activity which occurred during the *initial trials* in which the animal was behaviorally naive with that occurring during the *trained trials* during which the animal had learned the CR Initial trials consisted of the last 20 trials in the trial sequence during which the CR had occurred less than 20% of the time Trained trials began with the 11th CR and usually consisted of trials in which the CR occurred more than 80% of the time Fewer than 20 trials were used in each group in the occasional instances where it was not possible to obtain 20 trials which met these criteria

Initial trials were additionally compared with *transition* and *overtrained* trials Transition trials consisted of 20 trials between the initial and trained trials Overtrained trials were the 20 trials following the trained trial series

The data analysis of each experiment was identical except for the actual trial numbers comprising the 'initial', 'trained', 'transition' and 'overtrained' trial groups These were determined by the individual learning progress of each rabbit Since there was no behavioral performance with which to segregate trials in pseudoconditioned controls or in animals that failed to learn the CR, trial groups were determined by the trial numbers corresponding to a matched conditioning experiment

PST histograms of initial, trained, transition and overtrained trials were printed to provide a visual representation of the data Separate *t*-tests were performed for the 100 ms pre-stimulus period and for different portions of the stimulus interval to assess response alterations during the learning process Background activity was subtracted from stimulus-evoked values Sub-components of stimulus-evoked activity consisted of the 250 ms CS–US interval and the 250–400 ms interval during which the tone and puff stimuli overlapped The CS–US interval was subdivided into early (0–60 ms) and late (60–250 ms) components The 0–60 ms interval was further subdivided into 20 ms intervals

RESULTS

Complete behavioral and neural profiles were obtained from 35 neurons in 35 rabbits during conditioning Only one neuron could be recorded

from each conditioning rabbit because the animals were naive only once A total of 26 neurons were monitored in 4 rabbits during pseudoconditioning Another 15 experiments yielded neural data from 9 conditioned animals which didn't learn the CR

Behavioral data

Consistent with previous work[17], the 10th CR proved to be a reliable index of asymptotic CR performance CR performance was 75% or better in the 20 trials immediately following the 10th CR in 80% of conditioned animals The 10th CR ranged from trial 18 to trial 258 with the mean CR on trial 60 and the median on trial 48 Unlike the previous study, the total number of trials required for learning to take place varied considerably This discrepancy may have occurred because in the present study, the CS frequency differed from animal to animal depending on the CF of the cell under study and/or because two sessions of habituation to the puff were given before training CS frequency ranged from 1 to 34 kHz Pseudoconditioned rabbits showed no evidence of CR acquisition

Neural data initial vs trained trials

The major objective of this experiment was to compare the neural events recorded during the initial conditioning trials with those obtained during the trained trials The animal was behaviorally naive during initial trials and newly conditioned during the trained trials

Of the neurons in conditioned animals, 51% showed statistically significant ($P < 0.05$) changes in CS-evoked firing rates during the 250 ms CS–US interval In pseudoconditioned controls 19% of the neurons changed during this period

The 250 ms CS–US interval was subdivided into early (0–60 ms) and late (60–250 ms) components In

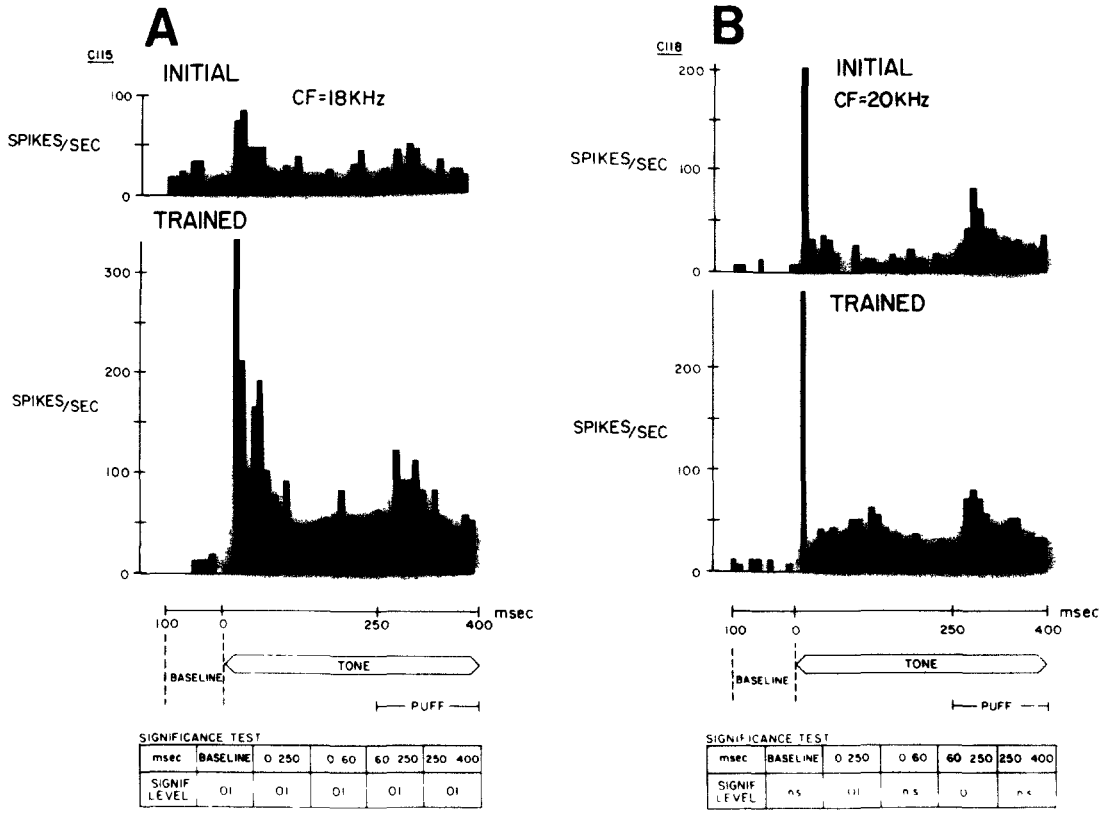

Fig 1 Neural activity occurring during 'initial' trials (rabbit behaviorally naive) is shown for comparison with that occurring during 'trained' trials (CR has been learned) Each histogram consists of firing rate summed over 20 trials The stimulus configuration and statistical changes in neural activity occurring during various components are illustrated The CF of each unit is indicated A generalized increase in CS-evoked firing rate with training Note the accompanying decrease in spontaneous firing rate B increase in CS-evoked activity selective to the late (60–250 ms) portion of the CS–US interval

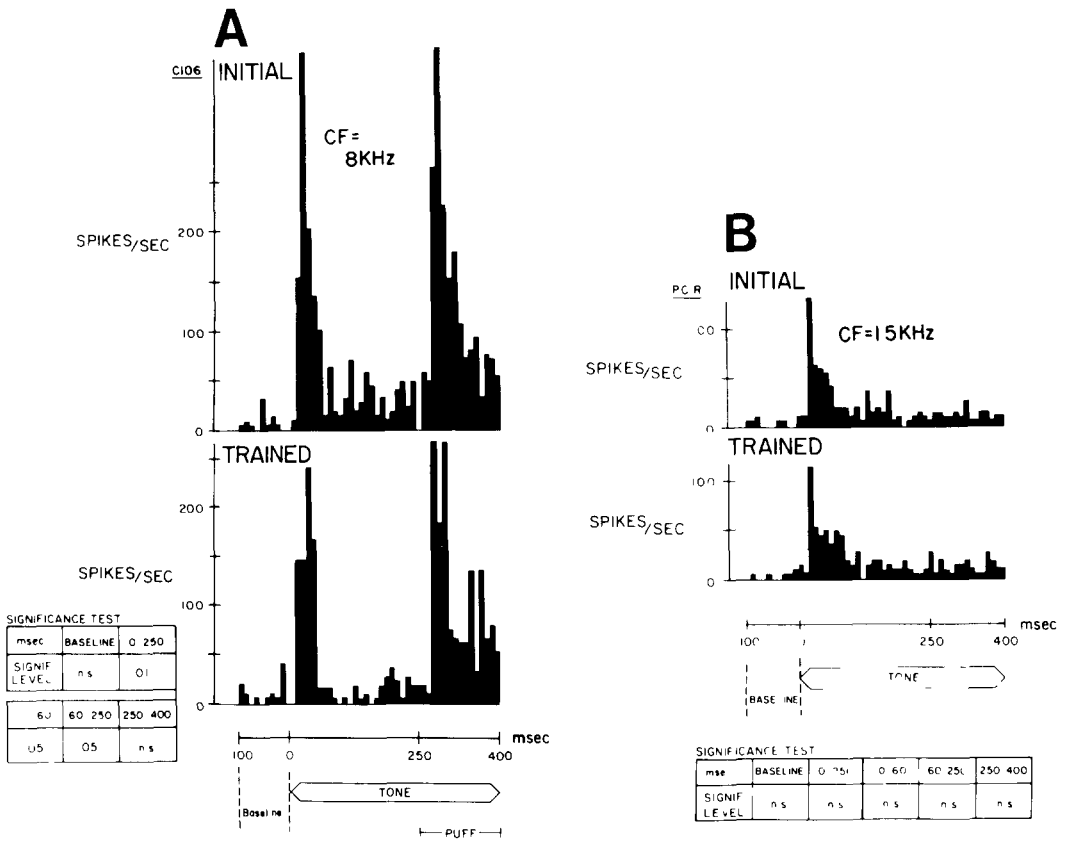

Fig 2 A generalized decrease in CS-evoked activity during the CS–US interval B pseudoconditioning control, no significant change in unit activity over time

conditioned animals 46% of the cells showed significant rate changes during the early period and 43% changed during the later portion In comparison, 15% and 23% of the neurons in pseudoconditoned controls changed, respectively Closer inspection of the neurons in conditioned animals during the 0–60 ms interval revealed that 6% changed in the 1–20 ms interval, 42% changed in the 21–40 ms interval, and 33% changed in the 41–60 ms interval In the pseudoconditioned group 11% changed in each interval in what appeared to be a random fashion

Response plasticity was expressed differently in individual neurons In 60% of the neurons that changed significantly, the shape of the PST histograms remained unaltered In these cells, changes in firing rate occurred in both early and late components and were of the same sign Fig 1A shows an example of an increased response A decreased response is shown in Fig 2A The remaining cells showed increases or decreases in firing rate selective to either the early or late portion of the CS–US interval (see Fig 1B) New components of a response pattern sometimes emerged during trained trials where they had been absent during the initial trial series Neurons in pseudoconditioned rabbits tended to remain constant (see Fig 2B)

In conditioned rabbits, CS-evoked changes during the 250 ms CS–US interval were evenly distributed between neurons which showed increases and those which showed decreases in firing rate 47% increased, 53% decreased In neurons recorded during pseudoconditioning, 80% of the changes were decreases In animals that did not learn, 75% of the changes were also decreases

Spontaneous rate measured during the prestimulus period varied considerably less than stimulus-evoked activity in both conditioned and control animals Shifts in spontaneous rate occurred in 14% of the cells recorded from conditioned animals and in 8% of neurons in the control group

TABLE I

Percentage of neurons showing significant change in firing rate

	Firing rate (ms)				
	Baseline	*0–250*	*0–60*	*60–250*	*250–400*
Conditioned animals (n = 35)	14%	51%	46%	43%	26%
Pseudoconditioned animals (n = 26)	8%	19%	15%	23%	8%
Animals that did not learn (n = 15)	27%	20%	7%	20%	27%

Since spontaneous rate was less likely to change than stimulus-evoked activity, in most instances, spontaneous rate remained constant while stimulus-evoked activity was altered There was no relationship between spontaneous rate and stimulus-evoked activity as evidenced by their correlation coefficient of +0 004

UCS-evoked activity in trained animals is most validly compared with that in conditioned animals that did not learn, since the puff stimulus was present during the 250–400 ms stimulus interval in both cases (There was no puff stimulus during this interval in pseudoconditioned animals) Significant changes in firing rate during this interval occurred to similar extents in conditioned animals that learned and those that did not (26% vs 27% respectively)

Table I is a summary of initial and trained trial comparisons Indicated are the percentages of neurons showing significant changes in firing rate across the various time periods The percentages of neurons that changed in control animals are shown for comparison Neurons recorded during conditioning showed a greater percentage of changes than controls across all of the time components examined

Initial vs transition and initial vs overtrained trials

Neural activity measured during initial trials was compared with that occurring during transition and overtrained trials The percentage of neurons showing significant firing rate changes during transition trials were about the same in both conditioned and unconditioned groups

During overtraining, the percentage of cells in which discharge activity was modified remained greater in conditioned than pseudoconditioned animals However, the difference between conditioned and pseudoconditioned groups was greatest during the trained trials Thus, changes associated

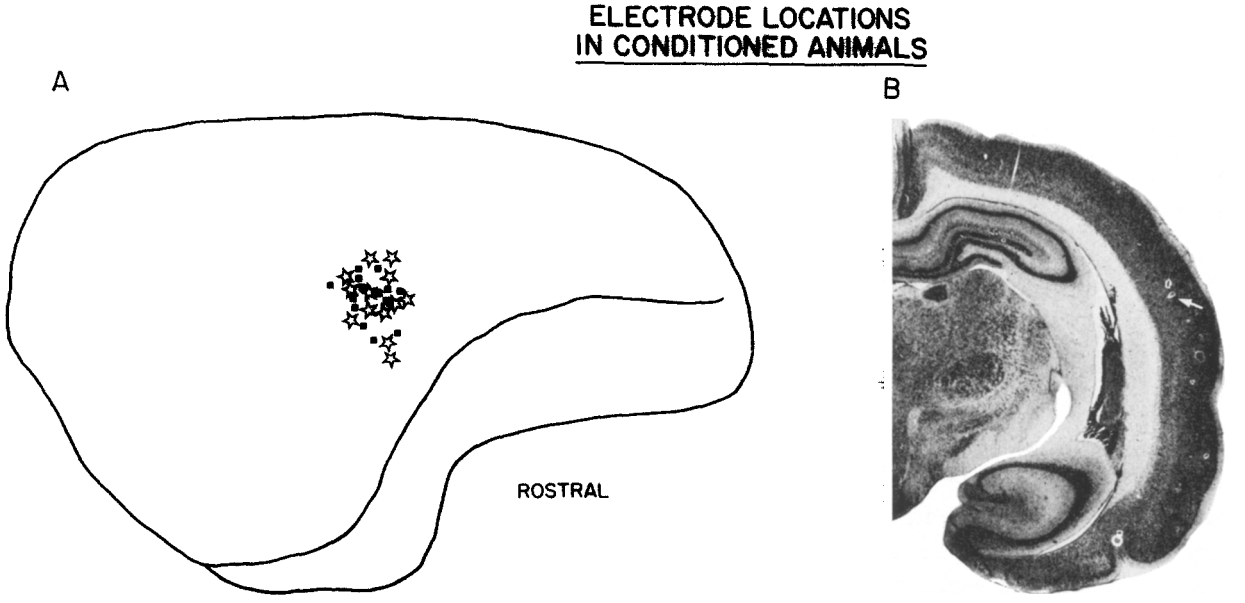

Fig 3 A electrode locations in conditioned animals are shown on the lateral surface of the right cerebral hemisphere Stars indicate neurons that showed significant changes in firing rate with learning Dots mark the location of neurons that did not change B coronal section of a rabbit's right hemisphere An electrode lesion, located deep in the granular layer (IV), is indicated by an arrow

with learning were most evident soon after the CR was learned

Animals that did not learn

Fifteen rabbits failed to learn the nictitating membrane response Unit data from these rabbits were treated in the same fashion as data from the pseudoconditioning group Significant changes in CS-evoked activity during the entire CS–US interval were about equal in animals which did not learn and in pseudoconditioned animals (19 % vs 20 %, respectively in the initial vs trained trials) In contrast, CS-evoked changes occurred in 51 % of the neurons in animals that learned Thus animals which did not learn provided an interesting, serendipitious control group insuring that changes observed during conditioning were dependent on CR acquisition Data from this group are included in Table I

Electrode location

Electrode lesions were identified in 28 of the 35 conditioned rabbits The position of these lesions did not deviate more than 2 5 mm in any direction across animals and were concentrated in cortical layers IV and V (see Fig 3B)

Recording sites in conditioned animals were reconstructed on the lateral surface of the right cerebral hemisphere (see Fig 3A) Neurons which changed were evenly interspersed with those which did not Both cytoarchitectural evidence and the position of recording sites on the cortical surface indicate that neurons were recorded from homologous regions of association AC across rabbits

DISCUSSION

Characterization of response plasticity

Multi-unit studies have shown that the acquisition of a tone-signalled behavior can systematically alter neural events in AC Now, these changes are characterized in single cells One of the most interesting findings was the diversity of alterations in stimulus-evoked activity observed A variety of response patterns was evident to begin with in the naive animal and this heterogeneity characterized the changes in neural activity during the acquisition process Both increases and decreases in CS-evoked firing rate accompanied behavioral acquisition Whereas 60 % of the changes consisted of generalized increases or decreases in stimulus-evoked firing rate, the remaining 40 % were specific to a subcomponent of the CS-US interval

Response alterations occurred as early as 11–20 ms after CS onset As units in AC respond to tones in the conscious rabbit with latencies as short as 10 ms[32], response plasticity was evident in AC in the earliest components of the tone CS evoked response, in agreement with previous studies of Olds et al [49]

Importantly, 49 % of the neurons showed no changes in stimulus-evoked activity with conditioning That is, response plasticity during learned association appears to be a property of a subgroup of AC neurons There may be a subgroup of cells whose specific function is to facilitate learned associations Or, different subgroups may change in different learning situations

Relevance to previous studies

The heterogeneity of stimulus-evoked auditory cortex single unit activity in this work is similar to that reported in awake, non-conditioned animals of other species[1,10,41] We also found that AC neurons exhibited a variety of response changes with learning While response heterogeneity during learning was anticipated based on single unit data gathered from trained animals in non-learning contexts[2,4,25, 31,40 53,62], it was not obvious from multi-unit conditioning studies during response acquisition Multi-unit studies reported a single typical pattern of activity in the naive animal and also during the conditioning process[11,15,16,50] For the most part, neural spike discharge, characterized by an excitatory response at stimulus onset followed by sustained excitation of a lesser degree throughout the CS–US interval, was reported to increase with CR acquisition Such a consistent pattern was not evident in our data

The most obvious explanation lies in the differences in physiological recording and data analysis employed Multi-unit and single unit recording techniques introduce different sampling biases based on cell size Another bias is introduced by the method of determining when the microelectrode has come in contact with a cell In AC, where the majority of neurons have spontaneous rates of less

than 5 spikes/s[21,32], the likelihood of recording from cells with excitatory responses to acoustic stimuli is greater than for cells responding in an inhibitory manner In multi-unit recording, there is little reason for inhibitory units to be excluded from a cell population under study The previous conditioning studies in AC which monitored neural changes during acquisition reported pooled data from many cells and animals so that the invariant pattern in multi-unit studies may have resulted from statistical averaging Another possibility is that the generalized increase in unit activity reported in multi-unit studies reflected the contribution of initially 'silent' neurons recruited during the conditioning process

A simple 'enhancement phenomenon', suggested by multi-unit studies[11,16,50] is insufficient to account for learning-associated bioelectric events in AC The assumption that increased firing rates constitute the neural substrate of learning is untenable since decreased rates also frequently accompanied conditioning Instead, conditioning may result in a complex alteration in the pattern of neural activity in auditory cortex Circuits involving both inhibition and excitation may be altered or newly formed

There have been several studies of single units in both cortical[29,42,43,46,47,60] and subcortical[3,6,30,65] non-auditory CNS regions during conditioning The work of Morrell and his associates[42,43] on sensory conditioning of parastriate cortical units in paralyzed cats and rabbits is of most relevance here because of its concentration on another sensory association cortical field They found that visual cortex neurons also showed considerable heterogeneity in conditioned stimulus evoked alterations with changes specified to a definite subpopulation of neurons studied (45 % of cells changed in their latest report) Their paradigm has not yet related single cell changes to a conditioned behavior, but the overall similarity of Morrell's data in visual association cortex to ours in auditory association cortex would suggest that common principles may underly plasticity of information processing in both sensory systems

Relationship between neural events and CR acquisition

Early in training (transition trials) neural response plasticity was equally present in conditioned and pseudoconditioned rabbits The finding that changes in AC activity were not evident early in learning suggests that AC is not directly responsible for CR acquisition Lesion studies[44,45] have shown that animals can learn a simple tone-signalled behavior without AC Berger and Thompson[6–8] have shown that hippocampal neurons, both individually and as a group, change very early in the trial sequence of rabbit NM conditioning before behavioral CR acquisition Our data indicate that auditory association cortex neurons, as a group, change later in the trial sequence than do hippocampal neurons Previous studies of cortical neurons during the acquisition of a gross movement CR in rats showed that the neurons changed later[16] or at the same time[15] as CR acquisition Conditioning-related changes in auditory cortex multi-units have been shown tc follow the acquisition of a pupillary dilatation CR in paralyzed cats[50] Thus several lines of evidence consistently indicate that auditory cortex neurons do change in tone-signalled learning paradigms but that they change after the initial behavioral conditioned responses have been elicited

While AC may not be necessary for learning to occur, it is nonetheless clearly active, particularly soon after the CR has been acquired In the present work, differences between conditioned and pseudoconditioned groups were greatest during trained trials, when the CR was newly learned There was little further change in unit activity during overtrained trials in conditioned animals In other words, the changes in CS-evoked responses in conditioned rabbits were closely tied to a particular stage in the animal's learning process A function of AC may be to integrate newly acquired information with that already stored in the brain Another possibility is that changes in AC activity reflect early memory storage processes

Spontaneous rate

The lack of correlation between stimulus-evoked and spontaneous activity and the fact that changes in spontaneous rate occurred to a similar extent during conditioning and pseudoconditioning supports the assertion that these changes reflected fluctuations in unit activity unrelated to learning This is consistent with single unit data in previously trained animals where stimulus-evoked changes

occurred in the absence of spontaneous rate changes[4,25,53] with one exception[63]

Recording location

The recording location in this experiment is outside primary auditory cortex (AI) Viewed from the cortical surface, the electrode locations correspond to an area which Woolsey[64] called an association region based on the tonotopic frequency array and evoked potential latencies recorded from the cortical surface Both Woolsey's findings and those by McMullen and Glaser[35] agree that in the rabbit AI lies ventral and caudal to our recording region Traditionally, the cytoarchitecture of AI has been described as containing densely packed cells, fused cortical layers III and IV and a broad, sparsely populated layer V[13,33,37–39,51,55] All electrode lesions in our experiment lay dorsal to the area fitting this description in the rabbit in a region with fused and densely populated layers II and III and with broad and sparsely populated layers V and VI Since primary auditory cortex differs in surface position among cats[37] and squirrels[38], the position of auditory association cortex may be expected to vary similarly, but both the cytoarchitecture and the location of the recording area on the cortical surface correspond to a non-primary region, PAR 5, described by Rose[56]

Response plasticity is not unexpected in an association region of AC While we do not know the extent to which neurons would have changed in rabbit AI, considerable evidence suggests that we were recording from an area where response plasticity is most striking Neurons in association cortex of cat[28,37] and owl monkey[27] have been described as more 'labile' than neurons in AI during passive listening Benson et al [5] reported that response plasticity was more prevalent outside primary cortex than in AI

There is little or no evidence that association cortical areas receive converging fibers from primary sensory areas or that learning takes place in such cortico-cortico pathways[14] Rather, it appears that association areas receive major projections directly from subcortical structures Graybriel[22,23] proposed a distinction between the 'lemniscal line' and 'lemniscal adjunct' pathways The higher centers of the auditory lemniscal line consist of the central nucleus of the inferior colliculus, the ventral division of the medial geniculate body (vMGB) and primary auditory cortex The adjunct system contains the external and pericentral nuclei of the inferior colliculus, the posterior nucleus of thalamus, the medial division of the medial geniculate (mMGB) and association AC

Disterhoft and Stuart[16] argued that the lemniscal adjunct pathway has the greatest capacity for response plasticity based on the finding that response plasticity was prevalent in posterior nucleus of thalamus Within MGB, conditioning-related changes were found in mMGB only, and not in the dorsal or ventral portions of the nucleus[9,57,61] Both mMGB and posterior nucleus of thalamus project to non-primary AC[14,22,23] We postulate therefore that response plasticity measured in auditory association cortex is mediated at least in part by such thalamic input

Conclusion

This experiment joins two heretofore separate lines of research on auditory cortex — studies which have monitored multi-unit activity during CR acquisition and single unit experiments on previously trained animals in non-learning behavioral contexts As in previous learning studies we found that physiological events in AC can be correlated with the acquisition of a tone-signalled behavior In examining the activity of the same neuron during various stages of the learning process, we were able to sample a single unit substrate underlying behavioral learning Consistent with previous work on single AC neurons, the neurons in this study differed from each other in response pattern, sensitivity to acoustic stimuli, and the way bioelectric activity was altered (or not) during CR acquisition Our findings suggest that only a subgroup of cells in AC is active during and immediately following behavioral learning Learning-associated response plasticity is manifested primarily by changes in CS-evoked activity, with minimal effects on spontaneous rate and neural activity during the UCS and CR performance CS-evoked activity can be either enhanced or diminished, creating an altered pattern of neural discharge In other words, a group of AC neurons appears to be specially designed to modify

their activity in order to effectively deal with a changing environment

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