

## Corticofugal Modulation of the Medial Geniculate Body

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The function of corticothalamic projections in the auditory system was investigated by reversible inactivation of primary auditory cortex. Changes in the discharges of multiple-unit "clusters" within the ventral division of the medial geniculate body were assessed following removal of normal descending influences by cortical cooling. Patterns of neuronal discharges to clicks or tones were classified as either reverberatory or nonreverberatory during precool control sessions. Cortical cooling suppressed the late reverberatory discharges of the former, but had no effect on the discharge rate or pattern of the latter. The short-latency (< 20 msec) response was unaltered for either type. In addition, cooling of primary auditory cortex produced a significant increase in the background activity of nonreverberatory neurons but had no consistent effects on background activity of reverberatory neurons. Two distinct corticothalamic pathways are postulated to account for these results.

It is well known that sensory systems have descending pathways which apparently parallel their ascending components (12, 18, 20, 21, 24, 28, 29). The existence of reciprocal anatomic connections between neocortex and thalamus has special relevance to the concept that sensory cortex modulates its own input (14, 19). In this regard, it has been demonstrated that the various subdivisions of auditory cortex in the cat are differentially connected to distinct subdivisions of the medial geniculate body (9-12, 30, 31). In particular, there exist reciprocal connections between discrete subregions of primary auditory cortex and discrete areas within the ventral medial geniculate body which include pars lateralis and pars ovoidea (8).

Despite the prominence of these corticofugal projections and the detailed anatomical accounts that are available, only a few published reports have

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focused on their possible physiological role in the modulation of afferent acoustic impulses in the medial geniculate body, and these are not in agreement. One line of evidence suggests that efferents from primary auditory cortex are inhibitory to the ventral medial geniculate body. Electrical stimulation of primary auditory cortex attenuates field potentials evoked by clicks (4) and reduces the response of single units to both click and tone stimulation (34). However, facilitatory effects have also been found. Stimulation of primary auditory cortex reduces the latency and increases the probability of single unit discharges of medial geniculate body (6). Finally, the absence of corticofugal modulation has been reported (2, 34). Aitkin and Dunlop (2) have advanced arguments that cortical stimulation effects are mediated antidromically. Furthermore, they found that massive ablations of primary auditory cortex failed to modify the recovery cycle of field potentials evoked by acoustic stimuli or to alter the pattern of "reverberatory" single-cell discharges in the medial geniculate body.

The purpose of the present study was to investigate the function of corticofugal input from primary auditory cortex to ventral medial geniculate nucleus. To insure stimulus constancy at the receptors, an unanesthetized, paralyzed preparation was used. To avoid the confounding effects of antidromic conduction, we have tried to assess the corticofugal influence by observing the effects of its temporary removal on response patterns of multiple-unit clusters in the ventral medial geniculate nucleus. Acoustic evoked responses were first recorded under normal conditions; then the primary auditory cortex was temporarily and reversibly cooled and, finally, rewarmed. The nonrandom changes in response patterning that occurred during cooling were attributed to the removal of the normal corticofugal influence.

## METHODS

*Subjects and Surgical Procedure.* Twenty adult cats of either sex served as subjects. Anesthesia was induced by an intravenous injection of 2.5% sodium thiamylal (Surital) and maintained through a venous cannula by subsequent injections when necessary during the surgical procedure. Atropine methyl nitrate (0.5 mg) was administered subcutaneously and an endotracheal tube with an inflatable cuff was inserted. Body temperature was regulated with a circulating warm-water pad. The cat was placed inside a sound-attenuating room and mounted in a stereotaxic instrument with hollow earbars. The skull was removed on one side overlying the portion of the medial geniculate body to be explored and the ipsilateral middle ectosylvian gyrus (primary auditory cortex). A dam of dental cement was formed around the edges of the skull opening which was filled with warm mineral oil. The dura was reflected and the cooling device was

positioned on the cortex. Then, a thermistor probe (Yellow Springs Instruments #514) and a microelectrode were placed at an angle just beneath the cooling device. The mineral oil was removed and the cement dam was filled with clear agar. All wound edges and pressure points were heavily infiltrated with either xylocaine jelly (Lidocaine) or mepivacaine hydrochloride (Carbocaine) and anesthesia was discontinued. The endotracheal tube was connected to the respirator located outside the room, and 35 mg/kg gallamine triethiodide (Flaxedil) was administered through the venous cannula.

*Stimulation and Recording.* Stimuli consisted of either clicks or pure tone bursts. The clicks were produced by 0.1-msec square-wave pulses generated by a Tektronix 161 pulse generator. Tones were generated by a Wavetek 114 oscillator. Either clicks or tones could be switched into both channels of a Grason-Stadler electronic switch (Model 829E) the onset and offset of which were controlled by Tektronix 161 and 162 modules. The tones had a 50-, 100-, or 200-msec duration and a rise-decay time of 5 msec. The output of each channel of the switch was fed through Hewlett-Packard model 350D attenuators into a TDH49 earphone mounted at the end of the earbar contralateral to the auditory cortex and medial geniculate nucleus. Stimulus intensities were approximately 85 dB re. 0.0002 dyn/cm<sup>2</sup> as determined by a Bruel and Kjaer 2205 impulse sound level meter.

Multiple-unit activity in the medial geniculate body and the auditory cortex was recorded from stainless steel microelectrodes insulated with EpoxyLite (tips, 2.0  $\mu$ m and impedance 0.5 to 5 M $\Omega$ ). The EEG and evoked potentials from primary auditory cortex were recorded from the cut end of a teflon-insulated tungsten wire 0.62 mm in diameter attached directly to the bottom of the cooling device. All neural activity was amplified by Tektronix 122 preamplifiers with bandwidths set at 0.8 to 10 kHz for units and at 0.8 to 250 Hz for the EEG and evoked potentials. Multiple-unit activity from primary auditory cortex was passed through a Schmitt trigger and output integrated and written out on a Grass 7PCP-B polygraph. The EEG and evoked potentials from auditory cortex were monitored on an oscilloscope. Unit activity from the medial geniculate body was monitored on an oscilloscope and recorded on one channel of a Sony TC-650 tape recorder; synchronizing pulses were recorded on the other channel.

*Cooling Technique.* The cooling device consisted of a hollow brass cylinder, 12.5 mm diameter and 68 mm in length, closed on one end. The tungsten wire which was used to monitor the cortical EEG and evoked potentials was affixed centrally on the bottom of the cylinder and extended 1.0 mm. The bottom of the cooling device was aligned against the surface of primary auditory cortex and held in place by a lucite bridge attached to

a stereotaxic electrode carrier. Cooling was effected by filling the cylinder with antifreeze and pellets of dry ice. This method rapidly reduced the temperature of primary auditory cortex to 8 to 14 C for approximately 10 min. The cortex was rewarmed to 37 to 38 C by removing the coolant and flushing the cylinder with warm water.

*Experimental Procedure.* Following initial preparations, the cat was left alone until EEG spindling stopped and the EEG exhibited low-voltage, fast activity. At this time, a microelectrode was lowered through the agar and into the brain directly above the medial geniculate body. As the electrode was lowered through the dorsal lateral geniculate body, activity could be evoked by the presentation of a light. When the light no longer evoked neural responses, contralateral acoustic stimuli were delivered and the electrode was lowered until clear evoked unit activity was observed.

The precooling trials consisted of acoustic stimuli delivered in blocks of 30 at a rate of 1/sec, alternating with an equal number of sham (or no stimuli) blocks. The acoustic stimulus was either a click or a pure tone of variable frequency, depending on which stimulus evoked the best response. After collecting several blocks of data, the primary auditory cortex was cooled to 8 to 14 C. It usually took approximately 3 to 5 min to reach this temperature and the cooling was accompanied by a depression of the EEG, diminution of evoked potential magnitude, and severe depression or abolition of unitary activity at the cortex. The lowered temperature of the cortex typically remained at this level for 10 to 12 min. The cooling trials consisted of alternating blocks of stimulation and sham trials. Following the collection of the cooling data, the cortex was rewarmed to 37 to 38 C. The cortex rewarmed quite rapidly, and this rewarming was heralded by the return of unit discharges just beneath the cooling device. The rewarming of cortex was judged to be complete when the EEG and the cortical evoked potential were recovered. Following rewarming, blocks of acoustic and sham stimulation were presented again. It must be noted that in nearly one-third of the cases, the evoked potential never fully recovered from cooling. Furthermore, cooling and warming could not be repeated indefinitely without indications of cortical deterioration. Not more than three multiple-unit clusters could be analyzed from a single subject. Marking lesions were made at the most ventral point of each penetration. At the end of each recording session, the cat was killed and the brain subjected to routine histological analysis.

*Data Analysis.* Unitary discharges for the medial geniculate body were analyzed off-line using a LAB-8 computer. The activity was played back into three identical Schmitt triggers, each set at a different level in order to investigate possible differences in neuronal patterning based on spike amplitude. The output of the Schmitt triggers was fed into the computer and poststimulus time histograms and levels of spontaneous activity were

computed. The histograms were constructed from 1 block of 30 acoustic stimulus presentations during the precool, the cool, and, when appropriate, the rewarm session. There was a resolution of 250 bins and each bin was 2 msec. To evaluate changes in the frequency of firing, the mean rates of discharges during the cooling period were compared statistically with mean rates observed during precool control conditions. The analysis of changes in neuronal patterning required a bin-by-bin spike count comparison (correlated *t*-test) between comparable segments of the histogram under the different conditions. The 500-msec histograms were divided into an initial segment (the first 10 bins) for evaluation of the short-latency onset response and a remaining segment (the last 240 bins) for examination of the "late" response. Changes in background activity were evaluated over the entire 250 bins. Complete recovery from cooling was determined when the rewarm histogram was not significantly different in spike rates or neuronal patterning from the precool histogram.

## RESULTS

*Cooling Controls.* The effects of cooling primary auditory cortex cannot be attributed to inactivation of corticogeniculate fibers unless alternate possibilities are eliminated. We addressed the problems of (i) direct cooling of the medial geniculate body, (ii) an actual activation of auditory cortex or the production of an epileptic focus by cooling, and (iii) nonspecific effects due to cortical cooling *per se* rather than to specific cooling of primary auditory cortex.

The temperature gradient during cooling of primary auditory cortex was determined in two cats (Fig. 1A). Auditory cortex was cooled to 12 C and maintained at this level for 45 min. A thermistor placed stereotaxically in the ventral medial geniculate body recorded no change from 36–38 C during the first 30 min; the temperature dropped thereafter to 25 C at 45 min. Experimental cooling sessions were never longer than 20 min, thus insuring that direct cooling of the medial geniculate body could be discounted.

Several investigators have reported that cooling may excite rather than depress cortical neurons (17, 26, 27). We monitored the functional state of primary auditory cortex beneath the cooling device in all animals. Cooling resulted in a depression of the EEG, a large reduction in the field potential to click, and virtual elimination of background unit discharges (Fig. 1B). These measures provided evidence that the auditory cortex had not been inadvertently excited by cooling.

Nonspecific effects were investigated by examining the cortical surface temperature gradient during cooling of auditory cortex and determining the effects on ventral medial geniculate activity of cooling nonauditory cortical

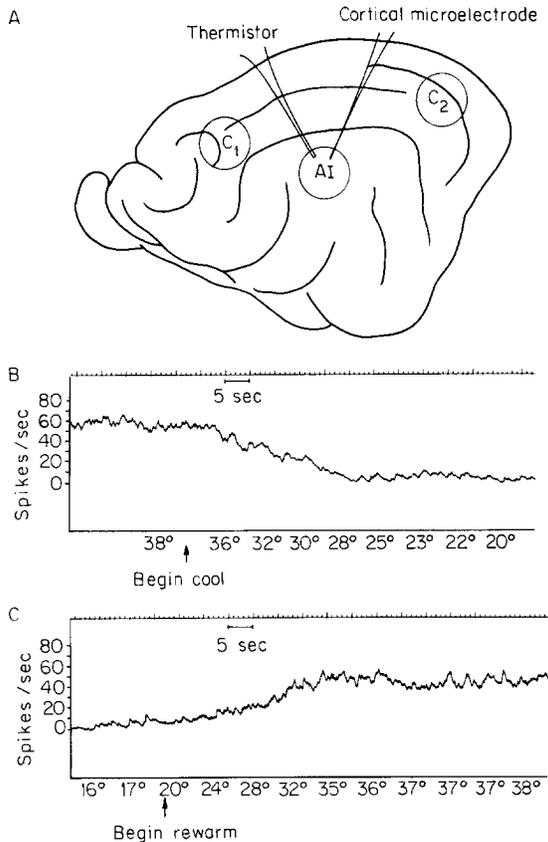


FIG. 1. View of cat cortex showing placements of cooling device, thermistor, and cortical microelectrode (A). Experimental cooling placements were over primary auditory cortex; control cooling placements were at C<sub>1</sub> and C<sub>2</sub>. Cortical temperature and multiple-unit activity were recorded continuously; cooling suppresses cortical activity (B) whereas rewarming reverses the effect (C).

regions. When the auditory cortex was cooled to 7 to 8 C, cortical temperature 5 mm from the edge of the cooling device was never lower than 27 C. Either the anterior suprasylvian gyrus or the posterior lateral gyrus was cooled in two cats (Fig. 1A) while recording from a total of three loci in ventral medial geniculate nucleus. In contrast to the effects of cooling auditory cortex (see below), cooling of nonauditory cortex was without consistent effect on neuronal activity in ventral medial geniculate (Fig. 2). All these findings indicate that the results reported below may be attributed to the inactivation of auditory corticothalamic fibers.

*Effects of Cooling on Ventral Medial Geniculate Body.* The effects of cooling were investigated for 34 points within the ventral medial geniculate body (Fig. 3). Multiple-unit activity recorded from each site is hereafter

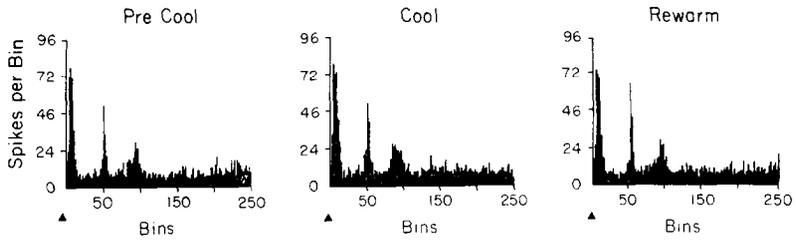


FIG. 2. Cooling of anterior suprasylvian cortex ( $C_1$ ) had no effect on click-evoked activity of ventral medial geniculate nucleus. Poststimulus time histograms generated from 30 stimulus presentations during the three different conditions. Arrow indicates click presentation.

referred to as the activity of “clusters” of neurons. The tone-burst and click poststimulus histograms of these clusters during precool conditions were similar to those reported for single units in this region (3, 13). Two major types of response to acoustic stimulation were discerned. The first type ( $N=21$ ) was a short-latency (8 to 14 msec) onset response (or in the case of tone bursts, a maintained response during stimulation or offset response at the end of stimulation) followed by a return to background levels of discharge (Fig. 4). The second type ( $N=13$ ) also had a short-latency onset response, which was followed by inhibition for 50 to 150 msec, and then excitation alternating with periods of low-discharge probability (Figs. 5 and 6). This pattern has been referred to as “reverbera-

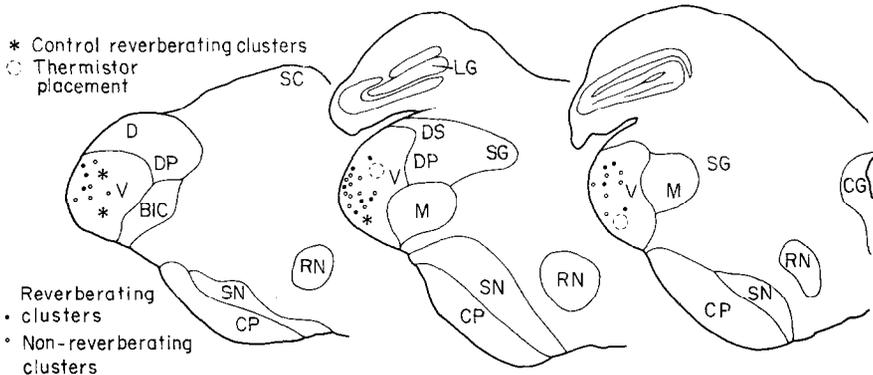


FIG. 3. Histological verification of thermistor and microelectrode placements in ventral medial geniculate as seen in coronal sections taken at A3, A4, and A5 [modified from (33)]. Reverberating and nonreverberating clusters appear randomly distributed throughout the nucleus. BIC—brachium of inferior colliculus, CG—central gray, CP—cerebral peduncles, D—dorsal division of medial geniculate body (MGB), DP—deep dorsal division of MGB, M—medial division of MGB, RN—red nucleus, SG—supragenicular nucleus, SN—substantia nigra, V—ventral division of MGB.

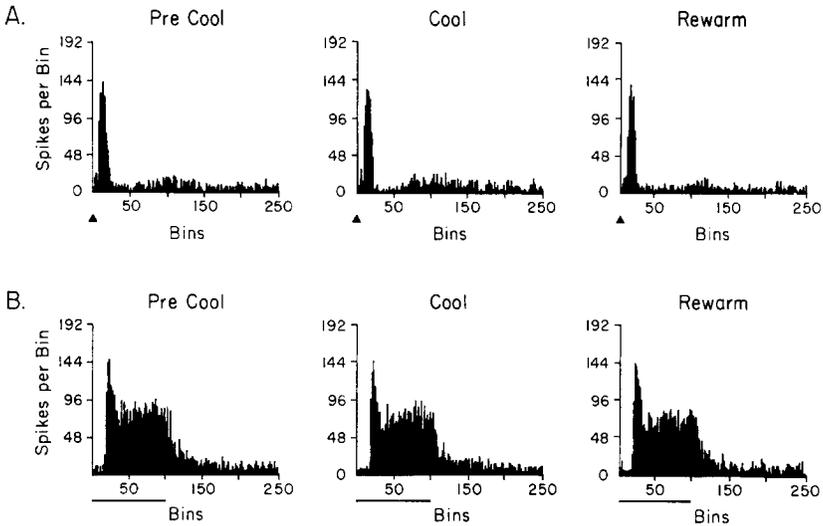


FIG. 4. Effects of cooling primary auditory cortex on nonreverberating cluster. Poststimulus time histograms generated from 30 presentations of clicks (arrow) and 2500-Hz tones (bar). Neuronal patterning is not affected by cortical cooling. Also notice the absence of any prominent late component.

tory", (3) and this terminology will be used here without any implications as to its physiological substrate. Reverberatory responses seemed to be associated with "bursting" background activity; however, no quantitative assessments were performed.

The evoked activity of the 13 reverberatory clusters was significantly reduced in all cases by cooling primary auditory cortex ( $P < 0.05$ ,  $t$ -test for correlated means). This effect was reversible during subsequent re-warming (Figs. 5 and 6). Inspection of poststimulus histograms indicated that cooling did not cause a reduction of all evoked activity, but was largely confined to the "reverberatory" discharges. Five of the 13 clusters received tone burst stimulation and exhibited either a sustained response ( $N=2$ ) or an offset response ( $N=3$ ) preceding the reverberatory response. These discharges did not appear to be attenuated by cooling (Fig. 5), but no separate statistical analyses were performed on these parts of the histograms. Statistical analyses of discharges during the first 20 msec of the poststimulus histograms revealed that cooling was without effect. Thus, the onset response of reverberatory-type clusters was immune from the effects of cooling the auditory cortex, whereas longer latency reverberatory discharges were always reduced or eliminated.

In marked contrast to the effects of cooling on evoked activity, no consistent effects were found for background activity. Background activity for three clusters was unchanged, for five clusters was significantly increased,

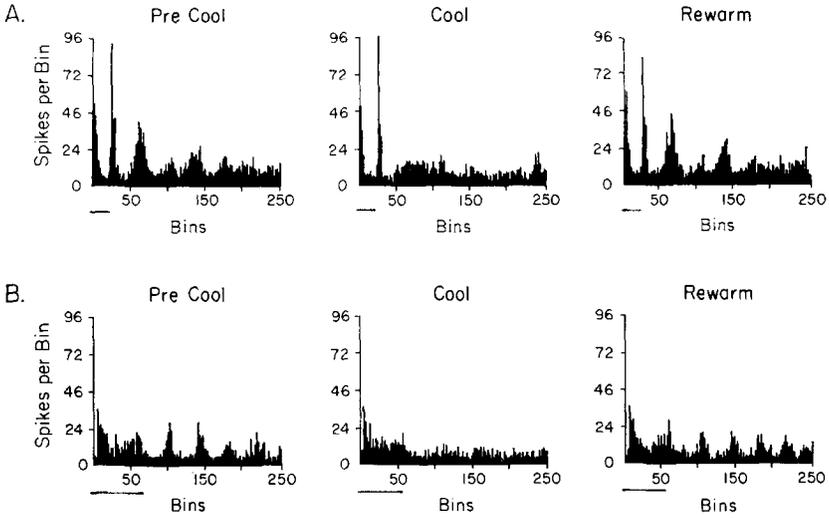


FIG. 5. Effects of cooling primary auditory cortex on two separate clusters (A and B) demonstrating different tone-evoked histograms. (A) has an "on-off" response pattern; (B) has a "sustained" response pattern. Cooling suppresses only the reverberation; neuronal activity returns to precool levels upon rewarming. Bar indicates tone duration.

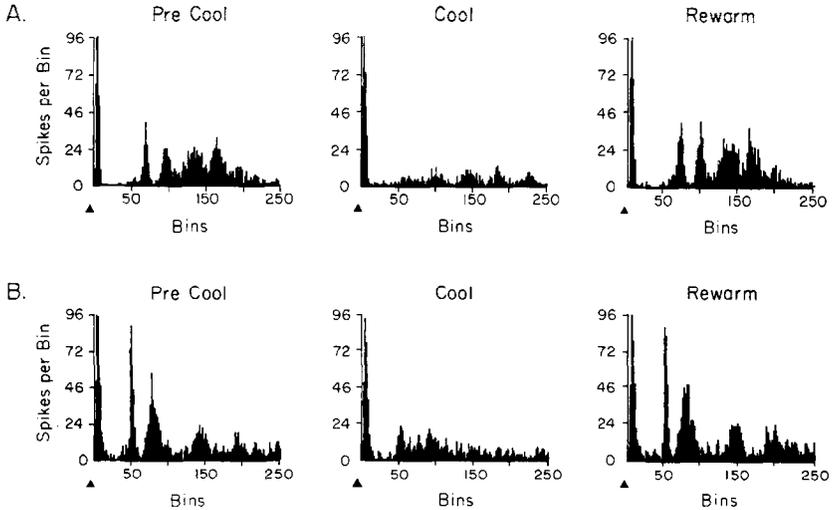


FIG. 6. Effects of cooling primary auditory cortex on two separate clusters (A and B) demonstrating different click-evoked histograms. Cooling suppresses the late reverberatory component of the histogram without affecting the initial onset response. Rewarming results in the return of neuronal activity to essentially precool levels. Arrow indicates click presentation.

TABLE 1

Changes in Firing Patterning of Ventral Medial Geniculate Neuronal "Clusters" during Cooling of Primary Auditory Cortex as Compared to the Precool Period

	Evoked Activity								
	Background Activity			Onset Response (<20 msec)			Late Response (20 to 500 msec)		
				NC	Inc.	Dec.	NC	Inc.	Dec.
<b>Reverberatory Response Pattern</b>									
Onset Excitation (n = 8)	1	5	2	8	0	0	0	0	8
Onset-Offset Excitation (n = 3)	1	0	2	3	0	0	0	0	3
Sustained Excitation (n = 2)	1	0	1	2	0	0	0	0	2
Totals (n = 13)	3	5	5	13	0	0	0	0	13
<b>Nonreverberatory Response Pattern</b>									
Onset Excitation (n = 2)	1	1	0	2	0	0	1	0	1
Onset-Offset Excitation (n = 11)	2	8	1	11	0	0	6	2	3
Sustained Excitation (n = 8)	1	5	2	7	1	0	3	4	1
Totals (n = 21)	4	14	3	20	1	0	10	6	5

<sup>a</sup> Number of clusters showing no change (NC), increased activity ( $P < 0.005$ ) (Inc.), and decreased activity ( $P < 0.05$ ) (Dec.).

and for the remaining five clusters was significantly decreased (Table 1).

The effects of cooling on nonreverberatory neuronal activity were quite distinct from the cooling effects on reverberatory neurons. Auditory cortical cooling had no consistent effect on long-latency ( $> 20$  msec) discharges; there was no change for ten clusters, an increase for six, and a decrease for five. On the other hand, background activity was often increased in these neurons; statistically significant increases ( $P < 0.05$ , *t*-test for correlated means) were found in 14/21 clusters. This number of increases is more than would be expected by chance ( $P < 0.02$ , chi-square). The responses of nonreverberatory clusters were the same as for reverberatory clusters in one respect: cortical cooling did not alter short-latency onset responses (Fig. 4, Table 1).

## DISCUSSION

*Specificity of Effects of Cortical Cooling.* The purpose of the present experiment has been to clarify the function of primary auditory corticofugal input to ventral medial geniculate body by temporarily and reversibly removing it and examining the effects of its withdrawal on the discharges

of the geniculate neurons. We determined that cortical cooling does not result in a direct spread of cooling to ventral medial geniculate body or in an actual activation rather than inactivation of cortical neurons. However, a thorough consideration of other possible nonspecific effects of the cooling procedure is required before we can conclude that our results are due to the inactivation of cortical neurons.

First, does cortical cooling produce changes in systemic blood flow which could alter the discharges of neurons in the medial geniculate body? Although we have no direct measure of blood flow through that region, two points argue against such vascular changes: (i) control cooling of non-auditory cortex had no effect on evoked reverberation in ventral medial geniculate body, and (ii) the short-latency geniculate onset response was unaffected by the cooling procedure. The stability of this onset response across different experimental conditions suggests a normal or at least stable blood flow through the nucleus.

Second, does the cooling procedure result in a "state" change by arousing the animal? Such changes in arousal level could have resulted from (i) the production of pain or cold sensations or both through receptors in the dura adjacent to the cooling device; (ii) the detection of the cold "lesion" in auditory cortex; or (iii) a dishabituation of the subject due to change in experimental conditions. Control cooling of nonauditory cortex should have produced the same degree of stimulation of the dura and "lesion" detection; the distinctly different neural effects resulting from the differential cortical cooling argue against the above nonspecific influences. Finally, if dishabituation of the subject due to changes in experimental conditions resulted in a change in evoked neuronal patterning, then the transition from the cool condition to the rewarm condition likewise should have constituted a change in experimental conditions and resulted in yet another pattern of evoked activity. The statistically indistinguishable patterns of evoked activity under conditions of precool control and rewarming suggest that a simple change in experimental conditions was not the crucial variable. The results of these control measures strongly suggest that our results were due specifically to the local cooling of primary auditory cortex.

*Effects of Cortical Cooling on the Ventral Medial Geniculate Nucleus.* The present findings provide compelling evidence that the primary auditory cortex does modulate the neuronal activity of the ventral medial geniculate nucleus. Furthermore, the effects are present regardless of whether clicks or tones are used. We cannot specify whether our recordings originated from principal neurons or Golgi type II neurons within the ventral medial geniculate nucleus. We suspect the former because of the similarity of the poststimulus histograms to previous data from single unit studies (3, 13). We also assume that both "reverberatory" and "nonreverberatory

cells" are principal neurons, observed under two slightly different levels of general arousal, because subtle changes in arousal level can occur in the absence of gross changes in the EEG (36). In addition, it is recognized that the present approach can give no definitive answer to the issue of the anatomic substrates of corticogeniculate control. Although we have emphasized the existence of direct corticogeniculate fibers, other cortical projections to the inferior colliculus (12, 15) and striatum (7, 15, 22, 23, 35) exist. Corticogeniculate effects could be mediated indirectly via such pathways. With these qualifications in mind, we turn to a consideration of the nature of cortical control of the ventral medial geniculate nucleus and the possible role of corticogeniculate circuitry.

Cooling of primary auditory cortex has two effects: (i) elimination or depression of long-latency rhythmic discharges in "reverberatory" neurons; and (ii) enhancement of discharges in nonreverberatory cells in the absence of specified acoustic stimulation. Therefore, it is apparent that corticofugal effects cannot be classified simply as either excitatory or inhibitory (see Introduction). Rather, the auditory cortex seems to have two processes for modulating its thalamic relay nucleus.

The first finding suggests that a corticofugal mechanism ordinarily supports rhythmic discharges following acoustic stimulation. This is not to suggest that the auditory cortex is absolutely necessary for rhythmic activity within the thalamus (8); there is abundant evidence that such rhythmicity may be intrinsic to the thalamus (1-3, 5, 16). Rather, it appears that primary auditory cortex can profoundly modulate rhythmic afterdischarges in the ventral medial geniculate nucleus. Previous reports which largely dismissed such a role for the sensory cortex (2, 3, 5) failed to record from the same neuron(s) under conditions of intact and ablated cortex. The present technique of functional "ablation" is apparently more sensitive to these effects. It is noteworthy that cortical cooling reduces long-latency reverberatory discharges without altering the initial short-latency response to auditory stimulation. Therefore, the normal cortical support of longer-latency rhythmic discharges may be triggered by the geniculocortical volley. It is known that primary auditory cortex neurons synapse upon the distal regions of dendrites of principal relay cells in the ventral medial geniculate nucleus (25). This pathway could account for the finding that stimulation of auditory cortex increases the excitability of principal cells without producing neuronal discharges (6). Increased excitability could promote continual afterdischarges following the firing of principal cells by a volley from the inferior colliculus. This would be the case regardless of whether the thalamic mechanism underlying rhythmic afterdischarges is based on recurrent collaterals (5) or dendrodendritic synapses (32).

The second finding suggests that another corticofugal mechanism ordi-

narily dampens the discharge of cells in the ventral medial geniculate nucleus because cortical cooling increases the background discharge rate of nonreverberatory cells. The functional significance of this mechanism is unknown, but the outcome of this tonic inhibitory influence would be to reduce the level of bombardment from the thalamus in the absence of changes in the acoustic environment. Such an effect could be mediated via corticothalamic fibers which are known to synapse on the proximal dendrites and somata of interneurons in the ventral medial geniculate nucleus (25). Such synapses are in a position to "drive" the Golgi type II cells, which in turn would inhibit the principal neurons.

These conjectures do not account for the failure of cortical cooling to consistently alter the background discharge rate of reverberatory cells or the evoked discharges of the nonreverberatory cells. Regarding the latter, it should be pointed out that, as in the case of reverberatory neurons, cooling did not affect the short-latency onset discharge to acoustic stimulation. As nonreverberatory cells exhibit no rhythmic afterdischarge, the presence of subliminal cortical excitation of principal cells (6) could not be revealed. With reference to the absence of effect on the background discharge rate of the reverberatory cells, it may be that the tonic descending inhibition operates only in the "nonreverberatory" state. It is recognized that these speculations are largely gratuitous pending more detailed knowledge of corticothalamic circuitry. For example, it would be vital to know whether corticogeniculate projections to principal and Golgi type II cells arise as branches from the same axons or from different cortical cells. The interpretations set forth here would require that two anatomically distinct corticogeniculate systems exist—one that dampens ascending activity in the absence of acoustic stimulation, the other which prolongs the ascending effects of auditory messages.

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