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# SUPPRESSION OF SINGLE UNIT ACTIVITY IN COCHLEAR NUCLEUS OF THE CAT FOLLOWING SOUND STIMULATION[1]

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A PERIOD OF SENSORY STIMULATION may alter the perception of subsequent sensory events. After sounds, for example, there may be changes in threshold (19), in the quality of acoustic sensation (20, 29), or tinnitus (20). The neurophysiological mechanisms underlying these alterations in sensation are not well understood. Working with cats, Starr and Livingston (30) described a profound and reversible suppression of summated spontaneous activity in subcortical stations of the auditory pathway as an aftereffect of sustained sound exposure. The period of altered spontaneous activity lengthened as the intensity or duration of the sound was increased. The experiments on single units in cochlear nucleus presented in this report were undertaken to define more precisely the relationship between the physical parameters of the sound stimulus and the period of altered spontaneous activity.

## METHODS

Experiments were performed on 12 cats, each weighing 3.5–5 kg. The animals were anesthetized with sodium pentobarbital (60 mg/kg., ip) and placed in a stereotactic apparatus with hollow ear bars. Body temperature was maintained with a heating pad and supplemental sodium pentobarbital was administered every 2–3 hours as needed. A portion of the bone and dura mater overlying the cerebellum was removed and a metal ring to support a motor-driven microelectrode carrier (3) was attached to the skull with dental cement; it was oriented toward the left cochlear nucleus in nine animals and toward the left trapezoid body in three animals.

*Microelectrodes.* Single unit activity was recorded with glass-insulated, platinum microelectrodes (15) with a tip diameter of 1–2 $\mu$. and an impedance of 1–12 megohms measured with a 1-msec. ramp pulse applied to the electrode via a small condenser (15).

*Sound stimuli.* Tones were generated by an audio-oscillator (General Radio, type 1301-A) having fixed frequency selections between 250 cycles/sec. and 15,000 cycles/sec., attenuated by an audiofrequency voltmeter (Philips, EL6082/10) attached to the left ear bar by a short plastic tube. The earphone responses were linear between 750 cycles/sec. and 2,000 cycles/sec., 55 db down at 5,000 cycles/sec., and 80 db down at 15,000 cycles/sec. Clicks were square-wave pulses, 0.07 msec. in duration, generated by a DISA stimulator and presented to the animal through the same attenuator and microphone. For studies on single units in the trapezoid body, another earphone (Oticon AFE4) was attached to the right hollow ear bar by a plastic tube similar in length to that used in the left ear bar. This

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microphone was used to present clicks obtained directly from the second output of the DISA stimulator (DISA Electronic, Copenhaven).

*Recording.* Electrical activity was recorded via a negative-capacitance cathode follower (16), amplified with a lower limiting frequency of either 10 or 100 cycles/sec. (3 db down) and an upper limiting frequency of 10,000 cycles/sec. (3 db down), displayed on a dual-beam Dumont oscilloscope and photographed on continuously moving film (5–20 mm/sec.). One of the beams was used for continuous recording while the other beam was used for single sweep recording once every second on a much faster time scale (1 msec/mm.–20 msec/mm.).

*Procedure.* The microelectrode carrier was mounted in the supporting ring and the electrode advanced through the cerebellum while clicks were presented at 1/sec. When an evoked slow-wave response was detected, the spontaneously active units were tested with tones and only those units responsive to clicks or pure tones were examined further. The tone which elicited an increase in unit activity with the lowest intensity was used in studying changes of spontaneous activity after sound stimulation. Each unit was stimulated with the frequency to which it was most sensitive at intensities between 4 and 23 db above threshold. At least four durations, from 5 sec. to 640 sec., were used. Sufficient time elapsed between each trial for the spontaneous activity to recover to pre-exposure levels, from at least 15 sec. after a 5-sec. tone to 20 min. after a 640-sec. tone.

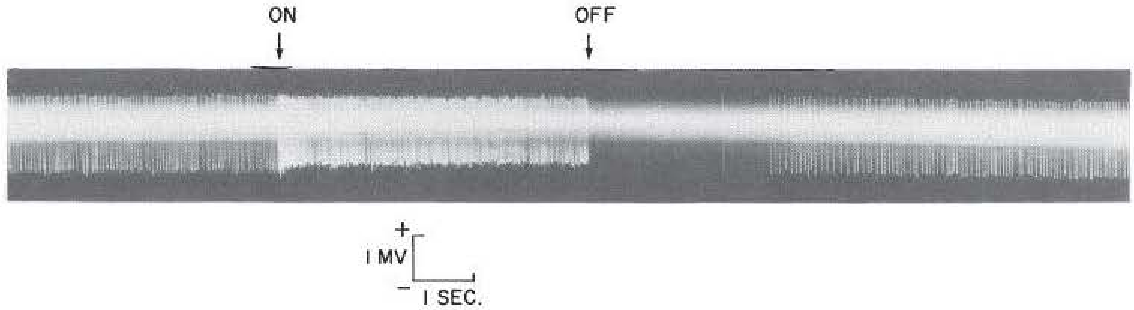


FIG. 1. To show the absence of spontaneous activity after cessation of an adequate stimulus and its gradual return to the prestimulus rate. The activity of a spontaneously active unit (*14–11*) before, during (from "on"), and after (from "off") 5 sec. of stimulation with a 2,500 cycles/sec. tone 4 db above the unit's threshold.

At the end of the experiment the electrode was left in place while the animal was perfused with 10% formalin and the head kept in the stereotactic apparatus overnight. Inspection of the middle ear revealed the ear bars to have disrupted the tympanic membrane in eight of the animals. In these animals the impairment of sound transmission across the middle ear necessitated the use of higher sound intensities to elicit aftereffects than in animals with intact middle ears. The cranium was removed so the course of the microelectrode could be determined by direct inspection and then the brain was placed in 10% formalin. Serial sections of the brain stem, stained with thionine, were examined for the electrode tracts. It was not possible to define where within the cochlear nuclear complex each unit was found since several tracks were made with each electrode and up to seven electrodes were used in a single experiment.

*Data analysis.* The film records were enlarged and the unit responses before and after each sound stimulation were counted. Cessation of activity after sound stimulation was defined as the time until a spontaneous discharge reappeared in each of two consecutive seconds. Unit activity during sound stimulation was determined by counting discharges on the interrupted sweeps since the rate of firing was often too high to be resolved on the continuous records.

## RESULTS

*Cessation of activity after sounds.* The 40 units examined showed a reversible depression of spontaneous activity on terminating the tone: spontaneous activity was absent during the first moments and reappeared only gradually to approximate pre-exposure rates (Fig. 1). In 35 of the 40 units the period of depressed spontaneous activity lengthened as either the intensity (Fig. 2*A*) or duration (Fig. 2*B*) of the tone was increased. Intensity effects were limited, however, to a 30–40 db range above thresholds; inten-

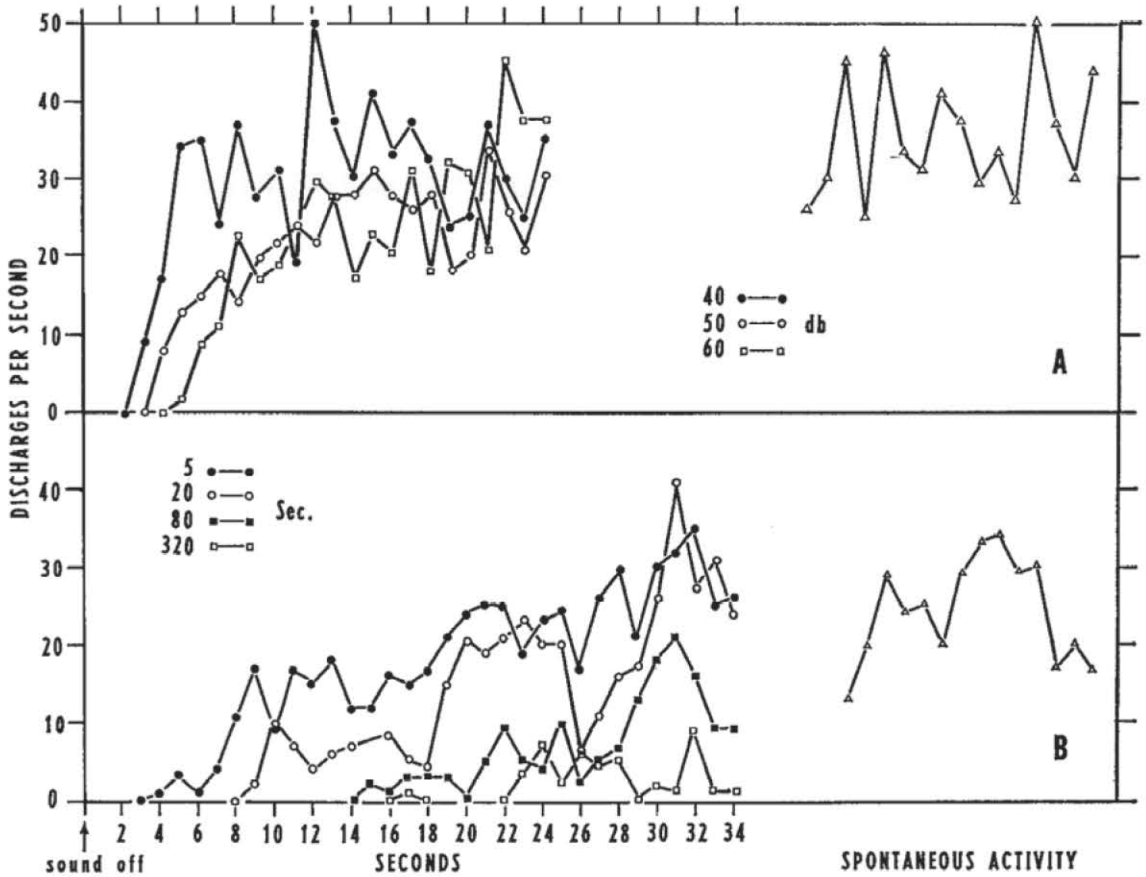


FIG. 2. To show the slower return to the pre-exposure rate of spontaneous firing with *A*, increasing intensity of the tone, and with *B*, increasing duration of the tone. *Ordinate:* frequency in spikes per second. *Abscissa:* time after cessation of the tone in seconds. To the right the variation in spontaneous firing is shown. *A: unit 3–6*, amplitude 1 mV., threshold 35 db. Stimulus tone 7,500 cycles/sec. for 10 sec. at 40 ●—●, 50 ○—○, and 60 □—□ db. *B: unit 5–6*. Stimulus tone 2,000 cycles/sec., 90 db, for 5 ●—●, 20 ○—○, 80 ■—■, and 320 □—□ sec.

sities above this level were not accompanied by any further lengthening of aftereffects. The duration of the absent activity was shortened if the unit's rate of spontaneous activity increased spontaneously during the course of study (Fig. 3). Therefore, the cessation of spontaneous activity as a function of the duration of the sound stimulus is given only for those units which fired at a constant rate (Table 1, Fig. 4). In 15 of 20 such units (types A and B), the period with absent spontaneous activity lengthened with increasing duration of the tone, ranging from 1.2 to 15.0 sec. for a 10-fold increase (20 db) in stimulus time. For 7 of the 15 units (type A, Table 1, Fig. 4) the aftereffect increased linearly with the logarithm of the duration of stimulation; the relation was not linear for the other 8 units (type B, Table 1, Fig. 4). In these 15 units the rate of adaptation was slow; their discharge frequency during the tenth second of stimulation being 59–95% of the rate during the initial second.

In five units, there was no prolongation of the aftereffect with increasing duration of the tone (type C, Table 1, Fig. 4). These five units did not differ from other units with regard to the rate of spontaneous activity or to the "best" frequency of the tonal stimulus. However, four of the five units were characterized by a relatively rapid adaptation to sound stimulation so that

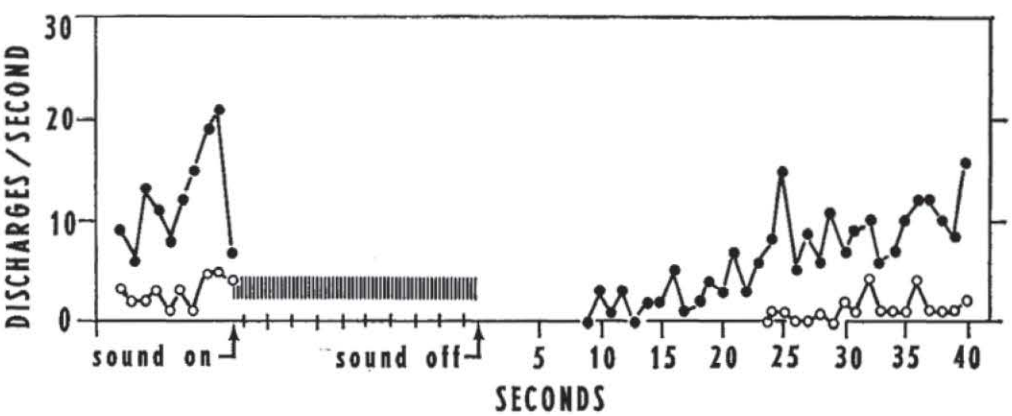


FIG. 3. To show the longer cessation of activity in a single unit (*14–10*) when the spontaneous frequency was low (1–4/sec., ○—○) than when it was high (6–25/sec., ●—●). *Ordinate:* frequency in spikes per second. *Abscissa:* time in seconds. Stimulus tone 10 sec., 1,000 cycles/sec., 10 db above threshold.

their discharge rate during the tenth second of stimulation was 19–39% of the rate during the first second of stimulation.

In six units (*12-3, 12-7, 13-7a, 14-4, 14-6, 25-1*), there were tones which, though not loud enough to increase the rate of firing, nonetheless were followed by cessation of spontaneous activity. These six units were unusually sensitive to the intensity of tones; their rate and pattern of firing being altered by slight changes in the intensity of the tones. In the example shown in Fig. 5, the frequency of firing decreased during a tone at 85 db and then increased briefly after cessation of that intensity to a rate higher than the pre-exposure rate. When the tone was increased by 10 db (to 95 db SPL), the firing rate remained essentially unchanged, but firing ceased for more than a second after cessation of the tone. A still louder tone (105 db) evoked an increase in activity and was followed by an absence of spontaneous activity for seconds. Thus, within a 20-db intensity range, this unit's responses changed from inhibition during the tone with rebound acceleration of activity after the tone to excitation followed by a temporary suppression of spontaneous activity. This type of dynamic response pattern has been described previously for single units in the cochlear nucleus (25).

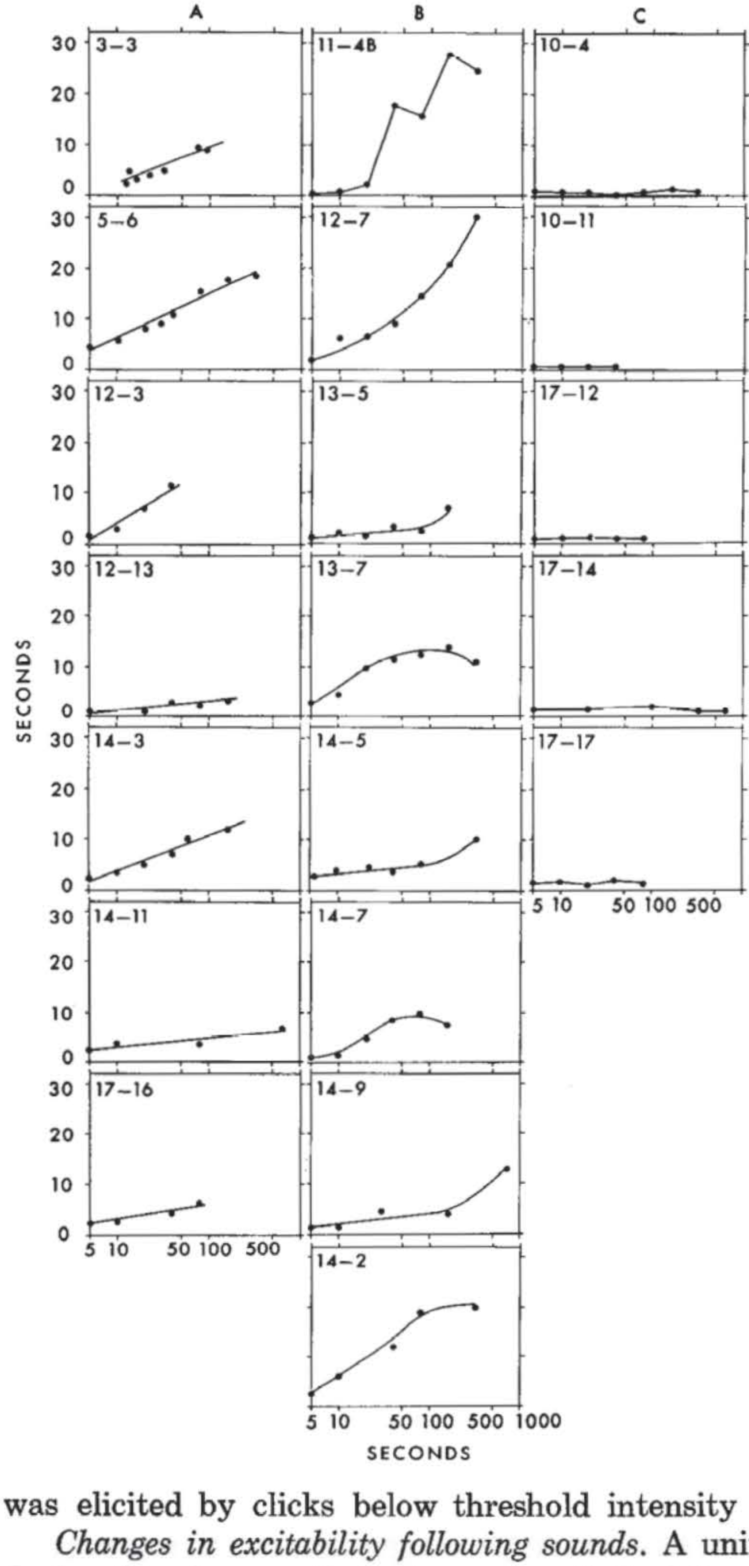


FIG. 4. The duration of absent spontaneous activity (ordinate, sec., linear scale) as a function of the duration of the tone (abscissa, sec., logarithmic scale). Twenty units are classified into three categories: *A*, a linear relation to the logarithm of duration, seven units; *B*, a nonlinear relation to the logarithm of duration, eight units; *C*, no effect of the duration of stimulation on the duration of absent spontaneous activity, five units. The data on these units are given in Table 1.

*Cessation of activity after clicks.* A depression of spontaneous activity followed clicks as well. In spontaneously active units, clicks elicited a short-latency discharge followed by a complete suppression of spontaneous activity for 20–120 msec. (Fig. 6); the more intense the click the longer was the suppression of spontaneous activity. In some units a suppression of spontaneous activity was elicited by clicks below threshold intensity for evoking a discharge.

*Changes in excitability following sounds.* A unit's excitability decreased during the absence of spontaneous activity following sound stimulation as demonstrated by testing its ability to respond to a click delivered after the tone. The unit shown in Fig. 7 failed to respond to a click for several seconds after a 10-sec. stimulation with a 103-db tone. This same click intensity had elicited a response with every presentation prior to the tonal stimulus. In this and in seven other units both the responses to clicks and the spontaneous activity returned sooner as the intensity or the duration of the tone was reduced. In all instances responses to clicks reappeared before spontaneous activity had resumed control rates.

*Trapezoid body.* Three units in the trapezoid body were examined to

*Table 1. Characteristics of single units graphed in Fig. 4*

| Unit | Amplitude, mV. | Form* | Spontaneous Activity, discharges/sec. | Best Frequency, cycles/sec. | Threshold,† db | Stimulus, db | % Adaptation‡ | Increase of Aftereffects, sec/10-Fold Increase of Stimulus Time |
|---|---|---|---|---|---|---|---|---|
| *Type A* | | | | | | | | |
| 3–3 | 8 | b | 10–19 | 3,000 | 65 | 75 | 95 | 7.6 |
| 5–6 | 6 | b | 20–30 | 2,000 | NT | 112 | 60 | 8.0 |
| 12–3 | 6 | b-n | 80–110 | 750 | 93 | 108 | 71 | 10.4 |
| 12–13 | 8 | b-n | 90–100 | 2,500 | 80 | 100 | 88 | 1.2 |
| 14–3 | 3 | b | 40–50 | 1,000 | 84 | 99 | 70 | 7.8 |
| 14–11 | 2 | m | 10–25 | 2,500 | 91 | 95 | 62 | 1.6 |
| 17–16 | 5 | b | 7–12 | 2,000 | 92 | 115 | 67 | 2.0 |
| *Type B* | | | | | | | | |
| 11–4B | 1.5 | b | 10–15 | 1,000 | 109 | 137 | 86 | 15.0 |
| 12–7 | 1.5 | m | 40–55 | 1,000 | 126 | 134 | 90 | 14.4 |
| 13–5 | 8 | b-n | 30–40 | 3,000 | 65 | 75 | 93 | 3.0 |
| 13–7 | 7 | b | 20–30 | 1,000 | 109 | 110 | 62 | 7.8 |
| 14–5 | 2 | b | 15–20 | 2,000 | 83 | 95 | 83 | 3.6 |
| 14–7 | 1 | b | 50–60 | 1,000 | 109 | 114 | 59 | 7.6 |
| 14–9 | 1 | b | 20–30 | 1,000 | 104 | 115 | 60 | 4.0 |
| 14–12 | 1.5 | b | 20–30 | 1,000 | 102 | 119 | 81 | 10.8 |
| *Type C* | | | | | | | | |
| 10–4 | 20 | b-n | 10–20 | 250 | 83 | 98 | 39 | 0.0 |
| 10–11 | 2.5 | b-n | 70–90 | 2,500 | NT | 111 | 19 | 0.0 |
| 17–12 | 1. | b | 60–80 | 1,000 | NT | 120 | 71 | 0.0 |
| 17–16 | 1.5 | b | 12–20 | 2,500 | 106 | 115 | 19 | 0.0 |
| 17–17 | 5 | b | 20–25 | 1,000 | 101 | 119 | 25 | 0.0 |

* b = biphasic; b-n = biphasic with notch on positive limb. † NT = not tested.

$\ddagger \frac{\text{discharges/sec. during 10th sec. of stimulation}}{\text{discharges/sec. during 1st sec. of stimulation}}$.

determine whether suppression of activity and altered click responsiveness after sound stimulation can be ascribed solely to changes in the peripheral receptor or whether it is an attribute of central mechanisms. Units in this station of the auditory pathway are suitable for such an analysis, since they may respond to sounds presented to either ear. For each unit the click intensity which always elicited a response was first determined. Then the unit's best frequency was delivered to the contralateral ear and clicks, delivered to the ipsilateral ear, were resumed as soon as the tone was discontinued. In the three units tested, responses to the test clicks did not reappear in the initial period of absent spontaneous activity. In these instances the cessation of single unit activity after a tone resulted from central mechanisms. Additional evidence for the participation of central mechanisms in aftereffects was the temporary suppression of the terminal injury discharges of single units following sounds (Fig. 8).

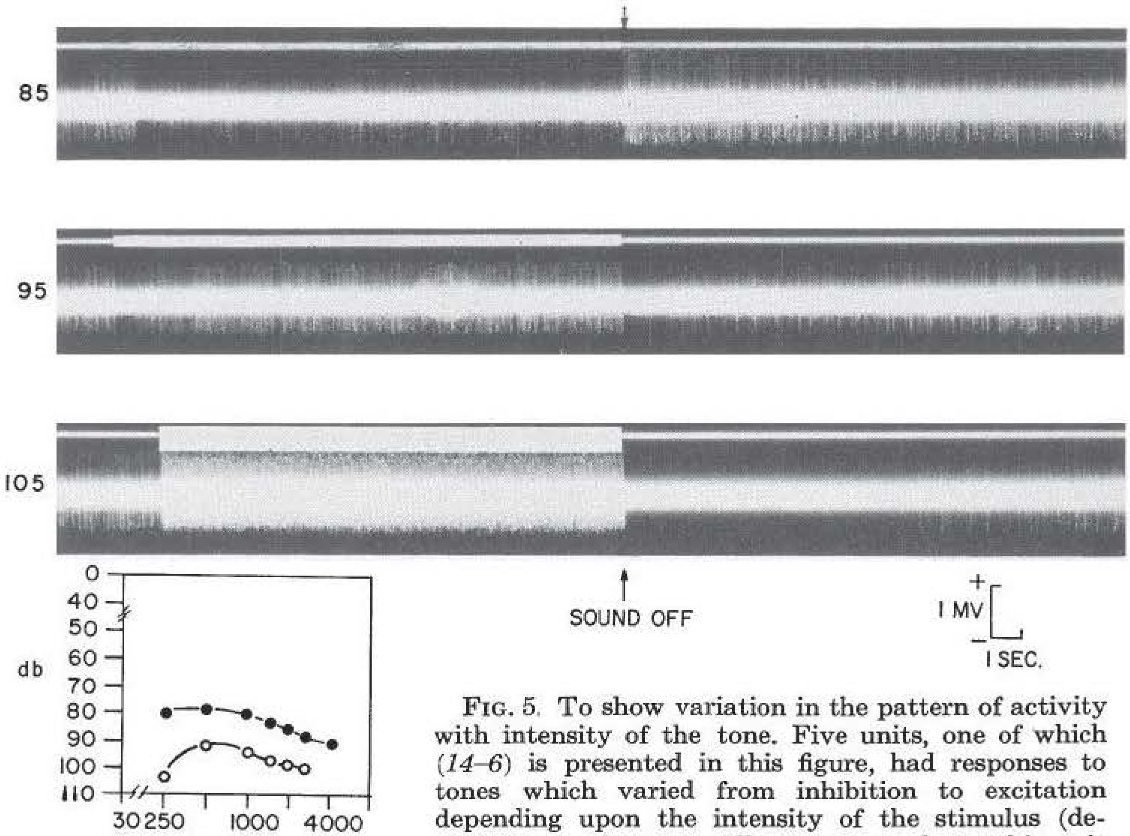


FIG. 5. To show variation in the pattern of activity with intensity of the tone. Five units, one of which (*14–6*) is presented in this figure, had responses to tones which varied from inhibition to excitation depending upon the intensity of the stimulus (description in the text). The insert graph for this unit demonstrates that 15–25 db separated those intensities inhibiting the unit (filled circles) from those activating the unit (open circles).

## DISCUSSION

*Aftereffects.* These experiments confirm earlier findings of a temporary suppression of spontaneous activity in single units in cochlear nucleus following sound exposure (28, 31) and establish that its duration lengthened with increases in both intensity and duration of the sound stimulus. Suppression of unit activity following tonal stimulation, however, did not depend solely on the parameters of the tone but was influenced, as well, by the unit's excitability both prior to and during stimulation. Thus, activity was suppressed for a briefer interval when the unit's rate of spontaneous activity accelerated suddenly or when there was a terminal discharge than when the

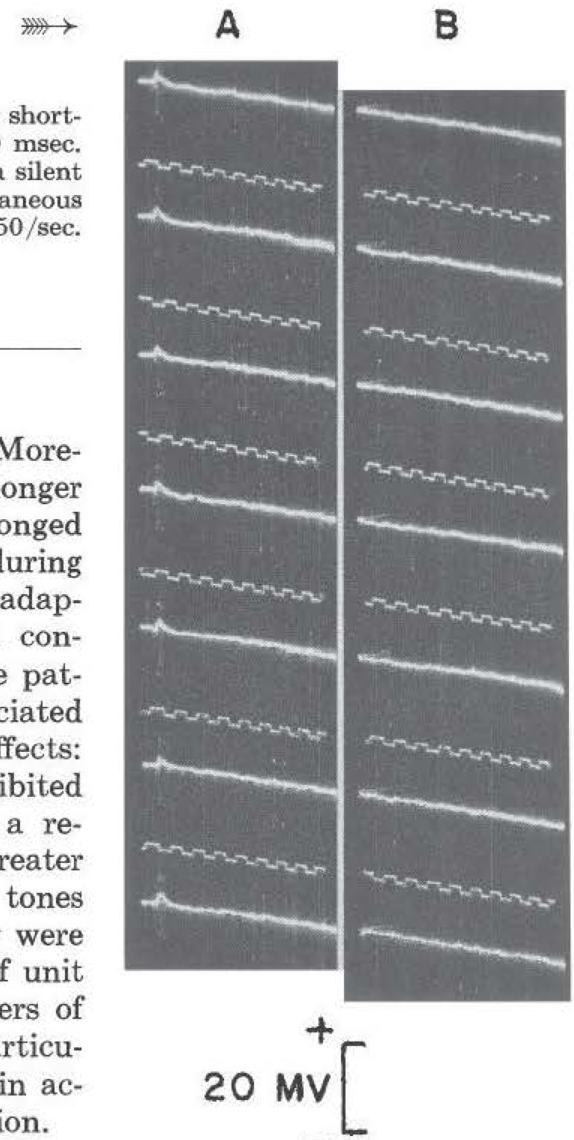


FIG. 6. Successive sweeps to show: *A*, the short-latency burst responses to clicks (signal 10 msec. after the onset of each sweep) followed by a silent period for 60–110 msec.; *B*, The unit's spontaneous activity (no clicks). Time calibration is a 50/sec. square wave.

rate of spontaneous activity was low. Moreover, activity was suppressed for longer periods as the tonal stimulus was prolonged in those units which adapted slowly during stimulation whereas units with rapid adaptation had aftereffects of brief and constant extent. Finally, changes in the pattern of evoked activity were associated with changes in the pattern of aftereffects: in the same unit, tones which inhibited ongoing activity were followed by a rebound acceleration of activity to greater than pre-exposure rates whereas tones which evoked an increase in activity were followed by a temporary cessation of unit discharges. Thus, both the parameters of the tone and the excitability of the particular unit contributed to the changes in activity appearing after tonal stimulation.

A temporary suppression of spontaneous activity following sound stimulation has been observed so commonly in other brain-stem stations along the auditory pathway (10–12, 18) that it may be considered as a typical aftereffect of sound. It may be seen both when studying single units and when recording the summed responses in brain-stem nuclei along the auditory pathway (30). That the cessation of activity represented an alteration of unit excitability was demonstrated by the failure of units to respond to clicks during the early absence of spontaneous activity; both the period of diminished click responsiveness and the period of suppressed spontaneous activity lengthened as the intensity or duration of the tone was increased. These results correspond to the findings of Rose *et al.* (28) and Enger (10) of decreased responsiveness of single units in cochlear nucleus and eighth nerve following sound stimulation. A change in excitability to clicks in cochlear nucleus after sounds might be accounted

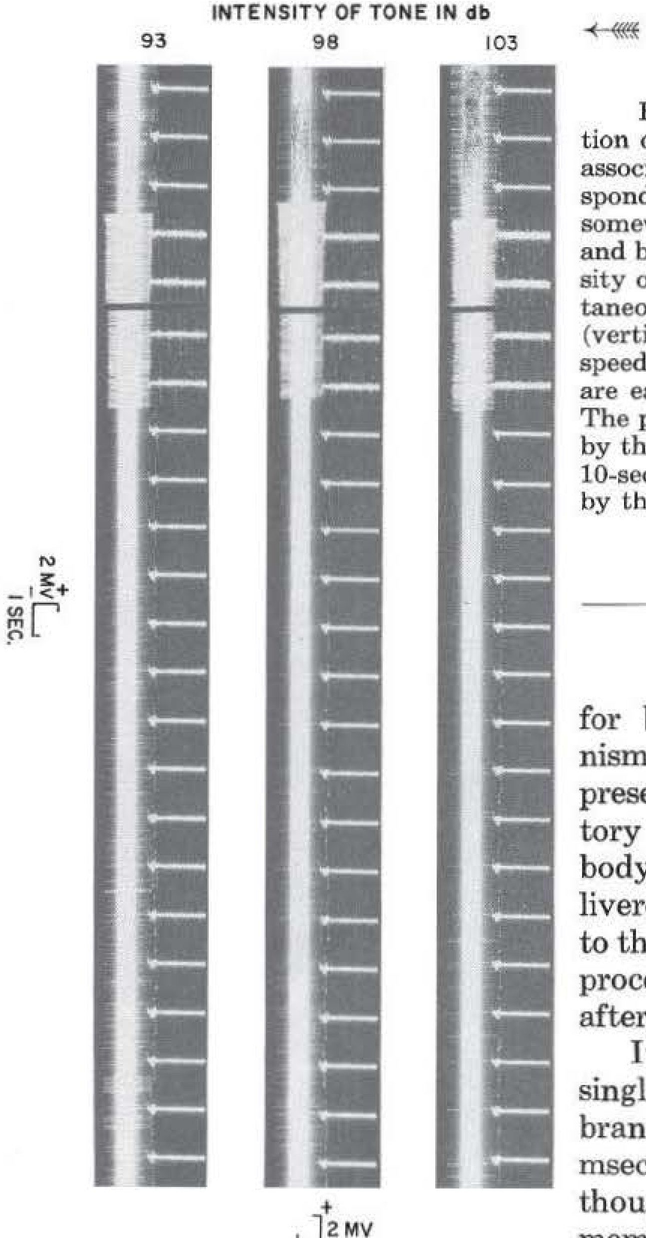


FIG. 7. To show that the initial cessation of spontaneous activity after a tone is associated with the unit's inability to respond to a click. Click responses return somewhat earlier than spontaneous activity, and both return sooner, the lower the intensity of the tone. Each column shows simultaneous continuous record at slow speed (vertical) and interrupted record at fast speed (horizontal). The interrupted records are each triggered by a click presentation. The period of tonal stimulation is indicated by the increase in activity. A portion of the 10-sec. tone has been deleted, as indicated by the black bar.

for by peripheral receptor mechanisms. The demonstration in the present experiments of a postexcitatory depression in three trapezoid body units when the tone was delivered to one ear and the test click to the other ear indicates that central processes also participate in the aftereffects of sound stimulation.

It has been shown that after a single excitation the neuronal membrane is hyperpolarized for up to 500 msec. (7). This hyperpolarization is thought to result from a change in membrane permeability, a metabolic effect of activity (14); the duration and extent of the hyperpolarization increase with increasing activity (8). Hyperpolarization as a consequence of excitation occurs in many neurons including those along the auditory pathway (26) and could account for the cessation of spontaneous activity and suppression of responses to clicks after tone stimulation.

A temporary suppression of spontaneous activity is not unique for the auditory system as it occurs in single units in the visual (4, 5, 13), olfactory (2), somesthetic (6, 21), and vestibular pathways (1, 23) following adequate sensory stimulation. In these systems, too, cessation of activity, or postexcitatory inhibition, depended upon the intensity and duration of the stimulus

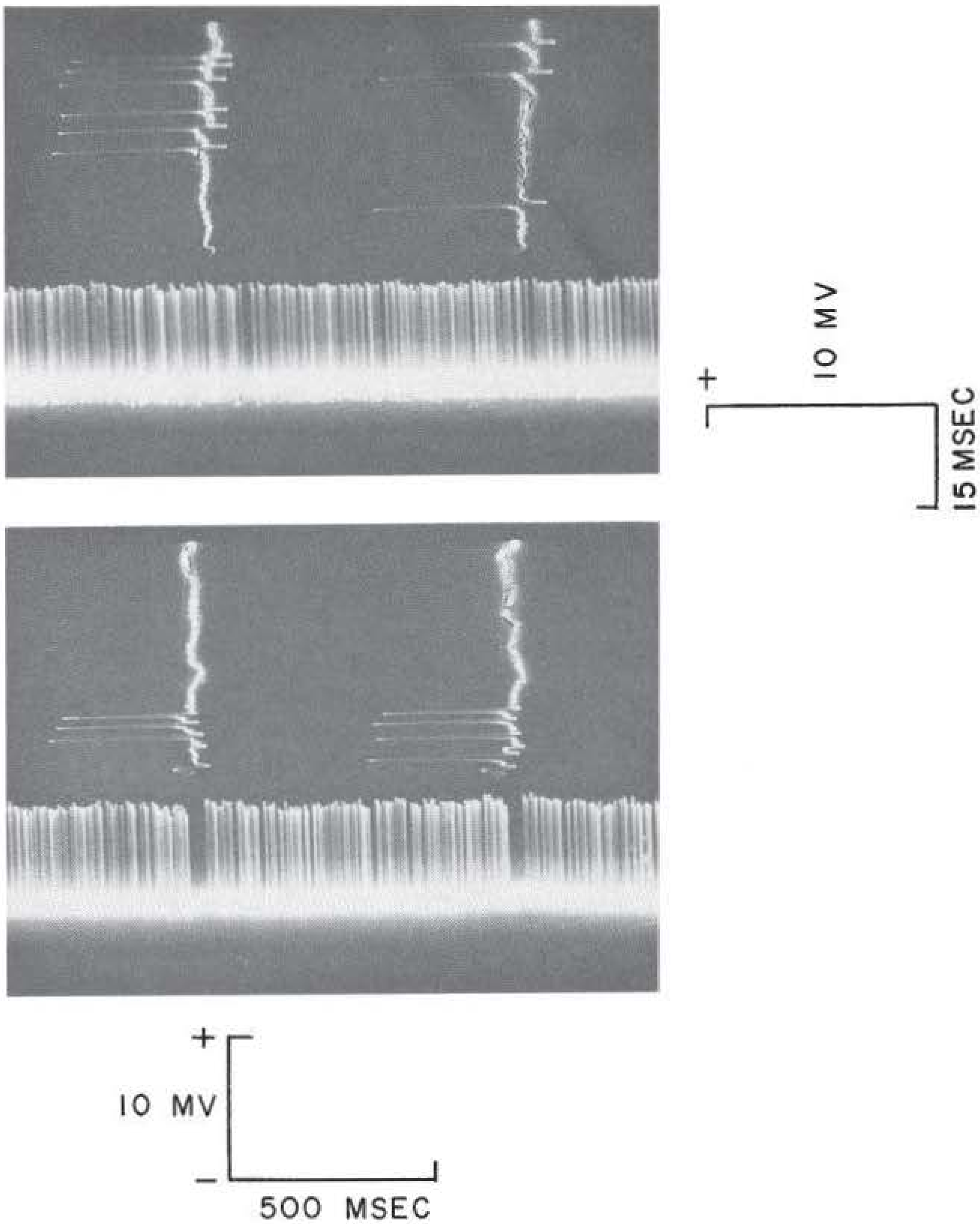


FIG. 8. To show the temporary suppression of terminal injury discharges following sound stimulation. (*Unit 8–3*, cochlear nucleus.) The unit's terminal activity is shown in the upper portion of the figure. The discharge rate of about 100/sec. and the predominantly monophasic (positive) potential contrasted with the slower spontaneous rhythm (5/sec.) and biphasic shape of the unit when initially observed. In the lower portion of the figure single clicks were presented coincident with the beginning of each vertical sweep. The evoked discharges were followed by a temporary absence of all injury activity. Unit responses were entirely lost shortly after these recordings.

(13, 23) and could differ from one unit to the next according to the unit's pattern of activity during stimulation (1, 13). The postexcitatory inhibition in these systems has, however, been limited to a few milliseconds, whereas postexcitatory inhibition was longer in the study reported here and could last for several hours when the summed activity was recorded (30). Possibly the use of longer stimuli in other systems would reveal similarly longer post-excitatory inhibition.

*Relation of aftereffects to auditory fatigue.* The aftereffects demonstrated in single units in the cochlear nucleus have several temporal and quantitative similarities to psychophysical results. In man, exposures to steady sounds are followed by change in acoustic thresholds referred to as auditory "fatigue" or "temporary threshold shift" ("TTS," 19). Both the recovery of thresholds (32) and the recovery of spontaneous activity in single units examined in the present experiments increase linearly as a function of the logarithm of the duration of sound exposure. In man, threshold changes are effected with sound intensities slightly above threshold (9) or even with sounds that are too faint to be perceived (22); the magnitude and duration of the TTS increasing with intensity (32). Correspondingly, in the present experiments sound intensities just a few decibels above a unit's threshold were followed by a temporary suppression of spontaneous activity, and increases of intensity within a 40-db range were accompanied by increases in the duration of suppressed activity. During recovery from TTS sounds may be perceived as unusually loud (17). Though the excitability of single units to clicks was reduced following sound exposure, responses reappeared at a time when spontaneous activity was absent or considerably reduced. If the nervous system functions in sensory estimations by comparing evoked to spontaneous activity as suggested by Mountcastle *et al.* (24) then the increased contrast between evoked responses and spontaneous activity could provide a mechanism for the perceptual aftereffect of increased loudness. Finally, there are psychophysical experiments suggesting that auditory fatigue cannot be attributed solely to changes occurring at the cochlea. The observation of a temporary threshold shift in the unstimulated ear following monaural stimulation (27) and the reports of tinnitus contralateral to the stimulated ear (19) suggest an involvement of central structures. The finding of alterations in spontaneous activity in subthalamic stations along the auditory pathway following sound exposure (30) is evidence for such central changes. These reversible alterations of spontaneous activity may be neurophysiological equivalents of perceptual aftereffects of sound stimulation.

## SUMMARY

1. Spontaneously active units in cochlear nucleus of sodium pentobarbital-anesthetized cats were studied during stimulation with steady tones for periods ranging from 5 sec. to more than 10 min.

2. In all of the units examined activity was absent on terminating the tone and reappeared only gradually to approximate pre-exposure rates.

3. The duration and intensity of the tone influenced the period of suppressed spontaneous activity. In 15 units absence of activity increased as the stimulus time was lengthened; in 7 of these units the increase was linear when plotted as a function of the logarithm of the duration of the tone and

in the other 8 units the relationship was nearly linear. Increasing intensity of the tone, up to 40 db above threshold, was followed by longer lasting suppression of spontaneous activity. Intensities above that range caused no further increase in aftereffects.

4. Unit activity after tones was also influenced by its discharge patterns both prior to and during stimulation. An increase in the rate of spontaneous activity shortened the period of absent activity after tones. In units with slow adaptation during stimulation the period of suppressed activity lengthened with increases in stimulus time. In contrast, units with rapid adaptation had brief aftereffects that were unaffected by stimulus length. In the same unit, aftereffects of excitatory stimuli and aftereffects of inhibitory stimuli differed. Following excitation there was a temporary suppression of spontaneous activity; following inhibition there was a temporary rebound acceleration of spontaneous activity to greater than pre-exposure rates.

5. The cessation of spontaneous activity after a tone is associated with a decrease in unit excitability as indicated by the inability of the unit to respond to clicks during most of that period. That the excitability change can be mediated by central nervous system mechanisms was demonstrated in three units recorded from the trapezoid body where tonal stimulation delivered to one ear caused an aftereffect of absent spontaneous activity and inability of the unit to respond to clicks delivered to the other ear.

6. The relationships of these findings to similar findings in other sensory systems, to the demonstration of postexcitatory hyperpolarization of the membrane potential, and to psychophysical observations on auditory fatigue in man are discussed.

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