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ACOUSTIC AND NONACOUSTIC FACTORS MODIFYING MIDDLE-EAR MUSCLE ACTIVITY IN WAKING CATS

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INTRODUCTION

MOST STUDIES OF THE MIDDLE-EAR MUSCLES have emphasized their reflex role in protecting the inner ear from damaging loud sounds (8, 12, 13, 24, 26, 30, 36). Recent demonstrations of middle-ear muscle activity during low-intensity sounds (29) suggest that these muscles may have a wider role than merely protecting against mechanical damage. The present experiments reveal that in waking cats middle-ear muscle activity may be modified by prior acoustic experience, by nonacoustic factors such as bodily movements, and by changing the significance of the sound for the animal. This paper presents an analysis of some of the mechanisms underlying middle-ear muscle activity and illustrates both sustained and transient contractions which are regulated according to complex central activities, rather than responding as a fixed protective reflex arc.

MATERIALS AND METHODS

Electrodes were implanted at the round window in adult cats according to the method of Galambos and Rupert (7). Animals with round-window electrodes have been divided into three groups: *1*) Fourteen "normal" animals; *2*) twelve animals with one or both tendons of the intra-aural muscles sectioned; and *3*) seven animals with one or both eighth cranial nerves sectioned but with intact intra-aural muscles. Operative procedures were performed on animals anesthetized with Nembutal, and utilizing aseptic technique. The surgical approach for electrode implantation of both the round-window and intra-aural muscles was from the posterior-lateral aspect of the bulla through a curved incision just behind the pinna. The tendons of the tympanic muscles were cut using a Zeiss binocular operating microscope at about 20× magnification. Division was done with the cutting circuit of a Birtcher electrosurgical unit, delivered to the bared tip of a 30-gauge stainless steel wire. This method permitted division of the tendons without damage to or dislocation of the ossicular chain. The eighth nerve was approached intracranially at the internal meatus of the internal auditory canal and crushed to avoid cutting the internal auditory artery and seventh nerve. At the end of all operations the bony defect in the bulla was closed with dental cement (Acralite). The animals were allowed to recover for a week prior to experimental observations.

Electrical activity of middle-ear muscles in seven cats was recorded with bipolar 36-gauge enamel insulated, stainless steel electrodes. The electrodes were made in the following manner. The insulation was stripped from the first 2 mm. of each electrode and the tip sharpened on a honing stone. Several coils were made 3 mm. from the sharpened tip. A single polyethylene tubing (PE 10) was threaded over two such wires from the unsharpened end and knotted just above the coils. The tips were placed in the muscle and the polyethylene tubing fixed to the bone with dental cement at the point where it crosses the opening

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made in the bulla. The coils were then spread, providing a slight spring-like tension to hold the electrode tips in place. The electrodes were easily inserted directly into the belly of the tensor tympani. However, to implant the stapedius muscle it was necessary to drill a small hole in the bone that covers it just dorsal to the round window (6).

Round-window responses were also recorded in ten animals following intercollicular decerebration. Decerebration was performed using ether anesthesia and the animals were allowed to recover for at least 2 hours before experimental observations. The ontogeny of middle ear action was studied in 11 kittens ranging from birth to 10 weeks of age. In these younger animals the bulla is poorly formed, and the round window was approached from the ventral aspect. A coiled electrode was used, smaller in size but similar in design to those employed in the adult. The kittens were implanted under ether anesthesia and allowed to recover for several hours. Recordings were made from most of these animals on the day of operation as well as on subsequent days.

Two hours of "white" noise stimulation of approximately 85 db (re .0002 dynes/cm^2) intensity was delivered to awake and unrestrained cats and also to the same animals while anesthetized with Nembutal. Frequency analysis of this noise revealed uniform energies between 500 cycles and 6,000 cycles, with a falling off of energies above and below these frequencies (33). The recording cage was constructed of chicken wire and located in a large sound-attenuating room separated from an adjacent observation room by a one-way mirror. The physical properties of the speaker and recording room result in sound field variations within the range of movement of the cat's head of only $\pm 3/4$ db (22). The sound was delivered as a free field stimulus from a speaker mounted 30 in. above the cat's head. The cage was mounted on small rubber grommets, allowing slight oscillation when the cat moved. A piezoelectric crystal (Astatic phonographic cartridge L-82-A), spring-mounted against the cage, served as a transducer for these oscillations (27). The output of the crystal was filtered to pass all frequencies between 0.2 cycle and 50 cycles (G. P. Frommer, personal communication).

The band-pass of condenser-coupled amplifiers (Tektronix type 122) was set at 0.2 cycle to 10 kc. for round-window responses and 0.2 cycle to 250 cycles for tympanic-muscle activity. These amplified potentials were monitored on a four-channel oscilloscope and photographed on 35 mm. and Polaroid films. The electrical activity was also summated and continuously recorded by techniques described by Starr and Livingston (33). The summator is essentially a recording a.-c. voltmeter, consisting of an attenuator, amplifier, full wave rectifier, and low-pass filter with variable time constants. The output of this circuit was recorded on Esterline-Angus type AW pen writers.

At post mortem the location of electrodes in the middle ear was verified using the Zeiss binocular operating scope. Petrous bones of animals with crushed eighth nerves were decalcified, serially sectioned, and stained with Luxol fast blue MBS and counterstained with cresyl violet to ascertain whether complete degeneration of cells of the spiral ganglion had occurred.

Results

At onset of loud sound there is a brief startle reaction which takes place even after many daily exposures to the same experience. Following this some cats cower and try to escape, while most look toward the loud-speaker before resuming a restful posture. During continuing stimulation cats may vocalize, groom, and shift position in the cage. These activities all vastly influence round-window and middle-ear muscle activity. Generally, however, cats remain quietly in the experimental cage before, during, and after white noise exposure.

1. *Round-window response in waking cats.* Round-window responses decrease sharply in amplitude between 30 and 75 msec. following onset of a steady, loud, white noise (Fig. 1). The average decrease for 14 normal round windows is 10 db with a range of 6–18 db. During continuing exposure responses gradually increase again, taking 30–90 min. to reach a stable high

plateau approximating the amplitude at onset. A typical record of an intact animal's summated round-window response during 1 hour of steady white noise is shown above the oscilloscopic responses in Fig. 1. (The high amplitude response at onset demonstrated on the oscilloscope does not appear as an independent event on the summated record because of its short duration.)

The gradual rise of the response is interrupted by large transient decreases regularly associated with bodily movement. The magnitude of attenuation reaches 7 db and is roughly proportional to the extent of movement. After movements cease, summated responses return to the premovement level during the next 30 sec. to 10 min. At termination of white noise,

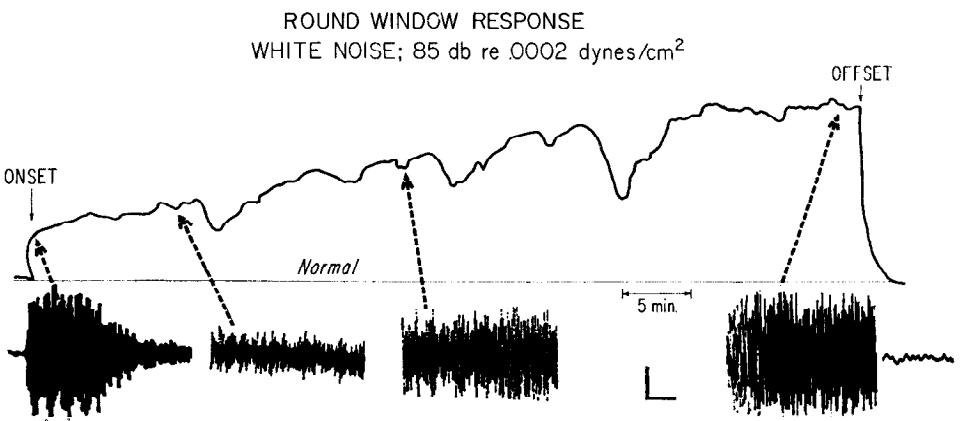

FIG. 1. Typical round-window response to prolonged white noise. Top: summated record; bottom: oscilloscopic photographs related to the summated response by arrows at onset and during noise. Time scale for summated response, 5 min. Time scale of oscilloscope records, 20 msec. Amplitude scale for oscilloscopic records, 200 μV.

summated round-window responses return promptly to pre-exposure level.

2. *Role of middle-ear muscles.* Activity of the middle-ear muscles is responsible for: *a*) the initial attentuation of round-window response shortly after onset of white noise; *b*) the gradual rise thereafter; and *c*) the intervening decreases associated with bodily movements.

The effects of severing the tendons of the middle-ear muscles are shown in Fig. 2, under "efferent mechanisms." With both tendons sectioned (three cats) the ipsilateral round-window responses are maximal at onset and do not show further rise with continuing exposure (Fig. 2*b*). (The small changes, ± 3 4 db, seen in the summated record reflect variations in sound field in different parts of the cage. Thus, the summated record for this animal is the same as that obtained by a moving microphone at the height of the cat's head.) The round-window response recorded from animals whose middle-ear-muscles are inactivated by Flaxedil is a plateau.

When the tendon of the tensor tympani is sectioned leaving the stapedius intact (five cats) animals attenuate round-window responses both at onset of

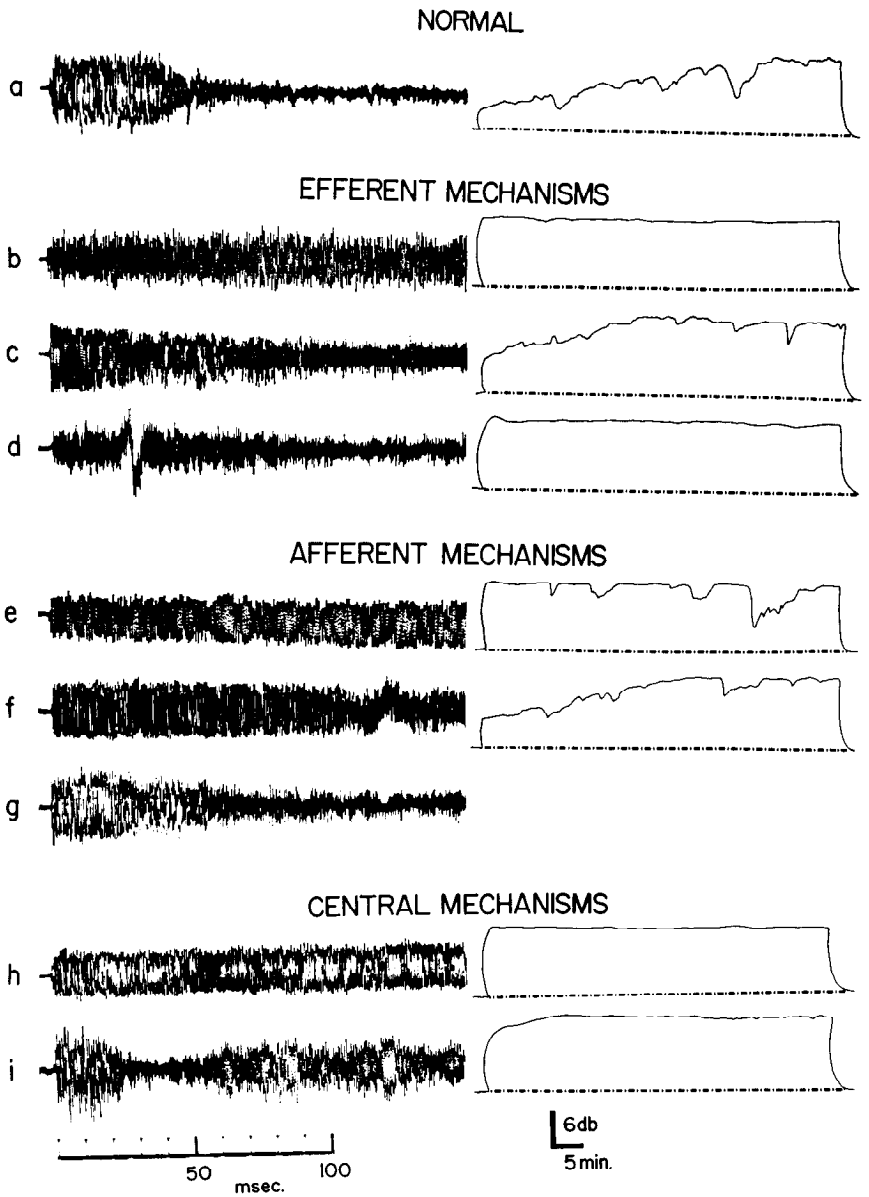

FIG. 2. Acoustic reflex responses. Left: round-window oscilloscopic responses at onset of white noise (85 db). Right: summated round-window response during 1 hour steady white noise. Comparisons are made between typical responses recorded from: (*a*) normal animal, and the effects of interfering with the efferent, afferent, and central acoustic reflex mechanisms. Efferent mechanisms: (*b*) animal with tendons of both middle-ear muscles sectioned; (*c*) animal with stapedius intact and tensor tympani tendon sectioned; (*d*) animal with tensor tympani intact and stapedius tendon sectioned. Afferent mechanisms: (*e*) animal with eighth nerves crushed bilaterally—cat behaviorally deaf; (*f*) animal with eighth nerve crushed unilaterally, responses recorded from operated side; (*g*) same animal as in *f* with eighth nerve crushed unilaterally, response recorded from unoperated side, summated record normal. Central mechanisms: (*h*) normal animal with intact middle-ear muscles during Nembutal anesthesia; (*i*) decerebrated and unanesthetized animal.

noise (4 12 db) and during bodily movement (up to 5 db) almost as well as normal animals (Fig. 2*c*). Summated activity rises to a stable plateau in 15–30 min., which is more rapid than normal. With stapedius sectioned, leaving the tensor tympani intact (four cats), round-window responses are attenuated only slightly at onset and during movements (Fig. 2*d*). In these animals onset attentuation is not always seen.

We conclude that the two intra-aural muscles act synergistically, their combined action producing a greater initial and more sustained attenuation of round-window responses than the sum of their individual actions.

3. *Acoustic reflex contractions of middle-ear muscles.* Middle-ear muscle activity at onset of sound is an acoustic reflex. An animal behaviorally deafened by crushing both eighth nerves (showing no neural components of round-window response) does not show initial attenuation of round-window response at onset of white noise (Fig. 2*e*). During continuing stimulation, responses do not gradually rise as in the normal animals. However, the transient round-window attenuation associated with bodily movements still occurs and is of slightly greater magnitude than normal, ranging up to 9 db.

Bilateral acoustic reflex contractions of the middle-ear muscles occur even though only one auditory nerve is left intact (five cats). In these animals, round-window responses differ on the side of eighth-nerve section as compered to the contralateral control; on the operated side, round-window responses are attenuated only after 70–300 msec. (Fig. 2*f*), about five times the latency of the control (Fig. 2*g*). Moreover, the amplitude of attenuation is about one-half that of the unoperated side, although falling within the upper part of normal range. These differences do not result from injury to the seventh cranial nerve supplying the stapedius muscle, as the middle-ear muscles contract equivalently on the two sides during bodily movements and vocalization. These results suggest that normal acoustic reflex functions of the middle-ear muscles depend on the intactness of sensory connections with the central nervous system. An analogous phenomenon may be the amaurotic mydriasis appearing after damage to one optic nerve. This is associated with the maintenance of normal pupillary dynamics during convergence (5).

The acoustic reflex contraction is very sensitive to Nembutal anesthesia. It is abolished at levels of anesthesia when other cranial nerve reflexes, such as the corneal and pinna reflexes, persist. Anesthetized animals show maximal round-window responses at onset of sound (Fig. 2*h*). There is no rise during continuing sound exposure and the transient decreases associated with movement are absent. During 2 hours of continuing exposure, summated responses may decrease slightly (about 1 db).

Intercollicular decerebration (ten cats), in the absence of anesthesia, is associated with profound alterations in round-window responses (Fig. 2*i*). The initial response attenuation of the round window is more rapid than in normal cats, developing within 20–30 msec. following onset of sound. (Normal

range is 30 75 msec.) Moreover, in decerebrated cats, this attenuation is not sustained for more than 100 msec. Summated responses rise within 10 min. to a stable plateau.

4. *Modifiability of acoustic-reflex contractions.* The level of arousal does not influence amplitude of round-window responses during steady sound stimulation. Summated activity remains similar in appearance whether the animal sleeps, sits quietly while awake, or gazes at a flashing light. When bodily movements occur during the initial orienting response to a novel stimulus, round-window responses decrease in amplitude in a manner not distinguishable from the effects of similar bodily movements occurring at other times.

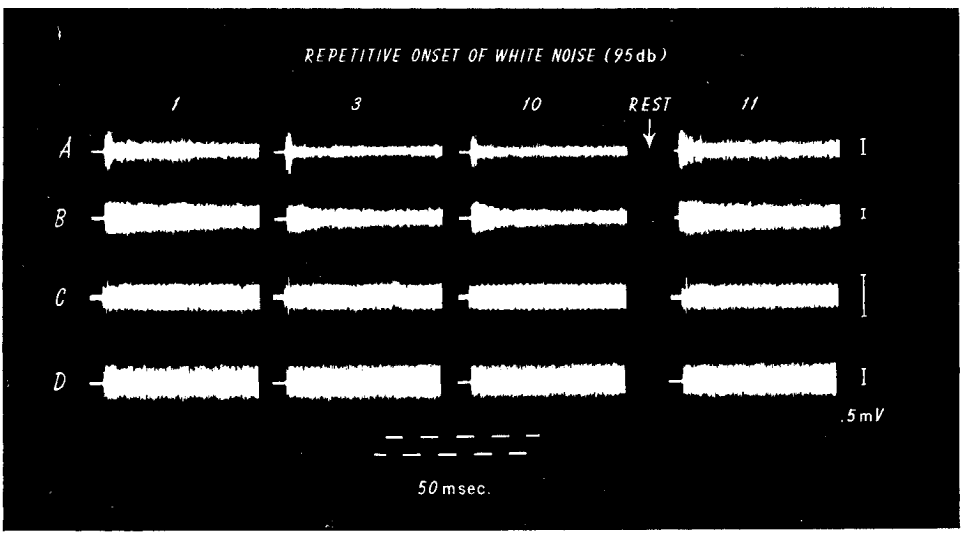

FIG. 3. Round-window responses at onset of repetitive white noise bursts. Bursts are 500 msec. in duration, repeated at 2-sec. intervals. Two minutes rest between tenth and eleventh trials. Comparisons are made between *A*, normal animal; *B*, animal with eighth nerve crushed unilaterally—recording from operated side; *C*, animal with tendons of both middle-ear muscles sectioned, but with nerves intact bilaterally; and *D*, normal animal under Nembutal anesthesia.

Round-window responses change during repetitive bursts of loud white noise. Figure 3*A* shows the results obtained in a normal animal when exposed to a 500-msec. burst of 90-db white noise repeated every 2 sec. for a total of 10 bursts. Cochlear microphonics recorded during a succession of sound bursts tend to become *1*) smaller in amplitude at onset, *2*) attenuated more rapidly, and *3*) attenuated to a greater degree than initially. These changes are temporary, as the response to the eleventh sound burst, given after an interval of 2 min. of rest, is similar to the first. With repetitive low-intensity (60 db) stimulation this effect is much smaller or absent. Animals with one eighth nerve crushed still show these dynamic changes bilaterally

(Fig. 3*B*). However, round-window responses on the crushed side show a longer latency and attenuate less with each sound burst than on the normal side. Animals with the tendons of both middle-ear muscles sectioned show no such dynamic changes in round-window response with repetitive exposure to a loud sound stimulus (Fig. 3*C*). This indicates that activity of efferent eighth-nerve fibers projecting to the cochlea do not seem to play a role in modifying round-window responses to such repetitive stimuli. Finally, this attenuation does not develop in intact animals anesthetized with Nembutal (Fig. 3*D*).

Acoustic reflex contractions can be modified by prior acoustic experience. Figure 4*A* shows a control round-window response to a 60-db white noise of brief duration. At the arrow the animal is exposed to a single burst of

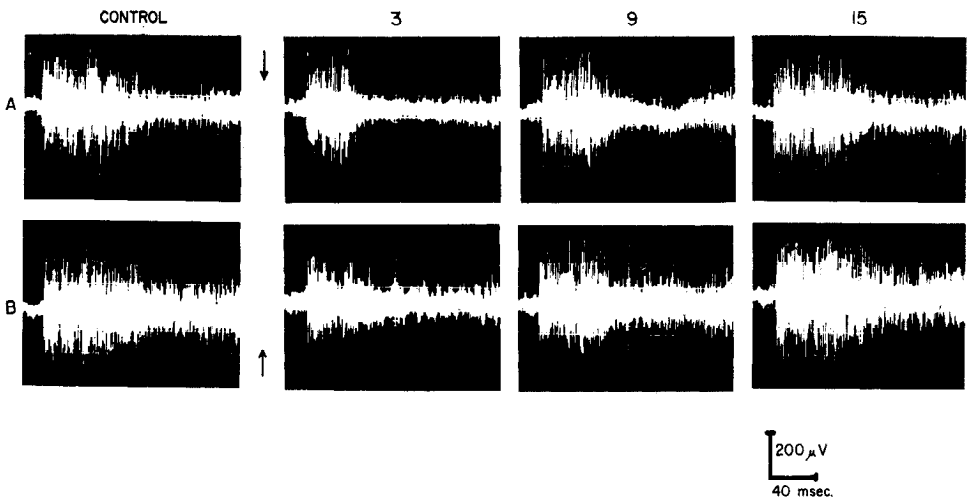

FIG. 4. Modification of round-window responses to low-intensity noise (60 db). *Series A* and *B* show round-window responses at the onset of low-intensity white noise. At the arrow in *series A* one short loud burst of white noise (300 msec., 95 db) is given; in *series B* the low-intensity sound is followed 500 msec. later by an electric shock to the animal's body. Two such conditioning trials are given. The numerals represent trials after the procedures at the arrows.

white noise at 95 db, lasting 300 msec. Subsequent round-window responses to the initial low-intensity sound are attenuated more rapidly and to a greater extent than in the control. These effects gradually disappear during repeated trials. They disappear more slowly if the trials are infrequent; but single test trials show that the effects entirely disappear within a few minutes.

Similar modifications of responses to low-intensity sounds are induced by associating the low-intensity sounds with electric shocks delivered across the body of the animal 500 msec. following sound onset. Two such pairings are shown at the arrow in Fig. 4*B*. During subsequent exposures to low-intensity

sounds, without associated shock, the altered round-window responses gradually return to normal. If a light flash is used in place of sound as the conditional stimulus for the same electric shock, round-window responses are unaffected. The animals indicate recognition of the association of the light with the noxious stimulus, but still do not show attenuation of acoustic responses following the conditioning shock-light trials.

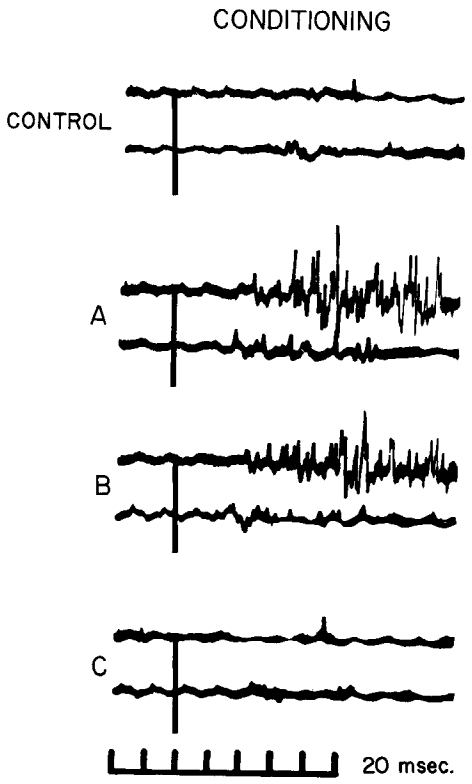

FIG. 5. Modifications of middle-ear muscle responses to low-intensity white noise (50 db). Vertical lines in control, *A*, and *B* indicate onset of low-intensity sound. Vertical line in *C* indicates onset of light flash in the absence of any sound stimuli. Stapedius EMG, upper trace; tensor tympani EMG, lower trace. Control responses show absent EMG to low-intensity sound. *A*, responses several seconds after one short burst of loud white noise (300 msec., 95 db); *B*, responses after a single pairing of the low-intensity noise with an electric shock (similar procedure as in Fig. 4); *C*, responses after several pairings of light flash with the same electric shock.

Electromyographic studies from animals exposed to even lower intensity sound (50 db) as a test stimulus are illustrated in Fig. 5. The control shows low-amplitude electromyographic activity following sound stimulation. However, this same intensity noise yields higher amplitude electromyographic activity following a single loud noise (*A*) and following a single pairing with shock (*B*). There is no such dynamic change when the shock is preceded by a light flash (*C*) instead of a sound stimulus.

5. *Electromyographic studies.* Acoustic reflex middle-ear muscle activity is initiated in the stapedius muscle at lower sound intensities and at shorter latencies than in the tensor tympani (Fig. 6). In both muscles the latency of response decreases with increasing sound intensity. Lorente de Nó demonstrated such a reduction in latency of ergogram from the tensor tympani of

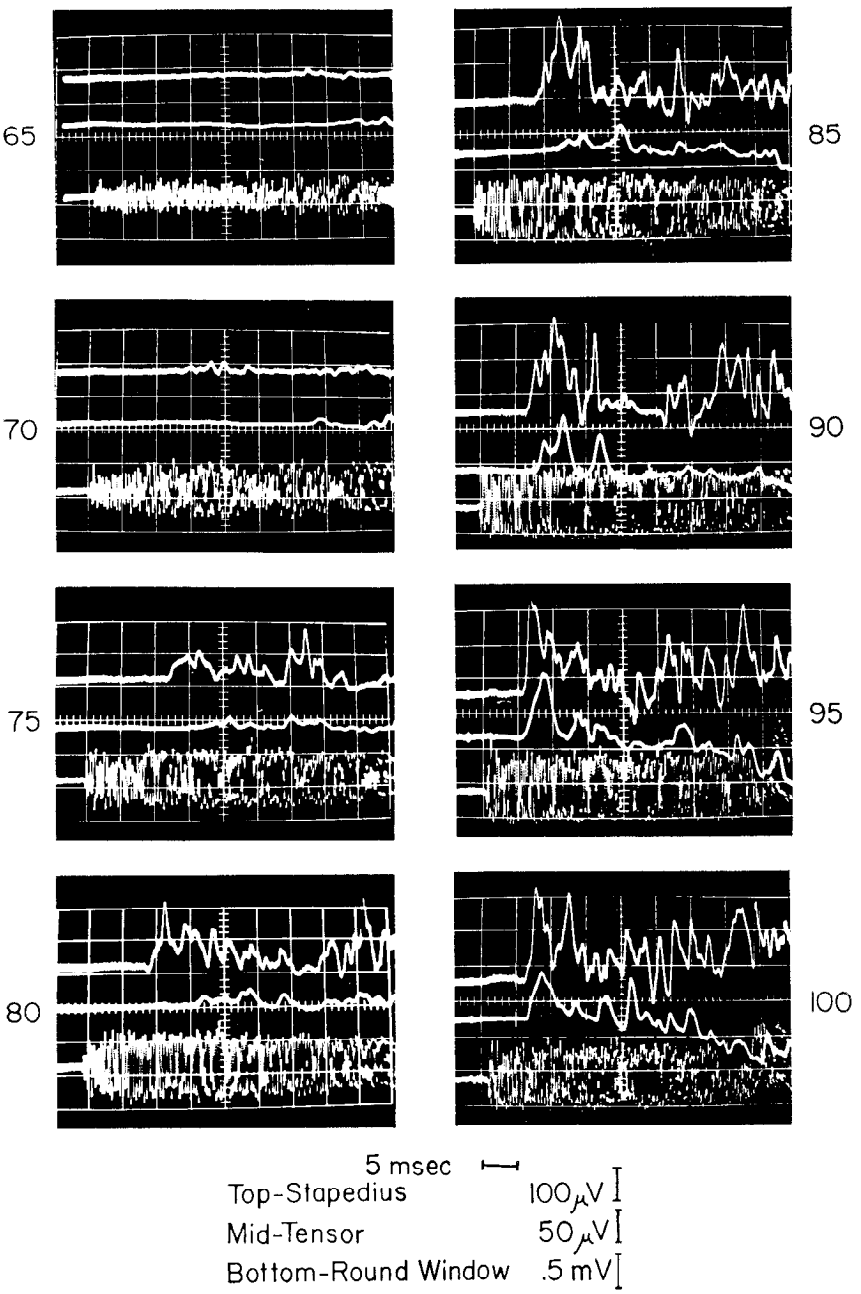

FIG. 6. Middle ear responses at onset of different intensities of white noise. Intensity of noise in db re .0002 dynes/cm² indicated by numerals. Top traces, electromyogram of stapedius. Middle traces, electromyogram of tensor tympani. Bottom traces, round-window responses. All recordings made from the same ear.

rabbits (20). The latency measurements are made from the onset of cochlear microphonics on the same side. Averages and ranges for at least 25 trials at each intensity are:

Sound Intensity, db	Average Latency, msec.	Range, msec.
	Stapedius Muscle	
75	13.3	8–22
80	11.8	7.5–19
85	9.1	6–12.5
90	6.7	5–9
95	6.2	4.5–7.5
100	5.8	4–7
	Tensor Muscle	
75	16.6	12.5–24.5
80	12.4	8.5–17
85	10.0	7–21
90	7.1	5–13.5
95	6.6	5–8.5
100	5.9	5–7.5

For the lowest intensities there were very few responses: Stapedius: 70 db, 16.7 msec. (10–29 msec.) n = 11; 65 db, 33 msec. (19–42 msec.) n = 7. Tensor: 70 db, 28.5 msec. (22–39 msec.) n = 6; 65 db, 42 msec. n = 1.

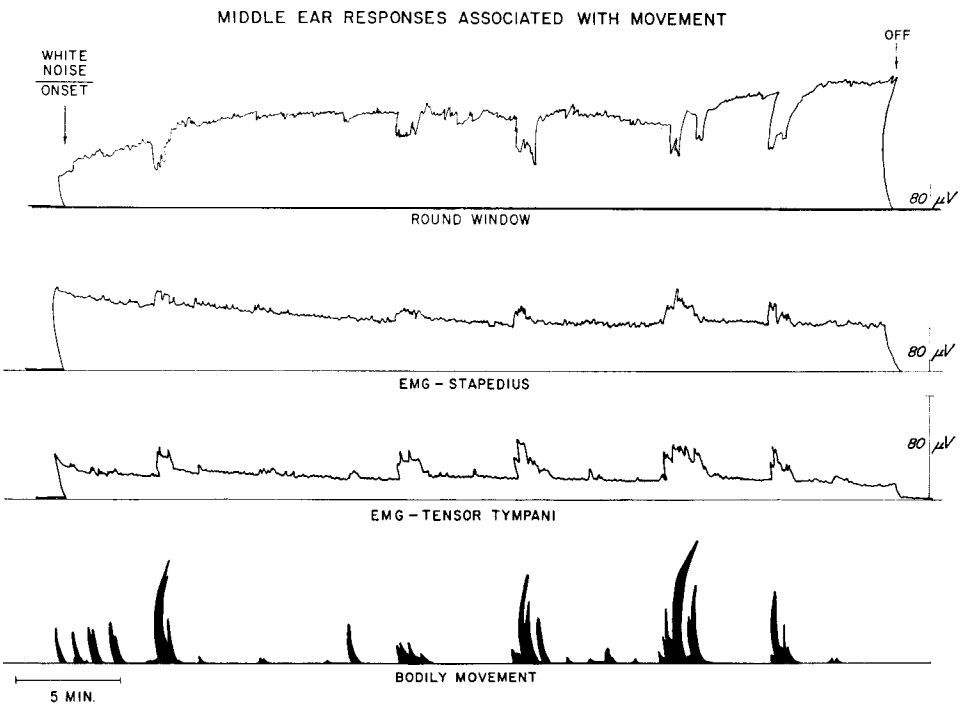

FIG. 7. Summated responses during prolonged white noise (85 db). Round-window, first trace; stapedius, second trace; tensor tympani, third trace; bodily movement, fourth trace.

Summated recordings of the tympanic muscles, round-window and bodily movements of an animal exposed to prolonged white noise are shown in Fig. 7. Photographs of oscilloscopic tracings taken at a much faster time base are shown in Fig. 8. Round-window activity approximately mirrors summated intra-aural muscle activity. At onset of white noise there is an abrupt rise in activity recorded from all middle ear electrodes. With continuing exposure, ear muscle activity decreases rapidly in amplitude during the first few minutes and gradually thereafter (Fig. 7 *B* and *C*). Tensor tympani responses decline to a plateau within a short time (ranging between 4 18 min. in four cats), while stapedius activity stabilizes only after a longer period (ranging from 15 75 min.) The transient decreases of round-window activity associated with bodily movements are paralleled by increasing

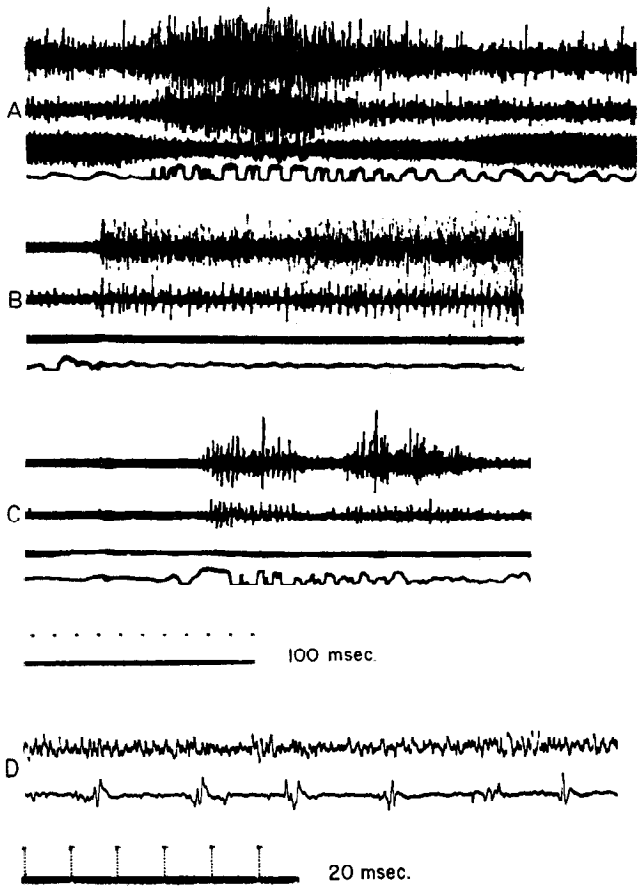

FIG. 8. Nonacoustic middle ear responses. *A:* responses with rapid movement during continuous stimulation with 85-db noise; *B:* responses to mechanical stimulation of external auditory canal; *C:* responses with rapid movement, no noise. Stapedius EMG, first trace; tensor tympani EMG, second trace; round-window response, third trace; piezoelectric response indicating movements, fourth trace; *D:* response during slow movement (at five times faster time base); stapedius, upper trace— tensor tympani, lower trace.

electromyographic responses (Fig. 7; Fig. 8*A*). Attentive behavior to a novel flashing light, in the absence of bodily movement, has no effect on intra-aural muscle activity or on round-window responses.

6. *Nonacoustic middle-ear muscle activity.* Many stimuli initiate intra-aural muscle activity on a nonacoustic basis. Cutaneous stimulation of the external auditory canal initiates contraction of the tympanic muscles bilaterally (Fig. 8*B*). Bodily movements are associated with contraction of both the tensor tympani and stapedius muscles. This middle-ear muscle activity is roughly proportional to the extent of movement; large rapid movements cause high amplitude, rapid activity in both ear muscles (Fig.

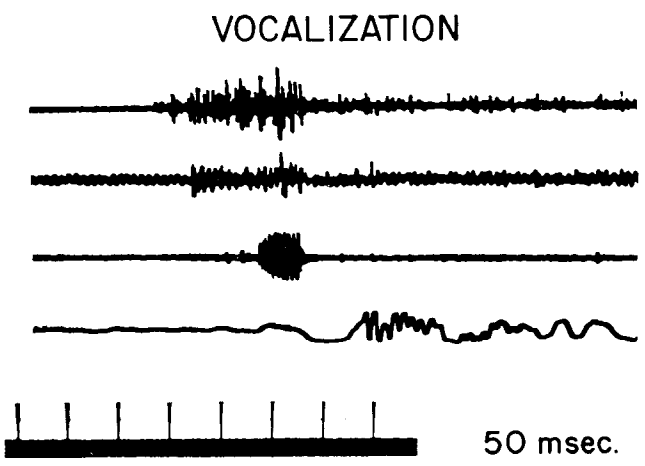

FIG. 9. Middle ear responses with vocalization. Stapedius EMG, first trace; tensor tympani EMG, second trace; round-window response, third trace; piezoelectric response indicating movement, fourth trace.

8*C*). During slow circling movements, large rhythmic potentials are often recorded from the tensor tympani, with relatively little activity from the stapedius (Fig. 8*D*). Such regular tensor tympani activity may also occur during tapping of the facial area, and occasionally spontaneously, without any apparent eliciting stimulus. This type of rhythmic firing is seen only in the tensor; stapedius activity is characteristically asynchronous.

Vocalization is associated with ear muscle activity (Fig. 9). The muscle action potentials precede by 75–500 msec. the onset of cochlear microphonics as recorded at the round window. Middle-ear muscle activity continues during vocalization, generally stopping simultaneously with the termination of the round-window response.

Crushing a single eighth nerve increases the latency of ipsilateral middle-ear muscle response to external sounds (Fig. 10*A*, upper beams). The cochlear microphonics appear simultaneously at both round windows, but the action potential of the stapedius on the operated side has a 15 msec. longer latency than the normal side (Fig. 10*A*, lower beams). These responses,

recorded at a slower time base, are shown in Fig. 10*B*. However, in the same animal, its own vocalization elicits simultaneous bilateral activity in the stapedius muscles prior to round-window responses (Fig. 10*C*).

7. *Developmental studies.* During the first 3 weeks of life the amplitude of round-window responses during loud white noise (100–105 db) is lower than that evoked by the kitten's own vocalization and much less than that recorded from adult cats. Recordings from two 3-day-old kittens exhibit no detectible response to loud environmental sounds, whereas responses to vocalization are apparent. Animals implanted when 3 days of age do not show acoustic reflex attenuation of round-window responses until the second to third weeks of life. The earliest such attenuation was obtained in a 15-day-old kitten. Attenuation of round-window responses during bodily movements and vocalization precedes the appearance of acoustic reflex attenuation by at least several days.

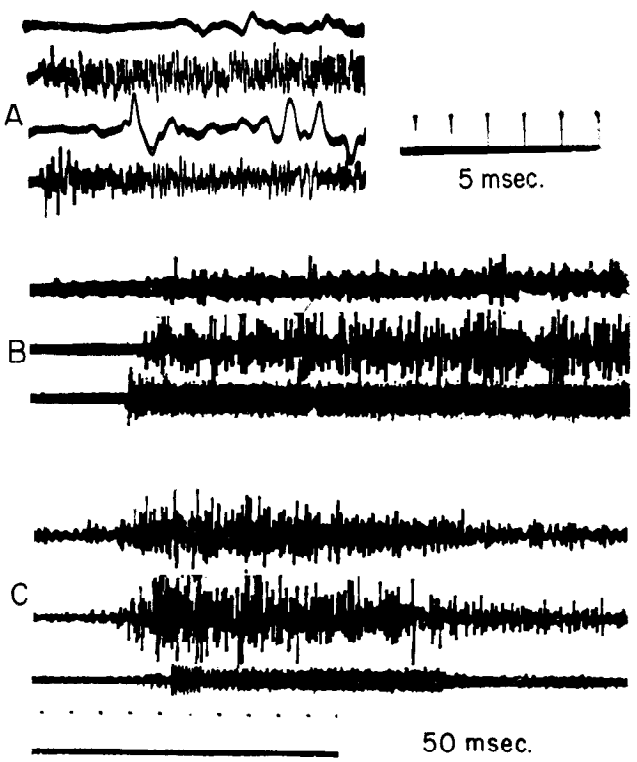

FIG. 10. Middle ear responses in animal with unilateral eighth nerve interruption. (*A*) responses at onset of white noise (85 db): operated side; stapedius EMG, first trace round-window, second trace. Normal side; stapedius EMG, third trace round-window, fourth trace. (*B*) response at onset of white noise: operated side; stapedius EMG, first trace. Normal side; stapedius EMG, second trace—round-window, third trace. (*C*) responses associated with vocalization. Order of traces as in *B*. Time scale for *A*: 5 msec /pulse. Time scale for *B* and *C*: 50 msec/pulse.

Discussion

1. *Acoustic middle-ear muscle activity.* Both middle-ear muscles contract shortly after onset of sound, and the latency of contraction decreases with increasing sound intensity. These contractions constitute the motor limb of an acoustic reflex and do not occur in deafened animals. The intensity of sounds reaching the cochlea is attenuated as a consequence of this middle-ear muscle activity.

In conscious animals, middle-ear muscle activity is maximal at onset of sound and decreases gradually during the next 30–90 min. of continuing stimulation. This muscle relaxation is not due to fatigue, since powerful contractions are reinstated promptly with either an increase or a decrease of sound intensity, or with bodily movements. As the middle-ear muscles relax, gradually increasing electrical responses are recorded from the round window and from the brain-stem stations along the auditory pathway (33).

Following discontinuation of prolonged sound stimulation, abrupt, profound, long-lasting, gradually reversible changes in electrical activity to below pre-exposure levels appear throughout all central subthalamic auditory stations. These aftereffects are due to central processes, inasmuch as they persist following section of the middle-ear muscles (33). These central processes affect the lower stations of the auditory pathway to a gradually increasing degree during prolonged sound stimulation, and become manifest in the form of aftereffects when the sound stimulation is discontinued. Both peripheral neuromuscular as well as neuronal control mechanisms confined to the central nervous system evidently modify the auditory pathway. These control mechanisms are probably interdependent.

The peripheral neuromuscular control is obviously capable of responding rapidly to acoustic transients, but exposure to prolonged sound stimulation is a prerequisite for the appearance of the internal neuronal effects. The neuromuscular mechanism provides rapid accommodation to abrupt environmental changes, whereas the central neuronal effects operate during longer lasting states. During prolonged sound there appears to be a shift from control via middle-ear muscles to control operating along the central auditory pathway. The decrement of one and increment of the other appear to be parallel in time.

This shift from a peripheral neuromuscular control to a completely central control may represent a metabolically more economical means for the organism to adjust to new, steady environmental states. Such a shift would have the further advantage of freeing the peripheral neuromuscular mechanism for its full range of activity against any new fluctuations in the level of background sound.

2. *Modifiability of acoustic middle-ear muscle activity.* In waking animals the threshold, latency, and strength of middle-ear muscle contractions can be temporarily modified by prior auditory exposure or by associating sounds with other significant stimuli. In the present experiments, a single brief,

loud, white noise (95 db) consistently induced a temporary exaggeration of middle-ear muscle response to subsequent low-intensity test sounds; cochlear microphonics were attenuated more rapidly and completely. Extremely faint sounds, which previously had been ineffectual in eliciting middle-ear muscle reactions were made temporarily effectual. Other workers have demonstrated the importance of prolonged auditory experiences in modifying middle-ear muscle contractions in both the cat (31) and in man (23). Our evidence indicates that in the cat central processes regulating acoustic reflex responses of the middle-ear muscles can be modified as well by single, brief sound exposures.

The importance of the central regulation of acoustic reflex responses is made clear also from similar modifications of ear muscle activity which develop if sounds are used as a warning signal for electric shock. These contractions are specifically acoustic responses because no middle-ear muscle activity develops when a light is used as the conditional stimulus for the same electric shock.

There is extensive evidence that the amplitude of click-evoked electrical responses can be altered by prolonged repetition of the stimuli or by associating the stimulus with novel or negatively re-enforcing events (4, 9, 10, 21, 28). The changes in click-evoked responses have been attributed to central processes regulating attention, habituation, and affective response to sounds. In the present experiments, modification of middle-ear muscle action by conditioning indicates the plasticity of middle-ear muscle responses. It may, however, be injudicious to assume that middle-ear muscle activity attenuating sound transmission through the middle ear necessarily effects a reduction in auditory "information."

3. *Nonacoustic middle-ear muscle activity.* Yawning (17), swallowing (35), and "defensive movement" in response to painful stimuli (16), and cutaneous stimulation of the external auditory canal, pinna, face (19), and neck (18) all are associated with middle-ear muscle activity. The stimuli initiating contraction are nonacoustic in nature and we find them to be still effective in deafened animals. Von Békésy suggests that the physical characteristics of the middle ear have evolved to permit the cochlea to be maximally sensitive to environmental sounds and minimally sensitive to self-produced sounds (3). Our finding that middle-ear muscle contractions occur in advance of the sounds of vocalization, thus attenuating cochlear responses, is consistent with Von Békésy's conception. However, vocalization is a complex motor act coordinating muscles innervated by cranial nerves five, seven, nine, ten, eleven, and twelve. Since the intra-aural muscles are innervated by two of these cranial nerves (five and seven) their activity in association with vocalization, as well as during yawning and swallowing, may represent nothing more than associated movements initiated during a complex motor performance and may not be auditory contributions per se. This interpretation is somewhat favored by the observation that these contractions persist in deafened animals.

Hugelin *et al.* induced middle-ear muscle activity in encéphale isolé animals by stimulation of the brain-stem reticular formation (14, 15). They interpret these contractions to be components of a generalized body motor response. Middle-ear muscle activity observed in our experiments to be associated with naturally occurring movements may be components of such a generalized body motor activity. These mechanisms appear to mature early in development because we find middle-ear muscle contractions appearing during bodily movements in kittens several days before the appearance of sound-induced contractions. Tsukamoto found that oscillations of the labyrinthine fluid in rabbits are reduced during contractions of the middle-ear muscles (34). We find that the amplitude of middle-ear muscle activity increases with more extensive bodily movements. During bodily movement this muscle-induced reduction of fluid oscillation may modify labyrinthine responsiveness to acceleration and spatial change.

Click-evoked cochlear nucleus responses have been reported to decrease in amplitude during presentation of a novel stimulus (11), and cochlear microphonics to decrease during conditioning to light stimuli (32). This kind of attenuation may turn out to reflect middle-ear muscle contractions imposed as a consequence of orienting movements rather than as a result of attentive processes per se. Experiments done in this laboratory indicate that in conscious cats bodily movements are associated with decreases in the amplitude of click-evoked responses all along the auditory pathway. However, attention of the animal to a novel flashing light in the absence of movement is not associated with noticeable attenuation of evoked responses (A. Starr, manuscript in preparation).

4. *Relation of middle-ear muscle activity and perception.* The amplitude of muscle-induced changes in round-window responses, in cats exposed to steady sound, range up to 18 db. We would expect similar dynamic changes to occur in man. Common sense experience indicates that we usually perceive no change in the intensity of steady sounds. However, shifts in cochlear responses of 18 db, resulting from modifications in the actual intensity of sounds similar to those used in the present experiment, are readily distinguishable. It may be that the central processes shown to control middle-ear muscle actions are integrated into sensory mechanisms in such a way as to enable a perceptual differentiation between internal and external modifications of cochlear activity and to compensate at perceptual levels for the internally controlled changes.

Perceptual disturbances are produced on interrupting the relationship between ear muscle activity and central control mechanisms. Patients with an acute paralysis of the stapedius and other muscles innervated by the seventh cranial nerve, as in Bell's palsy, often complain of hyperacusis (25). Sounds are heard more vividly and patients often find this uncomfortable. A similar complaint, that sounds at conversational intensities are uncomfortable, is occasionally volunteered by patients during exacerbation of myasthenia gravis (E. B. Schlesinger, personal communication). The symp-

tom is promptly relieved by giving prostigmine. These acoustic disturbances may result from a noncorrespondence between what one hears and what one expects to hear, based on previous central control experience with a properly functioning stapedius muscle. This interpretation would be consistent with the evidence from Ames' demonstrations which indicate the overriding importance in perception of factors of past experience and expectation (2).

Lorento de Nó (20) suggested that further understanding of the mechanisms underlying the acoustic reflex would be valuable in extending our understanding of general features of neuronal integration. We find middle-ear muscle activity to be related not only to the physical characteristics of the sound stimuli, but also to dynamic central processes affected by bodily movement, vocalization and prior acoustic experience. These findings suggest that actions of the tympanic muscles are not merely simple reflex actions protecting the inner ear from acoustic damage, but complex sensorimotor coordinations influenced by central nervous mechanisms which undoubtedly play a role in auditory perception.

SUMMARY

Recordings have been made in waking and unrestrained cats of cochlear round-window responses and of electromyograms from the stapedius and tensor tympani muscles during sound stimulation. Middle-ear muscle acoustic reflex contractions have been analyzed by selectively interfering with efferent, afferent, and central components of this reflex arc. New contributions to middle-ear muscle studies include analysis of: *1*) the gradual relaxation of middle-ear muscles during the prolonged sound stimulation, *2*) the contraction of middle-ear muscles during bodily movements and vocalization, *3*) the modification of middle-ear muscle reflexes by previous experience with loud noise, *4*) the modification of middle-ear muscle activity by associating test sound signals with electric shock, and *5*) the modification of middle-ear muscle activity by truncation of the central nervous system.

Ontogenetic studies show that contraction of the middle-ear muscles to nonacoustic factors precede the appearance of the acoustic reflex.

Results indicate that middle-ear muscle activities are not solely determined by the physical characteristics of sound stimuli but rather they appear to be governed by a wide variety of dynamic central nervous system processes.

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