The Middle-Ear Muscles

Tiny muscles behind the eardrum contract involuntarily when a person vocalizes or is exposed to a loud noise. This neuromuscular control system prevents sensory overload and enhances sound discrimination

by Erik Borg and S. Allen Counter

Modern industry has produced a noisy world. The din of jackhammers, the whine of jet engines and the blare of amplified electric guitars have become all too commonplace. It was therefore considerate of nature to have equipped the human ear with a rather sophisticated noise-reduction system: two small muscles that are attached to the ossicles, the tiny bones that connect the eardrum to the cochlea (the structure that houses the sound-receptor cells). When the muscles contract, they dampen the vibrations of the ossicles, thereby reducing the acoustic signal that ultimately reaches the inner ear.

Although they are skeletal muscles (in fact they are the smallest skeletal muscles in the human body), the middle-ear muscles are not under voluntary control. They contract reflexively about a tenth of a second after one or both ears are exposed to loud external sounds. Indeed, the characteristics of the reflex have become so well known that deviations from the normal response serve as a basis for diagnosing various hearing disorders and neurological conditions.

The muscles of the middle ear contract not only in response to loud external sounds but also immediately before a person vocalizes. This prevocalization reflex operates even when one speaks, sings or cries as softly as possible. Yet most evidence suggests that it is meant to protect the inner ear from the fatigue, interference and potential injury caused by one's own louder utterances, which can result in high sound levels in one's head. The shouting and wailing of children or babies, for example, can reach their own ears with the same intensity as the sound of a train passing nearby.

The middle-ear muscles do more than just indiscriminately attenuate internal or loud external sounds in humans. The muscles muffle primarily a loud sound's lower frequencies, which tend to overpower its higher frequencies. The net result of this frequency selectivity is to improve hearing—particularly of those sounds that contain many high-frequency components, such as human speech. In fact, the middle-ear muscles are what enables one to hear other people talking even while one is speaking.

Perceived sounds—regardless of their source—are air-pressure waves that have been funneled to the tympanic membrane, or eardrum, causing it to vibrate. The vibrations are transmitted through the three ossicles in the middle ear (the malleus, incus and stapes) to the cochlea. The middle-ear mechanism—the eardrum and ossicle linkage—serves to convert the movements of low-density air into analogous movements of the higher-density fluid in the cochlea. The movements of the fluid are transmitted to the stereocilia: fine, hairlike protrusions of receptor cells on the cochlea's basilar membrane. Mechanical forces on the stereocilia cause the cells to trigger electrical impulses in the auditory nerve that are then interpreted by the brain as sound.

Attached to the ossicles are the two middle-ear muscles: the tensor tympani and the stapedius [*see illustration on opposite page*]. The tensor tympani is connected to the neck of the malleus and is anchored in the wall of the eustachian tube (a ventilating tube connecting the throat, nasopharynx and middle ear). The stapedius originates in the wall of the middle-ear cavity and ends at the neck of the stapes, near its articulation point with the incus. The basic anatomy of the middle-ear muscles was described as early as 1562, by Bartolomaeus Eustachius (for whom the eustachian tube is named). Yet the function of the muscles in human hearing was a subject of speculation until this century, when laboratory experiments on animals and clinical observation made a comparative analysis of their physiology possible.

The middle-ear-muscle system is found in all classes of vertebrates, but it has distinctive features in certain species. In some species of frogs, for example, the hearing organ contains only a single ossicle that has a stapediuslike muscle attached to it. It is interesting to note that those frog species without a muscle or an ossicle in the middle ear tend not to vocalize.

Among lower vertebrates, birds possess the most elaborate systems for hearing and sound communication. In each ear they have a stapedius analogue, which is attached to both the tympanic membrane and a single ossicle, the columella. Because a bird's stapedius muscle lies mainly outside the middle-ear cavity, it can be studied more readily than the stapedius of mammals without damaging the delicate middle-ear structures.

We have worked with common domestic fowl, such as chickens, in a series of experiments on the physiology of the stapedius at the Karolinska Institute in Stockholm and at Harvard University. By attaching a strain gauge to the tendon of a bird's stapedius and then stimulating the muscle electrical-

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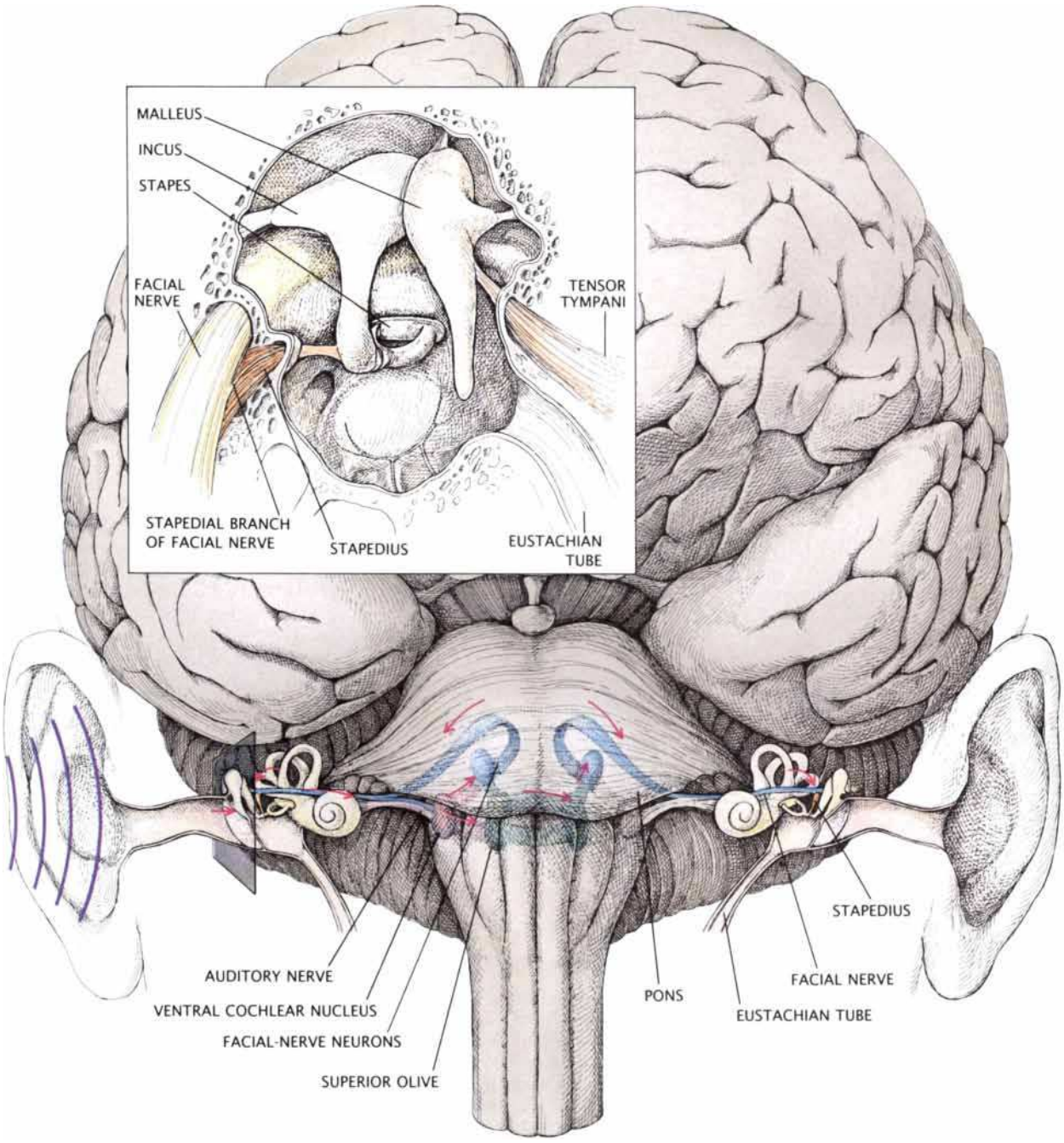
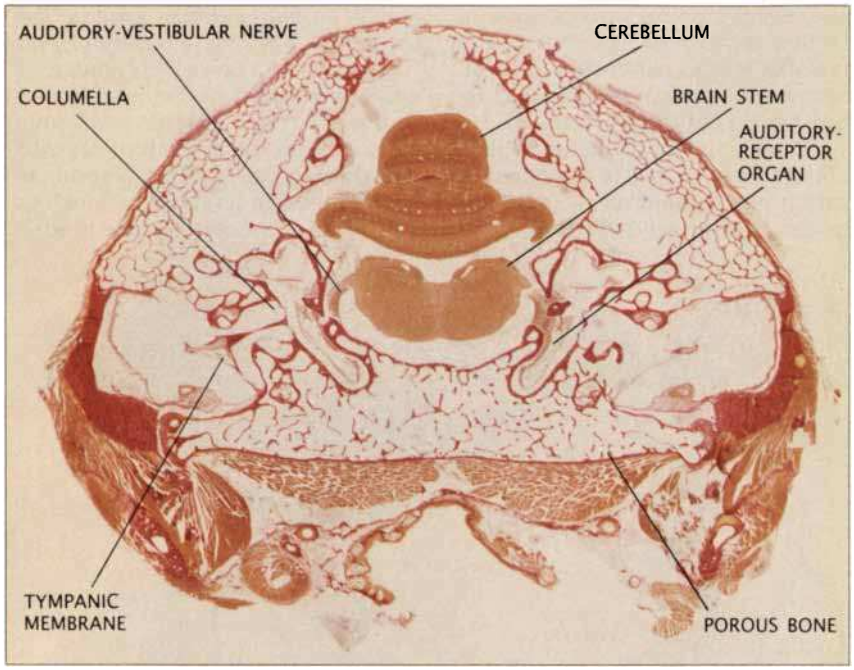
ly, we found that the stapedius was capable of contracting at rates in excess of 100 times a second.

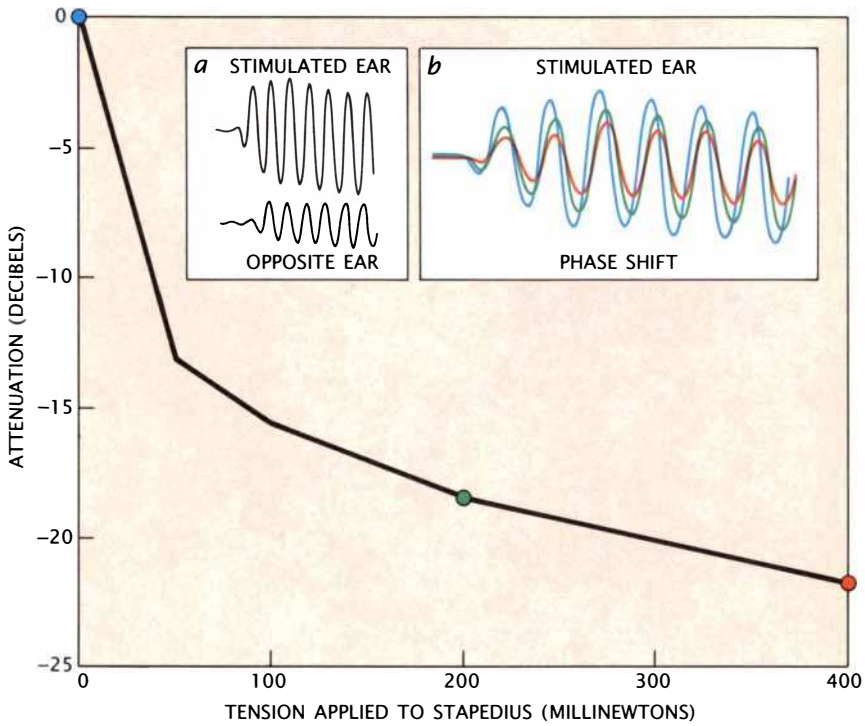
The muscle's inherent capacity for quick response and fatigue resistance is also evident from the microscopic appearance of its fibers. Electron micrographs show that the fibers contain abundant mitochondria (which provide energy), dense sarcoplasmic reticulum (which releases the calcium ions that trigger contraction) and numerous transverse tubules for the transmission of calcium ions.

Although the stapedius muscle of a bird always contracts during vocalization and swallowing, it does not appear to contract reflexively in response to loud external sounds. The primary role of the muscle in birds, then, seems to be the prevention of sensory overload of the auditory receptors during the birds' own loud cries. Indeed, the screech of seagulls and the crow of cocks can result in sound-pressure levels of as much as 130 decibels (measured at the head)—



MIDDLE-EAR MUSCLES (the stapedius and the tensor tympani) are seen in a view down an ear canal from which the eardrum has been removed (*inset*). When the muscles contract, they dampen the vibrations of the ossicles (the malleus, incus and stapes): the tiny bones that connect the eardrum to the cochlea, which houses the auditory receptors. The net result is a reduction of the sound transmitted to the cochlea. A loud sound (*purple*) in one ear activates the stapedius muscles of both ears through a neuronal pathway (*blue, red and green*) that lies in the lower brain stem (*above*). The pathway includes the auditory nerve, the ventral cochlear nucleus, the trapezoid body (not shown), the superior olive and the facial nerve.





STRUCTURE OF THE AVIAN HEARING SYSTEM is laid bare in a frontal cross section of a bird cranium (*top*). Birds have a single middle-ear muscle, the stapedius (not shown), and a single ossicle, the columella. Birds also have a porous-bone channel that connects both middle-ear cavities, which allows sound literally to go in one ear and come out the other. As a result the eardrum in the far ear normally vibrates out of phase with the eardrum in the ear directly exposed to sound. The phase difference is clearly evident (*bottom*) in a side-by-side comparison of the two ears' cochlear microphonics (*a*): the electrical output of their receptor cells, which mimics their acoustic input (in this case, a pure tone of 800 hertz). Experiments have shown that as tension is applied to the stapedius the sound reaching the inner-ear receptors decreases in intensity (*black curve*). Because the stapedius contracts vigorously while a bird vocalizes, the muscle's main function is probably to attenuate the sound produced at the bird's inner ear by its own cries. Also, tensing of the stapedius shifts the phase of an incoming sound wave slightly, as shown by cochlear microphonics (*b*). The attenuation and phase shifting might help birds to localize sound sources.

about the level of noise produced by a jet engine 15 meters away.

Direct evidence for the self-protection hypothesis came from recordings of a bird's cochlear microphonics: the electrical output of the cochlear receptor cells, which parallels the acoustic input to the cells. When we applied small amounts of tension to the stapedius muscle as we exposed the ear to a test tone, we observed a significant reduction in the amplitude of the cochlear microphonics and hence of the amount of sound energy reaching the receptors of the inner ear. The sound attenuation caused by stapedius-muscle contractions was essentially equal at all frequencies in the bird's auditory spectrum.

Another interesting observation was that tension in the avian stapedius muscle shifts the cochlear microphonics phase—the pattern of peaks and troughs making up its waveform—slightly with respect to time. What purpose might such a phase shift have? The answer has to do with the air-filled intracranial passageway that connects ears in birds and allows sounds literally to go in one ear and out the other. A sound wave crossing from one side of the head acts on the other side's tympanic membrane from the inside, generating cochlear microphonics in that ear that are slightly attenuated and almost totally out of phase with the microphonics of the ear through which the sound wave entered. We hypothesize that the contractions of a bird's stapedius muscles might modulate the binaural amplitude and phase relations in a way that helps the bird to locate sound sources.

One of the most specialized middle-ear-muscle systems is seen in echolocating bats. Their powerful stapedius and tensor tympani muscles contract at very high repetition rates as the bats make the rapid-fire click sounds that are their hunting cry. During each click a bat's middle-ear muscles reach peak tension quickly, but they relax just as suddenly so that the bat's ears will be sensitive to the click's echo from potential prey. The contraction-relaxation cycle lasts for only a few milliseconds (thousandths of a second) and can be repeated more than 100 times a second as the bat closes in on its prey. O'Dell Williams Henson, Jr., now of the University of North Carolina at Chapel Hill and Nobuo Suga of Washington University in St. Louis have confirmed that the bat's middle-ear muscles contract reflexively several milliseconds before the start of vocalization and, when contracted, can dampen the sound energy

reaching the inner ear by more than 20 decibels.

Experiments on individuals who have perforated eardrums show that the middle-ear muscles are active during vocalization in human beings as well. By inserting a harmless needle electrode through the perforation and into the stapedius, one can make an electromyogram (EMG) of the muscle as the subject vocalizes. An EMG records the electrical activity of muscle fibers, which increases as the fibers contract. Such EMG's have revealed that the electrical activity of the stapedius begins just before the subject makes a vocal sound; the activity increases as the vocalization becomes louder. Presumably, then, the stapedius contracts automatically in the ears of both whispering mothers and crying babies—from the softest to the loudest vocalized sounds. The tensor tympani probably contracts before vocalization as well.

The middle-ear muscles can also become active spontaneously, in the absence of any sound, resulting in the perception of a repetitive and often bothersome noise. In addition, tactile or electrical stimulation of certain skin zones on the face and ear can elicit a contraction in the stapedius.

In contrast to birds, human beings and mammals in general appear to have another type of reflexive contraction of the middle-ear muscles—one evoked by loud external sounds. In most mammals both the tensor tympani and the stapedius muscles are subject to such a reflex, but in humans only the stapedius is. For that reason the middle-ear response to loud sounds in humans is called the acoustic stapedius reflex (ASR).

The ASR causes the stapes to move some 50 microns (millionths of a meter) from its resting position, thereby increasing the stiffness of the ligaments holding the ossicle, which in turn reduces sound transmission to the inner ear by 20 decibels or more. Like the pupillary reflex (the contraction of both pupils in response to light shone in one eye), the ASR is normally observed in both ears, even if only one is stimulated acoustically. The reflex is generally elicited by sounds that are between 80 and 90 decibels above a person's hearing threshold—about as loud as the sound of a noisy street.

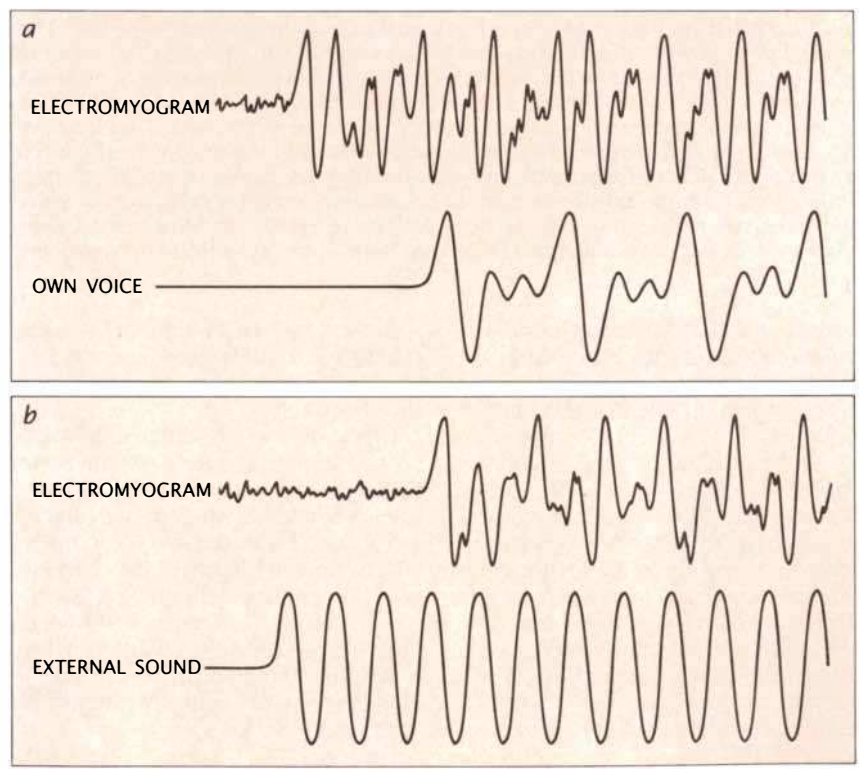
The importance of the middle-ear muscles in human hearing has been a matter of considerable debate over the years. Several single-function theories have been advanced, but the findings of many studies suggest that nature has been economical: it has given the muscles several separate but interrelated functions.

Animal-model studies of the middle-ear muscles conducted in the early 20th century by the Japanese otolaryngologist Toru Kato, along with more recent studies of the ASR by us and others, have made it fairly clear that the human stapedius is capable of protecting the inner-ear receptor cells from sustained, loud noise that might otherwise cause hearing loss—particularly in the frequency range that is most important for speech communication. Such hearing loss occurs when the inner ear suffers an acoustic battering that fractures stereocilia and thereby incapacitates receptor cells.

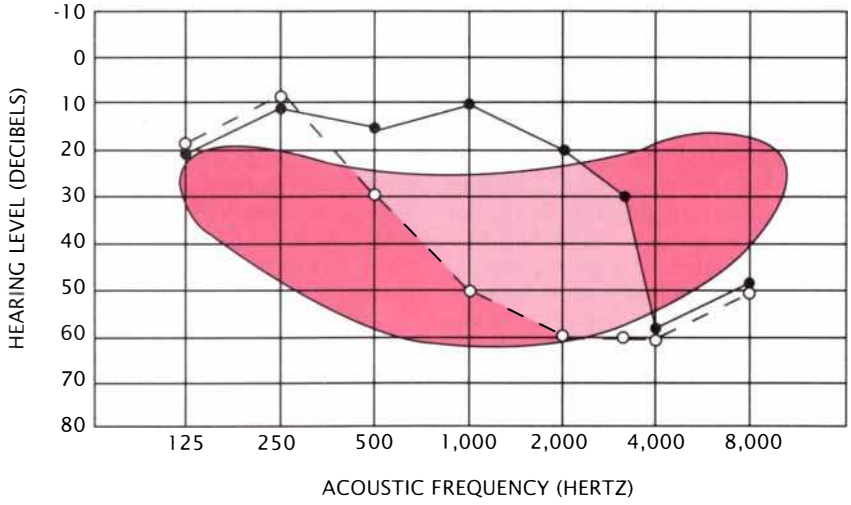
The stapedius cannot protect the inner ear from the damage that can be caused by an exceedingly sharp and intense sound pulse, however. The stapedius requires between 100 and 200 milliseconds to contract fully—a response time that is too slow to muffle, say, the sound of a gunshot before it reaches the inner-ear receptors. (Actually, one can probably reduce the risk of inner-ear damage from the bang of a gunshot by humming before shooting, since the middle-ear muscles are activated automatically during vocalization.) The stapedius can attenuate loud, abrupt sounds only if they come in quick succession, since the muscle then has a chance to build up tension. Apparently, the middle-ear muscles evolved to cope with the sounds of nature, such as thunder and loud animal sounds, which tend to rise slowly. Nevertheless, they do a remarkable job of protecting one's hearing from much of the noise of modern industrial society.

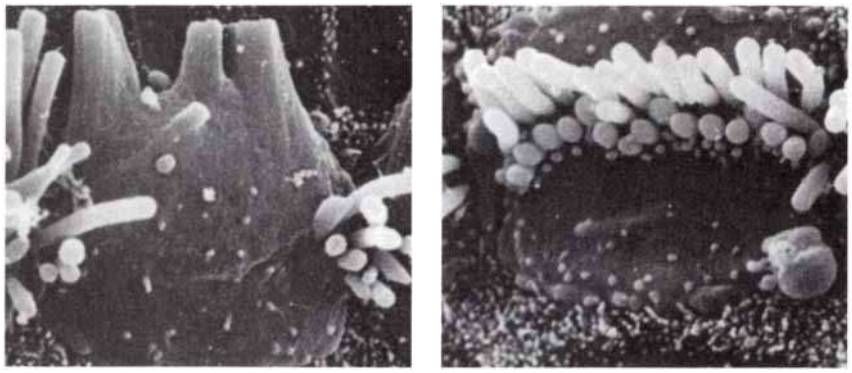
Other recent investigations have shown that the stapedius muscle has an even more sophisticated role in human communication. Audiologists have noted that people with nonfunctional stapedius muscles tended to have some difficulty in distinguishing speech sounds when loud background noise was present or when the sounds were amplified greatly. The lack of functional stapedius muscles appeared to have hampered the subjects' ability to discriminate spoken words. How could that be?

The answer becomes clear when one considers the way a sound wave is broken down into component frequencies in the long, spiral cavity of the cochlea. A sound wave propagating in the inner-ear fluid generates a traveling wave along the basilar membrane, which partitions the cochlear duct from beginning to end. Low-fre-



ELECTROMYOGRAM (a tracing of the electrical activity of muscle fibers) that is recorded in step with tracings of internal and external auditory stimuli shows that the human stapedius contracts involuntarily just before a subject vocalizes (*a*) and just after the subject is exposed to a loud sound (*b*). (The tracings are schematized.)





AUDIOGRAM (*top*) shows the clinical consequence of an inactive stapedius. The hearing of a person with a paralyzed stapedius who has had prolonged exposure to high levels of noise (*broken line*) is significantly worse than the hearing of a person in a similar auditory environment but who has functioning middle-ear muscles (*solid line*), particularly at frequencies ranging from 250 to 4,000 hertz. That frequency range (*light red*) includes many frequencies that form a part of normal speech (*red*). The physical cause of such hearing loss is the fracture of stereocilia (*bottom left*): the microscopic hairlike sensors of the auditory receptor cells. Normally the stereocilia are arranged in tapered bundles (*bottom right*). The micrographs were made by Berit Engström and Agneta Viberg of the Karolinska Institute in Stockholm.

quency sound components induce undulations in the membrane from the stapes all the way to the tip of the cochlear spiral, whereas the undulations of high-frequency components die out much more quickly and are confined to the area near the stapes. Because the low-frequency components dominate the undulations of the basilar membrane in the cochlea, low-pitched sounds of high intensity can drown out high-frequency sounds and even make them imperceptible.

Such "masking" of high-frequency sounds by low-frequency sounds is minimized by the ASR, since increasing the stiffness of the middle-ear linkage attenuates the low-frequency components of a complex sound more than it does the high-frequency components. Because many key speech sounds are generally high-pitched, the middle-ear muscles can actually enhance the perception of speech when they contract.

The ability of the stapedius muscle to maintain the ear's sensitivity to the frequencies encompassed by most speech sounds in spite of high sound levels was demonstrated experimentally by Roland Nilsson of the University of Gothenburg and John-Erik Zakrisson of Umeä University in Sweden. They showed that the ASR can improve the threshold for the detection of high-frequency sound in noise by as much as 50 decibels.

The stapedius also enhances one's ability to hear while speaking. A speaker's own ears are subjected to intense low-frequency vibrations that arise primarily from the enunciation of vowels. Fortunately, the prevocalization contraction of the middle-ear muscles prevents one's own speech from masking ambient high-frequency sounds. Indeed, the muscles are what makes it possible to hear soft sounds while one speaks.

The neural circuits that control the ASR and the prevocalization reflex are only now beginning to be understood. Modern techniques for the visualization of neurons in combination with the physiological studies have helped chart the intricate neuronal network that activates the middle-ear muscles.

The ASR relies on a complex pathway through several brain-stem nuclei (clusters of nerve cells) as well as the auditory and facial-nerve neurons [*see illustration on page 75*]. The primary neuronal pathway that controls the ASR in the ear being stimulated originates at the cochlear receptors, extends along the auditory nerve to the brain stem, where it includes parts of the ventral cochlear nucleus and the superior olive, and follows the facial nerve to its stapedial branch. There is evidence that the ASR is activated in the opposite ear by neurons from the ventral cochlear nucleus that communicate with the superior olive on the other side of the brain.

The close relation between the auditory neuronal pathway (which processes the signals from the receptor cells in the cochlea) and the motor neurons controlling the stapedius is particularly evident in experiments in which the stapedius is injected with labeling agents such as stains or certain viruses. These agents trace the neurons that make up the ASR pathway from the muscle to the brain stem. Such experiments have demonstrated that the cell bodies of the neurons innervating the stapedius in mammals (which exhibit the ASR) lie at the margin of the facial-nerve nucleus, very near the superior olive and the ascending auditory tract, from which they are activated. In contrast, birds (which exhibit no ASR) have stapedius motor neurons whose cell bodies lie within the facial-nerve nucleus, some distance from the ascending auditory pathway. It seems clear that these morphological differences in the brain stems of mammals and birds reflect the presence or absence of the ASR.

The neuronal pathway that controls the activation of the middle-ear muscles during self-vocalization has not been completely identified in mammals, but it probably has elements in common with the pathway that con-

trols the muscles of the larynx, or voice box, during speech. Indeed, a reflex circuit from laryngeal receptors to the stapedius muscles has been found in some mammals.

It turns out that the neuronal pathway of the ASR travels through an area of the brain stem containing control centers for many vital physiological functions. Hence, the reflex offers a way to test the integrity of such brain-stem centers. A noninvasive diagnostic technique based on the ASR, called the acoustic-impedance-change test, can pinpoint the site of lesions on the cranial nerves or in the brain stem.

The method, first developed by the Danish physician Otto Metz in 1946, measures the ASR's effect on the vibration of the tympanic membrane and ossicles. The membrane is set vibrating by a continuous pure tone emitted from a small probe that is inserted into the ear canal. (The tone is not loud enough to elicit the ASR.) The probe, whose rubber tip ensures an air-tight seal, carries a miniature microphone that can measure the sound level reflected from the eardrum in the sealed ear canal. A second, loud tone (called the activator or the eliciting stimulus) is then generated at either the same or opposite ear in order to induce the ASR. When the stapedius contracts, the eardrum becomes stiffer and the reflected-sound level in the ear canal changes. That change (which represents a change in what is known as acoustic impedance) is recorded by the microphone, whose output is presented graphically as a tracing [*see illustration on next page*].

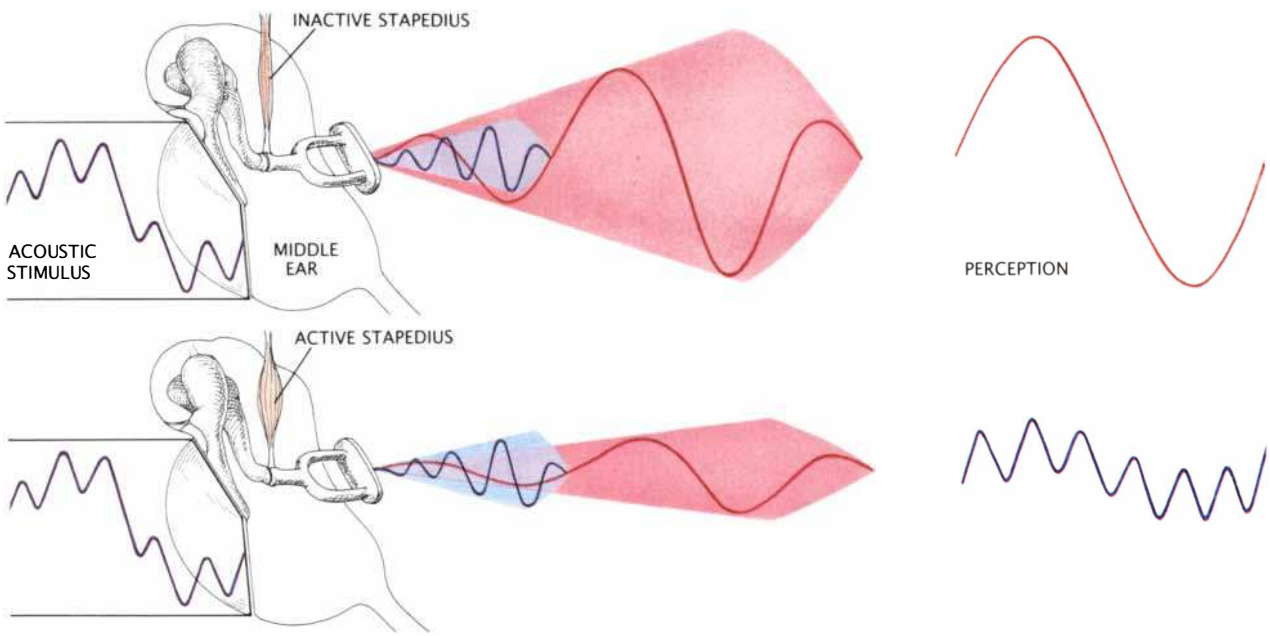
Normally the stapedius contraction produced in this fashion can be sustained for several seconds, during which the tracing shows little or no decrease in acoustic impedance. Henry Andersson of the Karolinska Hospital in Stockholm has shown that lesions on the auditory nerve can cause the ASR to decay at an abnormally rapid rate. Patients who have tumors near the nerve, for example, exhibit impedance tracings that decrease from the peak amplitude to prestimulus levels in a few seconds. The reason is that the tumor exerts pressure on the nerve fibers, which makes them more susceptible to fatigue.

Brain-stem lesions at the ventral cochlear nucleus are also manifested by abnormal decays in ASR amplitude and an increased ASR threshold in both ears. In contrast, lesions farther along the auditory pathway may block the reflex in the opposite ear but should not affect the response in the stimulated ear. Abnormal ASR magnitudes and decay rates are also exhibited by patients suffering from multiple sclerosis, a neurological disease.

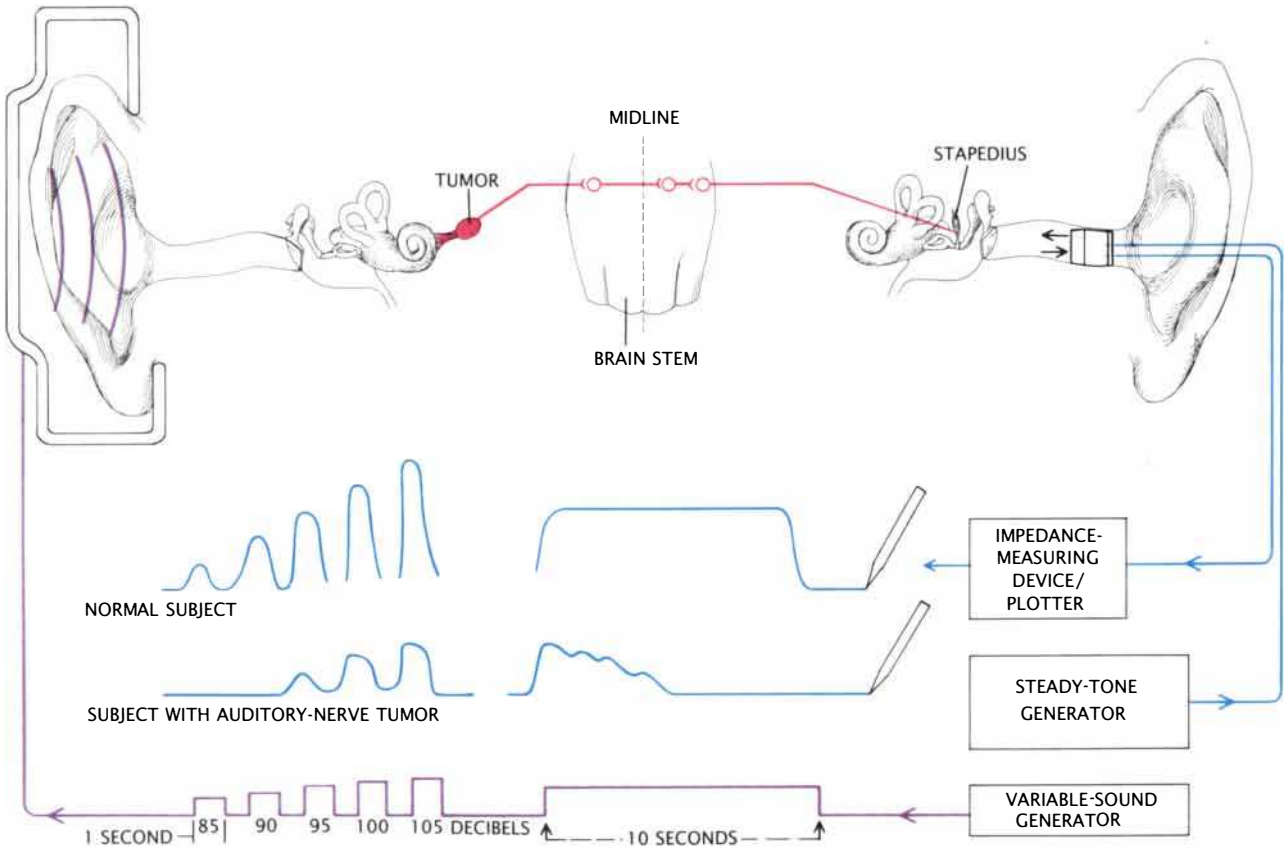
In patients afflicted with Bell's palsy (facial paralyis), the ASR is reduced or blocked entirely in one ear if the lesion lies between the facial nerve's origin and the branch innervating that ear's stapedius. In such a case, the acoustic-impedance measurements can serve to monitor the recovery of facial-nerve function during treatment of the condition or after spontaneous recovery. (Incidentally, patients who have a paralyzed stapedius muscle as a result of Bell's palsy have helped elucidate the muscle's role in human hearing: they complain that their ears are hypersensitive to loud sounds and that what they hear is often distorted.)

Measurements of acoustic impedance are also valuable in the diagnosis and monitoring of myasthenia gravis, an autoimmune disease that is characterized by muscular weakness and extreme susceptibility to fatigue. The disease is caused by the production of antibodies to the patient's own acetylcholine receptors on the muscle-surface membrane. Acetylcholine, a neurotransmitter, normally stimulates muscle activity; without functional acetylcholine receptors a muscle can quickly atrophy.

Acoustic-impedance recordings of patients with the disease typically show an increase in the ASR threshold, a reduction in its magnitude and an abnormally rapid decay. If more acetylcholine is made available by injecting the patient with a substance that inhibits the neurotransmitter's breakdown, the threshold drops consider-



ATTENUATION of a loud complex sound's low-frequency components by the stapedius helps to prevent the "masking" of its high-frequency components. Masking is thought to arise from the way a sound is broken down into high- and low-frequency components in the cochlea. In a person who has a nonfunctional stapedius muscle (*top*), the low-frequency components (*blue*) drown out the high-frequency ones (*red*), as is shown in a superposition of the two components' amplitude envelopes. Normally the stapedius minimizes such interference by dampening low-frequency sound components before they reach the inner ear (*bottom*), thereby allowing both high- and low-frequency sound components to be perceived.



ACOUSTIC-IMPEDANCE-CHANGE TEST takes advantage of the fact that the acoustic stapedius reflex (ASR)—the involuntary contraction of the stapedius muscle in response to a loud sound—can occur in both ears even when the sound is directed into only one ear. In the test, a headphone at one ear produces a sound loud enough to elicit the ASR in both ears. A probe then records the reflexive response in the opposite ear. The probe contains a small sound source that emits a steady, soft tone and a sensor that monitors the sound reflected from the eardrum. When the stapedius contracts, the ossicular chain and eardrum stiffen, and the sound reflected from the eardrum increases dramatically. In a typical run, tracings of the changes in the reflected-sound level (*blue*) are recorded as the ASR-eliciting sounds (*purple*) are progressively increased in intensity. The run is ended with a steady 10-second sound above the ASR threshold. The tracings for a healthy subject will mimic the pattern of ASR-eliciting sounds. In contrast, a person who has a tumor in the neuronal pathway on the side of the stimulus will exhibit weaker responses, an increased ASR threshold and a rapid decay in the response to the prolonged sound.

ably, and the ASR can be more than doubled in amplitude and duration.

The evolution of the middle-ear cavity and its associated structures endowed vertebrates with an increased sensitivity to sound. Yet that sensitivity, in turn, created a need for an efficient mechanism for coping with intense noises that would otherwise interfere with the perception of the sounds on which an animal's survival depends.

The middle-ear-muscle system is nature's elegant solution. This system of sound mufflers and tuners effectively suppresses loud internal and external noise, allowing relevant soft sounds to be separated from irrelevant loud ones. Specifically, the muscle's reflexive contractions prevent desensitization of the auditory receptors, interference between high and low frequencies in the perception of sound, and injury to the inner ear. It is quite likely that the significant evolutionary advantage conferred by the middle-ear muscles accounts for their existence throughout the phylogenetic scale, from the lower vertebrates to human beings.

The built-in reflexes of the middle-ear muscles have both enhanced the hearing of human beings and proved to be a reliable tool for determining the integrity of the ear and the neuronal circuits of the brain stem. Indeed, the acoustic-impedance-change technique has gained wide clinical application. It is likely that the application of the ASR will become even more important in the future for identifying individuals who are susceptible to noise-induced hearing loss as well as for rehabilitating those who have been outfitted with hearing aids.

FURTHER READING

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